Rotation projects available

Prader-Willi syndrome: Obesity and Hyperphagia

The lab studies the molecular basics of the Prader-Willi Syndrome, to most frequent genetic form of hyperphagia and obesity. It is now clear that the loss of small nucleolar RNAs located in the PWS critical region on Chromosome 15 is the cause of the disease. We showed that these RNAs function in pre-mRNA processing and discovered a new class of non-coding RNAs.

The rotation project will identify new target genes for these snoRNAs, determine their processing and analyze their composition. In addition, we test an oligonucleotide for its ability to stop mice from eating using wild-type and hyperphagic mice. **Techniques include RNAse protection analyses, RT-PCR, cell culture, transfection, mouse work and bioinformatics approaches**.

The project is funded by the National Institutes of Health until 2017 and is suitable for a Ph.D. project.

References:

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Pre-mRNA splicing as a cancer drug target

A deregulation in pre-mRNA splicing is a hallmark of cancer cells. Thus, manipulating pre-mRNA splicing could be of therapeutic value to treat cancer. Sudemycins are simpler chemical analogs of the pre-messenger RNA splicing modulator FR901464, derived from bacteria. Sudemycins causes the selective death of cancer cells through an unknown mechanism. Sudemycins bind to the U2 small nuclear ribonucleoprotein (snRNP) component SF3B1. The drug treatment causes a dissociation of U2 snRNP, which causes changes in alternative splicing, but also changes U2 snRNP interaction with chromatin.

Using cancer and control cells, the project will determine the effect of sudemycin on the formation of circular RNAs and chromatin modifications.

References:

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Contact: stefan@stamms-lab.net www.stamms-lab.net