German Genetics Society Meeting 2009: Session Plant Genetics II

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September 24, 2009

Another guest post by Alex Knoll reporting from the German Genetics Society Meeting in Cologne.

As promised, I attended the second Plant Genetics session of the meeting, thus missing the Epigenetics session.

I learned an important lesson at the first talk by Juliette de Meaux from the Max Planck Institute for Plant Breeding Research: Bring sweets for the audience, and you have everyone on your side! Her research is on the molecular basis of variation in the life cycle of the model plant species Arabidopsis thaliana. Natural Arabidopsis strains grow in very different climates, and therefore have to adapt their annual life cycle to the environmental conditions: germinating their seeds early or late, flowering quickly after germinating or growing vegetatively for months. The ability not to germinate although there are good conditions is called seed dormancy. This can be a very important trait, for example if the Arabidopsis strain grows at a location where a short favorable period is followed by a recurring period detrimental to the growth of the plant. Then the plants which did not germinate even during the short favorable period have an advantage. But such changes in seed germination time need the whole life cycle to be fine-tuned: For the seeds of the following season to be ready before winter, the growth rate has to faster, or the flowering time shorter.

Because of the varying locations where Arabidopsis grows, there should be variation in life history strategies to be found for selection to act upon. For the model system of germination rate, Juliette de Meaux' lab found a high variations in 180 natural genotypes they looked at. Some strains germinated at the day of harvest, while others didn't germinate at all even 250 days after harvest, and most somewhere in between. If seeds were exposed to a cold treatment 250 days after harvest (to simulate winter), the strains that didn't germinate after the 250 days suddenly started to germinate, while others decreased in their germination rate at longer times of cold exposure. Such and other measurements allowed for the analysis of a very complex dataset to derive the different strategies of life history in Arabidopsis. For example, there is a correlation of growth rate and flowering time to be found, but only in strains growing to the north (of Europe),

not in the southern strains. They also looked at the molecular basis of this life history variation, and the consequences of it, which were interesting parts of the talk in and of themselves.

The second talk was by Rüdiger Simon from the Institute of Genetics at the University of Düsseldorf. Plants also possess stem cells, and they are located in niches (in plants called meristems) at the tips of the shoot and the root. For the shoot apical meristem (SAM), it is already known that it is composed of the stem cells and an organizing center underneath. The stem cell proliferation is regulated by a negative feedback loop: Cells in the organizing center express Wuschel, which promotes stem cell fate, but stem cells express Clavata3, that represses Wuschel via the receptors Clavata1 and Clavata2. Apart from that, there is a second negative feedback mechanism of regulating Wuschel activity known. But according to computer simulations by Rüdiger Simon and his colleagues, there are further factors needed to explain the observed distributions of cells and gene expression. They looked for such factors, and found the coryne mutant, which resembles known clavata mutants; it must be in the same pathway.

Rüdiger Simon's lab also looked at the root tip stem cell niche, if it is perhaps also regulated by a Clavata-like pathway. Interestingly, they were able to find a Clavata3-related peptide in Cle40, that regulates the Wuschel homolog WOX5. While this is similar to the situation in the SAM, there are also differences: WOX5 is produced in the QC, which resembles the organizing centre that expresses Wuschel , but Cle40 is not produced by the stem cells, but by already differentiated cells derived from those stem cells.

Next, Bhupendra Chaudary from Gautam Buddha University in Greater Noida, India, gave a short presentation on the fate of duplicated genes in polyploid cotton. After a genome duplication event, the evolutionary constraints on duplicated genes are relaxed, and one way change is possible is by generating differences in expression, allowing for subfunctionalization or neofunctionalization. They looked at expression differences in homoeologous genes (pairs of genes duplicated by a polyploidization event) in species of the cotton Gossypium, where parental diploid genomes as well as allopolyploids can be found. Interestingly, none of the parental genomes showed a dominant expression in the hybrids globally, but some tissue-specific transcriptional subfunctionalization.

The last talk of the session was given by Frank Kempken from the University of Kiel on mitochondrial mRNA editing in plants. Mitochondria possess their own rudimentary transcription and translation machinery, and in between they change the mRNAs in a way the nucleus doesn't: they edit the mRNA sequence, usually by exchanging a C for an U to create novel stop codons, or to modify the amino acid sequence. How this is done is largely unknown.

Frank Kempken's lab developed a system to work with isolated mitochondria, because genetic engineering of them in plants is not possible, and in vitro approaches have serious drawbacks. In their 'in organello' system, they can electroporate the isolated mitochondria in buffer, and observe the editing done

to the expressed mRNAs. This allowed them to observe correct recognition of editing sites of Arabidopsis mRNAs in the mitochondria of maize, for example, or that mRNAs from chloroplasts are not edited in mitochondria. This would at least partially be expected, but somehow the plastid transcripts are not recognized by the editing machinery in the mitochondria.

Using a novel binding assay (which requires 10 liters of Arabidopsis cell culture per experiment, whew!) they were able to find proteins that bind to mRNAs at editing sites. A knockout mutant of one of them leads to growth retardation and smaller plants, exactly what one would expect when mitochondria don't function properly anymore.