maternal bacterial infection is mimicked in rodents by injection of lipopolysaccharide, an immunogenic bacterial component (*17*).

Although a genetic element clearly contributes to schizophrenia and other mental disorders, the maternal-fetal environment must also be taken into account. Environment can alter genetic outcomes and vice versa, and future research must both tease the two influences apart and consider them together to better understand the onset, progression, and treatment of mental disorders.

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10.1126/science.1150196

ECOLOGY

Thinking Long Term

Thousand-year records of animal population patterns and climate yield insights into the impacts of environmental change.

Robert A. Cheke

Ecologists seeking patterns in populations and environmental correlations dream of coming to grips with lengthy data sets. Usually, animal numbers are determined both by density-independent environmental factors and by density-dependent population processes involving time lags. Disentangling these different factors requires painstaking fieldwork and mathematical skills from the scientists and the patience of Job among funding agencies. Two new analyses of 1000-year-long data series illustrate how long series can reveal insights and improve predictions of pest outbreaks (*1*, *2*).

The value of long-term data. Recent studies of data sets spanning over 1000 years have shed light on the environmental factors that influence the population cycles of larch budmoths (**left**) and Chinese migratory locusts (**right**).

Caterpillars of the larch budmoth (see the figure, left) can reach densities of 30,000 per tree when they defoliate larch trees and inhibit tree growth, effects detectable as narrow growth rings. Esper *et al.* recently examined larch wood from the European Alps dating back 1173 years (*1*). The results show that budmoth outbreaks have occurred every 9.3 years on average since 844 C.E.; the authors attribute their absence since 1981 to contemporary warming, which stimulates early egg development and premature hatching. This may be good news for the trees, but is it yet another sign of the effects of anthropogenic climate change?

Thinking of insects' activities more than a thousand years ago recalls biblical accounts of plagues of desert locusts, but there is no continuous historical record of such plagues before the 20th century. However, a Chinese Emperor instigated the sporadic collection of data on Chinese migratory locusts (see the figure, right) as early as 707 B.C.E., and his successors maintained a continuous series of annual records from 957 C.E. (*3*–*5*). Stige *et al.* have now reanalyzed these data in the context of rainfall and temperature changes (*2*). As in time series of desert locusts (*6*), brown locusts (*7*), and Australian plague locusts (*8*), the data are not insect numbers but proxies based on numbers of administrative areas infested. Significant relationships with rainfall can be found in all of these locusts, but how rainfall affects the insects' survival may vary according to species, depending on whether they have eggs that can remain dormant for a year or longer and so survive droughts, and on the spatiotemporal distribution of the rain. For the Chinese locusts, Stige *et al.* show that both floods and droughts are important, with temperature and rainfall interacting to set the scene (*2*). The study also emphasizes the importance of low-frequency phenomena, which involve effects discernible at time scales longer than a year. These are known in many ecosystems and were detected in desert and brown locusts as unexplained 16- and 17-year cycles, respectively (*6*, *7*).

Previous studies of the Chinese locust (*3*–*5*) focused on interannual rather than longer-term variations, with one notable exception showing that population variability increased at longer time-scales (*9*). Stige *et al.* have now re-examined the data at lower frequencies than annual. In a kind of ecological archaeology, they used mean decadal temperature (derived from ice cores, tree ring data, lake sediments, and contemporary records) and mean decadal rainfall (based on samples of juniper that tally with precipitation indices) to show that there were more locusts when the climate was cold and wet and fewer when it was warm and dry.

The authors find that these climatic effects accounted for locust variability for periodicities of 30 years or more. Decadal frequencies of droughts and floods have a multiplicative effect on the locusts. Both droughts and floods are more common in cold, wet periods, conditions associated with high locust numbers because droughts allow the insects to lay eggs on riverbanks and lakesides; retreating floods also provide ideal breeding conditions. These responses detected at decadal scales have important practical implications: A projected warming Chinese climate would be expected to lead to fewer locusts as a result of a reduced breeding habitat, despite a positive association between locusts and temperature at the annual scale (*3*).

Frequency-dependent effects of this kind may need to be taken into account to correctly interpret other phenomena liable to disruption by global warming, such as wind systems

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that affect locust migrations and the mixing of swarms originating from different sources. Examinations at finer scales than the whole of China and further understanding of the interactions between subpopulations are needed. Desert locusts, for instance, have regional populations whose dynamics are cross-correlated (*10*).

Further insights from China are likely after the compilation of meteorological and ecological records from the past 3000 years (*11*). Science needs such long data sets and the financial commitments to provide them. Some series could be reconstructed, as in the larch budmoth case, but finding biological data sets on a par with those for the budmoths and locusts will need imagination and help from historians. The Chinese Emperors thought long term, and so should we, by maintaining current data collection programs essential for the understanding of contemporary phenomena in the short, medium, and very long term, perhaps 1000 years hence.

