at least some functional information about one of its domains is thus quite good. Genetic manipulation in yeast is easy and cheap, whereas such manipulation, even when possible in mammalian systems, is neither easy nor cheap. There is in addition the opportunity to exploit functional compatibility by the method described above for the RAS genes. At least 71 human genes complement yeast mutations; this is certain to be an underestimate (6). Thus, information about human genes learned from studying their yeast homologs comes at an excellent price.

Probably the best examples of the value of yeast as a model system concern human disease genes that have been mapped by linkage, positionally cloned, and then sequenced. Usually nothing is known of these genes beyond the fact that their inheritance results in disease. The sequence of the gene generally provides the first clue to function by way of homology to the genes of other organisms, commonly *S. cerevisiae* (7). Among the best matches are the human genes that cause hereditary nonpolyposis colon cancer (*MSH2* and *MLH1* in yeast), neurofibromatosis type 1 (*IRA2* in yeast), ataxia telangiectasia (*TEL1* in yeast), and Werner's syndrome (*SGS1* in yeast). Two of these have particularly illustrative stories.

Inherited nonpolyposis colon cancers have a cellular phenotype: instability of short repeated sequences in the tumor cells. Stimulated by this result, and even before the human genes had been cloned, yeast researchers isolated mutations in yeast genes with the same phenotype (including mutations in *MSH2* and *MLH1*), predicting that the colon cancer genes were likely to be their homologs (8).

Werner's syndrome is a disease with several hallmarks of premature aging. Again there is a cellular phenotype, which includes a reduced life-span in culture. The sequence of the human gene was found to be highly similar to that of the yeast *SGS1* gene, which encodes a DNA helicase. On page 1313 of this issue, Sinclair *et al.* (9) report that *SGS1* mutant yeast cells have a markedly reduced life-span and share other cellular phenotyes with cells from individuals with Werner's syndrome.

So yeast has indeed turned out to be a useful "model" for eukaryotic biology. There is ample justification for intensifying efforts to determine the functional roles of the remaining 60% of yeast genes whose function is still not known. There are as well many individual reasons to focus even more attention on genes such as *MSH2* and *SGS1*. These yeast genes may represent the most efficient path to understanding the colon cancer and the aging caused by mutations in their human homologs.

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ECOLOGY

Biodiversity and Ecosystem Function: The Debate Deepens

J. P. Grime

We continue to lose species and genetic diversity locally, nationally, and planet-wide. In deciding priorities for conservation, there is an urgent need for criteria that help us to recognize losses with potentially serious consequences. It would be naïve to assume that species-poor ecosystems are always malfunctional; some of the world's most extensive and ancient ecosystems—boreal forests, bogs, and heathlands—contain few species. For both species-rich and species-poor ecosystems, we need to establish whether current losses in biodiversity are likely to seriously impair functioning and reduce benefits to humans. This problem is serious enough that the United States and the United Kingdom have invested recently in costly ventures specifically designed to test experimentally the consequences of reduced diversity on ecosystems.

Model communities with controlled levels of species diversity have been created in the Ecotron at Silwood Park in southern England and at the Cedar Creek Reserve in Minnesota to assess the effects of diversity on various ecosystem properties such as primary productivity, nitrogen mineralization, and litter decomposition. Early publications from both sites (*1*, *2*) claimed to demonstrate benefits to ecosystem function arising from higher levels of biodiversity, and these have been highlighted by commentators (*3*, *4*) excited by the prospect of a scientific underpinning for conservation measures.

This view that "biodiversity begets superior ecosystem function" is not shared by all ecologists (*5*, *6*). There are obvious conflicts with published evidence from work on natural rather than synthesized ecosystems. As early as 1982, Leps *et al.* (*7*) had suggested that ecosystem processes were determined primarily by the functional characteristics of component organisms rather than their number. The same conclusion was drawn by MacGillivray *et al.* (*8*) who showed that differences between five adjacent ecosystems in northern England in their responses to frost, drought, and burning were predictable from the functional traits of the dominant plants but were independent of plant diversity.

