

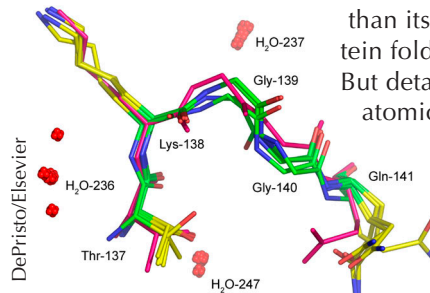
## Uncertainty in structures

Crystal structures may fit all the data, but a report from Mark DePristo and colleagues (University of Cambridge, UK) warns that, for any given structure in the Protein Data Bank (PDB), there will be many other overlooked structures that are equally consistent with the data.

Crystallized proteins retain the ability to move around, thus making interpretations of diffraction data an imprecise process. Crystallographers fit their data to models that pass quality controls, but they usually report only one such model. The Cambridge group generated alternate models that fit the data for several proteins. "We found a reasonable number of structures that are surprisingly different in their finer details," says

DePristo. And as the diffraction resolution decreased, the differences increased.

Most variability was found at the protein surface rather than its core, suggesting that a good idea of protein fold can be gleaned even at low resolution. But detailed conclusions that depend on precise atomic location, such as catalytic mechanism, may be misinterpretations. "We need a change in thinking of structures as less of a static, perfect model, but rather as models that have uncertainties," says Tom Terwilliger (Los Alamos National Laboratory, Los Alamos, NM). "Crystallographers need to develop a means for communicating the uncertainty in their atomic model." ■



Models based on the crystal structure of interleukin-1β vary significantly.

Reference: DePristo, M., et al. 2004. *Structure*. 12:831–838.

## Pollen spares all but self

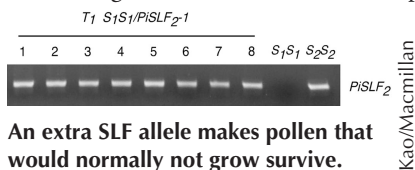
Many plants encourage genetic diversity by preventing self-pollination. Two groups now show that this system works by protecting only an RNase that destroys self. This RNase stops the growth of genetically identical pollen tubes, but RNases that would destroy nonidentical pollen tubes are themselves degraded.

The RNases are made by a part of the *S*-locus, a huge, intractable stretch of DNA. Although the female-specific product of the *S*-locus has long been known to be the *S*-RNase, the male-specific product (made by the pollen tube) has eluded scientists for a decade. It is now identified as a regulator of ubiquitination that seems to sentence all but self *S*-RNases to degradation.

Through a brute force sequencing approach, Paja Sijacic, Teh-hui Kao (Penn State University), and colleagues found that the petunia pollen *S*-component is the SLF F-box protein. Normally, a haploid pollen grain expresses only one *S*-allele. But forced expression of two different *S*-alleles alters pollen rejection. The group now shows that two different SLFs can likewise alter pollen rejection, thus confirming that SLF is the male incompatibility protein.

Hong Qiao, Yongbiao Xue, and colleagues (Chinese Academy of Sciences, Beijing, China) found that SLFs from snapdragon bind to both self and non-self *S*-RNases. But only the non-self enzymes were ubiquitinated and thus degraded.

How SLF prevents degradation of its own *S*-RNase is not clear. Kao guesses they may have matching interaction domains that either block the ubiquitination site or alter the F-box so that it cannot interact with other SCF components. ■



An extra SLF allele makes pollen that would normally not grow survive.

Kao/Macmillan

References: Qiao, H., et al. 2004. *Plant Cell*. 16:582–595.  
Sijacic, P., et al. 2004. *Nature*. 429:302–305.

## Cells step back in time

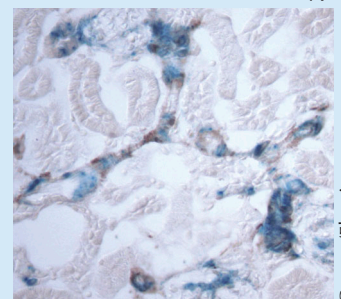
Many of us would like to be young again. Results from Maria Sequeira López, Ariel Gomez (University of Virginia, Charlottesville, VA), and colleagues show that a return to youthful activities is possible for at least some mature cell types.

These do-over cells are progeny of renin-secreting cells. The renin-angiotensin system controls body fluid and electrolyte levels. Although many cells make renin during development, those that hold this job in the adult are restricted to a small region of the kidney. During stresses such as dehydration, this population may be unable to make enough renin. To remedy the situation, more cells in and near the kidney begin to produce renin. Gomez's group shows that these helpers come from differentiated cells that had been embryonic renin producers.

The authors permanently marked any cell in a mouse that ever expressed renin. In the adult, marked cells included nonrenin-producing vascular smooth muscle, epithelial, and mesangial kidney cells. When fluid homeostasis was threatened, it was these marked cells that dedifferentiated and reverted to their renin-secreting ways.

Cells that had never made renin did not contribute. "At least for this system," says Ariel, "the change in cell identity is determined by the lineage of the cell. Not all cells can do anything." ■

Reference: Sequeira López, M., et al. 2004. *Dev. Cell*. 6:719–728.



Extra renin (brown) is made only by cells (blue) that previously made the hormone.

Gomez/Elsevier