Current Topics

Should Microbiologists Mourn Lost Commensals when Species Go Extinct?

Some biologists tracking biodiversity closely watch tropical rain forests, worrying that human encroachment, which shrinks these lush habitats, could lead to massive species losses, as could global warming. Other biologists, noting that the human population is no longer growing so fast, argue that projected species losses are too high. Amid this unrest affecting macro species, what about microbial biodiversity, whose fate also depends on habitats and hosts? The global threats facing macrobiota could well apply to microbiota as well, even if this issue was not a formal component of the agenda during the symposium “Will the Rainforests Survive? New Threats and Realities in the Tropical Extinction Crisis,” convened last January by the Smithsonian Institution in Washington, D.C.

“Are we losing 100 species per day?” asks Nigel Stork, a symposium participant from the University of Melbourne in Australia. He and others noted that a faction of biologists began speaking of alarming species loss rates several decades ago. Their direct estimates varied from one-quarter to one-half of all species, with some projecting losses of about 30% of species per decade. “If they were right, we should have seen losses of 50% by now,” he says. Insects, totaling as many as 5 million species, could account for perhaps as many as one-half of all species, while about one-fourth, or somewhere between 1 and 2 million, are beetles, he adds. “We’re trying to come up with better estimates.” Beetles are survivors, he says, whereas bird species are going extinct possibly seven times as fast as beetles, a figure that he calls “very speculative.”

Looking beyond the birds and the beetles, “hunting and disease are the unseen killers of conspicuous and prominent vertebrate species,” says Elizabeth Bennett of the Wildlife Conservation Society in Bronx, N.Y. As forest lands contract, diseases and hunting are more apt to deplete wildlife, enabling “empty forest” syndrome to develop. For example, Ebola outbreaks, hunting expeditions, and shrinking habitats have reduced gorilla populations by an estimated 95%, she says. Fungal diseases, particularly in Australia, are threatening many amphibian species with extinction.

Climate change may pose a greater threat to habitats and biodiversity, according to Joseph Wright of the Smithsonian Tropical Research Institute in Balboa, Ancon, Republic of Panama. Higher temperatures, particularly in the tropics, are slowing the growth rates of trees in old-growth forests, perhaps making them more susceptible to extinction, he says. Coming up with accurate estimates for the rate and extent of global deforestation is also no easy task, according to other symposium participants, including Gregory Asner of the Carnegie Institution of Washington, whose laboratory is in Stanford, Calif. Current uncertainties notwithstanding, he says, “In the face of deforestation, the safety net for biodiversity is frayed.” During the past several decades, deforestation in some places was aggressive, leading to extensive damage. “Selective logging is massive, with 5

Large-scale damage to and/or encroachment on areas such as this Brazilian rainforest may threaten the survival of diversity in both macrobiota and microbiota. (Photo © Gregory G. Dimijian, M.D./Photo Researchers, Inc.)
Recent Developments Affecting Malaria

Recent developments involving clinical and preclinical studies of malaria include:

- The efficacy rate for RTS,S/AS01E, an experimental vaccine to protect against malaria, was 53% when tested among children in Kenya and Tanzania, according to Philip Bejon of the Kenya Medical Research Institute in Mombasa and collaborators from several institutions, who recommend that the vaccine be evaluated in a phase-3 clinical trial; for details, see the December 11, 2008 New England Journal of Medicine (NEJM).
- A similar vaccine formulated with a different adjuvant has a “promising” safety profile and protected about two-thirds of vaccinated infants in a clinical trial in Tanzania against breakthrough malaria, according to Salim Abdulla of the Research and Training Centre, Ifakara Health Institute in Bagamoyo, Tanzania, and collaborators, who also recommend a phase III trial; their report is in the same issue of NEJM.
- Despite cases of malaria in which patients in Western Cambodia were slow to respond to artemesinin, resistance to this key antimalarial agent “does not seem to be a widespread epidemiologic phenomenon,” according to Harald Noedl of the Medical University of Vienna in Vienna, Austria, and his collaborators in Cambodia and Thailand, whose report also appears in the December 11, 2008 NEJM.
- A phosphinate dipeptide interferes with aminopeptidases and is a potent inhibitor of malaria parasites, blocking their use of hemoglobin proteins, according to James Whisstock of Monash University in Melbourne, Australia, John Dalton at the University of Technology in Sydney, and their collaborators, whose report appears in the February 5 online Proceedings of the National Academy of Sciences.
- Particular cytochrome P450 genes in mosquitoes were newly identified as conferring resistance to pyrethroid insecticides, a finding that suggests inhibitors targeting those gene products could help in combating malaria and other mosquito-borne diseases, according to Charles Wondji and Hilary Ranson of the Liverpool School of Tropical Medicine and their collaborators, whose report appears online February 5 2009 in Genome Research. doi:10.1101/gr.087916.108.

