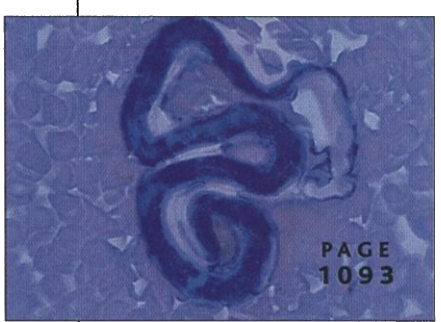
INTRODUCTION

Sex, Lineages, and Pathogenesis

For microbiology, times have never been so good; we are luxuriating in a surfeit of data. The more genomes we see, the greater the opportunity for comparative analysis. There is no shortage of leads for new drugs and vaccines and no lack of putative virulence determinants to explore. But to reap the full benefits of this research, we need to consider the big picture. People are not infected with one organism alone—we are host to communities of many species, most of which do us little harm. We need to spot the shift in the dynamics between microbe and host that tells us when harm might follow.

This issue of *Science* addresses some of the wider implications of the current renaissance in microbiology. Ochman and Moran (p. 1096) argue that sequencing the genomes of pathogens gives us a distorted view of bacterial diversity, because pathogens are a small, modern, and unusual proportion of bacterial species. Although a quick-change act is an advantage to a pathogen, the development of clonal lineages is the hallmark of many microbial infections. Read and Taylor (p. 1099) observe that within an afflicted individual, the competition between lineages will influence disease severity and transmission—factors that are often ignored in clinical decisions.

Baranowski *et al.* (p. 1102) point out that small changes in viral genomes can mean a significant change in the distribution of the virus in the host, which will have important repercussions for vaccine use and the risks from xenotransplantation. Although some retroviruses, such as HIV, are notorious for their high rates of sequence variation, Overbaugh and Bangham (p. 1106) explain that viruses that do adapt rapidly tend to be ecological generalists. As Woolhouse *et al.* (p. 1109) discuss, these include the many zoonotic human infections of increasing public health concern.



Although the mammalian gut is packed with microorganisms, inflammatory responses are rare. Levin and Antia (p. 1112) examine the immune responses to bacteria, comparing it to predator-prey dynamics and pointing out the risk of mounting a full inflammatory immune response to infection. Hooper and Gordon (p. 1115) expand on the cross-talk between gut commensals and the host, and Russell and Rychlik (p. 1119) describe the specialized symbioses of ruminants and how they are affected by animal husbandry.

In News, Carl Zimmer describes how genetic analyses of living pathogens are enabling researchers to reconstruct the origins and evolution of disease (p. 1090). Zimmer also details the extraordinary antics of *Wolbachia*—perhaps the most common infectious bacterium on Earth—and how it manipulates the sex lives of its hosts to ensure its own survival (p. 1093).

The plethora of new data should help answer long-standing questions about infection, revealing unique metabolisms vulnerable to drugs and vital surface molecules open to vaccine attack. More important, they will offer new insights into the ecology and evolution of infection. (For Web resources related to this issue, see www.sciencemag.org/feature/data/diseases/index.shtml)

–CAROLINE ASH AND LESLIE ROBERTS

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