Citation: Ewald, Paul W., "The Evolution of Virulence," Scientific American, April 1993, vol. 268, no. 4, pp. 86-93

SCIENTIFIC AMERICAN

April 1993 Volume 268 Number 4

Scientific American (ISSN 0036 8733), published monthly by Scientific American, Inc., 415 Madison Avenue, New York, N.Y. 10017-1111. Second class postage paid at New York, N.Y., and at additional mailing offices. Authorized as second class mail by the Post Office Department, Ottawa, Canada, and for payment of postage in cash. Canadian GST No. R 127387652. Subscription rates: one year $36 (outside U.S. and possessions add $11 per year for postage). Subscription inquiries: U.S. and Canada 800-333-1199, other 515-247-7631. Postmaster: Send address changes to Scientific American, Box 3187, Harlan, Iowa 51537. Reprints available: write Reprint Department, Scientific American, Inc., 415 Madison Avenue, New York, N.Y. 10017-1111, or fax: (212) 355-0408.

DEPARTMENTS

The Evolution of Virulence

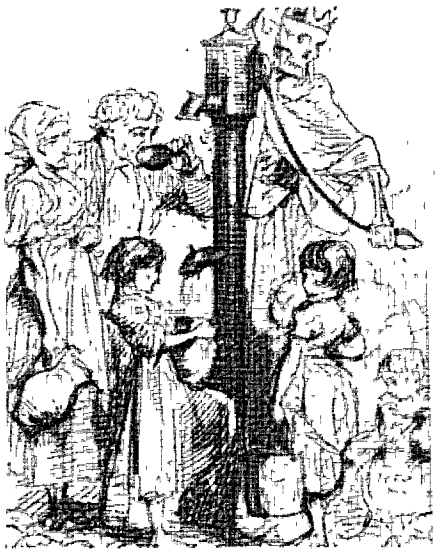
Human behavior appears to influence whether pathogens evolve into benign or harmful forms. Health policy should therefore include evolutionary considerations

by Paul W. Ewald

Some pathogens, such as those that cause cholera, smallpox, tuberculosis, malaria and AIDS, have quite severe effects. Others rarely inflict any damage beyond a cold or a sore throat. Recent studies suggest there are several evolutionary reasons for these varying levels of virulence. They include a pathogen's mode of transmission as well as its ability to survive outside a host organism for long periods. Human behavior may also play a significant, largely unrecognized role in the evolution of pathogens because it often determines the route and timing of transmission.

Understanding the forces that shape changes in virulence could become a powerful tool for medicine. By examining these variables, evolutionary biologists have already been able to predict patterns of morbidity and mortality in several diseases—including cholera, dysentery and AIDS. Using such an approach, medical scientists may be able to anticipate alternative evolutionary courses of pathogens and to tailor treatment and social behavior accordingly. They might even be able to transform virulent adversaries into mild ones.

Until recently, the understanding of how virulence evolves has generally been limited to one view. Most physicians and medical writers have concluded, unjustifiably, that the evolution of



host-parasite relations ultimately leads to benign coexistence. Their opinion is based on the idea that parasites that do not harm their hosts have the best long-term chance of survival: they thrive because their hosts thrive.

Some biologists, however, have arrived at a different conclusion. Evolutionary theory holds that what is best for a species may differ from what is best for its component individuals and that what is best for these individuals is defined by which genes are passed along most successfully. Even if a pathogen reproduces so extensively that it causes its host to become gravely sick, its host-impairing instructions may still win out over the less damaging instructions of a less aggressive competitor. The more virulent pathogen would achieve this success if its increased replication led to a level of transmission into new hosts that exceeded the loss of transmission resulting from the host's illness or death. In this scenario, the extinction of the host may eventually cause the demise of any pathogen that relies exclusively on one host. But the possibility of extinction does not inhibit the spread of the more virulent genes within the population of pathogens.

This perspective suggests that a parasite's virulence may reflect its mode of transmission. If the illness of a host impairs transmission, evolutionary biologists would predict that parasites would evolve to have milder effects. In contrast, if the host's disability does not inhibit transmission, pathogens could gain a competitive advantage by reproducing more rapidly.

Consider, for instance, a pathogen that relies on the mobility of its host. Rhinoviruses, causes of the common cold, reproduce in the cells that line the nasal passages. The viruses are shed in nasal secretions that trickle out as a runny nose or blast out during a sneeze. A person's finger may wipe away the mucus and may then touch the fingers of another person in the course of a handshake or by way of a borrowed pencil. The exposed individual may later touch fingers to nose or inhale the contaminated air, planting rhinoviruses on fertile ground.

Whichever route is taken, the ability of the host to move is critical. If the pathogen reproduces so extensively that the host is too unwell to leave home, thousands of rhinoviruses shed that day will die of exposure. A few might squeak by if they infect a family member, but such transmission could hardly be considered a great accomplishment, especially if the new host also becomes bedridden. For rhinoviruses, a person's movement improves prospects for transmission. Accordingly, rhinovirus replication is restricted to cells scattered in a sea of uninfected nasal mucosa, and virulence remains slight.

UNPROTECTED WATERS, including wells (*above*) and rivers, such as the Yamuna in India (*right*), have been infamous disseminators of disease. Purification limits the spread of infection. It also may provide an unrecognized benefit: it may force pathogens to evolve into mild forms.

PAUL W. EWALD is associate professor and chair of the biology department at Amherst College. In 1980 he received his Ph.D. in zoology from the University of Washington, where he studied the evolutionary and ecological aspects of aggression in birds. Two years ago the Smithsonian Institution named Ewald the first George E. Burch Fellow of Theoretical Medicine and Affiliated Science. When not researching evolutionary epidemiology, Ewald continues to investigate animal behavior. He also works as an unpaid carpenter with the hope of nursing his colonial-period farmhouse into its second quarter-millennium.