SFB 960-/BZR – Kolloquium

31. August 2017, 14.00 Uhr
H53

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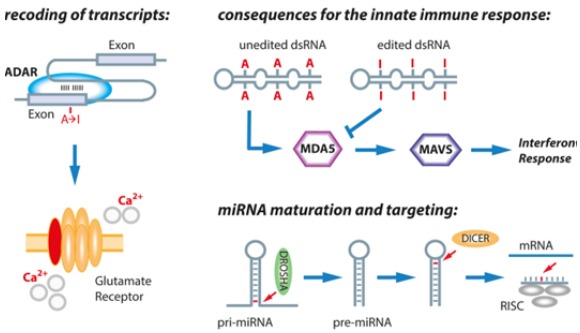
Co-transcriptional interplay between adenosine to inosine editing and mRNA-splicing

The sequence of the RNA is determined by the DNA template that encodes it. However, after synthesis it can be enzymatically altered. Adenosine deaminases acting on RNA (ADAR1 or ADAR2) enzymes catalyze the conversion of Adenosine to Inosine (A-to-I editing). As Inosine is interpreted as guanosine by cellular machines A-to-I editing changes the sequence information of the RNA. This can affect the coding potential of the RNA and impacts on e.g. innate immunity and miRNA maturation. Moreover, de-regulation of editing has been linked to several pathologies including epilepsy, depression, and cancer.

Editing sites in protein-coding parts of transcripts are frequently defined by exon-intron base-pairing, and hence RNA splicing was predicted to control the extent of editing. Konstantin will present data that demonstrate that reduced splicing efficiency leads to increased editing levels when the exonic editing site is coordinated by an intron. Thus, the efficiency of splicing is of great importance for the level of editing.

Moreover, it was recently found that lethality associated with the ADAR1 knockout in mice can be rescued when proteins involved in the innate immune response (MDA5, MAVS) are also deleted. This allowed the generation of viable, editing-deficient mice (ADAR1/ADAR2 double-null mice) that were used to create the first high-confidence map of editing sites which suggest extensive cross-talk between splicing and editing.



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