PRESS ALERT

"Mystery of stable transposon silencing across generations resolved in plants."

Using the model flowering plant Arabidopsis thaliana, a FWF-funded Austrian research team led by Hisashi Tamaru at the Gregor Mendel Institute of the Austrian Academy of Sciences, in collaboration with the laboratories of Robert L. Fischer and Daniel Zilberman at the University of California, Berkeley, USA, sheds light on one of the enduring mysteries of the genome: how are transposons stably silenced across many generations? In their new Science paper, "Active DNA Demethylation in Plant Companion Cells Reinforces Transposon Methylation in Gametes", the authors report that DNA de-methylation in the female and male gamete companion cells reinforces de novo DNA methylation of transposons in gametes, with the implication that DNA de-methylation and activation of transposons in gamete companion cells generates mobile signals - small RNAs - that move into the gametes to immunize against transposon activation over the plant sexual cycles.

What are transposons and what is transposon silencing through DNA methylation?

Transposons are parasitic mobile DNA elements, contained in large quantities in plant and animal DNA, which normally move from place to place within the genome, unless inactivated by defense mechanisms such as DNA methylation. But besides acting as harmful mutators, transposons contribute to important biological processes, such as genome evolution, gene imprinting, and chromosome segregation during cell division. One strict condition under which cells benefit from transposons is to limit to certain extent their activity and mobility. DNA methylation - a mark on genomic DNA made by the enzymatic addition of methyl group (CH_3) to cytosine bases - is associated with silencing of genes and transposons. Reversibly, methylated DNA can be de-methylated by enzymes that excise and replace methyl-cytosines with un-methylated cytosines. DNA de-methylation is associated with transcriptional activation of genes and transposons.

DNA methylation is catalyzed by enzymes, called DNA methyltransferases, that can be grouped by the two activities: maintenance methylation and *de novo* methylation. The maintenance DNA methyltransferase functions to replicate pre-existing methylation of DNA sequences to keep them silent throughout the cell cycle over generations, whereas the *de novo* DNA methyltransferase contributes to establishing methylation of previously un-methylated cytosines. However, the fidelity of maintenance methylation is not 100%, so that a fraction of cytosines in transposon sequences loses methylation during multiple rounds of cell divisions and embryonic development. Thus, cells must employ a security mechanism(s) to re-establish lost methylation of transposons and maintain genome stability.

What role does *de novo* transposon silencing play in sexual reproduction of plants?

Unlike the situation in animals where the gametes (egg and sperm) represent the direct product of meiosis, flowering plants form the female and male gametophytes, consisting of the gamete and its companion cell. Sexual reproduction in flowering plants involves two fertilization events. The pollen vegetative cell (the companion cell of the sperm) forms a tube that transports two haploid sperm cells to the ovule, where one fuses with the diploid central cell (the companion cell of the egg) to form the triploid placenta-like endosperm that nourishes the embryo, while the other fertilizes the haploid egg to form the diploid embryo.

DNA glycosylase enzymes catalyze active DNA de-methylation in plants. The *Arabidopsis thaliana* DEMETER DNA glycosylase, named after a Greek Goddess of the harvest, was first identified by its maternal role in seed development. The central and vegetative cells in the female and male gametophytes undergo DEMETER-mediated DNA demethylation, but the function, mechanism, and biological significance of this process was not known. In the *Science* paper, the researchers combine state-of-the-art cell sorting, next generation sequencing technology, and bioinformatics to demonstrate that DEMETER de-methylates thousands of similar transposons in the genomes of the central and vegetative cells, and a lack of DEMETER activity causes reduced *de novo* DNA methylation of complementary transposons in the egg and sperm.

What are the exciting implications of this discovery?

This new discovery is exciting because DEMETER functions to mediate two opposing processes, methylation and de-methylation of DNA, in two separate cells in both the female and male gametophytes. What are the mechanism and biological significance of these processes? The *de novo* DNA methylation machinery uses special types of RNA, 24-nucleotides small RNAs, to guide methylation of its complementary DNA sequences. Thus, the obvious deduction is that DNA de-methylation and activation of transposons in gamete companion cells generates mobile signals - small RNAs - that move into the gametes to immunize against transposon activation over the plant sexual cycles.

A dilemma of small RNA-directed *de novo* DNA methylation is that cells need to transcribe transposon DNA into RNA and dice them to generate small RNAs. Transcriptional activation of transposons increases the risk of transposon mobilization and mutations in the genome. Plant gamete companion cells provide a clever solution to this paradox: the companion central and vegetative cells, which are not transmitted to the next generation, risk their life to save the egg and sperm from being exposed to mobile transposons. Unlike in plants, animal gametes do not have companion cells. How mammals maintain transposon silencing across generations remains mysterious.

Links

Tamaru Group: http://www.gmi.oeaw.ac.at/research-groups/hisashi-tamaru
Hisashi Tamaru: hisashi.tamaru@gmi.oeaw.ac.at
GMI Media: http://www.gmi.oeaw.ac.at/pr

About the GMI

The Gregor Mendel Institute of Molecular Plant Biology (GMI) was founded by the Austrian Academy of Sciences in 2000 in the form of a company to promote research excellence within the field of plant molecular biology. It is the only international center for basic plant research in Austria. Research at the GMI is curiosity driven and covers many aspects of molecular genetics, from basic mechanisms of epigenetics, to chromosome biology, developmental biology, stress resistance, and population genetics. *Arabidopsis thaliana* is used as the primary model organism. The GMI has about 80 employees from 22 countries. The working language is English. The GMI is located at the Vienna Biocenter Campus within the purpose-built Austrian Academy of Sciences Life Sciences Center Vienna, completed in January 2006.

Über das GMI

Das Gregor Mendel Institut für Molekulare Pflanzenbiologie (GMI) wurde von der Österreichischen Akademie der Wissenschaften im Jahr 2000 gegründet, um Spitzenforschung in der molekularen Pflanzenbiologie zu fördern. Das GMI, organisiert als GmbH, ist die einzige internationale Grundlagenforschungseinrichtung auf diesem Gebiet in Österreich. Die Forschung am GMI gilt primär den Grundlagen der Pflanzenbiologie und umfasst vor allem molekulargenetische Aspekte wie epigenetische Mechanismen, Populationsgenetik, Chromosomenbiologie, Stressresistenz und Entwicklungsbiologie. Die Ackerschmalwand *Arabidopsis thaliana* ist die am meisten verwendete Versuchspflanze. Das GMI hat ca. 80 MitarbeiterInnen aus 22 verschiedenen Ländern. Die Umgangssprache ist Englisch. Das GMI befindet sich in einem modernen Laborgebäude der Österreichischen Akademie der Wissenschaften, das im Januar 2006 fertig gestellt wurde. Dieses gehört zum Vienna Biocenter Campus (VBC), auf dem mehrere Forschungseinrichtungen sowie Biotechnologie-Firmen angesiedelt sind.

Kontakt
Thomas Friese
Head of Science Support
Dr. Bohr-Gasse 3
1030 Wien
0043 1 79044 9101
thomas.friese@gmi.oeaw.ac.at

Wissenschaftlicher Kontakt
Prof. Magnus Nordborg
Scientific Director
Dr. Bohr-Gasse 3
1030 Wien
0043 1 79044 9100
magnus.nordborg@gmi.oeaw.ac.at

Büro für Öffentlichkeitsarbeit der Österreichischen Akademie der Wissenschaften
Dr. Ignaz Seipel-Platz 2, 1010 Wien

T +43 1 51581-1218, 1219, 1229, 1235
F +43 1 51581-1227
public.relations@oeaw.ac.at