THE MESSENGER

Newsletter No. 13

June 2020 — National Center of Competence in Research, RNA & Disease

NCCR
RNA & Disease

National Center of Competence in Research
The role of RNA in disease mechanisms

Dear colleagues

It is now 6 months since our last newsletter and as we are all slowly recovering from the coronavirus pandemic and the associated temporary shutdown of our labs, we are happy to finally return to the courant normal *and present you here a new issue of* The Messenger. *Besides a summary of two recent publications from the groups of Stefanie Jonas and Jonathan Hall, the main feature of this issue is an insightful interview with Mike Hall,* Grandseigneur *of TOR and a great and inspiring colleague in our network. As for the career paths of many other scientists, you will see that curiosity, an open mind, passion, hard work and a portion of luck were also main ingredients for Mike's seminal contributions to biology. When asked about his own role models, I started wondering how many of our younger researchers might actually have chosen Mike as their role model, which led me further to reasoning what is finally more important in a researcher's career, the scientific discoveries or the training and mentoring of younger researchers. I believe the latter is more important, as it is the training and mentoring through which one influences the values, the culture and the spirit of the current and future research community, while the discoveries would have been made anyway by someone else sooner or later. I am curious about your opinion.*

From the more philosophic to the profane: Our preparations for composing a strong phase 3 pre-proposal have started, thanks for your valuable contributions!

Oliver Mühlemann
Director NCCR RNA & Disease

Interview Michael N. Hall

"For a very long time, people did not even understand what we were saying, let alone accept or dismiss it."

Interview: Dominik Theler

In this interview, Michael N. Hall talks about scientific prizes, the discovery of regulated cell growth and his career.

On the long list of scientific prizes that you have received, the most prominent is missing. Do you expect to win the Nobel Prize?
Friends tell me that I can expect to win it, but that is what friends do. Do I personally expect it? I suppose that I am considered. However, I would be surprised if I won it. There are probably many who are considered every year. There are many deserving scientists. How can one expect it?

What do the prizes that you received mean to you?
They mean a great deal. On a personal level, prizes are a very nice compliment, but in the broader sense, they are important as validation of our work and that it is recognized internationally. As scientists, we work very hard and invest ourselves personally so it is very gratifying to receive such validation.

Do your colleagues treat you differently because of the prizes you received?
Not the long-term colleagues who know me well and who saw how the whole story developed. Others sometimes approach me as if I were a celebrity and probably treat me with a little too much respect. I dislike this because it creates a barrier between you and people. Sometimes, I say silly things just to remove that barrier and make people more comfortable. I do not like hierarchy among scientists.

"I do not like hierarchy among scientists."

How was the groundbreaking discovery that cell growth is controlled initially received by the scientific community?
For a very long time, people did not even understand what we were saying, let alone accept or dismiss it. We discovered that TOR (Target of Rapamycin) controls growth in the mid-nineties. If you at that time read a paper about cell growth, it meant cell division. When we went around telling others we discovered this controller of cell growth, people said "Come on, we have known about that for years. It is cyclin-dependent kinase". I realized that we had to re-define the term "growth" and make a

Interview Michael N. Hall

distinction: what cyclin-dependent kinases control is increase in cell number, but cell growth means increase in cell size.

A given space can be filled with many smaller parts or fewer larger parts.
Yes, it is so logical in retrospect. At that time, growth was not studied because it was not thought to be a regulated process. I also had to remind people that the process of getting more cells is a combination of cell growth and division. Cyclin-dependent kinases are extremely important and I would say TOR is also important, given that cell growth and division go hand-in-hand. TOR receives many inputs and then controls even more outputs – as if it were the brain of the cell.

(Image: University of Basel, Biozentrum, Matthew Lee)

Michael N. Hall

Biography

Michael N. Hall obtained his PhD from Harvard University in 1981 and conducted his postdoctoral research at the Institute Pasteur in Paris and the University of California in San Francisco. In 1987, he joined the Biozentrum in Basel as an Assistant Professor, where he was promoted to Full Professor in 1992. He is a member of the U.S. National Academy of Sciences, the European Molecular Biology Organization and a fellow of the American Association for the Advancement of Science. For his scientific achievements, he received numerous prizes and awards including the Louis-Jeantet Prize for Medicine (2009), the Marcel Benoist Prize (2012), the Breakthrough Prize in Life Sciences (2014), the Canada Gairdner International Award for Biomedical Research (2015), the Albert Lasker Basic Medical Research Award (2017) and the Sjöberg Prize (2020).

