tially uncouples the astrocyte syncytium. Identifying the molecular mechanism of astrocyte coupling through direct cellular connections (gap junctions) and its physiological modulators will be a challenge. A less exciting possibility is that the anesthetics used in the imaging experiments by Schummers *et al*. may have blocked gap junctions. Future experiments in awake animals should clarify this.


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MICROBIOLOGY

# How Bacteria Change Gear

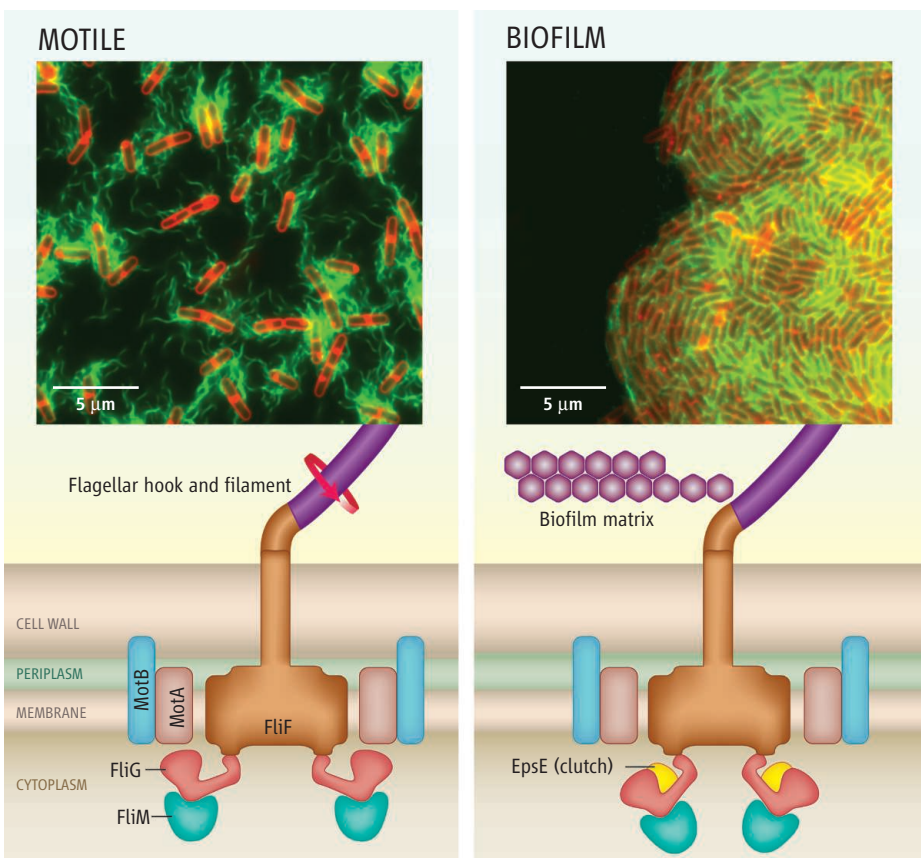
Bacterial motility is arrested when a protein that acts as a clutch disables rotation of the flagellar motor.


**Richard M. Berry[1] and Judith P. Armitage[2]**


Many species of bacteria form biofilms, slimy carpets a fraction of a millimeter thick that appear on rocks, leaves, pipes, teeth—pretty much any place that has a supply of nutrients and water. Cells must first attach to a surface, which in many species requires swimming propelled by rotating helical flagella (*1*). Two things typically happen next. Cells stop expressing genes that encode components of the flagellum, and they secrete a sticky matrix of polysaccharides that holds them together on the surface (*2*). Once at a surface, swimming may be a hindrance rather than a help, and an inverse relationship between swimming and attachment has been seen in many diverse species (*3*). However, the molecular details underlying this arrest in motility have not been fleshed out. Are flagella ejected or dismantled? If not, do they keep rotating until they are jammed by the newly formed matrix? On page 1636 of this issue, Blair *et al.* (*4*) show that in the bacterium *Bacillus subtilis*, the link between swimming and matrix formation is more subtle than either of these two extremes.

The flagellar motor in bacteria consists of a rotor ~45 nm in diameter that spins at several hundred revolutions per second, surrounded and pushed by a ring of protein complexes anchored to the cell wall and powered by an ionic current across the cytoplasmic membrane (see the figure). It is connected to a propeller consisting of a helical filament that extends for many micrometers outside the cell. In *B. subtilis*, motility and matrix formation are linked by SinR, a protein that up-regulates expression of flagellar genes and down-regulates expression of matrix-forming genes. Deletion of the *sinR* gene causes cells to form biofilm-like aggregates (*5*).

Blair *et al.* used fluorescent labels to show that flagellar filaments do not rotate in these aggregates. Furthermore, these bacteria do not swim even in the absence of a gene that is needed for matrix formation. A search for mutations that allow swimming without *sinR* revealed the off-switch for rotation: EpsE, a putative glycosyltransferase protein involved in building biofilms. The *epsE* gene is not found with other flagellar genes, but in an operon that encodes the proteins that




**Motor works.** A schematic of the *B. subtilis* rotary flagellar motor is shown. Motile cells are powered by interaction of the FliG protein with the MotA/B complex (which generates torque). The protein EpsE acts as a molecular clutch to disengage the rotary flagellar motor, leaving the flagellum intact but unpowered. This shuts down motility and facilitates biofilm formation. Fluorescence microscopy photos of *B. subtilis* show bacterial membranes in red and flagella in green, as described in (*4*). FliM and FliF are motor proteins.

