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The ecological roles of bacterial chemotaxis

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Brief summary
Chemotaxis is one of the best studied bacterial behaviours, but the underlying mechanisms are much better understood than the reasons and consequences of chemotaxis. In this Review, Keegstra, Carrara and Stocker discuss the costs and benefits both for individual bacteria and whole populations.

Abstract
How bacterial chemotaxis is performed is much better understood than why. Traditionally, chemotaxis has been understood as a foraging strategy by which bacteria enhance their uptake of nutrients and energy, yet it has been remained puzzling why certain less nutritious compounds are strong chemoattractants and vice versa. Recently, we have gained increased understanding of alternative ecological roles of chemotaxis, such as navigational guidance in colony expansion, localization of hosts or symbiotic partners, and contribution to microbial diversity by the generation of spatial segregation in bacterial communities. Although bacterial chemotaxis has been observed in a wide range of environmental settings, insights into the phenomenon are mostly based on laboratory studies of model organisms. In this Review, we highlight how observing individual and collective migratory behaviour of bacteria in different settings informs the quantification of trade-offs, including between chemotaxis and growth. We argue that systematically mapping when and where bacteria are motile, in particular by transgenerational bacterial tracking in dynamic environments and in situ approaches from guts to oceans, will open the door to understanding the rich interplay between metabolism and growth and the contribution of chemotaxis to microbial life.

[H1] Introduction

Chemotaxis is the widespread ability of motile microorganisms to direct their movement along chemical gradients. Although different strategies for motility exist[1], including twitching[2,3] and gliding[4] on surfaces, we concentrate here on bacterial swimming using flagella **[G]**, which is the hallmark strategy of chemotactic motility. Swimming cells typically explore the environment via random walks **[G]** that alternate straight paths (runs) and reorientations (such as tumbles or flicks). The chemotaxis signalling pathway enables cells to bias their movement in response to chemical stimuli by modulating the duration of the runs. The molecular and cellular mechanisms of responses to chemoattractants **[G]** are well characterized, especially the response of *Escherichia coli* but also of other species[1,2,5–10]. Surprisingly, the ecological role of bacterial chemotaxis is much less clear[11–13], along with our understanding of when and where bacteria adopt a motile lifestyle. We argue that it is now time to take the body of quantitative and mechanistic insights into how chemotaxis works and place it within the broader context of the contribution of chemotaxis to the lives of bacteria, to provide insights into why many bacteria have evolved this motility strategy.

Chemotaxis has mainly been interpreted as a foraging strategy in which the chemoattractants directly serve metabolism **[G]** as sources of nutrients[6,9] or energy[14,15] (Fig. 1a). However, some of the earliest chemotaxis experiments already showed that *E. coli* is also attracted to compounds that it does not take up, and that certain amino acids with low nutritional value are stronger chemoattractants than any sugar. It is still puzzling from an ecological perspective why bacteria would be attracted to compounds that do not or only poorly support growth and why, conversely, they are not attracted to certain compounds that support fast growth (Box 1). Furthermore, it has remained unclear how the cost-benefit relation of chemotactic foraging plays out in different environments. In parallel, an alternative explanation for chemotaxis has become more prominent, according to which a chemoattractant is not itself the objective but rather the means to an end (Fig. 1b), such as the colonisation of uncharted territory[16] or the location of partners by symbionts[17] or hosts by pathogens[18]. Chemotaxis enables pathogens to find hosts and locate specific entry points for infection[18]. Under this view, the chemoattractants can, but do not need to, be metabolizable to provide an ecological advantage. For example, *Helicobacter pylori* chemotaxes towards bleach (HOCl), an antimicrobial

produced by neutrophils), possibly as a signal for tissue inflammation[19]. *Bacillus subtilis* chemotaxes towards ethanol at concentrations that are detrimental for growth, and this behaviour might be beneficial for localizing ethanol-producing microorganisms that *B. subtilis* lyses as prey[20]. Accordingly, some chemo-attractants can have properties that would be usually associated with chemorepellents **[G]**.

Chemotactic bacteria are found in a wide range of environments, contributing to the health and disease of hosts[18,21] and playing a major role in biogeochemical cycles[22,23]. A key example is the gastrointestinal tract of warm-blooded animals, which is the main habitat of *E. coli*[24], among many other motile strains including pathogens[18]. Plants and soil are also favourable habitats for chemotactic bacteria, especially the microenvironments of leaves[25] (the phyllosphere) and around plant roots[26] (the rhizosphere). In aquatic environments[27], chemotactic bacteria are routinely found on marine particles[28] or associated with phytoplankton species[17] (the phycosphere), protists[29] or corals[30] (Fig. 1c-e). The diversity of these environments suggests that chemotaxis is an ecologically advantageous strategy for bacteria in a wide range of settings.

Determining precisely which strains perform chemotaxis and under which circumstances is not a simple task. Genetic analyses have revealed that genes for motility and chemotaxis are widespread, with core components conserved across distantly related bacteria and archaea[31,32]. But a motile lifestyle is not a universal trait and many habitats harbour both strains with and without motility genes[33]. Furthermore, genomic studies reveal only the potential for motility and not which strains really are motile. Many bacteria are known to switch between sessile and motile lifestyles[34], and bacteria can control motility at the post-translational level to cease swimming temporarily[35,36]. The complexity arising from interspecies diversity and phenotypic plasticity illustrates the challenge in establishing a framework to understand when bacteria adopt a motile lifestyle.

To understand the ecological roles of bacterial chemotaxis, we focus on a number of key characteristics that shape the environment on the microscale[37] (Fig. 1f-g). First, we consider spatial structure. Chemotaxis is advantageous for bacteria in situations in which chemical gradients guide bacteria within heterogeneous resource landscapes[37,38]. In addition, bacteria may also need to navigate landscapes with large areas lacking detectable gradients or with high viscosity that hinders swimming (for example, in the gastrointernal tract[39,40]) or complex microstructures such as intricate webs of channels, grooves or pores[41] (for example, on leaves and in soil). Second, we consider temporal structure. Different magnitudes and timescales of environmental changes impose very different constraints on the use of chemotaxis. Chemotaxis provides an advantage when responding to short-lived nutrient patches (such as sinking particles in the ocean) on timescales of seconds to minutes[38], but prolonged nutrient limitation between patches will impair motility and chemotaxis. Third, bacterial densities can be very different, ranging from extremely high densities (10^{10}–10^{11} cells g^{-1}) in the colon[42], to intermediate densities (10^{7}–10^{9} cells g^{-1}) in soil[43] and on leaves[44], to lower densities (10^{5}–10^{7} cells g^{-1}) in aquatic environments[42] (with the exception of nutrient hotspots such as particles[28]). Bacterial density ultimately determines whether bacterial cells use chemotaxis independently from each other (at low densities) or collectively (for example, by responding to environmental changes generated by the collective, at high densities). These microscale characteristics of bacterial habitats frame the ecological challenges to bacterial navigation and constrain the set of motility and metabolic strategies[45].

In this Review, we highlight how these aspects of bacterial density and spatial and temporal structure influence why and under which circumstances bacteria adopt a motile lifestyle. As any biological trait is the result of a cost-benefit relation embedded in its environment, we review first the quantitative understanding of the costs of motile behaviour, then we discuss how the environment influences the benefit of chemotactic behaviour by modulating the chemotactic performance of bacteria. We then contrast settings in which cells migrate individually versus collectively. Finally, we highlight the ecological roles of chemotaxis beyond nutrient finding, such as boosting range expansion in colonisation of new microenvironments and maintaining bacterial diversity.

[H1] Cost of chemotaxis

To understand under which conditions chemotaxis is advantageous and why some bacterial strains have adopted this strategy, the costs and benefits of chemotaxis need to be considered. For this, quantitative estimations of the energetic and proteomic costs of chemotaxis have become available

and recently it has become possible to directly compare these costs to the benefits of chemotaxis in simple environments.