Website Hall Lab

Why did your lab start working on Rapamycin in yeast?
It was an exciting time in medicine because of the development of transplantation surgery, which was made possible by immunosuppressive drugs such as rapamycin. There was a lot of interest in finding out how these drugs worked, particularly in mammalian cells. Joseph Heitman, who was a postdoc in the lab, got very interested in these drugs. He and Rao Movva, a collaborator at Sandoz, remembered that Rapamycin was initially shown to be an antifungal agent. So, they made the assumption that whatever the underlying mechanism is, it must be conserved from yeast to human. We viewed yeast as a better experimental system than mammalian cells because it was genetically tractable, unlike mammalian cells at that time.

Rapamycin was the first drug found to extend the lifespan of mammals. Did you consider using it for that purpose?
No, I do not consider anything like that. When that publication came out, the sales of Rapamycin produced for research purposes skyrocketed. Apparently, people started dosing themselves.

One meaning of "Tor" in German is fool, did you sometimes feel like a fool in the early days of TOR research?
Of course, this is part of the research process. You work very hard to get Mother Nature to reveal her secrets to you. You get it in little bits and pieces and often you feel like you are not making much progress.

Did you always want to become a scientist?
I wanted to do something that involved creativity. When I was a young student, I wanted to be an artist but then decided I could not be because it was too unstructured. Also, you need to be really courageous to be an artist. Then medical school was too structured, so I had to find another way. To me, science is a compromise that combines structured productivity and creativity.

"To me, science is a compromise that combines structured productivity and creativity."

What is for you the major difference between science and art?
An artist is unconstrained, whereas a scientist is constrained by truth. We are looking for the truth of how nature works. An artist can create anything and is limited only by the medium, for example, the edges of a canvas, but not by the concept.

"There is a lot of built-in anxiety there that you have to learn to deal with."

Did you ever have a Plan B to becoming a professor?
I briefly thought about a Plan B as a postdoc. My PhD studies were not a particularly stressful period because my experiments worked well and I was able to publish several papers. Everything came very naturally. I knew that I wanted to become a professor someday but was not thinking too far ahead. When I became a postdoc, I then realized the pressure was on. I knew I would have about four years to have a story that would allow me to sell myself on the job market. I had many sleepless nights as a postdoc, wondering whether I was going to make a discovery or not. There is a lot of built-in anxiety there that you have to learn to deal with. I did not want to take any chances and worked extremely hard. When things were not going that well during the postdoc, I was very fortunate to have friends and my wife, who helped me out and supported me.

How did your wife deal with your career?
If you are a scientist and married to a non-scientist, your spouse needs to be an extremely understanding person. Doing science is a passion so you invest many more hours than only from nine to five, and even when you are home, you are usually thinking about science. You are absent mentally a lot of the time, as well as physically due to traveling.

Why did you come from the US to the Biozentrum in Basel to start your research group?
I came here after my studies and postdoc in America, but I was much less American than most of my American colleagues. I felt more global because I grew up in South America, so I was more open to looking for positions outside of America. After my PhD, I did a mini postdoc in Paris and during this time I

Interview Michael N. Hall

met my wife who is Parisian. Then we went to San Francisco together, where I did my real postdoc, and when the time came to look for jobs, because my wife was European, I looked in Europe too. I was looking in French speaking areas in Europe and had offers from institutions in Paris and Lausanne.

Then Jeff Schatz asked me to look at a position at the Biozentrum in Basel and I could not decline his invitation. I went to visit with zero expectations. I spent a couple of days there and discovered that the Biozentrum was a great place in terms of science and the quality of the students and postdocs. It seemed like a fantastic place to do science and to develop one's career. However, I had to convince my wife and myself to come to German speaking Switzerland, which was a very different place in the mid-eighties than it is now. We decided to give it a try for three years and if we did not like it, go somewhere else. We ended up liking it and stayed.