Chemical Disrupts Biofilms, Enhances Antibiotics Used against Them

A fatty acid recovered from Pseudomonas aeruginosa potently prevents growth of and can disperse biofilms of this or other gram-negative or gram-positive bacteria as well as some fungi, according to David G. Davies of the State University of New York, Binghamton, and his collaborators. That relatively simple molecule not only disrupts recalcitrant biofilms but also enhances the activity of antibiotics that fail when used to treat infectious agents, including the notorious P. aeruginosa, particularly after they form biofilms.

That compound, cis-2-decenolic acid, operates apparently as a positive signal that is directly sensed by bacteria in biofilms. It then induces a cascade of responses, orchestrating the digestion of biofilm matrix polymers...
and retooling those cells for planktonic existance, Davies and his collaborators report in the March Journal of Bacteriology (191:1393–1403). This newly recognized chemical signaling agent thus “causes a switch in bacteria and in certain fungi from a biofilm to a planktonic phenotype, and acts much like a person shouting ‘fire’ in a crowded theater,” he says. Other factors that can trigger biofilm dispersal include nitric oxide, rapid changes in nutrient levels, surfactants, lysozyme, and soaps.

Efforts for Davies on biofilms date back nearly 20 years, and include his reporting a link between quorum sensing and biofilms about a decade ago. Like others studying these microbial assemblages, he was perplexed as to what controls traits such as their thickness, resilience, and durability. For instance, some biofilm experts thought that shear forces from fluid flow, perhaps combined with low nutrient levels, prevent biofilms from exceeding a maximum thickness. However, to the surprise of Davies and his collaborators, higher fluid flows, in tandem with low nutrients, increase biofilm thickness. Rather than growing to a certain thickness and stopping further growth in that dimension, he says, “the biofilm is a highly dynamic structure at steady state, with growth and dispersion occurring continuously.”

From experiments by Karin Sauer earlier this decade, Davies knew that phenotypic changes in bacterial cells occur when they are dispersed, rendering them less likely to form biofilms. Taken together with the more recent findings, he wondered whether a soluble factor acts as an antagonist to increases in biofilm thickness. If so, higher fluid flows would flush such a factor from growing biofilms, allowing them to thicken beyond the usual limits. Such factors, of course, might be metabolic waste products. The alternative—as proved the case with cis-2-decanoic acid—is that the factor belongs to the class of signal molecules that govern behaviors of microorganisms in biofilms.

“Over the past year, we have generated a considerable amount of data showing that susceptibility to disinfectants and antibiotics can be increased anywhere from half a log up to three logs and more when [antimicrobial] agents are used in combination with the dispersion autoinducer in vitro,” Davies says. “We may finally have an effective means to break the back of chronic infections that do not respond to antimicrobial therapies.” Moreover, he adds, this chemical communication system likely has deep evolutionary roots and may extend through many different taxa, and thus deserves further attention for other phenomena it may govern.

The implications are huge, according to others who study biofilms. “This is a big discovery considering the efficacy of the fatty acid against biofilms from gram-positive [and] gram-negative [bacteria] and yeast,” says Michael J. Schurr of the University of Colorado School of Medicine in Aurora. “The cis-2-decanoic acid may be used to treat all kinds of different infections that are recalcitrant to antibiotics. The fatty acid messenger may be used to break up established biofilms, which would allow antibiotics to be more effective. It may also help disrupt established biofilms in things like pipes and air conditioning units.”

“If you can disperse bacteria, you perhaps have the beginning of a novel and efficacious pharmaceutical that could treat infections, as most bacterial diseases in humans stem from biofilms,” adds Thomas K. Wood of Texas A&M University, College Station.