[H2] Energetic costs of motility and chemotaxis

Energy is an essential resource of a bacterial cell. Viscous forces dominate motion through liquid at small length scales[46] and the energy spent on propulsion increases linearly with the viscosity of the medium and quadratically with the swimming speed[47]. Motility by rotating flagella in water requires an estimated energy equivalent to ~10^4 ATP s^{-1} (assuming 8×10^{-20} W per ATP[48]) for *E. coli* swimming at ~25 μm s^{-1}, based on flagellar motor [G] output measurements [49–51](Fig 2a). Marine bacteria, which have swimming speeds of 40–200 μm s^{-1} (and reduced motor efficiency at high motor frequencies[52]) will expend one to two orders of magnitude more energy than *E. coli*[53]. For chemotaxis, in *E. coli* the energy expenditure is estimated[54] to be 10^3-10^4 ATP s^{-1} based on *in vivo* measurements of baseline pathway activity[55] and an additional 10^3 ATP s^{-1} for sensory adaptation[56]. As the costs for sensing are not expected to scale with swimming speed, this means that for high speeds chemotaxis only requires a small increase in expenditure relative to the energy required for propulsion, but that at moderate speeds (such as 25 μm s^{-1}) the costs of chemotaxis and propulsion can be comparable (Fig. 2a). There is theoretical evidence that cells could devote even more energy to chemotaxis for a marginal improvement of sensing accuracy, but that they have settled for relative higher noise levels to reduce energy expenditure on sensing (Box 2).

In the energy budget of a cell, the fraction of the total energy expenditure for motility and chemotaxis depends on the growth rate (ln(2)/doubling time) of the cell. *E. coli* requires approximately 4×10^{10} ATP to double its biomass[57], which corresponds to 10^7 ATP s^{-1} for a doubling time of 1 h. This means that at this (or higher) growth rate, propulsion at moderate speed requires less than one percent of the cell's total energy budget[46,58] (Fig. 2c). This fraction increases for lower growth rates. In the absence of growth the energy expenditure of the cell reduces to its maintenance level, estimated[59,60] to be ~10^4 ATP s^{-1} for *E. coli* and species with similar size. This means that during starvation, motility would become the dominant energy-consuming process (Fig. 2c). In some environments, such as deep-sea sediments[61], energy limitation makes motility impossible, but if bacteria have sufficient energy for fast growth, they will certainly have enough energy for swimming.

Although chemotaxis is energetically costly at low growth rates, its potential benefits under such conditions can be high, making it a high risk–high reward investment. Bacteria have developed strategies to adjust their motile behaviour to the challenges imposed by energy limitation. Under prolonged nutrient starvation, it has been observed in several strains that the number of motile cells decreases over timescales of hours to days[61]. Although this behaviour could be a simple consequence of energy loss, it could also point at an energy saving mechanism. Many taxa, including *E. coli* and *Salmonella enterica,* are able to stall flagella, using proteins that bind to the motor and act as a molecular brake[36,62,63]. This points to the ability of bacteria to control energy expenditure by actively turning motility on and off[64]. Another possible strategy is the use of endogenous energy storage compounds[65,66], which could fuel swimming for hours using only a small fraction of the cellular biomass[67,68]. Finally, bacteria might use the widely distributed light-absorbing proteorhodopsin proteins to fuel motility, which would decouple the energy supply from the chemical landscape[69].

[H2] Proteomic cost of motility and chemotaxis

Bacteria need to allocate resources for the production of the proteins that enable motile behaviour, which is dominated by the expression of flagella. In *E. coli*, with an average of 3-4 flagella per cell, each cell expresses in the order of 60,000 subunits of the flagellin FliC protein in building flagella[70,71] (Fig. 2b) and an additional 10,000–40,000 proteins, mostly chemoreceptors[72,73] **[G]**, for the chemotaxis pathway. The synthesis cost of these proteins (assuming an average length of 300 amino acids per protein) can be estimated as 8×10^8 ATP per cell cycle[60,74], which means that even for growth rates as low as 0.07 h^{-1}, the energetic costs for synthesis dominate over the cost of swimming and sensing[70]. As these resources could have been invested in the production of other proteins, it is important to consider the proteomic costs.

To understand proteomic expenditure by bacteria, a coarse-grained model of proteome allocation has proven successful (Box 2). The model assumes that the total number of proteins per cell volume is constant and therefore cellular processes are fundamentally limited by the fraction of proteins devoted to each task. For *E. coli*, the expression of motility and chemotaxis proteins amounts to 2–4% of the total cellular expressed proteins[72,73,75]. Since these proteins could have been invested in other

processes that directly contribute to growth rate, a trade-off [G] between motility and growth can be postulated. However, because the benefit of chemotaxis to growth is environment-dependent, it cannot be understood as a strict universal trade-off between motility and instantaneous growth rate.

Experimental evidence for a trade-off between motility remained indirect until recently. Experimental evolution had shown that selecting for faster migrating phenotypes was associated with reduced growth rates in a well-mixed environment[76,77]. More direct evidence for a trade-off came from a recent study of gene expression[71]. The promoter activity of two of the main motility proteins (the flagellin FliC and the chemoreceptor Tar) of *E. coli* decreased linearly with increasing growth rate, in agreement with predictions from the proteome allocation model. Cells had different growth rates growing on different carbon sources. This relation indicates that *E. coli* invests more in motility proteins when the growth rate, and thus the nutrient quality, is low (Fig. 2d). Alternatively, this negative correlation between the growth rate and the investment in motility may be a result of changes in cell size associated with different growth rates. Faster growing cells are larger, and if, as it has been proposed, *E. coli* maintains a fixed number of flagella per cell, this would yield a decreasing investment in motility proteins with increasing growth rate[78]. Regardless of what mediates the observed expression differences of motility proteins with growth rate, the balance of cost and benefit of expressing these proteins may shift with growth rate. To compare the cost and benefit of expressing motility and chemotaxis proteins at different growth rates[71], co-culture experiments were used of wild-type *E. coli* with a mutant deficient for motility and chemotaxis, growing on different carbon sources that support different growth rates. In a well-mixed environment, the mutant outcompeted the wild type for all nutrient qualities, demonstrating a trade-off between motility and growth. This setting was contrasted with a spatially structured environment, where in addition to the primary carbon source, a bead was added, that slowly released a mixture of amino acids (each estimated[79] at 10–100 μM on the surface of the bead), thereby establishing nutrient gradients. In the structured environment, the wild type dominated for carbon sources with low growth rates (<0.6 h^{-1}), revealing that the trade-off between growth and motility diminishes when the additional nutritional benefit of chemotaxing to higher concentrations of the amino acids is large. For carbon sources that support high growth rates, the mutant outcompeted the wild type. These experiments suggest that *E. coli* invests more resources into chemotaxis if the anticipated benefit is high, but an actual benefit of chemotaxis towards amino acids was only observed in environments that support low growth rates (<0.6 h^{-1}).

It is noteworthy that the actual benefit of chemotaxis for *E. coli*, in terms of increased uptake of amino acids, does not outweigh the cost at higher growth rates (>0.6 h^{-1}). However, at these growth rates, *E. coli* still expresses genes for chemotaxis and motility. Assuming the expression levels of the motility and chemotaxis genes are close to optimal, this suggests that *E. coli* in natural environments anticipates an additional benefit that makes chemotaxis beneficial even for high growth rates. What could this benefit be? One possibility is the anticipation of a future decrease in growth rate and loss of protein synthesis capacity, so that cells pre-emptively start the gene regulation program to build flagella, which they do on a timescale of approximately one hour[80,81] (for a Review on motor and flagella assembly, see Ref. [82]). This may partly explain why *E. coli* shows high expression of motility genes during exponential growth, and a decrease in the expression of motility genes upon entering stationary phase[16,83], when the resource demand for motility is arguably too high. Another possibility is that the main benefit does not originate from the nutritional value of these chemoattractants, but that they are instead used as navigational cues to favour the colonisation of more favourable environments, similar to the hypothesized role of the response of *E. coli* to hormones and secondary metabolites[85,85] within the gastrointestinal tract. Importantly, natural environments could also include explicit disadvantages of a motile lifestyle, such as potential mortality costs arising from increased encounters with predators[86] or specific flagella-targeting immune cells[87]. Quantitative comparisons of the anticipated benefit of chemotaxis (as the fractional investment in motility and chemotaxis proteins) and the actual benefit (increased biomass or yield) in controlled microenvironments are important for establishing under which conditions bacteria should invest in a motile lifestyle, and shine light on the different ecological roles of chemotaxis.