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10.1126/science.1150636

MICROBIOLOGY

Deadly Priming

Roberto Kolter

Bacteria behave like cells of a multicellular organism, secreting a molecule that primes a subpopulation to commit suicide during stressful conditions.

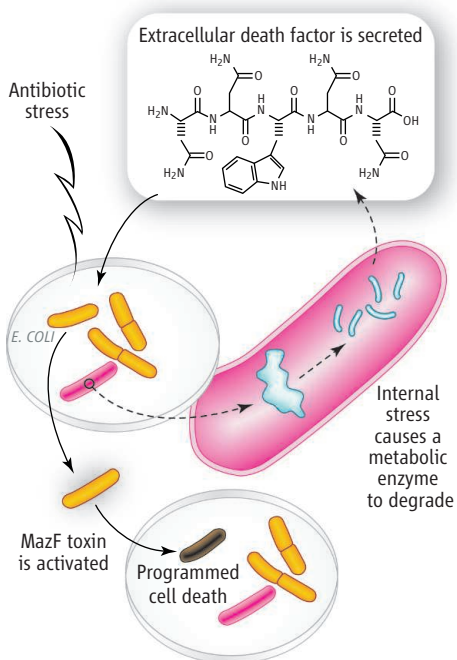
Constituent cells of an organism communicate with each other through chemical signals to coordinate growth and differentiation. Cells also perish along the way, their programmed death benefiting the organism's survival (*1*). Bacteria also "talk" to each other through chemical signals and, on occasion, kill themselves (*2*, *3*). This suggests that they are capable of multicellular behaviors (*4*). On page 652 of this issue, Kolodkin-Gal *et al.* show that the bacterium *Escherichia coli* releases a signaling molecule that activates a programmed cell death pathway, supporting the concept that multicellularity is a general bacterial trait (*5*).

Programmed cell death mechanisms in bacteria often follow a common theme: A lethal toxin is constitutively produced and, at the same time, an antitoxin is made for protection (*3*). When synthesis of both is maintained, the bacterial cell survives even though the antitoxin is usually more labile than the toxin. But when their synthesis is arrested, the greater lability of the antitoxin eventually unmasks the toxin's activity, leading to the cell's demise. The lethal effect of these toxins can be due to a variety of activities, such as inhibiting DNA replication or translation.

Bacterial toxin-antitoxin systems were first found encoded in plasmids, autonomously replicating extrachromosomal elements whose inheritance is not always guaranteed upon cell division. However, any cell that fails to inherit the plasmid will soon find itself without any antitoxin and dead. The surprise came when similar toxin-antitoxin coding modules were found in bacterial chromosomes. Why would a bacterium want to kill itself? Such a response makes little sense for an individual cell. But when that cell is a member of a multicellular aggregate, its death could benefit the rest. For example, the development of fingers in a human embryo requires the cells between the fingers to undergo programmed cell death.

Many of the known stimuli that trigger programmed cell death in bacteria are environmental stresses, such as exposure to antibiotics or ultraviolet light. Kolodkin-Gal *et al.* now demonstrate that for these stresses to be effective, a self-generated signal must also be present. Prior results indicated that toxin-mediated cell death could only be observed in cultures with high population density (*6*). Kolodkin-Gal *et al.* determined that cell-free medium from a high-density culture can induce programmed cell death in cultures with low population density. The culprit turned out to be a linear pentapeptide in the medium.

Perhaps the most exciting finding in this report is the source of the pentapeptide. Many self-generated, secreted signaling molecules in bacteria are small molecules, including peptides (*2*). So the fact that the "extracellular death factor" discovered by Kolodkin-Gal *et al.* is a peptide should come as no surprise. But the peptide signals described in earlier studies are encoded in small genes that generate prepeptides, which are processed to yield the signaling molecule. What is striking about the *E. coli* peptide is that it is derived from the degradation of glucose-6-phosphate dehydrogenase, a metabolic enzyme (see the figure). Although the exact pathway leading to the production of this pentapeptide remains to be defined, it seems reasonable that it is made when the bacterium, for reasons unknown, begins to destroy this enzyme. Thus, an apparent internal stress, such as the onset of starvation, could generate this signal, which is secreted (perhaps by some moribund cells in the population) and primes the population such that some cells can be killed through the pro-



Spreading the message. A bacterial metabolic enzyme is degraded, yielding a peptide that is released. When sensed by cells in conjunction with environmental stresses, such as antibiotic exposure, toxin-mediated programmed cell death is triggered.

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