› NEWS UPDATES

A 'filmier' way to grow mold

An improved *in vitro* method for growing the fungus *Aspergillus fumigatus* better resembles the *in vivo* attributes of this sometimes deadly mold. The ability to better approximate *in vivo* conditions in the laboratory may allow researchers to study aspects of *A. fumingatus in vitro* that were not available using previous protocols.

Every day, humans inhale hundreds of *A. fumigatus* spores from the air. In most people, the immune system quickly clears the spores; but, as the number of immunocompromised individuals has increased in recent years, so has the number of *A. fumigatus* infections; it is now the most common airborne fungal pathogen.

In the past, researchers have grown *A. fumigatus* in shaken flasks of liquid, but this system does not mimic key *in vivo* features of *A. fumigatus* growth. Using a new technique more similar to conditions in the lung, Anne Beauvais and coworkers at the Pasteur Institute (Paris, France) grew the mold on static gel open to the air (*Cell. Microbiol.*, doi:10.1111/j.1462-5822.2007.00895.x). The researchers found that colonies grown by this method were surrounded by a sugary biofilm. Lung biopsy of *A. fumigatus*-infected Swiss mice demonstrated that the biofilm exists *in vivo*. Further investigation of the fungus using this model will hopefully yield insight into its pathobiology.

Un-clefting palates *in utero*

Using a 'chemical genetics' approach, researchers have rescued *in utero* mice engineered to have cleft palates, shedding light on the genetic underpinnings of the condition and demonstrating the feasibility of chemical prevention of developmental defects.

A cleft palate results from failed fusion of the two skull plates that form the hard palate. The condition, which occurs in 1 in 2,000 births, may result in feeding problems, speech irregularities, and ear disease, but is generally corrected by surgery in infancy.

Now, in the 1 March issue of *Nature*, Michael T. Longaker and colleagues at Stanford University School of Medicine (Palo Alto, CA) report work that may lead to the ability to arrest development of the defect before birth. They created a line of mice carrying a chemically regulated allele of *glycogen synthase kinase 3-β* (*GSK-3β*), a gene known to be involved in a slew of biological processes, including neuronal cell development and body pattern formation. The allele, tagged with FRB*, a small molecule that alters protein function, results in an unstable chimeric protein; $GSK\text{-}3\beta^{FRB^*/FRB^*}$ mice are phenotypically indistinguishable from $GSK\text{-}3\beta^{-/-}$ mutants, displaying cleft palates and sternal defects. Treatment of pregnant dams with a drug that stabilizes the protein during the developmental window of palate formation—thus restoring GSK-3β activity—rescued $GSK\text{-}3\beta^{FRB^*/FRB^*}$ pups from developing cleft palates.

Reversing Rett syndrome

Researchers have reversed the neurological defects associated with Rett syndrome in a murine model. Although the results do not herald immediate elimination of the symptoms of Rett syndrome in affected girls, they suggest that the condition may someday be treatable.

Rett syndrome is a severe disorder on the autism spectrum that affects 1 in 10,000 girls. It is usually caused by a mutation in the X-linked *MECP2* gene, which encodes a protein that is essential for nerve cell function. The symptoms of Rett syndrome include loss of mobility and stalled cognitive development. Previous research has shown that lack of functional MeCP2 causes abnormal neuronal morphology, but not cell death, begging the question of whether damage to the defective neurons is irreparable or can be repaired by re-expression of MeCP2.

To answer this question, Adrian Bird of Edinburgh University (UK) and colleagues used a mouse model of Rett syndrome in which the *Mecp2* gene is silenced by insertion of a removable cassette. The resulting neurological symptoms in mice include inertia, tremor, irregular breathing, and abnormal gait (*Science*, 23 February). The researchers were able to reactivate the gene by treating the mice with a drug that deletes the cassette. In female mice exhibiting neurological symptoms, switching on the *Mecp2* gene resulted in reversion to wildtype