Cannibals Defy Starvation and Avoid Sporulation

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The term “programmed cell death” (PCD) refers to any form of cell death mediated by an intracellular death program. Although PCD is generally associated with multicellular eukaryotic organisms, PCD has also been found in prokaryotes (1–3). Well-characterized PCD systems in bacteria include the “toxin-antitoxin” modules located on extrachromosomal elements (such as plasmids or phages) (2, 3) and homologous modules located on the bacterial chromosome that are activated by a decrease in nutrients (3, 4). Under starvation conditions, it may be advantageous for a fraction of a bacterial population to undergo PCD, thus providing nutrients for the remaining cells (1, 3). On page 510 of this issue, González-Pastor, Hobbs, and Losick describe a new PCD system in the bacterium Bacillus subtilis (5) that is crucial for spore formation (sporulation) (6, 7). In a process that the authors term “cannibalism,” some cells resist sporulation by killing other sister cells, enabling them to feed on the released nutrients.

Nutrient limitation triggers spore formation, and entry into this process is governed by the regulatory protein SpoOA (8). In earlier work, Losick and co-workers discovered that SpoOA regulates two additional operons: skf (sporulating killing factor) and sdp (sporulating delay protein) (see the figure), which are strongly induced at the beginning of sporulation (9). Now, González-Pastor et al. (5) show that the skf operon is involved in the production of an extracellular killing factor during sporulation. The operon, through its products SkfE and SkfF, also confers resistance to the killing factor. SkfE resembles an adenosine triphosphate (ATP)–binding cassette, and SkfF resembles a transport complex (ABC transporter). It is possible that together these two factors form an export pump that pumps the killing factor out of the cells.

The second operon controlled by SpoOA is sdp (see the figure). SdpC is responsible for producing a 5-kilodalton extracellular factor that acts as a signaling protein among bacteria. SdpC strongly controls the transcription of a two-gene operon, yvbA and yvaZ, located immediately downstream of the sdp operon. Artificially inducing yvbA is sufficient to delay sporulation. A search for genes that could be under the control of the YvbA transcription factor turned up the ATP synthetase operon, which is responsible for ATP production, and the yusLKJ operon, whose inferred products are similar to lipid catabolism enzymes. High levels of expression of yusLKJ were dependent on both the signaling protein SdpC and YvbA. Thus, the signaling protein switches on synthesis of YvbA, which in turn causes an downstream delay in sporulation, which is consistent with the delay in sporulation caused by artificially inducing yvbA.

Defying starvation. When faced with a decrease in nutrients, a subpopulation of a B. subtilis culture delays sporulation (A) by activating the regulatory protein SpoOA, which switches on a series of operons (B). In SpoOA-active cells, SpoOA delays sporulation by switching on two operons, skf and sdp. The skf operon is involved in the production of an extracellular killing factor. Its other two products, SkfE and SkfF, antagonize the lethal action of the killing factor, probably by forming an export pump that pumps the factor out of the SpoOA-active cells. In contrast, cells containing active SpoOA are killed by the secreted killing factor, resulting in the release of nutrients that can be consumed by SpoOA-active cells. In this way, SpoOA-active cells can postpone sporulation. In both active and inactive SpoOA cells, a gene product of the sdp operon (SdpC) switches on expression of the transcription factor YvbA. This factor seems to delay sporulation in SpoOA-active cells, probably by activating lipid catabolism and ATP-producing enzymes. In contrast, in SpoOA-inactive cells, YvbA contributes to skf-dependent cell death, probably by inhibiting sigma factor σ^HI, which is responsible for detoxification and antibiotic resistance. Question marks and dashed lines represent sections of this model that are yet to be confirmed.
Global Climate Change Strikes a Tropical Lake

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Global circulation models predict that global warming caused by greenhouse gases will be particularly marked at high latitudes. Possibly so, but lower latitudes do not escape unscathed. On page 505 of this issue, Verburg et al. (1) demonstrate a profound effect of global warming on Lake Tanganyika, just south of the equator in tropical Africa.

Lake Tanganyika is one of the oldest and deepest lakes in the world. Its great mass of water gains and loses so little through rivers that its water renewal time is measured in millennia rather than years or decades. One might expect the great thermal inertia of so much water to buffer the lake against the vagaries of climate change. The work of Verburg et al. contradicts that expectation.

Many animal species that live nowhere else are endemic to Lake Tanganyika. There are hundreds of endemic species of fish, not all of which are in the notoriously fast-evolving cichlid family, and scores of endemic molluscs and ostracod crustaceans (see the figure). Even many genera are restricted to this single lake. Among the endemic species are bizarre specialists, such as a caddis fly larva that gyrates on the surface like a whirligig beetle, and a mastacembelid eel that swims on its side like a flatfish (2–4).

The lake contains two genera of small herrings, and many of its gastropod molluscs have spiny shells like those found on tropical seashores. This led early zoologists to postulate a previous connection to the sea, but such a possibility was disproved by geological mapping between the lake and the ocean. The spectacular fauna evolved to meet the challenge of a large and stormy lake during the millions of years since its formation.

Because of its great depth, Lake Tanganyika is perennially stratified. Dead organic matter sinks from the illuminated surface zone and decays as it falls, releasing nutrient materials such as silica and phosphorus. Organisms near the surface are chronically short of these nutrients, which they need for photosynthesis; in the depths, nutrients are plentiful but there is...

References