Laboratory of RNA Biology

pre-mRNA splicing, spliceosome, epigenetics, retinitis pigmentosa

David Staněk

david.stanek@img.cas.cz

www.img.cas.cz/research-groups/david-stanek

Our long-term interest is to determine how cells decode information stored in the genome. We focus on molecules called RNAs that serve as messengers between DNA and proteins. Information in RNAs is fragmented and we analyse how the different fragments of RNA are recognized and joined together. This process is called RNA splicing and we mainly focus on splicing variations among different cells and assembly of the machinery that catalyses RNA splicing. We also aim to determine why mutations in the splicing machinery cause retinitis pigmentosa, a human genetic disease characterized by photoreceptor cell degeneration. As we study all these fascinating processes directly in living cells, we widely employ various microscopy techniques (e.g. fluorescent microscopy, live cell imaging, fluorescence cross-correlation microscopy, and other).

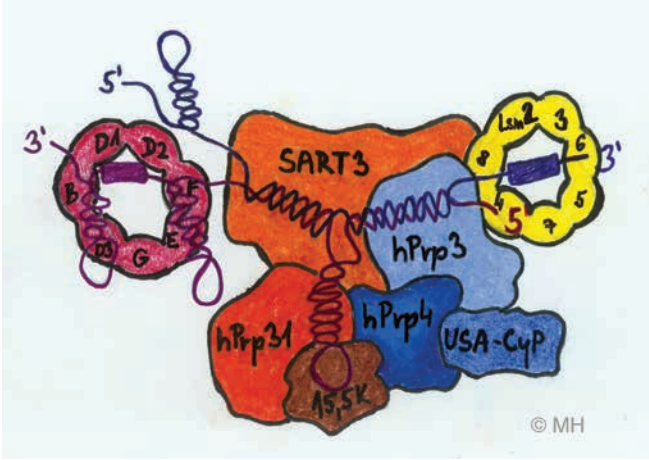

Fig. 1. Schematic representation of a small nuclear ribonucleoprotein particle, a basic building block of the spliceosome (drawing by Martina Huranová).

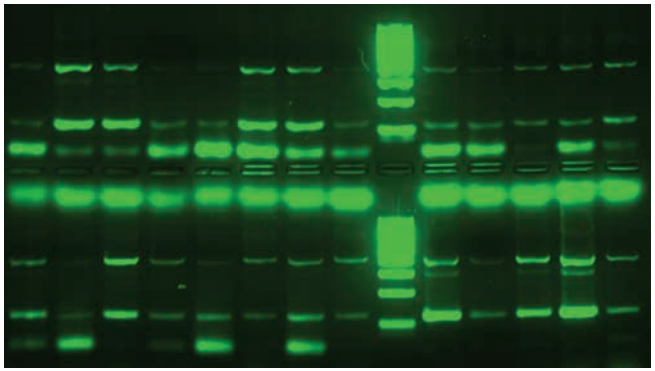

Fig. 2. Analysis of alternative splicing by RT-PCR after inhibition of histone deacetylases (Jarmila Hnilicová).

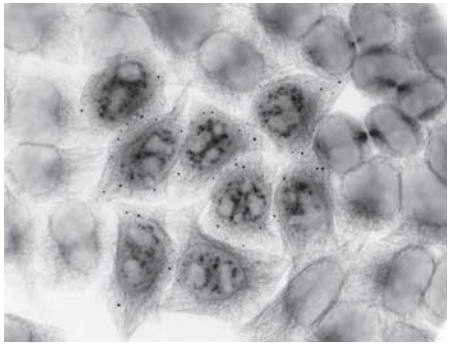

Fig. 3. Human carcinoma cells expressing LSm4 tagged with GFP and stained for microtubules (negative staining by Ivan Novotný).

- AS CR, KAN200520801 - Targeted expression and transport of bioactive molecules, 2008-2012, D. Staněk
- GA CR, GAP305/10/0424 - Regulation of alternative splicing via chromatin acetylation, 2010-2013, D. Staněk
- GA CR, GAP302/11/1910 - Formation of splicing machinery in the context of the cell nucleus, 2011-2014, D. Staněk
- GA CR, P305/12/G034 – Centre for RNA Biology, 2012-2018, D. Staněk
- GA CR, GPP301/12/P425 - Functional analysis of hBrr2 mutations linked with retinitis pigmentosa, 2012-2014, Z. Cvačková
- AS ČR, M200521206 – Functional organization of nuclear Cajal bodies with focus on formation of small nuclear ribonucleoprotein particles, 2012-2014, D. Staněk

1. Cvačková Z, Staněk D. Retinitis pigmentosa linked mutations of hBrr2 reduce splicing fidelity. submitted
2. Novotný I, Podolská K, Blažíková M, Valášek LS, Svoboda P, Staněk D. Nuclear LSm8 affects number of cytoplasmic P-bodies via controlling cellular distribution of LSm proteins. **Mol Biol Cell 2012** 23(19): 3776-3785.
3. Hnilicová J, Hozeifi S, Dušková E, Icha J, Tománková T, Staněk D. Histone deacetylase activity modulates alternative splicing. **PLoS One 2011** 6(2): e16727.
4. Hnilicová J, Staněk D. Where splicing joins chromatin. **Nucleus 2011** 2(3): 182-8.
5. Novotný I, Blažíková M, Staněk D, Herman P, Malinsky J. In vivo kinetics of U4/U6·U5 tri-snRNP formation in Cajal bodies. **Mol Biol Cell 2011** 22(4): 513-523.

From the left:
Samira Hozeifi, MSc / PhD Student · David Staněk, PhD / Head of Laboratory · Klára Klimešová / Diploma Student · Adriana Roithová / Diploma Student · Ivan Novotný, PhD / Postdoctoral Fellow · Eva Šimková, MSc / PhD Student · Eva Dušková, MSc / PhD Student · Daniel Matějů / Diploma Student · Anna Malinová, MSc / PhD Student

Not in the picture:
Zuzana Cvačková, PhD / Research Associate · Martina Huranová, PhD / Postdoctoral Fellow (until March 2012) · Jarmila Hnilicová, PhD / PhD Student (until December 2011) · Jaroslav Icha / Diploma Student (until June 2012) · Viola Hausnerová / Diploma Student (until June 2011) · Jakub Novák / Diploma Student (until June 2011)