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 interpreta-
tions 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.”

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

**Uncertainty in structures**

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**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, thus
confirming that SLF is the male incompatibility protein.

Hong Qiao, Yonghia
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.

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

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**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 re-
gion 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 differen-
tiated cells that had been embryogenic 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 threat-
ened, 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.