German Genetics Society Meeting 2009: Session Cellular Genetic Mechanisms

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

Both sessions had some interesting talks, so it was really hard to decide between attending Cellular Genetic Mechanisms or Human Genetics, but in the end I went to the former.

The first talk by Marina Rodnina from the Max Planck Institute for Biophysical Chemistry is not easy to put into words. Not because of a bad presentation, I should add! Her work on biochemical properties of translation involves kinetic data of a row of proteins involved in the phases of translation: initiation, elongation, termination and ribosome recycling.

I'll give you a short summary of the initiation of translation in bacteria: First the 30S subunit of the ribosome binds a mRNA and the initiator tRNA with the help of effector proteins to form the 30S initiation complex. The 50S subunit then binds to that to form the 70S initiation complex. By fluorescently labelling all of the components of the complex, they were able to measure the rate constants (how do they bind and dissociate from the ribosome) and then calculate the binding rates to find the order of binding. This is hardest to establish for the mRNA, because of control elements like the composition of the Shine-Dalgarno sequence or secondary structures. But ignoring all of these elements, the most significant factor at this step is the mRNA concentration. Looking at secondary structure, the unfolding of the mRNA is the slow step after a quick binding. This represents a kind of stability switch to enable correct start codon recognition. Not only during initiation, but also for elongation Marina Rodnina's lab could show that a lot of checks are kinetic in nature. Essentially, this can be summarized by the concept of induced fit: correct substrates are selectively stabilized by accelerating their forward steps.

Following was a short presentation by Matthias Schäfer on unpublished research. At the ETH Zürich in Switzerland, he is looking into the molecular role of protection against reactive oxygen species (ROS) in the epidermis.

Aria Baniahmad fom the Jena University Hospital, Germany, talked about an

interesting connection of telomerase activity and androgens in prostate cancer. There, telomerase is usually active, as in many cancers to allow the cancer cell to divide indefinitely. Androgens repress telomerase activity, which is probably one reason why prostate cancer occurs later in life, when androgen levels decrease in men.

The decrease of telomerase activity is based on binding of the androgen receptor to the promoter of the catalytic telomerase subunit hTERT. In many prostate cancers, an androgen receptor with a specific point mutation can be found. This mutated AR's binding to the telomerase promoter is weaker, which results in increased telomerase expression.

The session was closed with a talk by Thorsten Hoppe from the Institute for Genetics of Köln University, who presented intruiging, but unpublished data about his research on ubiquitin and ageing in C. elegans.