This edition of *Science* (pages 1296, 1300, and 1302) includes three contributions (*9*–*11*) to this important debate. One is a report of results from the Cedar Creek synthesized plant assemblages, whereas the two others describe biodiversity-ecosystem studies conducted on natural systems (mediterranean grassland in California and northern forest in Sweden). In all three, variation in ecosystem properties is found to be related to differences in the functional characteristics, especially resource capture and utilization, of the dominant plants, and there is no convincing evidence that ecosystem processes are crucially dependent on higher levels of biodiversity. The evidence presented by

The author is in the Unit of Comparative Plant Ecology, University of Sheffield, UK. E-mail: j.p.grime@sheffield.ac.uk

Wardle *et al.* (*10*) is particularly compelling because it involves an extensive study of ecosystem properties on 50 relatively pristine forested islands of varied size and plant biodiversity. It is clearly shown that a suite of ecosystem properties—including higher microbial biomass, high litter quality, and more rapid rates of litter decomposition and nitrogen mineralization—coincide with the lower botanical diversity and the earlier successional state of the vegetation on larger islands (both consequences of the higher incidence of lightning strikes and more frequent fire history of larger islands). On small islands, succession proceeds uninterrupted to more species-rich vegetation, but here the dominant plants, *Picea abies* and *Empetrum hermaphroditum*, are extremely stress tolerant and produce litter of poor quality, thereby slowing the rates of ecosystem processes. This strongly supports the contention of MacGillivray *et al.* (*8*) that it is the biological characteristics of the dominant plants rather than their number that control ecosystem productivity and biogeochemistry. This same conclusion is prompted by the new data presented by Tilman *et al.* (*9*) and Hooper *et al.* (*11*). Both of these groups have adopted a more experimental approach and created ecosystems in field plots where they can control both the functional composition and species richness of the vegetation. Here again, there is strong evidence that productivity and nutrient cycling are controlled to an overwhelming extent by the functional characteristics of the dominant plants, and evidence of immediate benefits of species-richness within functional groups remains weak.

Biodiversity. *By a Shady Track in Coombs Dale* [Adapted from a drawing by Gail Furness]

Why is a different perspective emerging from these more recent studies conducted on model systems and under more natural conditions? In a penetrating critique of earlier work, Huston (*12*) has pointed out that several of the apparent benefits to ecosystem function reported in the model experiments can be explained as consequences of inappropriate experimental design and faulty interpretation of data. In particular, he believes that the supposed benefit to productivity associated with greater biodiversity in the Ecotron experiments is attributable to the fact that the more diverse communities that were created contained larger and more productive plant species that were omitted from the experimental assemblages of the less diverse communities. A key publication (*1*) from Cedar Creek claimed that both the resistance and resilience of vegetation to drought were increased by species richness. Huston reminds us, however, that the drought-sensitive vegetation involved in these experiments was not only species poor but was also very different functionally as a consequence of heavy and sustained applications of inorganic fertilizer. A recent reanalysis of this work (*13*) recognizes that drought resilience (recovery) was not more rapid in the unproductive but more diverse ecosystems; this brings the Minnesota findings into closer agreement with the earlier results from Leps *et al.* (*7*) and again points to an interpretation in which the functional characteristics of component species take precedence over their number.

It could be argued that the tide is turning against the notion of high biodiversity as a controller of ecosystem function and insurance against ecological collapse. However, such a stance would be as premature as that of the commentators who rapidly embraced early evidence of its supposed benefits. It is obvious that for all ecosystems a point could be reached at which further loss of key species could impair functioning and usefulness to humans. The most immediate problem is to identify irreplaceable species and functional types and to discover whether there are situations in which ecosystem viability depends on unusually high biodiversity. We might speculate that high biodiversity may be vitally important in structurally diverse ecosystems such as layered forests or in ecosystems that experience drastic fluctuations (*14*) on a seasonal or longer time scale (for example, flooded forests, lake shores, and semi-arid ecosystems).

What lessons can be learned from the recent history of research on the significance of biodiversity? First, I suspect that we need a more integrated approach with greater input from those scientists with specialist knowledge of the functional biology and resource dynamics of key plants and animals. Both laboratory experiments and studies of natural ecosystems must be informed by a knowledge of resource dynamics and should be designed as tests of predictions on the basis of the functional attributes of component plants and animals.

Perhaps most important of all, we should reconnect recent endeavors on the functional significance of biodiversity with an older and extensive literature on the mechanisms controlling biodiversity itself. This would be to reassert a more Darwinian perspective in which high species-richness is viewed not as an attribute of certain ecosystems but instead as a function of population processes associated with special circumstances that hover precariously between two different forces for extinction (extreme habitat conditions and competitive dominance) (*15*). So far, neither evolutionary theory nor empirical studies have presented convincing evidence that species diversity and ecosystem function are consistently and causally connected.

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