How was living in Basel after living in San Francisco?
In the very beginning, there were not that many positive sides and we were in a kind of a shock. At that time, there were like three official days per year when people moved apartments. We arrived just after one of these and it was challenging to find a good apartment. There were all these rules like not taking a shower after a certain time, which we were not used to coming from San Francisco. It was also within months of the Chernobyl explosion and the Schweizerhalle accident that polluted Europe and the Rhine, respectively. Things were bleak in the beginning.

You grew up in South America until age thirteen when your parents sent you to boarding school in New England. How was this transition for you?
That was a far bigger shock than moving to Basel. I was leaving my parents for the first time, moving to a country where there was winter and going from a completely carefree childhood in the tropics to a very regimented environment in that school. This changed me a lot and it was sort of an intellectual awakening because when I was growing up in the tropics, school was not that important to me. I did well in school, but it was more of a place where I met my friends. This idea of going to school to learn something and to acquire knowledge, and having a life of consequence … I did not think about these issues in the tropics as a child.

Did you encourage your children to become scientists?
No and I am not disappointed that they did not. My parents never pressured me to do anything and never said you should become a doctor or go into business, etc. Somehow, they had this deep-seated trust that I would find out by myself what I wanted to do. This is the same approach I have with my children. I want them to find their own interests to pursue.

You once said that the rational scientific approach can lead to difficult situations outside of science. Can you elaborate on this statement?
I think, as scientists, we do not get super happy when good things happen and also not very sad when things go the wrong way. We analyze and move on. In the real world, people want you to show emotions and, as scientists, we do not do that very much.

What was a useful career advice you received?
I cannot think of any explicit advice. I have many role models who gave me advice via how they behaved and handled things. My role models also changed during my career. My immediate role models were my advisors and as more distant role models I would name François Jacob and Jacques Monod.

"Follow your passion. However, that is not the hard part. The hard part is finding it."

What advice would you give young scientists?
My advice for anybody young is very cliché: follow your passion. However, that is not the hard part. The hard part is finding it. So, expose yourself to as much as possible, trying to figure out what you are going to do for the rest of your life. If you find what you like to do, your life will be a wonderful experience.

Throughout your career, did you notice the students changing?
Yes, they are less focused because there are more distractions in their lives. We did not have social media and smartphones and all these types of things, which add up and distract. To me, it seems that our human brains are not wired to handle all the distractions that are out there now.

I tell my graduate students that the phase that they are going through is the transition from a student to a professional. This big transition requires a high degree of dedication. To me, it is like a Buddhist monk going to the mountaintop to meditate until he achieves enlightenment. He is up there with no distractions and focused. This is the way I went through graduate school and I then stayed focused my entire life.

What was one significant change in research you witnessed over your career?
When they sequenced the genome, this sort of took all the mystery out of biology to me, because then we knew the boundaries. We would never again isolate a gene which had not been seen before and would never discover a protein that had not already been known. When I was a student and you would isolate a mutant or a protein, it was something that had never been seen before and then you had to figure where it fit in the big picture.

Have you been tempted to have your genome sequenced to know more about your disease risks?
Yes, but not for that reason. I have been tempted from the perspective to know more about my family background. My grandparents immigrated to America and before that there is little family history recorded. It would be interesting to know what our roots are.

Another significant change in research was the development of CRISPR-Cas gene editing. Can you comment on the CRISPR babies?
I think this is a disaster and completely unethical. We need the trust of the public because the public finances our research. If things like this become more common, it will be hard to convince the public to fund science and funding science is essential.

How should scientists react in a debate when confronted with non-factual arguments?
You should never use a condescending or insulting tone of voice. This precludes being convincing. You have to be calm and go by the numbers.

Research Highlights

Keeping RNA helicases in check

Veronika Herzog

RNA helicases are a highly conserved class of proteins that play central roles in regulatory processes and are fundamental in nearly every aspect of RNA metabolism. On the molecular level, RNA helicases bind and remodel RNA-protein complexes in an ATP-dependent manner. The DEAH RNA helicases, an essential family of RNA helicases, have pivotal roles in pre-mRNA splicing and ribosome biogenesis. Like many helicases, they harbour low intrinsic activity and no target specificity, and thus, they require dedicated adaptor proteins to fulfil their specific functions and get simultaneously activated. G-patch proteins are one broad class of such DEAH adapters and activators. With their eponymous glycine-rich motif these proteins hook the helicases to a target RNP and trigger RNA unwinding. The molecular mechanism of how G-patch proteins activate helicases, however, remained enigmatic.

