March 28, 2006 | vol. 103 | no. 13 | 4795–5242

PNAS

Proceedings of the National Academy of Sciences of the United States of America www.pnas.org

# In This Issue



DEVELOPMENTAL BIOLOGY

## Transcription factor directs *Lotus japonicus* floral symmetry

Xianzhong Feng *et al.* report that *LjCYC2*, a homolog of the TCP-box transcription factor *cycloidea* (*CYC*), determines petal shape and symmetry during flower formation in the legume *Lotus japonicus*. An evolutionary adaptation, floral bilateral or dorsoventral asymmetry facilitates outcrossing in plants by attracting pollinators. Research has shown that TCP-box genes, such as *CYC*, control floral symmetry. Feng *et al.* isolated four TCP-box genes from *L. japonicus* and observed that *LjCYC2* had the most similar expression pattern to *CYC*. Both transcription factors are expressed in the dorsal region of floral meristems and dorsal organs of developing flowers. *LjCYC2* expressed constitutively in transgenic plants conferred a dorsalizing effect in lateral and ventral petals. Mutagenized *L. japonicus* screened for abnormal floral symmetry resulted in the identification of two mutants, *squ1* and *kew1*. Plants with the *squ1* mutation possessed abnormally shaped dorsal petals and carried a point mutation in *LjCYC2*. In *kew1* plants, lateral petals resembled ventral ones in both petal shape and epidermal cell type. Flowers of the *squ1/kew1* double mutant exhibited enhanced ventralizing phenotypes on the dorsal and lateral petals. These data demonstrate that *CYC* and its homologs are conserved dorsalizing factors, contributing to the wide variation in flower morphology. — F.A.

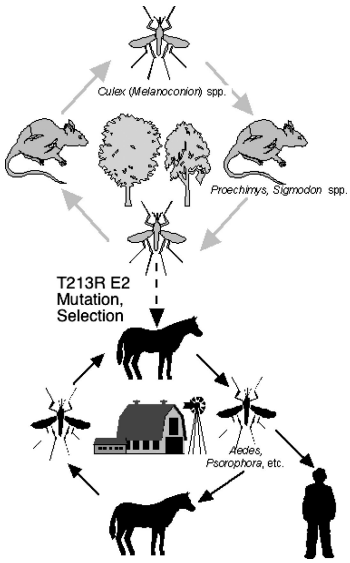
Three types of zygomorphic wild-type flower of *L. japonicus*.

*"Control of petal shape and floral zygomorphy in* Lotus japonicus*" by Xianzhong Feng, Zhong Zhao, Zhaoxia Tian, Shilei Xu, Yonghai Luo, Zhigang Cai, Yumei Wang, Jun Yang, Zheng Wang, Lin Weng, Jianghua Chen, Leiying Zheng, Xizhi Guo, Jianghong Luo, Shusei Sato, Satoshi Tabata, Wei Ma, Xiangling Cao, Xiaohe Hu, Chongrong Sun, and Da Luo* (see pages 4970–4975)

EVOLUTION

## Phylogenetically predicted mutations in horse–human virus

Michael Anishchenko *et al.* have identified how the Venezuelan encephalitis virus (VEEV) periodically mutates to cause disease in horses and humans. The finding pinpoints a mutation that causes the disease and shows the power of phylogenetic analysis to predict and identify important mutations. An RNA virus, VEEV flares up every few decades in the Americas, with its natural reservoir in forest-dwelling rodents. Mosquitoes transmit the virus between rodents and horses, with spillover into humans. Anishchenko *et al.* studied mutations in samples from a 1992–1993 outbreak of Venezuelan encephalitis. A mutation in an envelope glycoprotein of VEEV, which changes its virulence for horses, was identified. The mutation was found where phylogenetic analyses predicted it should occur. The authors say ecological and epidemiological factors probably constrain the frequency of Venezuelan encephalitis outbreaks, more so than the generation via mutation of more infective viral strains. The results underscore the general ability of RNA viruses, such as HIV and SARS, to alter host range, virulence, and epidemic potential via minor genetic changes. VEEV also demonstrates the unpredictable risks to human health that can come with the introduction of horses and humans into habitats that harbor zoonotic RNA viruses. — P.D.



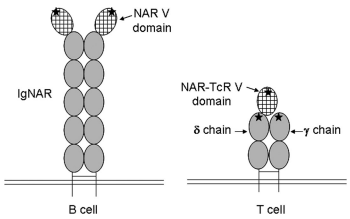

Emergence process of epidemic VEEV strains.