[H1] Chemotactic performance of individuals and collectives

In certain scenarios, the advantage of chemotaxis (the why) emerges at the population level, due to the phenotypic diversity [G] in motility[88] and signalling[54,89–91] behavior within a population[92,93]. Understanding why the maintenance of a broad spectrum of chemotactic behaviours is advantageous for a population may provide clues to the ecological relevance and evolutionary advantage of chemotaxis under different conditions[11]. This includes the enhancement of chemotactic migration

towards food sources through cell-cell communication[94], bacterial stress resistance[95], systemic infection within a host[18], and efficient population navigation of complex topologies such as mazes[96] or fractal landscapes[97]. In this section, we discuss the ecological role of chemotaxis in two scenarios: populations in a low-density regime in which bacteria only sense and respond to the environment, and in a high-density regime in which they may shape the environment and behave as a collective.

[H2] Bacteria as independent searchers

To evaluate the chemotactic performance of a population and the fitness benefits in its environment the perspective of a single cell remains crucial, because of the large phenotypic diversity within bacterial populations. Phenotypic diversity can increase the population performance even in the simple environment of a steady linear chemoattractant profile. When population density is low ($<10^8$ cells g^{-1}), cells are far from each other (in liquids, mean spacing >22 μm) so that direct interactions through chemical signalling and indirect interactions through nutrient consumption are unlikely to occur, and each individual climbs gradients independently. Tracking bacteria in simple static gradients have revealed that cells with a low tumble bias **[G]** climb gradients much faster than individuals with the average tumble bias (Fig. 3a), meaning that in a population displaying a large phenotypic diversity in motility traits the average chemotaxis speed is higher than the chemotactic speed of the average individual[92,90]. Therefore, it has been hypothesized that evolution has not only acted on the mean tumble bias, but also on the shape of its distribution across the population[90,98].

Behavioural differences in motility strategies due to phenotypic diversity are particularly beneficial under dynamic and heterogeneous conditions[99,100] that force a population to balance different challenges. Natural environments are more complex than single steady gradients, and spatial structure at the microscale, such as the network topologies in the phyllosphere[25], in the rhizosphere[101], in corals[102] and in the human lungs[18], introduce great physical complexity in the environments that many bacteria must navigate. This affects bacterial navigation and transport, the spatial range of interactions between individuals or symbiotic partners, and the ability to find and infect hosts (Fig. 1). Recent work using microfluidics to mimic spatially structured microenvironments has analysed the decision-making of thousands of cells navigating chemical gradients in complex geometries[96] (Fig. 3b) such as branching T-mazes[103] or fractal topologies[97]. The likelihood that a bacterium successfully navigates a gradient depends ultimately on the sensitivity of its chemotaxis pathway[104], and this sensitivity displays large variation within a population in both the tumble bias and the pathway gain **[G]** [103]. The tumble bias is most relevant for the response speed when cells need to climb gradients from unsteady sources, whereas the pathway gain is most relevant in the tight accumulation around persistent sources and for the navigation in complex geometries[90,103]. The maintenance of a wide distribution of phenotypes therefore enables effective gradient climbing by a part of the population in dynamic and heterogeneous environments[90,98].

Spatial exploration is a main ecological challenge for motile organisms in sparse environments. In environments characterized by patchy nutrient sources against a very dilute nutrient background, large regions contain no detectable chemical gradients[11]. The task thus shifts from climbing to finding gradients, and bacteria can increase encounters with nutrient patches by increasing their effective diffusivity **[G]**. Theoretical analysis and numerical simulations indicate that random motility alone provides a 100–1000 fold increase in encounter rate compared to non-motile bacteria[105,106]. Chemotaxis can provide a further advantage to the search behaviour by increasing the region of space around the nutrient patch where a cell can encounter the chemical gradients emanating from the source[107], however the exact benefit is likely environment-dependent (including fluid flow, size and velocity of the nutrient patch) and strain-dependent (including swimming speed, tumble bias, pathway gain).

In sparse environments, such as the phyllosphere or the bulk ocean, the performance of a population can depend predominantly on the performance of only a small fraction of its individuals, because a low chance of success is compensated by a high reward, such as the encounter of a bacterium with a rare but extremely rich nutrient patch or with the entry point to a host organism. This form of stochastic foraging inequality is exemplified by bacterial foraging on marine particles in the ocean[35,45,105], which represent rich hotspots of nutrients and of associated microbial activity[22]. Bacteria–particle encounters are probabilistic events: bacterial motility increases on average the chance of encountering a particle by increasing bacterial diffusivity[108]; however, the waiting times between encounters within a population are exponentially distributed, where a small number of bacteria encounter a particle disproportionally earlier than the average bacterium[105] (for example, the

first 1% encounter a particle 100 times faster than the average). As a result of this stochasticity, strategies involving chemotactic motility to forage on particles can be viable even in nutrient-poor environments in which a bacterium that experiences the average search time would be expected to starve to death before an encounter. This argument suggests that nutrient-poor marine environments, traditionally thought to be dominated by non-motile bacteria[33], might in fact afford important niches also for motile bacteria. When evaluating why chemotaxis is a viable strategy in the ocean as in many other environments, the single-cell perspective is essential to evaluate the performance of a population.

A cell can enhance spatial exploration by exploiting molecular fluctuations in the chemotaxis pathway[109] (Fig. 3c), but at the cost of reducing sensing accuracy. Slow (~10 s) temporal fluctuations in the activity level of the chemotaxis pathway transform a Brownian random walk **[G]** into a Lévy flight **[G]** [109–112] (Fig. 3c), in which the power-law distribution of run lengths greatly increases the chance of encountering a nutrient patch. Recently, intracellular measurements of chemotactic pathway activity using Förster resonance energy transfer (FRET) in single *E. coli* cells[113,114] revealed that the temporal fluctuations vary substantially among cells[113]. This suggests that bacterial populations can hedge their bets between increasing the encounter with nutrient patches and the ability to retain position after an encounter[115]. *E. coli* has been shown to suppress bet-hedging behaviour when more environmental information is available and the benefit of bet-hedging is lost. This is achieved by modulating expression levels[116] as well as using post-translational modifications of signalling proteins[117], revealing that bacteria can adjust their phenotypic distribution dynamically to optimize performance.

[H2] Chemotaxis and collective behaviour

To understand certain ecological roles of bacterial chemotaxis we need to move beyond the single-cell perspective of how individual bacteria respond to chemical cues, because some important forms and functions of chemotaxis (the why) only emerge at the level of a population of interacting individuals. At high bacterial densities (>10^8 cells g^{-1}), such as those found in the large intestine[42], around nutrient hotspots in the soil such as roots[43], or on marine particles[28], cells are no longer decoupled agents because of the increased importance of chemical and physical interactions. Bacteria are social organisms, capable of communication via chemical signalling[118]. The most iconic example of cell–cell communication is quorum sensing, the process that allows groups of bacteria to synchronise changes in behaviour in response to changes in population density. Quorum sensing relies on the production, detection and response to extracellular signalling molecules called autoinducers[119]. The coupling between the release of signalling molecules and chemotactic motion towards these molecules may generate spatial patterning and bacterial spontaneous aggregations in otherwise homogeneous and static environments[120,121], and could also be used by bacteria to narrow their distribution around nutrient hotspots[95] (Fig. 3d).

Some classic forms of collective behaviour in bacterial chemotaxis, such as the formation of traveling bands[16,93,122–124] do not rely on the secretion of signalling molecules but rather are the emergent consequence of interactions between bacteria and their limiting resources[93]. At high bacterial density, the local consumption of a chemoattractant generates a gradient that the population starts to chase, generating a travelling band. Using a microfluidic device that enabled the simultaneous tracking of individuals and observation of collective migration, a recent study[93] revealed the formation of cohesive travelling bands, despite the population being composed of phenotypically diverse individuals. Within the travelling bands, local bacterial consumption of chemoattractant, initially distributed homogeneously, introduces a self-generated gradient, which is sensed by bacteria (Fig. 3e). This density-dependent mechanism contributes to levelling-off the gradient in front of the band, sorting for the better chemotaxers, and to sharping it at the back of the band, where the weaker performers are. Density-dependent mechanisms mediated by consumption represent a subtle and inexpensive form of indirect communication among phenotypical diverse bacterial cells, which in this example enables populations to maintain diversity while travelling as a cohesive group.

