PERSPECTIVES: ECOLOGY

Invariants, Scaling Laws, and Ecological Complexity

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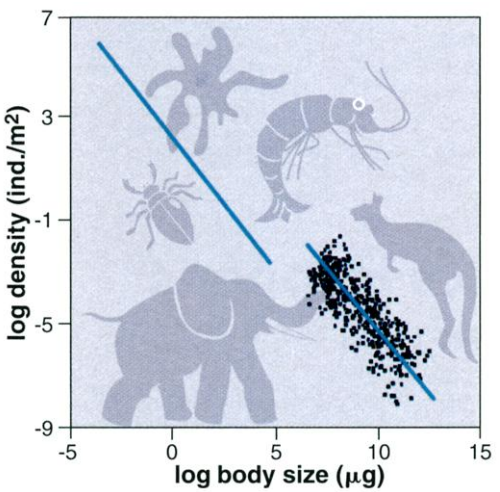
Life on Earth has diversified to occupy every kind of environment. Within these environments, diffuse associations of organisms form ecological communities. To understand the structure and dynamics of these communities, ecologists have adopted two major approaches. The experimental, microscopic approach emphasizes the highly variable and idiosyncratic nature of communities (*1*). The alternative macroscopic, nonexperimental (macroecology) approach emphasizes the existence of statistical patterns in the structure of communities that seemingly reflect the operation of general principles or natural laws (*2*). The report by Schmid *et al.* (*3*) on page 1557 of this issue sheds new light on a well-known and much debated macroecological pattern: the relation between population density and body size. These investigators thoroughly analyzed the relation between population density and body size in more than 400 invertebrate species—from insects to tiny single-cell organisms (such as the testate amoeba, which weighs about 10^{-4} μg)—in two local stream communities, one in Austria and the other in Wales. Their study is among the first to take a statistically rigorous look at the density–body mass relation across a large number of invertebrate taxa that includes microorganisms. They demonstrate that the inverse scaling law (population density decreases as body size increases) holds across all the taxonomic groups and that it is not limited by energy requirements. (Given a fixed amount of available energy, species of small body size that require less energy to survive should be more abundant than larger species.)

As a colleague of mine likes to put it, the diversity of life is largely a matter of size. And he is right, for life spans more than 21 orders of magnitude. Macroecologists are well aware of this diversity and have commonly searched for empirical statistical patterns relating the size of organisms, usually expressed in mass units, with ecological and evolutionary traits (*4*). These relations take the form of a power law: $Y = Y_0 \times M^b$ where Y is the dependent variable (usually a physiological, ecological, or evolutionary attribute); Y_0 is a normalization constant; M is the independent variable, usually body mass; and b is the scaling exponent. These scaling relations have the valuable property of becoming linear after logarithmic transformation, and the scaling parameter b can be estimated as the slope of the regression between log Y and log M. The scaling relation between population density and body mass has been the subject of intensive empirical research for more than 60 years, but, as yet, there is no consensus over its form, let alone the mechanisms that generate it (*5*).



Big is beautiful but lonely. The inverse scaling relation between population density and body size. Small organisms typically attain greater population densities than do larger species. The upper line describes the relation between population density and body size fitted to data collected from two totally separate local stream communities (*3*), where organisms ranged in size from insects to the testate amoeba. For comparison, the bottom line depicts the same relation for all mammals worldwide (*8*). Lines are fitted with the OLS-bisector method, and in both cases they have a slope or regression coefficient that is not different from –1.0.

The study by Schmid *et al.* (*3*) demonstrates that in local stream communities there is a strong negative scaling relation between population density and body size, in agreement with previous work in rocky intertidal habitats (*6*). Taken together, these findings make a strong case for the existence of a simple inverse relation between population density and body mass in local communities (see the figure). This is in contrast to the notion that polygonal relations—characterized by ample scatter restricted to a defined region of the population density–body mass plot—should be the common outcome at local scales (*7*). Furthermore, the thoroughness of Schmid *et al.*'s sampling methods makes the existence of sampling biases against small, rare species unlikely and also controls for artifacts that may arise from different census areas (*5, 7*).

The Schmid study also conveys the need for caution. To assess scaling relations in local ecological systems, the full diversity of the community must be represented together with carefully designed and standardized sampling schemes. The observed scatter in the relations reported by Schmid and coworkers suggests that if they had restricted their analysis to a smaller range of body sizes (by excluding the amoeba), they would have obtained a more polygonal and still negative but nonlinear scaling relation.

