German Genetics Society Meeting 2009: Session VI

Martin Fenner, Gobbledygook

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

Saturday had two more sessions before the end of the meeting. Irina Stancheva from the Wellcome Trust Centre for Cell Biology at the University of Edinburgh started us into the day with a talk on epigenetics in mouse development.

One form of epigenetic silencing is methylation of cytosine bases in DNA. This is done by DNA methyltransferases like Dnmt1 for maintenance methylation, or Dnmt3a and Dnmt3b for de novo methylation, at CpG sequences enriched at promoters. During mouse embryonic development, DNA methylation is essential, for a loss of either of the DNA methyltransferases is embryonically lethal. Methylation and demethylation during development is surprisingly dynamic, with loss and new gain, and differences between the embryo and the trophectoderm. Besides the Dnmts, there are further proteins involved in the regulation of methylation levels. One interesting protein here is Lsh, a chromatin remodelling ATPase belonging to the SNF2 family. It seems to have a low ATPase activity in vitro, but it cooperates with several known factors like the DNA methyltransferases and Histone deacetylases. With mouse promoter microarrays, Irina Stancheva's group compared wildtype and Lsh knockout cells, and found a reduction in promoter methylation when Lsh is missing. The patterns of DNA methylation observed in Lsh null cells suggest that this protein has a role in developmentally-programmed methylation events in the early mouse embryo.

Dominique Soldati-Favre from the University of Geneva in Switzerland gave an interesting talk about her work on apicomplexan parasites. This phylum includes important human and animal pathogens such as Toxoplasma gondii and Plasmodium falciparum causing toxoplasmosis and malaria respectively. They belong to the clade of Chromalveolates, whose ancestor acquired a plastid organelle by the secondary endosymbiosis of an alga. While some members of this clade are free living organisms or predators the Apicomplexa have evolved as obligate intracellular parasites. These parasites have developed an elaborated strategy to actively penetrate the host cells by a mechanism distinct from phagocytosis and involving gliding motility. This active mode of entry is favorable

to the parasites, because this way they can evade the host cell defense mechanisms. To identify and study the molecular components of the vital process leading to host invasion, the Soldati's group has developed genetic tools beginning from reliable DNA transfection up to the establishment of an inducible expression system for the malaria parasites based on transcription machinery elements similar to those found in plants. Studies performed on T. gondii have established that the mechanism of host cell entry is the result of a concerted action of adhesins involved in the attachment of the parasite to the host cell, the actin cytoskeleton and myosin motors that relocalize these adhesins from anterior to posterior pole of the parasite, and proteases that finally release these adhesins from the parasite surface.