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make the matrix and is repressed by SinR.

How does EpsE work? A search for mutants that could swim even with EpsE expressed found several mutations in *fliG*, a gene distant from the matrix-encoding operon that codes for a protein involved in torque generation in the flagellar rotor. Induced expression of EpsE in *B. subtilis* stopped cell motility. Fluorescent-labeled EpsE localized at spots corresponding to individual motors, suggesting direct interaction with FliG, but not in cells with the mutations rendering FliG insensitive to EpsE.

To determine whether EpsE acts as a brake that locks the motor, or a clutch that leaves the rotor freely spinning, Blair *et al.* tethered bacteria to a substrate by their filaments and observed rotation of the cell bodies around single flagellar motors. Under the influence of EpsE, cells stopped spinning but continued to undergo free rotational Brownian motion, indicating a clutch mechanism.

The direct inhibition of motor rotation by EpsE represents a newly discovered control mechanism for bacterial swimming. Bacterial flagella are large protein complexes that require about 40 to 50 genes to assemble (*6*). Thus, the most obvious advantage of the EpsE mechanism over transcriptional control of flagellar genes is speed. In *B. subtilis*, only one protein, EpsE, needs to be expressed to stop the motor. Presumably, this is important if cells are to stay put in the early stages of biofilm formation. However, the advantages of a clutch over a brake mechanism are not so clear. Perhaps free rotation of flagella—or, alternatively, reduced motility during the transition to the EpsE-inhibited state—is important for the formation of well-structured biofilms (*7*). Or maybe a clutch is simply easier to make than a brake.

Whether this is a universal mechanism or a peculiarity of *B. subtilis* remains to be discovered, as do the details of how the clutch works and how it helps biofilm formation. More experiments like this are needed, not just to find genes and proteins, but to learn what they do and how.

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GEOCHEMISTRY

# What Drives Iron Isotope Fractionation in Magma?

Unlike other metals, magmatic melting and recrystallization fractionate iron isotopes, possibly because of the different oxidation states of iron.

**Stefan Weyer**

The isotope composition of natural material can vary, either through the decay of a radioactive parent that results in radiogenic ingrowth of a particular isotope, or as a product of chemical reactions driven by physical changes, for example, during the interactions between biosphere, hydrosphere, and rocks (*1*). Stable-isotope fractionation can reach levels of several percent for light elements, such as hydrogen, carbon, nitrogen, oxygen, or sulfur. However, the magnitude of isotope fractionation drastically decreases with the nuclear mass $M$ (as $\sim 1/M^2$) and also with temperature. Accordingly, magmatic fractionation of (heavy) metal isotopes was long considered to be insignificant. Early studies on samples that formed at high temperatures, such as meteorites, used the isotopic composition of metals [such as iron (Fe), copper, and others] to detect heterogeneities from the origin of our solar system or to address processes of planetary accretion [see, for example, (*2*–*4*)]. However, the study by Teng *et al*. on page 1620 in this issue (*5*) strongly indicates that Fe-isotope fractionation during magmatic differentiation needs to be considered when investigating planetary materials.

A remarkable range of Fe-isotope variations [on the order of ≈1 per mil (‰)] in high-temperature environments, such as Earth`s mantle, was first observed by Williams *et al*. (*6*, *7*). Their findings could not be explained by the recycling of material into the mantle that had isotopically fractionated previously during low-temperature processes on Earth's surface. Rather, their results indicated that these isotopic variations in the mantle were produced by metasomatic processes, in which alteration is driven by interactions of the mantle with small amounts of melts or fluids.

Subsequently, Weyer *et al*. (*8*, *9*) observed that Fe appears to be isotopically lighter in Earth's mantle than in the crust, by ~0.1‰. Additionally, Weyer and Ionov (*10*) observed that in several suites of mantle rocks, Fe-isotope fractionation was coupled with the amount of melt that was extracted from these rocks.

These findings indicated that Fe isotopes can fractionate during partial melting in the mantle, at temperatures of 1200°C or higher (see the figure). However, because most mantle rocks that were brought to Earth's surface originate from the uppermost mantle, which is commonly modified by fluids and recycled material, it was still highly debated whether these isotopic differences between mantle and crust (and isotopic trends in the mantle) stem from partial melting or from metasomatic processes (*8*, *11*, *12*). This question is intriguing, because isotope fractionation during magmatic processes has not yet been observed for any other metal.

Teng *et al*. now provide convincing evidence that Fe isotopes fractionate during magmatic differentiation. Because mantle rocks in equilibrium with melt are difficult to find, these authors studied the opposite process, the fractional crystallization of olivine from a magma. They investigated the Fe-isotope composition of a suite of basalts from a lava lake in Hawaii and that of corresponding olivine grains that crystallized from the lava. They observed that Fe in basalts becomes isotopically heavier as more olivine has crystallized, and that olivines are always isotopically lighter than the coexisting basaltic melts from which they formed (see the figure).

Studies similar to the investigations of Teng *et al*. have been performed already for lighter metal isotope systems, such as lithium (Li) and magnesium (Mg) (*13*, *14*). However, no isotopic differences between basalts and olivine crystals were observed. Why do the isotopes of Fe fractionate during magmatic

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