Even if these findings inspire new business plans, they seem unlikely to be headed toward practical means for treating infections with biofilm-forming pathogens or to other applications, according to Hans-Curt Flemming of the University of Duisberg, Germany, who also praises the quality of the research. For one thing, he says, “This fatty acid messenger is very specific for Pseudomonas, [and] it will have the same fate as the massively oversold quorum sensing molecules that were first thought to be the wonder weapon against [other] biofilms.” For another, the results “cannot be extrapolated to any environmental biofilm.”

David Holzman
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Epigenetic Changes of Several Viruses Tied to Cancer Development

Gradually developed epigenetic changes to the genomes of several distinct types of virus that infect humans appear to lead to development of cancers among those who are infected long term with those viruses, according to Manel Esteller of the Bellvitge Institute for Biomedical Research in Barcelona, Spain, and his collaborators. Thus, the genomes of Epstein-Barr virus, the human papilloma virus, and the hepatitis B virus become progressively methylated in those patients who developed cancer, distinguishing them chemically and epigenetically from the viral genomes of individuals who were asymptomatic carriers of those viruses, he says. These findings could lead to new means for studying, diagnosing, and treating viral-related diseases and cancer, he and his collaborators report. Details appear in the February 9, 2009, Genome Research doi:10.1101/gr.083550.108.
RNA Polymerase Trio from 1918 Influenza Strain Might Explain Its Lethality

Scientists continue to investigate factors that could account for the high lethality of the Spanish influenza virus, which killed at least 20 million people during the 1918 pandemic. Recent findings point to a set of RNA polymerase genes plus a nucleoprotein that apparently enabled the 1918 virus to move from nasal passages and grow in the lungs, causing severe edema and hemorrhage among the infected, according to virologist Yoshihiro Kawaoka at the University of Wisconsin (UW), Madison, and his collaborators. Their findings could make the flu RNA polymerase complex a marker for pandemic strains as well as a target for new drugs.

Ordinary “seasonal” flu viruses that circulate globally each year replicate mainly within the upper respiratory tract of infected individuals, setting them apart from the 1918 flu strain, whose victims experienced severe tissue damage from the virus in their lungs, according to autopsy findings from that era. What viral genes account for such differences? That question, which for decades was purely rhetorical, became approachable about 12 years ago when Jeffrey Taubenberger at the Armed Forces Institute of Pathology in Rockville, Md., determined the likely genomic sequence of the 1918 flu virus based on segments recovered from preserved specimens of lung tissues from pandemic victims.

More recently, Kawaoka and his UW colleagues used viral gene sequence data, plasmid-driven reverse genetics, and an ordinary circulating flu virus strain, designated K173 (A/Kawasaki/173/2001), to produce a set of recombinant viruses with different mixes of genes corresponding to its genome and the genome of the 1918 pandemic strain. These different viruses were inoculated under enhanced BSL3 conditions into the noses of ferrets, whose responses to flu infections closely resemble those of humans.

After three days, the recombinant that closely resembles the 1918 strain was actively replicating in the nose, trachea, and lungs of ferrets, while also causing bronchopneumonia, leading to severe lesions and hemorrhages in the lungs. In contrast, the K173 wild-type virus replicated only within the nose, as did other strains of the recombinant set that contained only single genes from the 1918 virus. However, a recombinant that contained a set of three 1918 polymerase genes, PA, PB1, and PB2, plus a 1918 nucleoprotein gene, grew in the trachea and lungs nearly as well as did the all-1918 virus. The nucleoprotein acts as a scaffold that the polymerases wrap around, and any further contribution of the nucleoprotein remains to be explored.

Thus, “the viral RNA polymerase complex played an important role in the spread of the 1918 virus from the upper to the lower respiratory tract,” according to Kawaoka and his collaborators, whose report is published in the January 13, 2009 Proceedings of the National Academy of Sciences. “We want to find out why the 1918 viral replication complex allows the virus to grow in lungs,” he says.

“This is a tremendously important study because now we can start to understand pathogenesis,” says virologist Robert Webster at St. Jude Children’s Research Hospital in Memphis, Tenn. Moreover, the findings point to “the polymerase gene complex as the next antiviral target.” Drugs that target the influenza RNA polymerases in theory could be effective against both seasonal flu and pandemic strains. With viral RNA polymerases as its target, investigators at Toyama Chemical Company in Tokyo, Japan, developed T-705, an orally administered candidate drug that now is being evaluated in Phase 1 clinical trials, Kawaoka points out.

