Editorial

Via media

The gene for the ergosterol biosynthetic enzyme C-14 reductase is named *ERG24* in *Saccharomyces cerevisiae* and *erg-3* in *Neurospora crassa*. [While we're on the subject, I have to correct the misspelling of Sydney Brenner's name in the previous editorial. I mixed it up with Sidney Poitier's, who has a daughter named Sydney. Sorry.] *ERG24* and *erg-3* research has taken different trajectories, driven by mutant/wild-type differences on variously supplemented media, bringing to mind Marshall McLuhan's famous aphorism (coined in another context), 'the medium is the message'.

ERG24 was cloned by Leo Parks and colleagues. First, they selected a fenpropimorph-resistant (*fen*^{r}) mutant on YPD (yeast extract + peptone + dextrose) medium supplemented with the C-14 reductase inhibitor. Wild-type yeast accumulates ignosterol in the presence of fenpropimorph, and its growth is inhibited. The *fen*^{r} mutant also accumulated ignosterol in the presence of fenpropimorph, but its growth was not inhibited. This suggested that an *erg24* mutant would be lethal in the wild-type background but viable in the *fen*^{r} background. Then, anticipating the mutant sterol would confer resistance to nystatin, they selected the *erg24* mutant on YPD + nystatin. *ERG24* was cloned by complementation of the mutant (*DNA Cell Biol.* **11** 685–692, 1992). Disruption of *ERG24* in the wild type was lethal, but the disruptant could be maintained by anaerobic growth on ergosterol-supplemented YPD (*J. Bact*. **178** 2991–2993, 1996). Adding 1.0 mM calcium alleviated the aerobic growth inhibition on YPD, and revealed the relationship between ergosterol biosynthesis and calcium homeostasis (*Curr. Genet.* **34** 93–99, 1998).

The Neurospora *erg-3* mutant was selected for nystatin-resistance by Morris Grindle and shown to be C-14 reductase deficient (*J. Gen. Microbiol.* **137** 2627–2630, 1991). We found it was super-sensitive to the pea isoflavonoid pisatin, cloned the wild-type allele via complementation, and demonstrated its orthology with *ERG24* (*J. Genet.* **73** 33–41, 1994). Although *erg-3* was not noticeably fastidious about calcium, mutant ascospores produced colonies with a distinctly different morphology on sorbose-supplemented medium (*Fungal Genet. Biol.* 31 91–97, 2000). Given that calcium gradients underlie hyphal tip growth (*Microbiology* **149** 2475–2485, 2003), this might well reflect abnormal calcium homeostasis at hyphal tips.

Minimal medium midwived molecular biology. 'Medium No. 3' enabled George Beadle and Ed Tatum to hunt for Neurospora nutritional mutants. David Perkins recounted that medium no. 3 could be made up only to a 2× concentration without precipitating, and therefore it was replaced by Vogel's Medium N, which could be made up as a 50× stock. 50× Medium N calls for 10 % ammonium nitrate, a potentially explosive salt. Robert Metzenberg's alternative recipe for Medium N noted that 'the tendency of the dry salt to become caked in its container invites foolish actions to free up a sample, for example, banging the jar on the edge of a benchtop' (*Fungal Genet. News.* **50** 14, 2003).

Mites and insects are sterol auxotrophs. Mites consume 10–50 μg of Neurospora mycelium per mite per day (*Ann. Entomol. Soc. America* **53** 549, 1960). Since testing the *erg-3* mutant for mite-resistance ran the risk of infesting the laboratory, we reared Drosophila on an agar medium made with powdered lyophilized Neurospora mycelium and glucose. Although Drosophila did well on 'wild-type Neurospora' medium, things got more interesting when the flies were challenged with *erg-3* medium (unpublished results).

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