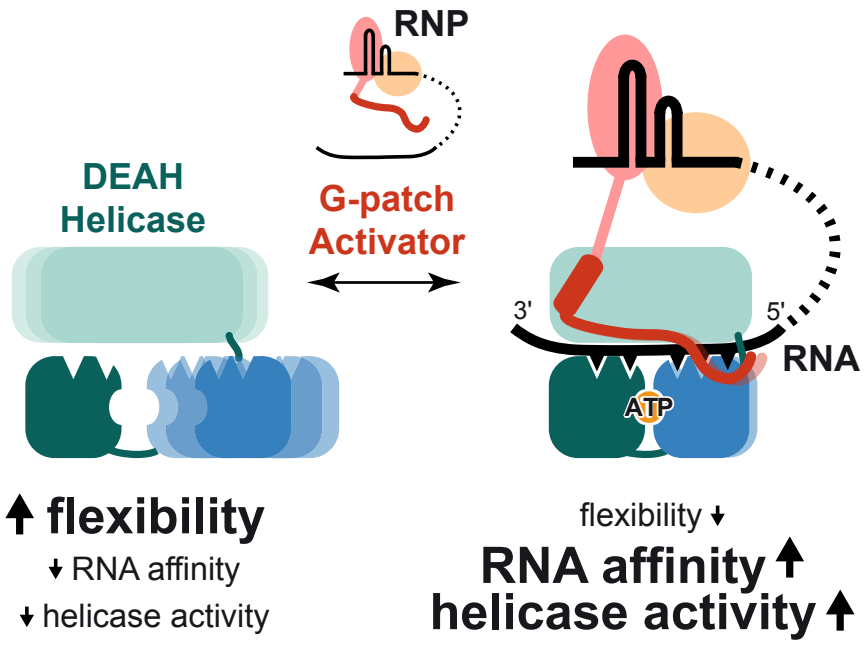
In a recent study published in the *Proceedings of the National Academy of Sciences*, the Jonas Group at ETH Zurich investigated the structural basis of how the human DEAH helicase DHX15 is activated by the G-patch protein NKRF, which is involved in human ribosome biogenesis. To dissect the molecular mechanism of how the G-patch motif stimulates the helicase activity, the authors solved the crystal structure of DHX15 in complex with the G-patch motif of NKRF and followed up the structural analyses with rigorous biochemical assays.

The crystal structure revealed how the G-patch peptide binds to DHX15: The mostly unstructured G-patch motif stretches along the DHX15 surface, almost like a molecular clamp that holds together two parts of the helicase that are otherwise highly flexible. Stefanie Jonas explains that the crystal structure was the key result of the project: *"It was one of the rare events, where the structure already hinted strongly towards a molecular mechanism that we were able to confirm in a series of in vitro assays"*.

The authors compared the structure with published structures from a fungal DHX15 ortholog that were solved in presence and absence of RNA. This analysis revealed that the conformation of the human DHX15 protein in presence of the G-patch peptide harbours an intact RNA channel, proposing that this conformation has a high affinity for RNA. Furthermore, a comparison of the solved DHX15-G-patch complex structures in presence or absence of ATP suggested that the tightened conformation of DHX15 still permits motions in the catalytic core. The researchers confirmed these observations in *in vitro* experiments: The restricted conformation of DHX15 induced by the G-patch peptide greatly enhances RNA affinity, ATPase and helicase activity.