Meanwhile, the Centers for Disease Control and Prevention announced

Survey Reflects Scientists’ Concern over Biodefense Research Misuse Risks

Some U.S. scientists are so concerned that microbiology and other research in the life sciences might be misused for bioterrorism that they have taken preemptive actions, “even in the absence of guidelines or government restrictions,” according to survey findings that were released in February by the National Academy of Sciences (NAS) in Washington, D.C. Such actions included broken collaborations, halted research projects, and results that were kept undisclosed, according to the report, “Survey of Attitudes and Actions on Dual Use Research in the Life Sciences: A Collaborative Effort of the National Research Council and the American Association for the Advancement of Science.” The survey results suggest that some scientists might be “responsibly responding to reduce the risk of misuse,” or that some of them could be “overreacting to the perceived threat,” says NAS committee chair Ronald Atlas from the University of Louisville in Louisville, Kentucky. Such uncertainties plus the low response rate from U.S. scientists who were contacted for the 2007 survey indicate follow-up investigation is warranted here and abroad, he notes. The report is available from the National Academies Press, http://www.nap.edu.
recently that the H1N1 influenza A strain that was circulating during the 2008–2009 flu season is resistant to Tamiflu, which targets the influenza neuraminidase. Such resistance underscores the need for alternative drugs for combating influenza infections.

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Vibrio Consortia, Warming Seas Aggravate Yellow Band Disease in Corals

A consortium of 10 Vibrio species, some well known for causing an epidemic of yellow band disease (YBD) among Caribbean coral reefs, also infects coral in the Pacific, raising questions as to how it traveled from one ocean to the other. Although six species within the consortium are novel, all members are like two shellfish pathogens, V. alginolyticus and V. harveyi. Moreover, the pathology in both oceans is the same “at the morphological and cellular levels,” and virulence rises in parallel with increases in ambient temperatures, according to James Cervino of Pace University, New York, N.Y., who is also a visiting scholar at Woods Hole Oceanographic Institute in Woods Hole, Mass., and his collaborators.

“In a global warming world, this disease is the worst possible scenario that can happen to corals,” he says. Details appear in the November-December Journal of Applied Microbiology (105:1658–1671).

Coral reefs are among the richest and most diverse habitats on earth. Coral are members of the phylum cnidaria, which also comprises jellyfish and sea anemones, and they live in symbiosis with the alga Symbiodinium zooxanthellae. In return for housing, these algae supply chemical energy to meet the needs of the reefs, which are stationary herds of animals, some of which live for hundreds of years.

This stability can be lost, however, when YBD strikes. YBD, or yellow blotch disease, produces characteristic pale yellow lesions and eventually kills symbiotic algae by impairing mitosis and damaging the photosynthetic apparatus. This condition now affects coral reefs throughout the Caribbean along with a rising percentage of reefs in the Pacific, including near Southeast Asia, says Cervino, who has tracked coral pathology for two decades. In controlled experiments in aquaria, the consortium of vibrios causes YBD in the Caribbean coral genus Montastraea, which is a major coastal reef builder, he says. Single Vibrio species are far less virulent than are clusters of such species.

Although YBD infections start at ambient temperatures, they spread with increasing efficiency as temperatures rise from 29–33°C, according to Cervino. Despite the importance of YBD, however, thermal stress remains the number one killer of coral through damage inflicted when algae, in response to rising temperatures are expelled from corals, bleaching and killing them, he says. In addition to heat stress and pathogen-inflicted damage, coral is sensitive to, and YBD is exacerbated by, two- to five-fold increases in nitrogen and phosphate levels. Those nutrients also exacerbate Aspergillus infections of coral. “If global warming-induced thermal stress and YBD infections continue to proliferate in the tropics, this will contribute significantly to the collapse of the remaining reef habitats in the next decade,” he and his collaborators warn.

Excesses of nonsymbiotic algae, a condition aggravated by overfishing, also can damage and kill coral, according to Eugene Rosenberg of Tel Aviv University in Israel. With the impact of overfishing in mind, Cervino plans to test whether microorganisms that grow in aquaculture pens also contribute to the progress of YBD when coral is exposed experimentally to Vibrio consortia.