*"Venezuelan encephalitis emergence mediated by a phylogenetically predicted viral mutation" by Michael Anishchenko, Richard A. Bowen, Slobodan Paessler, Laura Austgen, Ivorlyne P. Greene, and Scott C. Weaver* (see pages 4994–4999)

IMMUNOLOGY

## Shark antigen receptor variable region in Igs and TcRs

Michael Criscitiello *et al.* have identified an antigen receptor chain in sharks with two variable (V) domains shown to be used in both Igs and T cell receptors (TcRs). Cartilaginous fish such as sharks are the oldest animals that have an adaptive immune system centered on rearranging antigen receptors. Shark Ig loci exist as clusters throughout the genome. Criscitiello *et al.* examined the TcRδV region in the nurse shark and found transcripts encoding an additional V domain, termed new antigen receptor (NAR)-TcRV. Four NAR-TcRV families were identified in the nurse shark through cDNA cloning. The NAR-TcRV domains were found to be supported by dedicated TcRδV domains membrane-proximal to domains highly similar to IgNARV. The highest expression of the antigen receptor was in the thymus, followed by the spleen, spiral valve, and peripheral blood. Phylogenetic analysis of vertebrate V domains confirmed the close relationship between NAR-TcRV and IgNARV. According to the authors, the ancestral NAR V gene may have recombined with the TcRδ locus in a cartilaginous fish ancestor >200 million years ago, to encode the first V domain used in both Igs and TcRs. — F.A.

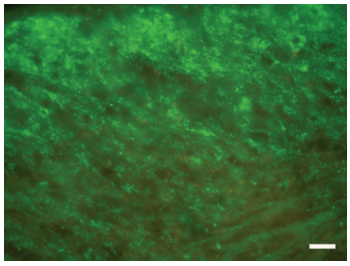


IgNARV and NAR-TcRV.

*"An evolutionarily mobile antigen receptor variable region gene: Doubly rearranging NAR-TcR genes in sharks" by Michael F. Criscitiello, Mark Saltis, and Martin F. Flajnik* (see pages 5036–5041)

MEDICAL SCIENCES

## Self-assembling nanofiber scaffold rebuilds rodent brain


Axons in superior colliculus of regenerated hamster brain.

Rutledge Ellis-Behnke *et al.* report that self-assembling nanofibers injected into the brains of hamsters can promote axonal regeneration and functional recovery after traumatic brain injury. Scar tissue formation and large gaps in brain tissue that occur after traumatic brain injury make treatment by conventional medical and surgical methods difficult. Ellis-Behnke *et al.* attempted to circumvent these obstacles by using peptide nanofibers in young and adult hamsters with severed optic tracts, an injury resulting in blindness. A solution of peptide nanofibers was injected into the area of brain injury (superior colliculus) in the hamsters. The injected solution was shown to self-assemble into a molecular scaffold that permitted axonal regeneration across the area of damage. The regenerated axons reconnected with their target tissues, allowing vision to be restored in 75% of the animals, as tested by a behavioral visual task of orienting toward a small object. The nanofibers were immunologically inert and broke down into L-amino acids excreted in the urine. The apparent safety and efficacy of the nanomaterial make it a candidate for future use in reconstructive brain surgery, the authors suggest. — M.M.

*"Nano neuro knitting: Peptide nanofiber scaffold for brain repair and axon regeneration with functional return of vision" by Rutledge G. Ellis-Behnke, Yu-Xiang Liang, Si-Wei You, David K. C. Tay, Shuguang Zhang, Kwok-Fai So, and Gerald E. Schneider* (see pages 5054–5059)

MICROBIOLOGY

## Pyrimidine catabolism pathway identified in *Escherichia coli*

Kevin Loh *et al.* report that the b1012 operon of *Escherichia coli* K12 bacteria encodes an unidentified pathway for pyrimidine degradation. For uracil and thymine catabolism, previous research had identified a reductive and an oxidative pathway, but the gene products of the b1012 operon had not been characterized. Sequence study by the authors showed that genes of the b1012 operon were not homologous to those of known pathways for pyrimidine catabolism, but b1012 gene products were similar to proteins of known function. Loh *et al.* screened a parental *E. coli* K12 strain and strains with transposon insertions in genes of the b1012 operon for nitrogen catabolism. Mutant strains could not respire with uracil or uridine as the nitrogen source. The researchers grew the strains on minimal medium with glycerol as the carbon source and either ammonium or uridine as the sole nitrogen source. Mutant strains could only utilize ammonium for growth, whereas the parental strain used both. In addition, mutant strains grew to a lower yield than the parental strain with cytidine as the sole nitrogen source. The b1012 operon thus appears to code for proteins constituting a previously unknown pathway for pyrimidine breakdown. — F.A.

*"A previously undescribed pathway for pyrimidine catabolism" by Kevin D. Loh, Prasad Gyaneshwar, Eirene Markenscoff Papadimitriou, Rebecca Fong, Kwang-Seo Kim, Rebecca Parales, Zhongrui Zhou, William Inwood, and Sydney Kustu* (see pages 5114–5119).