Taken together, *Studer et al.* propose a new model of how DEAH RNA helicases are stimulated by the G-patch motif. In the absence of G-patch, DHX15 is highly flexible, has low RNA affinity and low ATPase activity. Upon G-patch binding, the helicase is restricted in its flexibility, which enhances RNA binding and catalytic activity DHX15. Thus, the specific activation of DHX15 by an adaptor protein restricts unproductive helicase movements on inadvertent targets in the cell. This detailed dissection of the mode of DHX15 activation sheds new light into how RNA helicases can fulfil their dedicated cellular functions. The mechanism of G-patch mediated DHX15-activation seems to be a general principle of helicase activation. Stefanie Jonas is excited about the broader significance of this study: *"By taking our data and reanalysing the published structures of other helicases, we can draw conclusions beyond the DHX15 helicase and predict the mode of activation for a multitude of other RNA helicases."*

Studer et al. (2020) PNAS, 117(13):7159-7170



G-patch-mediated helicase activation. G-patch proteins impede the flexibility of RNA helicases, thereby increasing RNA affinity and helicase activity. Picture kindly provided by Stefanie Jonas.

Research Highlights

Towards a treatment of Erythropoietic protoporphyria

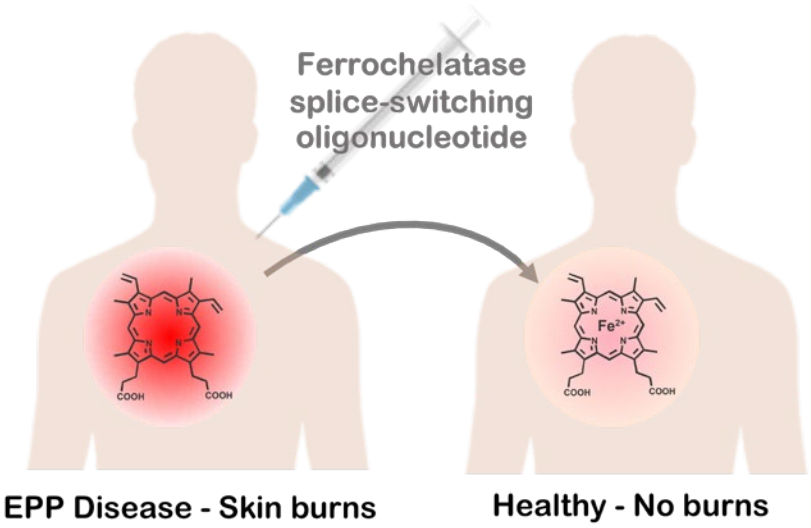
Veronika Herzog

Erythropoietic protoporphyria (EPP) is a rare genetic disease, in which patients suffer burn-like injuries of the skin and severe pain after exposure to sunlight. A subgroup of patients will go on to develop hepatic complications with the risk of liver failure. EPP is caused by a deficiency in the enzyme ferrochelatase (FECH), which catalyses the insertion of iron into protoporphyrin IX (PPIX) to generate haem. This results in PPIX accumulation in red blood cells. Upon exposure to light, PPIX produces reactive oxygen species that trigger lipid peroxidation, cell membrane damage and inflammation.

In nearly all patients, one *FECH* allele contains a deleterious mutation whilst the other carries a T>C single nucleotide polymorphism in the third intron. This polymorphism causes the usage of an alternative splice site, yielding an mRNA that harbours a premature termination codon. Consequently, the mRNA is degraded by a nonsense-mediated decay mechanism, resulting in reduced levels of FECH protein and activity.

A research team from the ETH Zurich and the University of Bern, in collaboration with clinical researchers and patients from the Triemli Hospital and the University Hospital Zurich has now published a study in *Nucleic Acids Research,* in which they describe the development of a potent splice-switching oligonucleotide (SSO) that serves as a proof-of-concept for a future new treatment for EPP. *"The management of the EPP condition largely relies on avoiding exposure to sun, severely impacting patients' quality of life. There is an urge to develop a therapeutic strategy that directly addresses the pathological mechanism"*, Jonathan Hall, one of the corresponding authors of the study, explains.

The application of oligonucleotides represents a promising strategy for EPP, especially given their recent success employed to treat other genetic diseases, such as spinal muscular atrophy. SSOs are a class of antisense oligonucleotide that modify the splicing of pre-mRNA to which they hybridize with high affinity and selectively in the cell nucleus. This creates a "steric block" to the binding of alternative splicing factors, thereby altering the outcome of splicing. Since the splicing-altering T>C polymorphism is present in nearly all EPP patients, a single SSO could provide therapeutic benefit to almost the entire EPP patient population.