Compilation analyses of published data for closely related species worldwide (*8*) typically report that the slope of the relation between population density (D) and body mass (M) is about –0.75 using ordinary least squares (OLS) regression. Because metabolic rate (MR, an estimate of the energy required by an individual for the basic processes of living) increases with body mass raised to the 0.75 power, the scaling exponent of this relation has been taken as evidence that the abundance of species is limited by energetic requirements (*8*). Similarly, the total energy consumed by a species population per unit area (EU) can be assessed by multiplying the energy used by an average individual by the number of individuals per unit area. Thus, if both variables are related to body mass with similar exponents, but opposite sign, then the energy used by different species should be roughly equal and independent of body mass (that is, $EU = MR \times D \propto M^{0.75} \times M^{-0.75} \propto M^0$). This is the so-called "energy equivalence rule" (*8*). Hence, depending on the estimated scaling exponents, larger, smaller, or none of the species use a disproportionate amount of the available energy within ecosystems. Although these kinds of algebraic operations have been questioned (*9*), the statistical issue of what is the best method to

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estimate scaling relations still remains contentious. Several studies have recommended alternatives to OLS regression, such as reduced major axis regression (RMA) (*10*), because both population density and body mass are subject to measurement error. Schmid and colleagues go one step further and apply a new method, the OLS-bisector: This method calculates the line that bisects the OLS (X versus Y) and OLS (Y versus X) best-fit lines. With this approach they demonstrate that the scaling exponent of population density is different from that of metabolic rate, and that the former varies across functional and taxonomic groups, implying that the pattern cannot be explained solely by the energy equivalence rule.

The OLS-bisector method is commonly used by astrophysicists and has been shown to outperform other approaches if both variables are subject to measurement error and it is not clear which variable should be treated as the independent and which as the dependent (*11*). The usual convention in allometric studies is to use body size as the independent variable (*12*). However, there is no way to logically prove the independent nature or causal role of body size (*13*) because body size and physiological or ecological traits do not evolve in isolation, but affect each other. In fact, plant ecologists have traditionally treated the sizes of individual plants as though they were determined by population density (the Thinning Law) (*14*).

The study of whole communities containing a wide array of taxonomic groups is a daunting task. Such an approach is badly needed, however, if we are to glean insights into the structure of ecological systems. The identification of invariant scaling relations as reported by Schmid *et al.* (*3, 6*) and, more generally, the existence of simple scaling laws (*15*) suggest that general principles underlie the complex organization of ecological systems.

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PERSPECTIVES: GENOMICS

Genomics Happens

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Modern epidemiology is rooted in the work of John Snow, an English physician whose careful study of cholera victims led him to discover the waterborne nature of this disease. Cholera also played a part in the foundation of modern bacteriology—40 years after Snow's seminal discovery, Robert Koch developed the germ theory of disease following his identification of the comma-shaped bacterium *Vibrio cholerae* as the agent that causes cholera (see the figure). Koch's theory was not without its detractors, one of whom was so convinced that *V. cholerae* was not the cause of cholera that he drank a glass of it to prove that it was harmless. For unexplained reasons he remained symptom-free, but nevertheless incorrect.

A notable addition to *V. cholerae* research comes from Heidelberg *et al.* (*1*) who report in a recent issue of *Nature* the complete genome sequence of its two circular chromosomes (*2*). The sequencing team discovered a total of 3885 open reading frames (lengths of DNA that encode proteins): 2770 on the larger chromosome 1 (2.9 million base pairs) and 1115 on the smaller chromosome 2 (1.1 million base pairs). Slightly more than 50% of these open reading frames encode proteins homologous to proteins of known function; the remainder encode proteins without ascribed functions or that are not homologous to any known protein.

Cholera continues to be a scourge throughout much of the world with seven global epidemics (pandemics) recorded since 1817. In 1991, for the first time in 100 years, cholera arrived in the Western Hemisphere from its focal center in Asia. Cases were first reported in Peru, and epidemics throughout South and Central America rapidly followed. The sequenced *V. cholerae* isolate, El Tor strain N16961, is representative of the strains causing the current pandemic. Between epidemics, *V. cholerae* lives in aquatic environments, often in association with marine invertebrates. Although most isolates of *V. cholerae* do not cause disease, some carry genes that have enabled the microbe to adapt to humans and to produce virulence factors. Upon infection of humans, these strains colonize the gut mucosa and produce cholera toxin (CT), which stimulates secretion of water and electrolytes by gut epithelial cells leading to severe diarrhea that can be fatal within hours (see the figure).

Even before completion of the *V. cholerae* genome sequence, it was well established that virulence genes directing the interaction between this pathogen and its human host were acquired by pathogenic *V. cholerae* from bacteriophage and from large gene clusters called pathogenicity islands by horizontal gene transfer. Two major virulence factors, CT and a colonization factor called toxin-coregulated pilus (TCP), are each encoded on genetic elements in chromosome 1 that are not universally found in *V. cholerae* strains; yet they are regulated by genes that appear to predate entry of these elements into the genome (*3*). The genes encoding CT are acquired from the genome of bacteriophage CTXϕ (*4*); those encoding TCP are carried on an element called the *Vibrio* pathogenicity island (VPI), also thought to be of bacteriophage origin (*5, 6*). The genome sequence data challenges the notion that the VPI is of bacteriophage origin because none of the VPI genes encode phage structural or morphogenetic proteins. An integron island (a system for gene capture and dissemination) on chromosome 2 is also likely to be im-

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