EPIGENETICS

An epigenetic cause of adaptive variation

Understanding the molecular basis of adaptive variation is one of the main goals of ecological and evolutionary genetics. Recent work from Shindo and colleagues reveals that variation in epigenetic silencing controls variation in flowering time in *Arabidopsis thaliana* — a key adaptive trait.

Arabidopsis thaliana plants need to be exposed to a period of cold before they can flower — a response called vernalization. The species grows in a wide range of environments, and plants that come from different environments show considerable differences in flowering times and vernalization responses. To understand the molecular basis of this adaptation to different environments, Shindo *et al.*

assessed expression levels of two markers: flowering locus C (*FLC*), the molecular target of vernalization, and vernalization insensitive 3 (*VIN3*), an early component of the response. To their surprise, there was no correlation between RNA levels of these two markers and the length of exposure to the cold.

Next, the authors looked at the stability of *FLC* repression — when expressed, *FLC* represses flowering; vernalization alleviates this effect by repressing *FLC* expression through an epigenetic mechanism. In this case they found a difference — in plants that respond more slowly to vernalization (collected from northern locations), *FLC* RNA levels increased significantly once the period of cold was over, whereas in plants with faster responses to vernalization (collected from more southern locations) these RNA levels essentially did not change. In fact, the authors found that increasing the length of the cold period quantitatively enhanced the stability of *FLC* repression. Chromatin immunoprecipitation (ChIP) experiments showed that, in molecular terms, variation in the response to vernalization translates into differential accumulation of histone H3 lysine 27 methylation.

QTL analysis in four *A. thaliana* populations revealed that genetic determinants of the variation in vernalization response mapped to chromosomes 1, 4 and 5, distributed

across four or five distinct loci. Notably, however, these loci do not correspond to any of the three known *trans* factors (*VRN1*, *VRN2* and *VIN3*) that are thought to directly affect the stability of *FLC* silencing. The mapping data also indicate that the *FLC* locus itself might contribute to the variation. Although the 5' region and the first intron of *FLC* have been shown to be important for the maintenance of *FLC* repression, key *cis* elements remain to be identified.

On the basis of the ChIP data, flowering time and *FLC* expression analysis, the authors propose that vernalization occurs in three phases: an insensitive phase is followed by a phase in which silencing occurs; when the levels of histone modification are sufficient, the third phase begins, in which *FLC* is fully silenced. The time taken to accumulate sufficient histone methylation levels determines the speed of the response to vernalization. The hunt for the genes that underlie the identified QTLs and that control this natural variation in vernalization response is on.

Magdalena Skipper

ORIGINAL RESEARCH PAPER Shindo, C. *et al.* Variation in the epigenetic silencing of *FLC* contributes to natural variation in *Arabidopsis* vernalization response. *Genes Dev.* **20**, 3079–3083 (2006)

FURTHER READING Orr, H. A. The genetic theory of adaptation: a brief history. *Nature Rev. Genet.* **6**, 119–127 (2005)

RESEARCH HIGHLIGHTS ADVISORS

MICHAEL AKAM
University of Cambridge, UK
SEAN B. CARROLL
University of Wisconsin, USA
NANCY J. COX
University of Chicago, USA
SUSAN FORSBURG University of
Southern California, USA

RALPH J. GREENSPAN
The Neurosciences Institute,
California, USA
YOSHIHIDE HAYASHIZAKI
Riken Genomic Sciences Center,
Japan
MARK JOBLING
University of Leicester, UK

PETER KOOPMAN
University of Queensland, Australia
LEONID KRUGLYAK
Fred Hutchinson Cancer Research
Center, USA
BARBARA MEYER
University of California, Berkeley,
USA

JOHN QUAKENBUSH
Dana-Farber Cancer Institute and
Harvard School of Public Health,
Boston, USA
JANET ROSSANT
Mount Sinai Hospital, Toronto, Canada
MARC VIDAL Dana-Farber Cancer
Institute, Boston, USA

VIRGINIA WALBOT
Stanford University, USA
DETLEF WEIGEL Max Planck Institute
for Developmental Biology, Germany
PHIL ZAMORE
University of Massachusetts, USA
LEONARD I. ZON
Children's Hospital, Boston, USA