rection by means of a rotating electric vector of light (see figure). Conservation of angular momentum determines how the light pulse is coupled to the electron spin. With the input into the device and the measurement both accomplished by optical means, it might be possible to limit the time of each operation to the nanosecond range. The spin relaxation time found by Kikkawa fits in well with this range. The authors are careful to note that their method of measuring the long spin memory time, while establishing limits to the external sources of spin decoherence, does not rule out decoherence of individual spins involving mutual electron spin interaction.

In metals the conduction electron spin relaxation time decreases drastically with increasing temperature because of increasing lattice vibration, which affects the electron spin coherence through the spin interaction with its orbital motion (6). It is then a puzzle why the spin relaxation time in the semiconductor system of Kikkawa *et al.* (*1*) is so weakly temperature dependent.

Kikkawa *et al.* find that doping (that is, the introduction of electrons into the system) increases the spin relaxation time by a factor of 10 to 100. This effect occurs only if the excited electron density is a sizable fraction of the doped electron density. How does this sea of electrons, which has no net spin, help to maintain the spin coherence of the excited electrons? One possibility is the polarization of the electron sea by the optically excited electrons with a common spin direction. The Coulomb interaction among the electrons creates a collective spin that is then not easy to perturb. The spin polarization of the electron sea had earlier been observed (*7*), but the relaxation time was of the order of 100 ps at 10 K.

The long spin relaxation time found by Kikkawa *et al.* means sufficient time for controlling the spin dynamics. This is an important first step toward using semiconductor systems for coherent quantum devices. The fundamental physics of this phenomenon is interesting and could be very rich because it depends on a variety of interactions effects. The next step is to see if quantum coherence control is feasible. The construction of a quantum logic gate would be an encouraging demonstration of the possibility of coherent quantum semiconductor devices.

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GENETICS

Yeast as a Model Organism

David Botstein, Steven A. Chervitz, J. Michael Cherry

The first complete DNA sequence of a eukaryotic genome, that of the yeast *Saccharomyces cerevisiae*, was released in electronic form more than a year ago (*1*). No doubt, each member of the international consortium of yeast biologists made the argument to his or her own funding agency in Europe, Japan, Britain, Canada, or the United States that this yeast would be a fine "model organism," useful for interpreting and understanding human DNA sequences. How right were they?

It was clear long before the systematic sequencing of genomes began that there are genes in yeast and mammals that encode very similar proteins (*2*). Some homologies—including proteins of molecular systems (for example, the ribosomes and cytoskeletons)—were no surprise. Some were quite unexpected, however. A particularly arresting early example was the discovery in yeast of two close homologs (*RAS1* and *RAS2*) of the mammalian *ras* proto-oncogene; yeast cells lacking both genes are inviable. In 1985 this system was the occasion for the first of many deliberate tests of functional conservation: The mammalian H-*ras* sequence was expressed in a yeast strain lacking both RAS genes, with the remarkable result that viability was restored, indicating a profound conservation not only of sequence, but also of detailed biological function (*3*).

With the entire yeast genome sequence in hand, we can estimate how many yeast genes have significant mammalian homologs. We compared (*4*) all yeast protein sequences to the mammalian sequences in GenBank [EST (expressed sequence tag) databases were not included]. The result (see the table) is encouraging: For nearly 31% of all the potential protein-encoding genes of yeast (open reading frames, or ORFs), we found a statistically robust homolog among the mammalian protein sequences (*5*). This is clearly an underestimate, as the databases surely do not yet contain the sequences of all mammalian proteins or even representatives of every protein family. Many of these similarities relate individual domains, and not whole proteins, no doubt reflecting the shuffling of functional domains characteristic of protein evolution.

Mammalian homologs (based on *P* value)			
P value	Number of ORFs at *P* value or lower	Percent of total ORFs (n = 6223)	Percent of ORFs with unknown function
1×10^{-10}	1914	30.8	34
1×10^{-20}	1553	25.0	30
1×10^{-40}	1083	16.8	26
1×10^{-60}	784	12.6	23
1×10^{-80}	576	9.3	22
1×10^{-100}	442	7.1	21
1×10^{-150}	221	3.6	23
1×10^{-200}	101	1.6	25

Even though *S. cerevisiae* is among the best-studied experimental organisms, 60% of its genes still have no experimentally determined function. Of these, the majority nevertheless have some similarity or motif suggesting possible functions, leaving about 25% (by actual count) with no clue whatever. In compiling the data in the table, we observed that genes with homology to mammalian sequences are much less likely to have nothing experimental known of their function. Only 34% of the entire set of yeast genes with mammalian homologs have no function listed in the *Saccharomyces* Genome Database; compared to less than 25% of the genes having the strongest homology. We do not know the reason for this, although we do not rule out the optimistic idea that yeast biologists have succeeded in concentrating on the most important genes (those most likely to be conserved).

The likelihood that a newly discovered human gene will have a yeast homolog with

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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.

References and Notes

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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

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