[H1] Beyond foraging

Chemotaxis has ecological roles beyond finding nutrients[125] and serves additional ecological functions in which the chemoattractant is not the target but rather the means to another end. Recent work sheds light on the apparent paradox of chemotaxis to compounds associated with low growth rate (Box 1) by highlighting the wider ecological and evolutionary implications of chemotaxis, such as

boosting colonisation of new environments[16] and promoting diversity by stabilizing bacterial communities[70,126].

[H2] Chemotaxis boosting range expansion

An important, recently proposed function of chemotaxis is boosting range expansion—the migration of a species into new habitats—and to enhance population growth in nutrient-replete environments. As described above, at high bacterial densities the consumption of nutrients creates a gradient that cells at the leading edge of the population chase, resulting in a traveling band of bacteria[122,123] (Fig. 4a). Surprisingly, recent experiments with *E. coli* on soft-agar plates showed that adding small amounts of serine or aspartate homogeneously in the agar as a chemoattractant to plates with glycerol or glucose as primary carbon sources caused bacterial populations to expand twice as fast as populations in the same environment without the addition of the chemoattractant[16]. The increase in the expansion speed was dependent on a division of labour within the bacterial population: the colonisation is led by a group of pioneering cells that pull the population front by chemotactic motion, and as these cells replicate they leave behind offspring that settle and occupy the environment by growth (Box 3). The primary carbon source is used to sustain growth while the chemoattractant provides a cue for navigation, which drives migration before the onset of the nutrient limitation, which is typically encountered by cells during range expansion on a carbon source alone. Importantly, under initially replete conditions, chemotactic motility is not triggered, as in the classical Keller-Segel model of chemotactic migration (Box 3), by nutrient depletion to find better environmental conditions[71,127]. The addition of a chemoattractant makes a fast-growing population rapidly generate a gradient at the expansion front through uptake of that chemoattractant, which acts as a cue for individuals and accelerates population growth and expansion[16]. The mathematical description of the boosted range expansion could also qualitatively and quantitatively describe the expansion in the case of rich undefined semisolid media[16], showing that colony expansion in complex media can be understood as driven by a growth on a primary carbon source and expansion using a chemoattractant distinct from the carbon source.

The phenomenon of boosted range expansion may thus help solve the puzzle of why bacteria have high chemotactic sensitivities for less beneficial growth substrates, such as serine or aspartate. In batch culture, *E. coli* is can only increase growth rate relative to a baseline minimal medium supplemented with glucose to a limited extent (10–20%) through catabolysis of serine or aspartate[79,128]. However, during range expansion, the colonisation rate increases more than 100% when serine or aspartate are added to the primary carbon source, because of the effective decoupling between growth and chemotaxis, each of which relies upon different chemical compounds (Box 3). *E. coli* does not grow very well on serine or aspartate as its sole carbon source[129]. However, *E. coli* takes up serine or aspartate first when cells are fed a mixture of many degradable compounds in a well-mixed environment[79]. A flux balance analysis indicated that these amino acids are taken up at a higher rate than required for protein synthesis and catabolized to generate energy[79]. Together with the fact that they are small and abundant, this makes them good candidates to be used as sensory cues in situations in which another carbon source is the main driver of growth. Taken together, these observations raise the possibility that the chemotactic preference for serine and aspartate did not evolve only to directly supply metabolic demands of a cell, but also to increase the colonisation rate of a population in new environments.

[H2] Motility-based mechanisms for coexistence

Chemotaxis can promote bacterial diversity through trade-offs between chemotactic motility and other performance traits, such as growth or competitive abilities. The competition–colonisation trade-off, in which better competitors are inferior colonizers and vice-versa, was first tested in a study quantifying interactions in protist microcosms[130], and has long been considered a plausible explanation for species coexistence in environments characterized by a continual turnover of new patches[131,132]. In the ocean, seascapes of organic particles introduce microscale heterogeneity that can drive ecological differentiation of microbial populations through the competition–colonisation trade-off[133]. Theoretical analysis suggests that trade-offs in motility performance may create alternative niches even within initially uniform environments[90]. However, verification of the growth–motility trade-off in natural ecosystems is rare because environmental heterogeneity and direct competition are usually confounding factors. Recently, work unifying themes from microbial spatial ecology and evolutionary theory with systems biology and biophysics has highlighted the concerted contributions of growth, motility and chemotaxis to generating and stabilizing community diversity[70,126] (Fig. 4).

In work to test the role of motility and spatial competition in driving bacterial coexistence, a growth–motility trade-off was consistently found in co-existing wild strains of *E. coli* that were isolated from a single host[70], implying that these traits were the evolutionary outcome of stable coexistence (Fig. 4c). The growth and motility parameters were measured for each strain when cultured alone. The trade-off emerges because swimming requires the expression and operation of flagella and so can limit growth by inducing proteomic costs (Box 2). When two such strains were co-inoculated in the centre of a plate, mimicking the onset of competition for a freshly available nutrient patch, spontaneous spatial segregation happened. The fast-moving (but slow-growing) strain prevented the slow-moving (but fast-growing) strain from moving outwards and accessing nutrients available in the periphery (Fig. 4d). The indirect inhibitory interaction between species that drives spatial segregation is mediated by the coupling between nutrient consumption, which sculpts the nutrient landscape, and motility, which enables the population with higher motility to expand faster. Although the spatial exclusion can also be observed for motile but non-chemotactic cells[70], chemotaxis enhances the segregation by enabling cells to climb self-generated gradients and colonise the newly available environment faster[70] (Fig. 4e).

Evolution experiments have shown that chemotactic motility is an evolvable phenotype even within initially homogeneous environments[126] (Fig. 4f). In experiments with *E. coli*, a strain evolved in sequential expansion experiments in which bacteria were inoculated and allowed to grow to fill a plate, then five selection series were formed by picking at one of five different distances from the initial seeding point over 50 selection cycles (about 600 generations). This created five lines with different behavioural characteristics, namely two with reduced expansion speed, one stable, and two with increased expansion speed. These evolved lines were then inoculated with the ancestor strain in the centre of a plate to engage in competitive spatial expansion, as done in ref[70], leading to spatial segregation. This approach revealed that rapid expansion to colonise new habitat in the presence of competitors is not the optimal strategy if space is limited, as is the case for the surface of resource patches[125]. Rather, in colonising a habitat of a specific size, the bacteria with the winning strategy are those that expand at a speed that scales as the product of their growth rate and the habitat size. The unique optimal expansion speed for a given habitat size warrants that each distance from an inoculation site occupies its own niche. Consequently, balancing the expansion of the bacterial wave front into virgin territory and the colonization of the territory behind the wave front is an evolutionarily stable strategy facilitating coexistence. Like the growth-motility trade-off, this expansion-colonization trade-off leads to spatial segregation of species (Fig. 4g) with different motility characteristics[126] in multispecies communities (Fig. 4h).

While the growth-motility trade-off and the expansion-colonization trade-off generate visually similar spatial segregation when expanding on soft-agar (Fig 4 d,g), the underlying mechanisms are different (Fig 4 e,h). In contrast to the growth-motility trade-off, the expansion-colonization trade-off requires chemotaxis for the boosted expansion speed of the traveling wave[134]. However, the expansion-colonization trade-off does not require that the strains that expand more slowly have a faster growth rate, and in fact the fast expanders may also grow faster without destabilizing coexistence[126]. Therefore, both trade-offs may contribute to bacterial diversity in natural environments.

The recent findings presented here, in which chemotaxis promotes bacterial coexistence during repeated cycles of competitive colonisation of new habitat, may be relevant for environments such as the gut, with regular arrival of nutrients with low bacterial density, or for patchy environments such as the ocean, where bacteria participate in colonisation–exploitation dynamics of nutrient-rich particles[28,35,133]. Importantly, these studies suggest that microbial populations that perform chemotaxis can act as micro-ecosystem engineers that shape and respond to chemical gradients in their immediate surroundings can contribute to foster bacterial diversity, and highlight the fundamental role of motility and chemotaxis in niche formation and community dynamics.

