Ecological recovery after the Mount St. Helens eruption in 1980 has proceeded with minimal human interference and with notable success; however, the recovery of tropical marine ecosystems such as coral reefs after large natural disturbances "is almost invariably confounded by anthropogenic stresses." The idea that *Pseudomonas aeruginosa* combines the genetic strategies of mutation and recombination, evolving hypermutable and hyperrecombinant strains, to adapt to the complex and harsh environment in the lungs of cystic fibrosis patients is discussed. And the relative contributions of nongovernment and government scientists, researchers in the biotechnology industry, and policy-makers to the agricultural biotechnology debate are examined.

Confounding Factors in Coral Reef Recovery

The effects of the Mount St. Helens eruption on the surrounding forest and the processes affecting recovery from this major disturbance, which Franklin and MacMahon discuss in their Perspective "Messages from a mountain" (*Science*'s Compass, 19 May, p. 1183), are similar to the effects of hurricanes on coral reefs. For both the forest and the reef, major natural disturbances leave most of the ecosystem structure intact, and recovery can begin from surviving organisms (or, in some cases, portions of organisms) (*1*). Both the species composition and the physical structure of these ecosystems influence the course of recovery.

Buck Island Reef National Monument, St. Croix, after Hurricane Hugo in 1989.

In the case of Mount St. Helens, the recovery process has had little interference from humans. In contrast, the recovery of coral reefs from hurricanes is almost invariably confounded by anthropogenic stresses, most notably fishing. Overfishing can delay reef recovery by removing herbivorous fishes that keep algal growth in check. If grazing is not intense enough, algae (particularly "fleshy" seaweeds) can inhibit both growth of adult coral colonies and settlement or survival of coral recruits (*2*). Humans have now degraded tropical marine ecosystems to the point where our ability to evaluate ecological theories about succession and effects of disturbance has been compromised.

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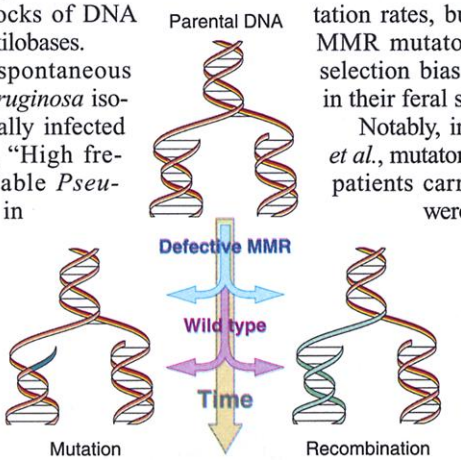
Pseudomonas Survival Strategies in Cystic Fibrosis

Pseudomonas aeruginosa is a versatile species exhibiting variable colony morphologies, multiple ecological niches, and diverse disease consequences. Several laboratories have observed this adaptability at the molecular level, especially when the cystic fibrosis (CF) lung is a reservoir for chronic infection by the organism. Römling *et al.*, for example, compared 18 CF isolates of clonal strain C of *P. aeruginosa* and showed that the chromosome of each isolate had a hypervariable structure (*1*). The complex pattern of DNA rearrangements in the chromosome included large inversions and acquisition of blocks of DNA ranging from 1 to 214 kilobases.

In their study of spontaneous mutation rates of *P. aeruginosa* isolates from 30 chronically infected CF patients (Report, "High frequency of hypermutable *Pseudomonas aeruginosa* in cystic fibrosis lung infection," 19 May, p. 1251), Oliver and colleagues found that the lungs of 11 out of the 30 CF patients were colonized by a hypermutable (mutator) strain of the organism. In contrast, a mutator phenotype was not found in 75 non-CF patients acutely infected with *P. aeruginosa*. Oliver *et al.* and Rainey and Moxon, authors of the accompanying Perspective "When being hyper keeps you fit" (*Science*'s Compass, p. 1186), ascribe the evolution of these mutator phenotypes to the hitchhiking of mutator alleles within the adaptive mutants they spawned—seemingly, an inherent consequence of being a haploid organism. Moreover, Oliver *et al.* suggest that selection pressure (the unfavorable environment within the lungs of CF patients subjected to antibiotic challenge and other undefined stresses) culls and enriches mutators of *P. aeruginosa*, analogous to the specific selection schemes used for in vitro enrichment of *Escherichia coli* (*2*) and *Salmonella enterica* (*3*) mutators.

The studies by Oliver *et al.* complement the molecular and genetic studies highlighted above. Specifically, their results suggest that, in addition to the hypermutable phenotype, promiscuous recombination in mutator strains might be a significant source for adaptive change in *P. aeruginosa*. When we reported on the high incidence of mutators found among pathogenic strains of *E. coli* and *S. enterica* (*4*), we proposed that the hyper-recombinagenic nature of particular mutators contributed to the evolution of these enteric pathogens, indicating that horizontal transfer and homeologous recombination (recombination between diverged DNA sequences) helped frame the genetic diversity of these populations. All mutators found (predominantly with mutations in the *mutS* gene) in our studies were deficient in methyl-directed mismatch repair (MMR), a pathway that plays a central role in blocking the recombination of mismatched DNA. We reasoned, therefore, that it was not (solely) the hypermutable phenotype, because defects in any of 25 genetic loci in *E. coli* increase spontaneous mutation rates, but the promiscuity of MMR mutators that provided the selection bias for mutator bacteria in their feral settings.



Mutation and recombination occur on a shortened time frame in MMR-deficient bacteria.

Notably, in the report by Oliver *et al.*, mutators from at least 4 of 11 patients carrying mutator strains were defective in the *mutS* gene. The *mutS* mutator, unlike any other mutator alleles, plays an exacting and singular role in homeologous recombination (*5*). These MMR mutator strains could be the mixing pools where chromosomal changes—from ei-

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