were declining and now restricted to a significantly smaller range (Proc. Natl. Acad. Sci. USA *108*, 662). Looking for possible causes, the researchers found that the declining species had a significantly higher prevalence of infection with *Nosema*. They also discovered that the declining populations had reduced genetic diversity compared with the stable ones. Establishing causal links between these observations, however, will require further research, the authors say.

The authors also observe that the species affected by decline in North America have previously had a wide climatic range. By contrast, studies in Europe have found that species with a narrow climatic range are most at risk. This contrast suggests that different causes and mechanisms may be behind the decline on both continents.

The simultaneous threats to both the domesticated honey bees and the wild pollinators are bound to have repercussions throughout the natural environment and are also putting agricultural production and food supplies at risk. George McGavin commented: "The global threat to bees is a greater threat to our survival than global warming. This is a total ecological disaster we can avoid." Considering the scale of the industries affected, government spending on bee health has remained minuscule. McGavin calls the £1 million support that bee researchers get from the UK government "laughable". The EU has so far been inactive, but in January the European Commission acknowledged the importance of the problem and announced the installation of a European reference laboratory for bees' health to be based in France.

Tennekes concludes his analysis of the impact of neonicotinoids on wildlife in the Netherlands: "Ground and surface water contamination with persistent insecticides that cause irreversible and cumulative damage to aquatic and terrestrial (non-target) insects must lead to an environmental catastrophe. The data presented here show that it is actually taking place before our eyes, and that it must be stopped."

More research and political action is required to ensure that we don't, after all, experience what Rachel Carson anticipated 50 years ago: a silent spring.

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Quick guide

Neurospora

Eric U. Selker

What is* Neurospora*? *Neurospora* is the genus of a group of filamentous fungi but the word is most often used as a nickname for the best studied species, *N. crassa*, which has served as a model eukaryotic organism for nearly a century. The name *Neurospora* apparently came from the nerve-like stripes found on its sexual spores ('ascospores'; Figure 1). *Neurospora* is easily recognizable by its orange aerial asexual spores ('conidia').

What is its life style? The haploid vegetative filaments ('hyphae'), which look somewhat like axons (Figure 2), weave together to form a mat ('mycelium'). *Neurospora* grows at a prodigious rate — the mycelium advances at ~4 mm per hour in a reasonably warm environment if given some sugar, simple nutrients, and one vitamin (biotin). *N. crassa* is 'heterothallic' meaning that it has different subtypes ('mating types') that must find each other to enter the sexual phase of the life cycle. About 10 days later, its fruiting bodies ('perithecia') shoot the ascospores towards light. Germination of ascospores requires heat (for example, 65°C for an hour), which kills other cells in the neighborhood and accounts for reports of *Neurospora* in French bakeries in the 1800s and for the presence of *Neurospora* in burned sugar cane fields and burned forests in modern times.

What was* Neurospora *first known for? Research in the 1920s and 1930s revealed *N. crassa* to be a convenient and powerful genetic system; indeed it became a textbook example of first-division and second-division segregation, with easily demonstrable crossing over at the four-strand stage, and provided the first proof of gene conversion. The fact that it could be easily grown on defined media led to its adoption for the Nobel-prize winning 'one gene–one enzyme' work of Beadle and Tatum in the 1940s, which demonstrated that genes

Figure 1. A dissected perithecium of *N. crassa* with octets of ascospores (stripes not visible at this magnification) showing segregation of a color marker (courtesy of N. Raju).

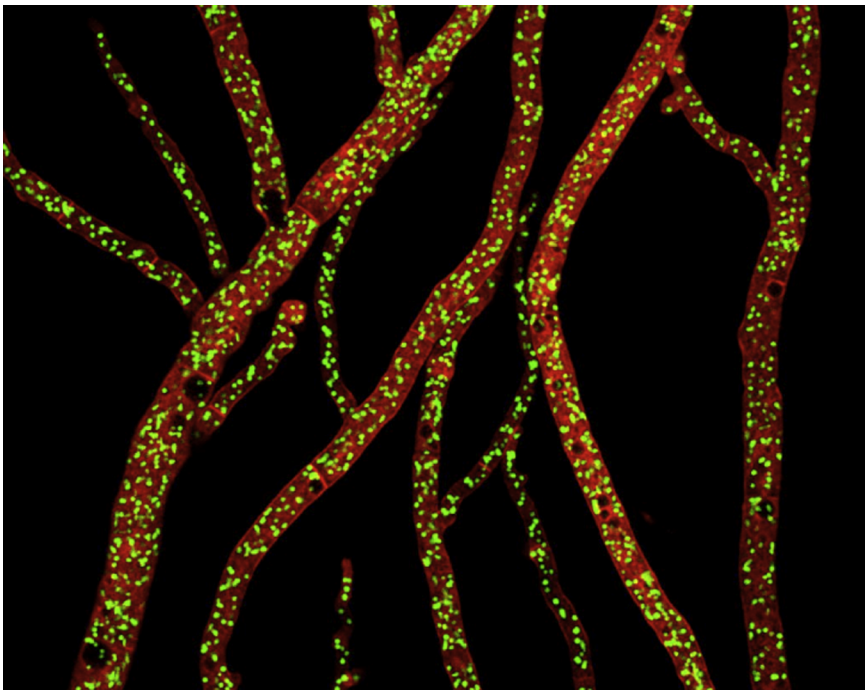

Figure 2. GFP-labeled histone H1 in *N. crassa* hyphae (courtesy of P. Hickey).

control biochemical processes. This and similar studies established the field of biochemical genetics and effectively initiated the discipline now known as molecular biology. Since then, *Neurospora* has served as a useful model in a large number of studies on problems in biochemistry, genetics, cell biology, development, physiology and population genetics, generally predating similar work with bacteria, yeasts, *Drosophila*, worms, mice, plants and other model systems.