[H1] Outlook

Recent research is revealing a much wider set of ecological roles for chemotaxis in the lives of bacteria than originally postulated, yet much work remains to be done to discover the full extent of the ecological functions of chemotactic responses of bacteria. In many natural environments, such as on plants, in soil, on marine particles or within animal hosts, surfaces containing degradable polysaccharides provide an important niche for motile bacteria, whose lifestyle often combines a motile planktonic phase with a sessile, surface-attached phase. Although motility-driven accumulation towards polysaccharides has been observed[30,135,136], the role of chemotaxis in this process is still poorly understood[135] and most studies have focused on chemotaxis towards low molecular weight

substances. The response to oligosaccharides can be very different depending on the environment. On the one hand, chemotaxis towards highly diffusible oligosaccharides could direct bacteria to polysaccharide substrates[135]. On the other hand, cells can depart from a surface before nutrients are completely exhausted[34,35,137], indicating that oligosaccharides, although often serving as chemoattractants, can also mediate departure from a substrate[137,138]. To further understand sessile-motile transitions in bacteria, more work is needed to characterize the role of chemotaxis in substrate colonisation, but also the rich interplay of chemotaxis with other pathways that control substrate adhesion such as the secondary messenger cyclic-di-GMP[139].

Interesting future directions gravitate around the relation between bacterial physiology and chemotactic motility under nutrient fluctuations. In a recent study, *E. coli* cells were observed when nutrients were abruptly switched from glycolytic **[G]** carbon sources to fermentation products, revealing long lag times after this switch[140]. By expressing different metabolic enzymes bacteria could have changed their pre-switch growth rate but at the cost of a longer post-switch lag time. These results suggest that growth rate is not a mere reflection of the nutritional value of a compound, and cells may lower the growth rate on a compound to anticipate a nutrient shift. We suggest that this ability to tune growth rate specifically for dynamic environments[141] could be relevant for bacterial motile behaviour because anticipation of change could in a similar way increase the potential benefit of chemotaxis in responding to those changes. We therefore expect that more can be learned from viewing motility, uptake, and metabolic preferences as co-evolved rather than independent traits.

There have been important recent advances in the understanding of microbial processes, from cellular motility to the assembly of complex communities, thanks to the increased ability to observe dynamics and manipulate ecological settings at the microscale[142–144]. This progress, in combination with conceptual advances in understanding cellular resource allocation, phenotypic diversity, and spatial self-organization, has led to studies on the model system *E. coli* that inform our understanding of the ecological role of motility and chemotaxis in the natural settings experienced by bacteria. Ultimately, the ability to answer why bacteria perform chemotaxis also hinges on deepening our understanding of when and where bacteria are motile and chemotactic in their natural environment, for example through modern micro-engineering and molecular technology to sample *in situ* microbial activities at the micro- and mesoscale[145,146]. At the same time, in dynamic heterogeneous landscapes in which encounters with nutrient hotspots impact the motile behaviour on the level of an individual bacterium[38], it is important to study aspects of chemotactic decision-making and behavioural strategies across multiple generations[147]. Long-term tracking of bacterial individuals[148] in heterogeneous landscapes marks the next challenge in combining microscopy and microfluidics, paving the way for a more quantitative, single-cell understanding of the multiple contributions of chemotactic motility to bacterial fitness.

Breakthroughs in the understanding of chemotaxis, which build upon a backdrop of a detailed understanding of the molecular mechanisms of chemotaxis in model organisms, are beginning to connect the underlying molecular biology and biophysics with microbial ecology. This is an exciting development in view of the rapidly growing interest in microbial community assembly and function, and its consequences on a wide range of environments from the human gut, to soils and oceans. Chemotaxis thus represents a blueprint for advances at this nexus of microbial physiology and ecology, catalysing the interest of a broad scientific community from molecular biologists and microbial ecologists to physicists and mathematicians.

Glossary terms

Random walk – a type of movement in which steps are taken in random directions. It can be biased if step length or orientation favour a certain direction.

Metabolism – the chemical reactions required to sustain living systems: breakdown of chemicals to release energy (catabolism), the synthesis of biomass (anabolism), and elimination of waste chemicals.

Trade-off – a situation in which a certain trait cannot increase without decrease in another trait because of certain physical or biological constraints. When the constraint is lifted, the trade-off disappears.

Flagella – elongated, thin, and stiff filament that generates forward thrust by rotating. Multiple filaments together may form a flagellar bundle.

Flagellar motor – transmembrane protein complex connecting to the flagellar filaments, which converts a protonic or ionic gradient into rotary motion.

Chemoreceptors **–** elongated transmembrane proteins in which binding to a ligand molecule induces a conformational change that affects downstream pathway activity.

Chemoattractant – a chemical that attracts an organism, inducing movement towards higher concentrations of the chemical.

Chemorepellent – a chemical that repels an organism, inducing movement towards lower concentrations of the chemical.

Phenotypic diversity – variation in the biological traits among members of an isogenic population due to biochemical noise.

Tumble bias – the relative proportion of time that a bacterium spends reorienting during motility. Cells with high tumble bias re-orient more frequently.

Pathway gain – how strongly a cell amplifies the signal from a given chemical gradient. The amplification is determined by the properties of the signal transduction machinery.

Brownian motion – a type of random walk of small particles in a fluid, driven by thermal effects, which results in diffusive behaviour.

Effective diffusivity – the rate at which a randomly swimming cell explores space.

Lévy flight – a type of superdiffusive random walk in which the step-length distribution is heavy tailed, leading to increased spatial exploration compared to Brownian motion.

Glycolytic – using the glycolysis pathway to generate energy.

Sensory adaptation – (partial) restoration of pre-stimulus behaviour during prolonged stimulation.

Adaptation time – the time required for the pathway activity and tumble bias to restore to pre-stimulus levels after prolonged stimulation.

Allee effect – positive density dependence of individual fitness which arises from cooperation or facilitation among individuals in the population.

