





ANDREW IERMY



uch like researchers, microorganisms suffer from stress. one source of which is the ultraviolet (UV) radiation in sunlight. But, as discussed on page 791 by Gao and Garcia-Pichel, various microorganisms, including bacteria, have developed the means to protect themselves from UV stress through the synthesis of compounds that act as microbial sunscreens. These compounds are often secondary metabolites, such as mycosporins. carotenoids and scytonemins, as well as melanin. Some of these compounds are already in use commercially as sunscreens for humans, and additional compounds could by applied similarly. Another way in which nearly all free-living bacteria respond to the presence of particular cellular stress signals involves toxin-antitoxin modules, as described by Yamaguchi and Inouye on page 779. Escherichia coli K-12 alone encodes at least 36 such modules. In the absence of the antitoxin, the toxin slows the growth of the cell, in some circumstances leading to cell death. Although the exact role of many of the toxin-antitoxin modules remains unclear, they can affect biofilm formation, drug resistance and entry into stationary phase. The stress response in microorganisms also often involves elaborate signalling cascades and regulatory networks that result in an altered transcriptional response. However, many post-transcriptional and post-translational effects can contribute to this response, thereby conferring ultrasensitivity and robustness to these systems. Ray, Tabor and Igoshin provide an overview of such non-transcriptional regulatory processes on page 817.

Finally, on page 771, Colby and Prusiner describe the rapidly moving research on the generation of infectious prion strains in the laboratory, which has provided insight into the structure of prions and the mechanisms that underlie their infectivity and propagation.

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