Using a *FECH* reporter system in cells, the authors identified one SSO from a small library of oligonucleotides that binds to the T>C polymorphism and reverses the splicing defect. To generate an SSO that is effective in vivo, the team conjugated the oligonucleotide to different chemical groups that were expected to increase its stability and the delivery to selected tissues. They administered the conjugated SSOs to a first generation EPP mouse model and studied its distribution, its metabolic stability and, most importantly, its splice-switching ability at the FECH locus in different organs.

Jonathan Hall explains that one of the main challenges for the development of an SSO to treat EPP is its delivery to erythroblasts in the bone marrow, the organ in which the great majority of PPIX is synthesized. *"We really didn't know whether there would be any effect in vivo at all, since historical data from the literature has shown that SSOs in the bone marrow were ineffective."* Remarkably, the authors demonstrated that an SSO conjugated to cholesterol group boosts the levels of the correctly-spliced FECH transcript by 80% in the bone marrow of EPP mice.

Future work will aim to further improve cholesterol-conjugated SSOs, so as to maximize their stability for systemic delivery and potentially further increase their splicing-correction. In parallel with these efforts, the team is currently working on a new EPP mouse model to help determine whether these FECH SSOs can alleviate the painful skin lesions suffered by EPP patients. The development of an SSO delivery strategy to the bone marrow is not only a promising treatment for EPP but also for other diseases that arise from splicing deficiencies manifested in cells of the bone marrow.

Halloy et al. (2020) NAR, 48(9):4658-4671

Events

5th NCCR RNA & Disease Retreat

After last year's extended joint retreat with the Vienna RNA Biology Network near Salzburg, this year's retreat took place in its usual format and place. Over 140 researches gathered in Kandersteg from January 27–30, 2020.

The exciting research ongoing in the network was presented in over thirty talks and over sixty posters. The scientific program was complemented by keynote lectures by the NCCR's Scientific Advisory Board Members Adrian Krainer and Sarah Woodson. The retreat provided again an excellent opportunity for the participants to foster existing and initiate novel collaborations as well as to build their professional network.

We are grateful to the Scientific Advisory Board Members Jørgen Kjems, Adrian Krainer, Witold Filipowicz, Robert Schneider and Sarah Woodson for attending the retreat and providing the network with their valuable advice and input.

At this year's retreat, we had to wish farewell to Larissa Grolimund, who was the driving force behind all the NCCR's retreats so far. We are grateful to her for all her excellent work for the NCCR and wish her all the best for her new position.

Scenery in Kandersteg

Talk Session

Poster Session

Events

1st bench2biz Workshop

The 2019 bench2biz workshop took place on December 11, 12 & 16 at ETH and the Technopark in Zurich. Ten teams, each consisting of aspiring entrepreneurs and experts from different fields, participated in the fast-paced two and a half days workshop. The participants evaluated various aspects regarding the commercialization of their business idea and were supported by experts with their know-how and advice. The participant's feedback was very positive, as exemplified by the following statement: *"The meeting was really intense, but was extremely instructive. It was a great use of our time and very helpful to start thinking about the project differently than just as a research project"*.

The 2019 bench2biz workshop was a joint initiative by the NCCRs Bioinspired Materials, Chemical Biology, PlanetS, RNA & Disease (local organizer), QSIT and Transcure. The organizers are grateful to Innosuisse and the Swiss Intellectual Property Institute for their financial support, the team experts and the two workshop facilitators Christian Moser and Mark Wilson.