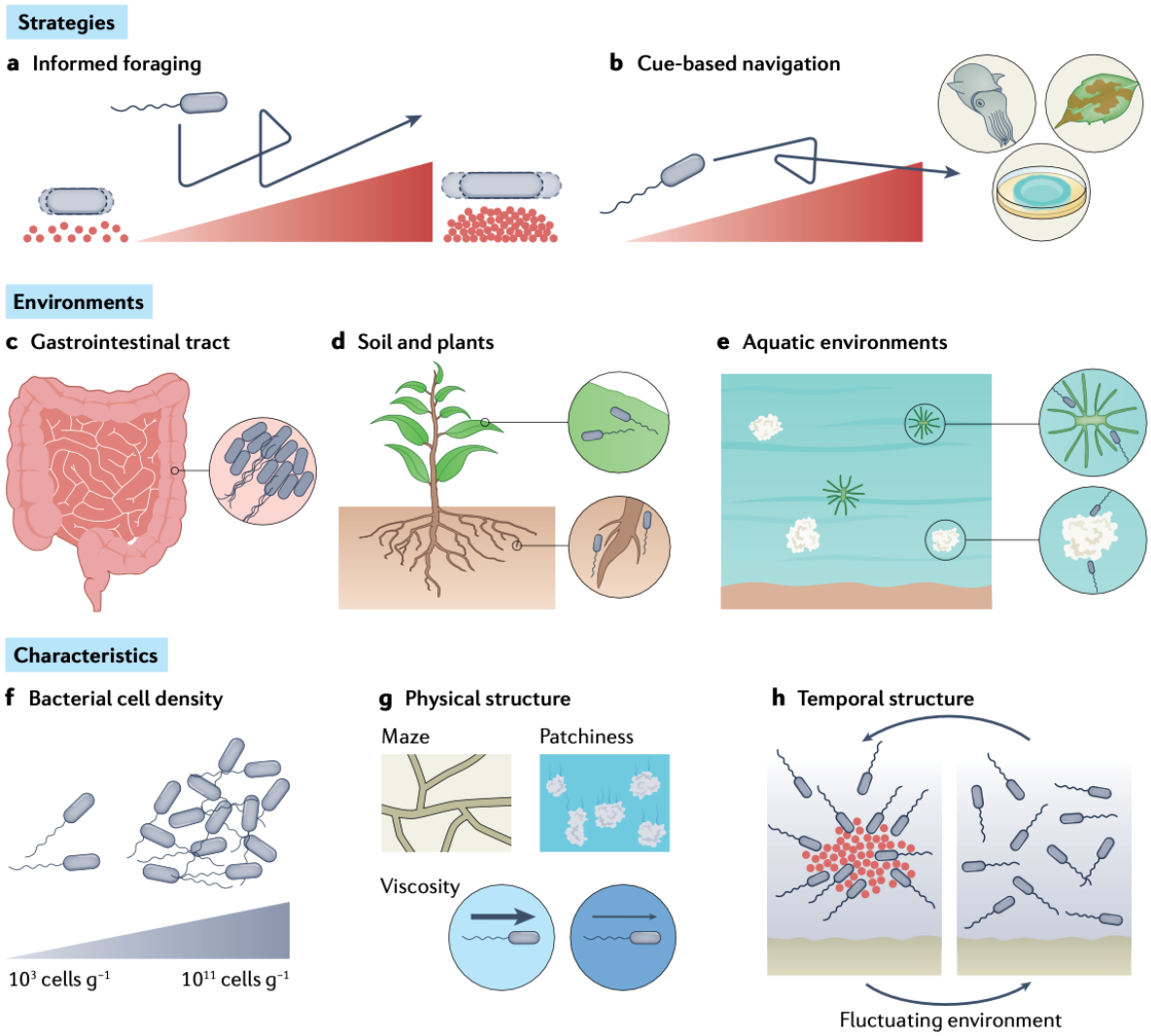


Fig. 1 | Chemotactic bacterial motility across different environments. a | Chemotaxis can act as a mode of informed foraging in which a nutrient is an attractant, enabling cells to climb the nutrient gradient to increase growth rate. **b** | Alternatively, the chemoattractant gradient acts as a cue for a favourable target other than the attractant itself. **c-e** | Environments in which chemotactic bacteria are commonly found: (c) the gastrointestinal tract of warm-blooded animals; (d) soil and plants, including areas with high microbial activity around plant roots ('rhizosphere') and on leaves ('phyllosphere'); (e) aquatic environments including nutrient hotspots around particulate organic matter and around eukaryotic cells ('phycosphere') **f-h** | Characteristics of environments that influence the ecological role of chemotaxis: (f) bacterial cell concentration (in cells per gram of substrate)[28,42,44]; (g) physical characteristics of the environment such as complex topography, patchiness and viscosity, and (h) temporal structure of the chemical environment.

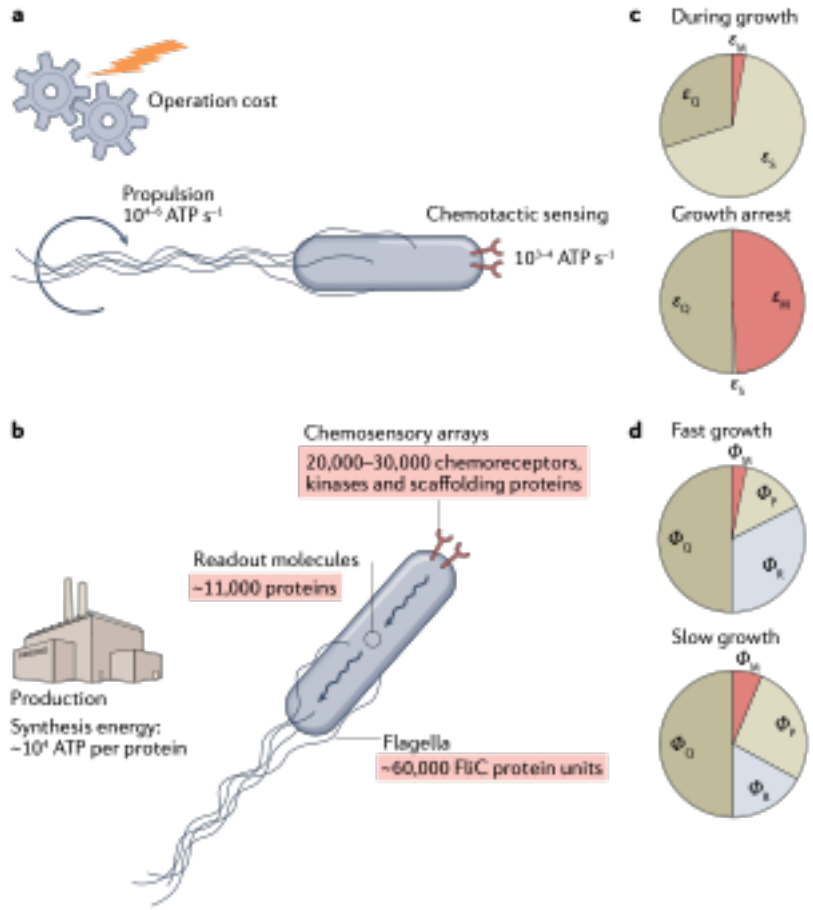


Fig. 2 | The relative cost of bacterial chemotaxis depends on the metabolic state of a cell. **a** | At the scale of a bacterium, the drag scales linearly with the swimming speed, therefore the power required to rotate the flagellum scales quadratically with the swimming speed. For high bacterial swimming speeds (>40 μm s^{-1}), rotation of the flagellar motor[50] consumes the majority of the energy compared to expenditures for sensing[54], adaptation[56], and maintenance. Maintenance cost is a lower estimate based on metabolic scaling laws[59]. **b** | Resource allocation of the proteome is the limiting factor during fast growth. Bacterial chemotaxis requires flagella for propulsion (blue) and the chemosensory network components for directed navigation (red). **c** | The energetic allocation for motility and chemotaxis (ε_M) is relatively low compared to the energy required for synthesis and growth (ε_S) when growth is fast (doubling time of few hours)[58,70], but high compared to synthesis (ε_S) and maintenance cost (ε_Q) for very slow growth (doubling time of days) or growth arrest. **d** | An increased fraction of the proteome is allocated to motility and chemotaxis (ϕ_M) proteins during slow growth at the expense of the fractions for ribosomes (ϕ_M), nutrient uptake (ϕ_P) and maintenance (ϕ_M) [71] (see Box 2).