Why is* Neurospora *still a favored model organism? Although *Escherichia coli* and yeast ultimately became more popular than *Neurospora* for studying many basic problems in molecular biology and genetics, *Neurospora* offers features not found in these and other eukaryotic systems. Thus, *Neurospora* is still regarded as an exemplary system for numerous genetic and molecular studies. Some of the reasons that it is an excellent model organism are: 1. *Neurospora* is easy to grow (in either liquid or solidified medium) and to store in suspended animation; 2. the haploid vegetative tissue is handy for scoring genetic traits and generating heterokaryons (strains with genetically distinct nuclei), useful for complementation tests; 3. *Neurospora*'s rapid and well-defined sexual cycle, compact genome with small, but cytologically recognizable, chromosomes that can be readily modified using either classical or molecular techniques makes the organism well-suited for genetic studies; 4. thousands of genes/mutations have been characterized and a high-quality genome sequence is available; 5. extensive collections of wild and constructed strains are readily obtainable, including knock-out mutants for the majority of known and predicted genes; 6. *Neurospora* sports features of higher eukaryotes that are absent from many other simple systems, for example DNA methylation and other 'epigenetic' marks, photobiology, circadian rhythms, gene silencing systems, cytoplasmic streaming, vegetative incompatibility reactions, morphogenesis; 7. modern tools are available for *Neurospora*, such as materials and methods to silence genes, to introduce genes at either homologous or non-homologous genomic sites and to perform proteomic studies; 8. the *Neurospora* community is unusually friendly and cooperative. It is noteworthy that a few talented, dedicated and altruistic *Neurospora* researchers, including the late David Perkins and the late Bob Metzenberg, were instrumental in publicizing the virtues of *Neurospora* while demonstrating that the use of *Neurospora* as a model system can be highly productive and fun. *Neurospora* meetings, which take place at Asilomar conference grounds in even years, typically draw 150–200 participants and welcome newcomers.

Can you tell me something wild about* Neurospora*? Starting with the discovery of 'repeat-induced point mutation' (RIP) more than two decades ago, *Neurospora* has revealed several remarkable and unexpected genetic mechanisms that serve to counter invasive DNA. RIP, which operates in the sexual phase of the life cycle in the period between fertilization and nuclear fusion, scans the haploid genome for duplicated sequences, such as those commonly resulting from the activity of transposable elements. Such sequences are then inactivated with multiple C to T mutations as well as with methylation of remaining cytosines. In addition, in vegetative cells, repeated sequences commonly generate aberrant RNAs that trigger silencing via an RNA interference (RNAi) mechanism called 'quelling'. Finally, in meiosis, unpaired sequences, such as those resulting from insertions or deletions in one parent, cause temporary silencing by another RNAi-based mechanism known as 'meiotic silencing by unpaired DNA' (MSUD).

Where can I find out more?

Borkovich, K.A., Alex, L.A., Yarden, O., Freitag, M., Turner, G.E., Read, N.D., Seiler, S., Bell-Pedersen, D., Paietta, J., Plesofsky, N., *et al.* (2004). Lessons from the genome sequence of *Neurospora crassa*: Tracing the path from genomic blueprint to multicellular organism. Microbiol. Mol. Biol. Rev. *68*, 1–108.

Davis, R.H. (2000). Neurospora: Contributions of a Model Organism (Oxford University Press).

Davis, R.H., and Perkins, D.D. (2002). Timeline: Neurospora: a model of model microbes. Nat. Rev. Genet. *3*, 397–403.

Galagan, J.E., Calvo, S.E., Borkovich, K.A., Selker, E.U., Read, N.D., Jaffe, D., FitzHugh, W., Ma, L.J., Smirnov, S., Purcell, S., *et al*. (2003). The genome sequence of the filamentous fungus *Neurospora crassa*. Nature *422*, 859–868.

Griffiths, A.J.F., and McCluskey, K. *Neurospora* information website. http://www.fgsc.net/Neurospora/neurospora.html.

Perkins, D.D. (1992). Neurospora: the organism behind the molecular revolution. Genetics *130*, 687–701.

Perkins, D.D., and Davis, R.H. (2000). Neurospora at the millennium. Fungal Genet. Biol. *31*, 153–167.

Rountree, M.R. and Selker, E.U. (2009). Genome defense: The Neurospora paradigm, in: Epigenomics, edited by A.C. Ferguson-Smith, J.M. Greally, and R.A. Martienssen (pp. 321–341).

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