Cervino’s findings are “important in the field because there has been a great deal of debate between microbiologists, who believe that bacteria are largely causing bleaching and eventual death in corals, and marine biologists, who believe that abiotic factors such as temperature, UV, and pollution...
cause coral bleaching,” says Rebecca Case of the Harvard University Center for the Environment, who was not involved in the research.

David Holzman

International Phobos-Sampling Mission Risks Putting Microbes on Mars

Russia is going back to Mars for the first time since losing a probe in 1996. However, the new probe being planned for launch this October, the Phobos-Grunt (Grunt from the Russian word for soil) is considerably more complex than was the mission that failed 13 years ago. Moreover, it risks transferring microorganisms being carried as part of the mission from Earth to Mars—a risk that has some experts worried that longstanding international agreements might be broken.

Once near Mars, Phobos-Grunt will launch a Chinese-made satellite into Martian orbit, then perform maneuvers before landing on the moon Phobos orbiting some 6,000 kilometers above the Martian surface. Once on the tiny 27-km by 19-km moon, the spacecraft will collect samples for return to Earth on a built-in rocket.

“The Phobos-Grunt mission will be the next step toward accomplishing a full Mars sample return mission and is a dress rehearsal for it,” says Louis Friedman, executive Director of the Planetary Society, a space advocacy organization that he founded with the late Carl Sagan.

The Planetary Society supports plans for returning samples from Phobos for analysis and also has its own experiment on the Phobos-Grunt probe consisting of a capsule carrying the living interplanetary flight experiment (LIFE). “LIFE is a container resembling a hockey puck that is 56 mm in diameter and 18 mm thick and weighing about 100 grams,” Friedman says. “It will hold 30 small tubes, each 3 millimeters in diameter, of terrestrial microorganisms.” The experiment will test the survivability of these microbes during 34 months in space.

“Between us and our European collaborators that include the Russian Academy of Sciences, Moscow State University, the American Type Culture Collection, and the Institute for Aerospace Medicine in Germany, we are sending three strains of Bacillus subtilis, Deinococcus radiodurans, Saccharomyces cerevisiae, seeds of Arabidopsis thaliana, Haloarcula marismortui, and tardigrades [microscopic, water-dwelling, segmented animals known as water bears], each in triplicate to obtain better science results,” says David Warmflash, a member of the Planetary Society and an associate with the National Astrobiology Institute and National Aeronautics and Space Administration (NASA) Johnson Space Center in Houston, Tex. “We have included representatives of the three domains of life on Earth: bacteria, eukaryota, and archaea.”

However, not everyone is enthusiastic about the experiment, including Catharine Conley, who is the NASA Planetary Protection Officer. “There is considerable discomfort within the planetary protection community regarding the advisability of sending living Earth organisms to Mars, even though under nominal conditions they would never reach the Martian surface,” she says.

The problem is that missions can fail, releasing pieces that land in unexpected places. For instance, the NASA 1999 Polar Lander as well as the 1988 Russian Phobos 2 spacecraft and the 2003 UK Beagle 2 likely crashed on Mars, dispersing their respective cargoes. When the Columbia Space Shuttle broke apart in 2003 upon entering Earth’s atmosphere, it included containers of microorganism that were strewn along with other shuttle debris during its fiery breakup.

In 1967 the United Nations Committee on the Peaceful Uses of Outer Space drafted The Outer Space Treaty, which was signed and ratified by key nations, including China,
Metal Balance Helps Explain Survival of Microbial “Superhero”

“If there’s a superhero microbe, it’s Deinococcus radiodurans,” says Michael J. Daly from the Uniformed Services University of the Health Sciences in Bethesda, Md. For this bacterial extremophile to withstand massive doses of radiation and other physical insults, he adds, “what really counts is not just Mn$^{2+}$ accumulation, but the balance between Mn$^{2+}$ and Fe$^{2+}$ as well as the ability of manganese to form free-radical-devouring chemical complexes.” He reports in the March issue of Nature Reviews Microbiology (7:237–245) that this and other microbial species with high manganese-to-iron ratios are extremely resistant to $\gamma$-radiation-induced protein oxidation, while those with low manganese-to-iron ratios are hypersensitive.