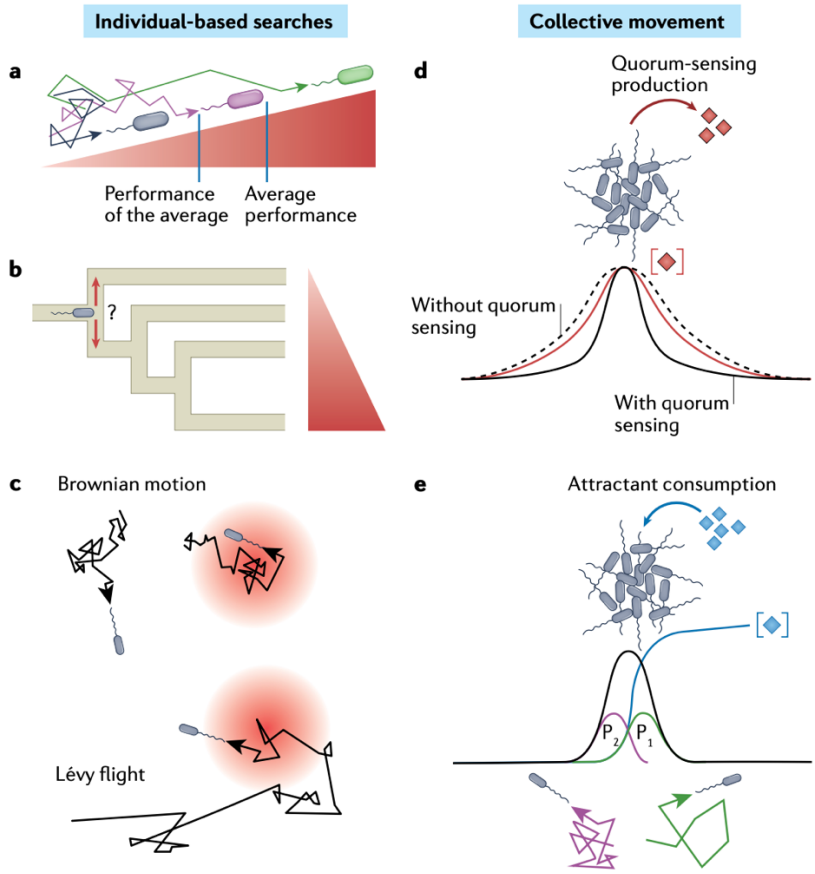


Fig. 3 | The importance of chemotaxis in individual and collective motility.
a | At low bacterial densities, bacteria behave as independent searchers. Even in the simplest scenario of a static chemical gradient, cells exhibit a large variation in climbing performance due to variation in the phenotypes of the chemotaxis pathway, including adaptation time [G] or tumble bias[92]. This diversity can increase the average chemotactic performance of the population compared to the performance of the average phenotype. **b** | Bacteria in nature often navigate within structures that provide obstacles or branching geometries, as mimicked by recent microfluidic T-maze experiments[103]. These experiments revealed that phenotypic variability in parameters such as the pathway gain directly impacts population performance. **c** | In patchy landscapes, cells can exploit molecular noise in the chemotaxis pathway to explore space more effectively by using a Lévy flight with extended runs instead of a Brownian random walk[109]. This is achieved at the expense of sensing accuracy, leading to less tight accumulation around nutrient sources. **d** | Bacteria can release aggregation compounds such as quorum-sensing (QS) molecules[95] to actively enhance the compactness of a population while moving. **e** | At high bacterial concentrations, cellular activity such as nutrient consumption or chemical signalling can induce collective movement. A classic example is the phenomenon of bacterial travelling waves induced by the self-generation of a chemoattractant gradient due to consumption at the leading edge of the wave (see Box 3). Within such a wave, subpopulations (P1 and P2 in the figure) sort spatially according to their motility characteristics[93].

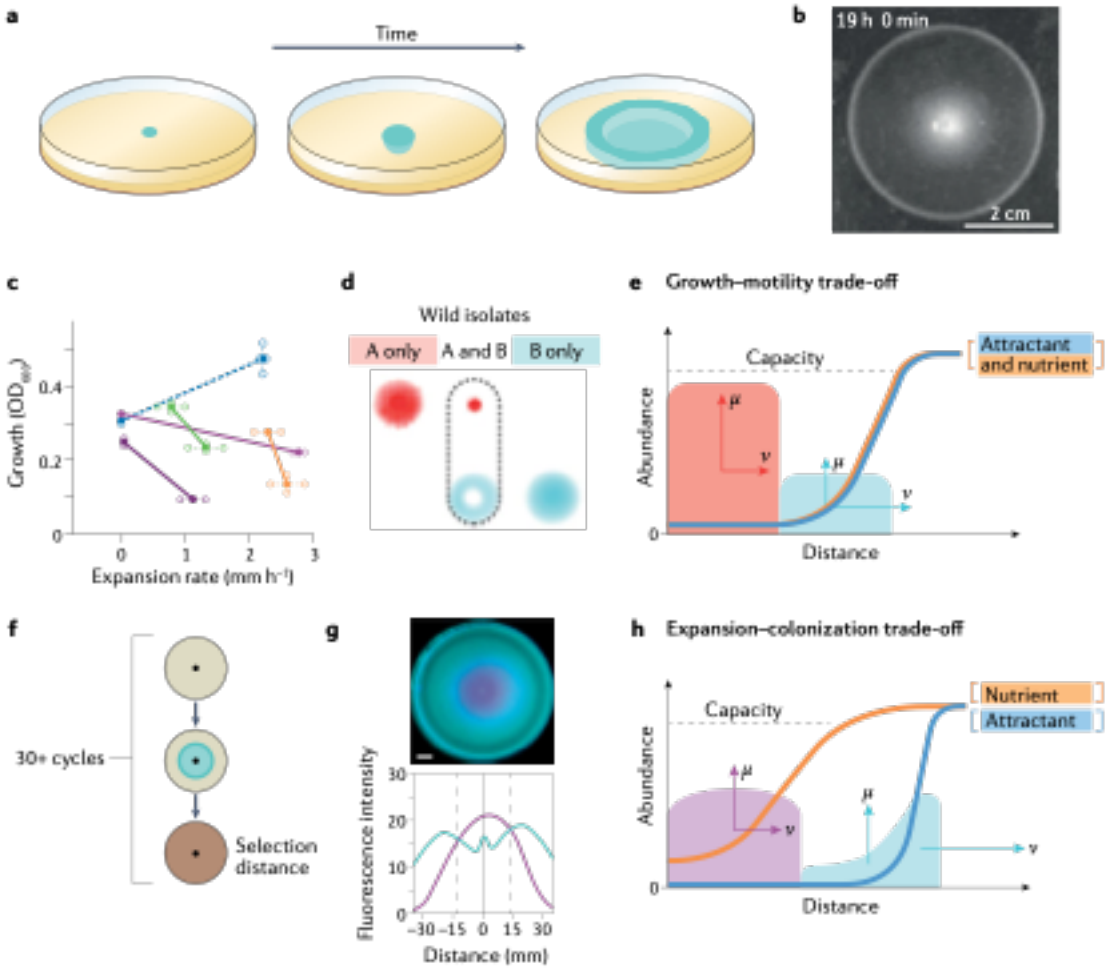
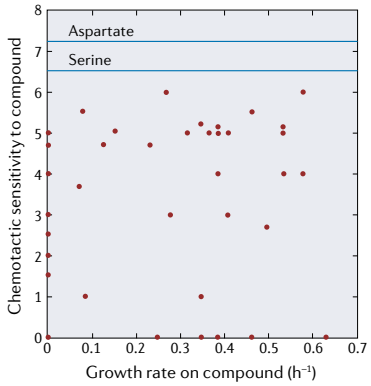


Fig. 4 | **Motility and chemotaxis can drive community diversity. a** | Growth and motility of a bacterial population (cyan) in soft agar (yellow) generate typical expanding rings as the bacteria track a chemical gradient generated by nutrient consumption. **b** | Example of expanded colony of *E. coli* migrating in soft agar. Image reproduced from Ref. [16]. **c** | Co-existing *E. coli* isolates from the same host individual engage in a trade-off between growth and motility (measured by expansion rate). **d** | When one such species pair is inoculated together, spontaneous spatial exclusion is observed, in which the slow-moving but fast-growing strain is constrained to the centre (A, red) and the fast-moving but slow-growing strain inhabits the periphery. Image c and d reproduced from Ref. [70] (B, blue). **e** | Illustration of bacterial diversity enabled by spatial exclusion dynamics, where a fast-moving strain (blue) confines a fast-growing strain (red) by moving outwards and inhibiting nutrient transport (orange) to the centre. Image adapted from Ref. [70] **f** | Evolution experiments show that motility is an evolvable phenotype even within spatially homogeneous environments. Sampling repeatedly at different distances from the plate centre ultimately selects for genotypes with different expansion speeds after ~300 generations. **g** | When an isolate selected for an expansion speed greater than its ancestor is co-inoculated with its ancestor, spatial separation is observed, where the fast-moving strain dominates the periphery (cyan) and the slow-moving ancestor occupies the centre[126] (purple). Images f and g reproduced from Ref [126]. **h** | A trade-off between expansion and colonization generates spatial exclusion and ultimately coexistence. In this case, cells chase a non-nutritional gradient (blue) that enhances migration speed, while growing in nutrient-rich (orange) conditions (Box 3). This mechanism of spatial expansion enables rapid domination in the periphery (cyan) at the expense of excessively diluting the rear-guard, leaving the central area to be colonised by the slow-moving strain (purple).

Box 1: Relation between chemotaxis and metabolism in *E. coli*

Historically, the question of why bacteria perform chemotaxis became overshadowed by research to understand how they perform it[149]. Right from the pioneering work of Julius Adler on chemotaxis in *E. coli* in the late 1960s, it was clear that cells also use chemotaxis to swim towards chemical compounds that are not used as a nutrient or energy source. A correlation between nutrient quality and influx rate has been demonstrated for the PTS sugar influx sensing system[15], but this only partly determines the chemotaxis response because many sugars are also sensed through chemoreceptors. In general, for sugars there is no clear relation between the growth rate afforded by a given sugar and its strength as an attractant[150]. In the figure below we replot data from capillary assay studies performed in the 1970's[150,151] where the chemotactic sensitivities, defined as the negative of the logarithm of the threshold concentration inducing chemotaxis, to many sugars was measured, as well as the growth rate on those sugars when used as carbon source (red dots). This reveals no clear correlation between measured growth rates and chemotactic sensitivities. Furthermore, the best studied chemotaxis response is that of *E. coli* to the amino acids aspartate and serine[6,151]. While *E. coli* is most attracted to the amino acids it consumes first[129], serine and aspartate, it is attracted to more strongly to those than to any sugar (blue lines)[151] although these amino acids only sustain low growth yields when provided as the sole carbon source[129]. The chemotaxis preferences of *E. coli* point to much wider ecological functions of chemotaxis beyond simply tracking the sources of a compound to fuel metabolism.