$D.\ radiodurans$ is “endlessly fascinating but very stubborn and malodorous,” Daly continues noting that “it’s also virtually impervious to desiccation and easily survives massive exposures to ionizing radiation, both X-rays and $\gamma$-rays, ultraviolet light, and chemical oxidizing agents.” In 2007, Daly showed that the hardiness of $D.\ radiodurans$ comes from protecting its proteins with accumulated manganese (Mn$^{2+}$) ions, thus sparing a sufficient number of enzymes critical for repairing its genome (Microbe, July 2007, p. 327; http://www.asm.org/microbe/index.asp?bid=51529).

Now, Daly and his collaborators report that $D.\ radiodurans$ and other similarly gifted microbes depend on particular metal ions as part of their protein-sparing apparatus. Notably, $D.\ radiodurans$, which has very efficient systems for Mn$^{2+}$ uptake, typically accumulates 100 times more manganese than do radiation-sensitive bacteria. “Unlike ferrous ions (Fe$^{2+}$), Mn$^{2+}$ ions are innocuous in aerobic environments with virtually no negative redox consequences,” he says. “Fe$^{2+}$ but not Mn$^{2+}$ catalyzes the Fenton reaction, one of the most powerful oxidizing reactions known.” Further, extreme radiation and desiccation resistances depend on formation of superoxide-scavenging Mn$^{2+}$-phosphate complexes and accumulation of hydroxyl radical-consuming small organic molecules.

“X-ray fluorescence microspectroscopy has just shown that manganese is dispersed throughout $D.\ radiodurans$, but much of its iron is partitioned between dividing cells, which helps explain how global enzyme protection is accomplished,” Daly says. “Because the hydrogen peroxide (H$_2$O$_2$) generated during irradiation diffuses widely, manganese and iron partitioning serves to minimize the Fenton reaction.” In contrast, iron-rich and manganese-poor bacteria suffer a torrent of reactive oxygen species (ROS) during irradiation, which inactivates many enzymes. “Unless an irradiated cell can protect its enzymes from oxidation, even the most minor DNA damage will kill it,” he notes.

Knowing that diploid yeast cells can recover from exposure to $\gamma$-radiation, Daly is developing “Deinococcus-inspired” radioprotectants—combining Mn$^{2+}$ with ligands such as phosphate and other small molecules. “The right mix, when delivered into human cells, could spontaneously form intracellular complexes that scavenge superoxide and related ROS,” he says. Potential applications include “making radiation therapy more tolerable for cancer patients, protecting astronauts from radiation during long-duration space travel, cleaning up the ‘slumgullion’ of radioactive waste left over from the Cold War, and developing ways to slow down the aging process. I’m excited that in the last few years, this research has moved from the realm of science fiction to plausible reality.”

Daly’s results are being closely monitored by other scientists, including cell biologist Colin Dingwall at Kings College London. Dingwall has shown that BACE1, or beta-secretase, a principal component of senile plaques, is linked to copper in the brains of patients with Alzheimer’s disease. “Substituting Mn$^{2+}$ for Cu$^+$ to prevent redox chemistry is an interesting idea and, if it works, small mol-
ecules such as peptides might be used as delivery agents,” he says.

“Daly’s convincing demonstration that simple manganese complexes protect proteins from oxidative damage in vivo makes me wonder if manganese is acting similarly in more complex organisms,” says biochemist Joan S. Valentine from the University of California Los Angeles, adding that “perhaps the antioxidant effects of manganese supplementation that we’ve been attributing to increases in manganese superoxide dismutase enzymes might really be due to simple manganese complexes.”

Commenting on how <i>D. radiodurans</i> evolved its manganese-based resistance to high-dose radiation, Rodney L. Levine from the National Institutes of Health in Bethesda, Md., says “it’s unlikely that it evolved to survive high-dose radiation as such, it’s more likely an example of cross-resistance, probably acquired as a consequence of its ability to survive desiccation; organisms which evolve or induce a resistance to one stress are more often than not resistant to multiple other stresses.” But, he adds, “as Daly points out, we live in a DNA-centric world which holds that cells die because of genome injury, and this is not entirely correct. <i>Deinococcus</i> DNA is as diced and sliced by irradiation as that of <i>E. coli</i>, but <i>Deinococcus</i> survives when <i>Escherichia</i> dies. Daly’s experimental data show why this happens; it’s all about the proteins.”

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