Box 2: Optimality and energetics of bacterial chemotaxis

Proteome allocation during growth

The proteome allocation framework[54,75,152] describes how cells adjust their proteome to optimize growth rate, using a coarse-grained description of nutrient fluxes in the cell that contribute to growth, most importantly nutrient uptake and conversion of nutrients into biomass. The fundamental assumptions are that the amount of protein per cell volume is constant and that the specific growth-related metabolic fluxes are limited by the amount of protein, specifically the number of ribosomes (R) responsible for producing proteins and transporter and catabolic proteins generating nutrient flux (P). Other proteomic fractions include those devoted to motility and chemotaxis (M) and to other processes that do not directly contribute to growth (Q). Cells can only adjust the proteome fraction ϕ devoted to each task, so that:

$$\phi_R + \phi_P + \phi_M + \phi_Q = 1.$$

This means that an increase in one proteomic fraction occurs at the expense of other fractions. It has been shown empirically that the growth rate depends linearly on the proteomic fraction devoted to ribosome synthesis ($\lambda \propto \phi_R$) and to nutrient uptake ($\lambda \propto \phi_P$). Optimal allocation requires that proteomic fractions are such that all fluxes are equal and equally limiting. When external conditions (such as nutrients) change, this can result in a new growth rate, and with it a new array of optimal proteomic fractions. For example, a reduction in nutrients requires a larger fraction of the proteome to be devoted to influx (such as transporters) at the expense of ribosomes. This framework has been successfully used to explain growth dynamics at the transition between two carbon sources[153] as well as overflow metabolism[75], and provides a valuable framework to understand the trade-offs between chemotaxis and growth.

Proteome and energy allocation in signal transduction

The task of any signal transduction system is to transfer information about the environment into the cell. In the case of the chemotaxis pathway, this information concerns the concentration of attractants or repellents. A body of theoretical literature[54,154,155] on the limits of accuracy in cellular sensing has established that bacteria are fundamentally limited by concentration fluctuations of molecules diffusing within the small volume of a bacterial cell, which gives rise to sensory error. Experiments in which *E. coli* and the marine bacterium *Vibrio ordalii* responded to short-lived, low-concentration pulses of attractants have shown that these bacteria can sense gradients close to the theoretical optimum of sensing accuracy set by diffusion[154,156]. A systematic analysis of single bacterial trajectories in shallow gradients have revealed that the pathway of *E. coli* has evolved to near-optimally use the limited information coming from the environment[157].

Cells can approach the fundamental sensing limit by increasing the number of measurements (for example, by increasing the sampling frequency or the receptors and signalling molecules), but the sensing error decreases at best with the square root whereas the cost increases linearly with the number of signalling molecules or molecule turnover events. Suppressing molecular noise with active feedback is an even more costly enterprise: the resource expenditure scales inversely with the fourth root of the relative error, meaning that a 20% reduction in noise would require a 2-fold increase in resource expenditure[89]. Bacteria may thus tolerate certain noise levels from a resource-saving perspective[54,156].

One can assume that the chemotaxis pathway is optimized to maximize its information transmission. This assumption has been used to infer the types of gradients that *E. coli* typically encounters given measured input–output relationships[158]. However, molecular noise is not always detrimental. Bacteria may exploit molecular noise to enhance their search behaviour in shallow gradients[159] or in situations in which sensory cues are missing (see main text)[10] or facilitate behavioural bet-hedging by generating phenotypic diversity[98,113].

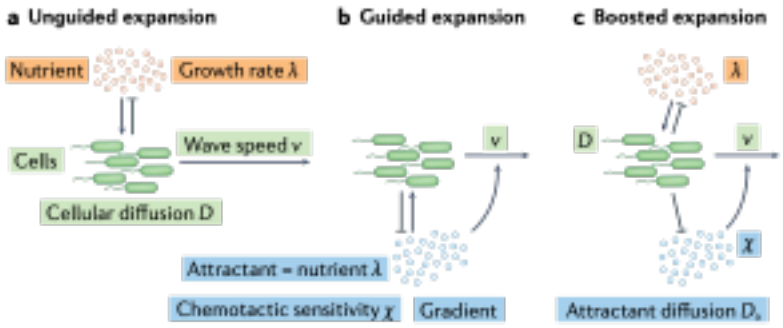
Box 3: Life in motion: movement and reproduction

Biological dispersal shapes the spatial distribution of species and ultimately their ability to coexist. In the ecological literature, classical diffusion, advection–diffusion, reaction–diffusion, and reaction–advection–diffusion models have been used to establish the link between individual, short-term movement and long-term, broad-scale population patterns, such as the dispersal and invasion of animals[160]. The study of bacterial motility, chemotaxis, and growth has proceeded in parallel through the combined work of microbiologists and biophysicists. This body of work has contributed to characterizing the behavioural and metabolic strategies of motile microorganisms and the underlying mechanisms. We present here the basic models proposed in the literature to characterize motility, chemotaxis and growth.

Unguided expansion. Fisher[161] was the first to model the spread of an advantageous gene in a population and Kolmogorov[162] obtained the basic analytical results for the reaction–diffusion model. This model couples a logistic term describing the reproduction of individuals with growth rate λ and carrying capacity K, and a diffusion term accounting for local movement, characterized by the diffusion coefficient D. These species traits define the characteristic spatiotemporal scales of dispersal, which proceeds via a travelling wave moving undeformed at a constant speed $v = 2(D\,\lambda)^{1/2}$ (Figure, part a, all reproduced from Ref. [16]). This form of spatial expansion, where individuals move randomly in space and reproduce until saturation of resources, was first applied to describe the dispersal of animal populations. It has recently been observed in microorganisms, both for systems in which the wave is pulled by individuals diffusing at the leading edge[163] and in systems characterized by a strong Allee effect **[G]** in which the wave is pushed by individuals behind the front[164].

Guided expansion. Chemotactic migrations are well captured by reaction–advection–diffusion models, known as Patlak–Keller–Segel (PKS) systems[165]. In this type of expansion, individuals perform chemotaxis towards a single compound that enables growth. This behaviour leads to the formation of a travelling wavefront moving faster than the Fisher wave, but the exact scaling remains unknown (Figure, part b). The seminal model of Keller and Segel[123] was developed to study aggregations of slime moulds and migrations of chemotactic bacteria, where the production of new cells is negligible and an instability in the uniform distribution of the population leads to the formation of travelling bands. Recently, the classic PKS model has been extended to account for phenotypic diversity[93] and to characterize spatial exclusion dynamics in multispecies communities[70].

Boosted expansion. A richer set of dynamics compared to the PKS model emerges when the chemoattractant is not the main nutrient source. This scenario can lead to the formation of complex, transient spatial patterns[120] or it can boost the range expansion of bacterial populations[16]. A recently proposed model of bacterial expansion[16,134] describes conditions in which a medium containing saturating amounts of a primary carbon source also contains a chemoattractant at low concentration. In this model, the concentrations of the major nutrient source and of the attractant are two distinct variables driving bacterial growth and motility, respectively, which results in an expansion–colonisation process with speed
$v \approx (\chi - D)\,(\lambda\, a_0/a_m)^{1/2}\,(\chi - D + D_a)^{-1/2}$ (Figure, part c)[134], with D_a the diffusion coefficient of the attractant, χ the chemotactic sensitivity coefficient and a_0 and a_m as respectively the initial attractant concentration and the lower limit of attractant sensitivity. Importantly, the population growth rate λ and the chemotactic sensitivity coefficient χ are associated with different compounds, so that to sustain a high migration speed the nutrient does not have to be a strong attractant and vice versa. By responding in this way to a compound that is not the primary nutrient, in what appears to be a paradoxical chemotactic response, bacteria achieve an effective dispersal mechanism and a fitness advantage, namely faster colonisation of virgin territories[126], compared to unguided expansion (part a) or the classic travelling bands where the chemoattractant is the main nutrient source (part b).



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