Group picture after the last session

Mark Wilson sketching out the road to a startup company

Christian Moser giving an input talk for the next assignment

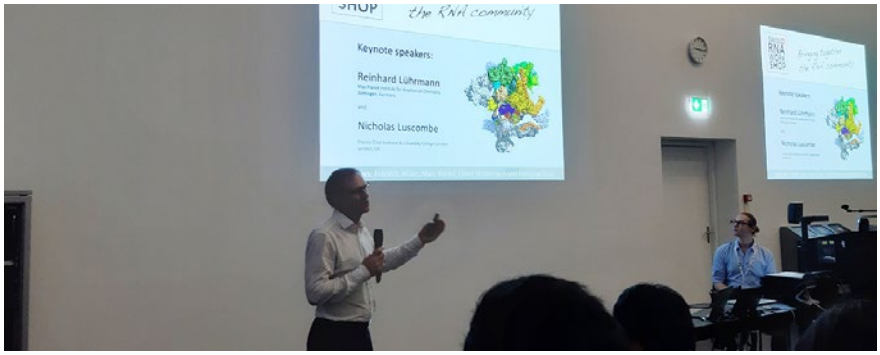
Events

21st Swiss RNA Workshop

The 21st Swiss RNA workshop took place on January 24, 2020, at the University of Bern. Over 200 RNA aficionados from Switzerland and abroad came together for a full day of exciting RNA research and interactions.

This year's keynote speakers were Nicholas Luscombe (Francis Crick Institute & University College London, London, UK) on *"Using hiCLIP to discover long-range loops in mRNAs: what do these loops do?"* and Reinhard Lührmann (Max Planck Institute for Biophysical Chemistry, Göttingen, Germany) on *"Structural insights into major design principles of the human spliceosome"*. Thirteen talks, selected by the organizers from submitted abstracts, were given and over forty posters were presented.

We would like to thank the RNA Society for its continued financial support and this year's company sponsors Axonlab, Huberlab, Lexogen, Merck, Microsynth, Qiagen, Takara and VectorBuilder. The 22nd Swiss RNA Workshop is planned to take place on Friday, January 29, 2021, at the University of Bern.



Opening remarks by Oliver Mühlemann

Keynote speakers Reinhard Lührmann (left) and Nicholas Luscombe (right)

Announcements

Awards

We congratulate Michal Hall for being awarded the 2020 Sjöberg Prize and the 2020 BBVA Foundation Frontiers of Knowledge Award in Biology and Biomedicine.

Congratulations to the NCCR's Scientific Advisory Members Sarah Woodson for receiving the 2020 RNA Society Lifetime Service Award and Adrian Krainer for being awarded the 2020 Ross Prize in Molecular Medicine and being elected as a member of the U.S. National Academy of Medicine.

New Principal Investigators

As of May 2020, Volker Thiel joined the NCCR RNA & Disease as a Full Principal Investigator and Ataman Sendoel as a Junior Principal Investigator.

Volker Thiel is Full Professor for Veterinary Virology at the Vetsuisse Faculty, University of Bern and Head of Virology at the Institute of Virology and Immunology (IVI). The Thiel lab researches Corona virus-host interactions and developed a reverse genetics system for investigating Corona viruses.

Ataman Sendoel is SNSF Assistant Professor and recipient of an ERC Starting Grant at the Institute for Regenerative Medicine, University of Zurich. Research in the Sendoel lab focuses on translational control in health and disease.

Welcome to the network!

Principal Investigator Promotions

Jeffrey Chao and Magadlini Polymenidou were promoted to Full Principal Investigators of the NCCR RNA & Disease as of May 2020. They both received tenure at their respective institutions in 2019. Congratulations!

Leaving Principal Investigators

Ana Claudia Marques will move to a Research & Development position in industry and close her lab in autumn 2020.

Rory Johnson will gradually move his lab to the University College Dublin, where he accepted a tenured Associate Professor position.

We are grateful to both for their contributions to the network, especially in their respective roles as Delegate for Equal Opportunities and Co-Delegate for Knowledge & Technology Transfer, and wish them all the best!

Announcements

New Associate Member

We welcome Roger Geiger as a new Associate Member of the NCCR RNA & Disease.

Roger Geiger leads the Systems Immunology group at the Institute for Research in Biomedicine in Bellinzona and is assistant professor at the Università della Svizzera italiana. His lab researches T cell biology in the context of liver cancer applying mass spectrometry, functional assays and microfluidics.

Scientific Officer Change

We bid farewell to Larissa Grolimund, who worked as a Scientific Officer of the NCCR RNA & Disease since its inception. As of March 2020, she took up a new position in the private sector. We are very grateful for all her initiatives and efforts for the NCCR and wish her all the best!

As of May 2020, Veronika Herzog took up her position as Scientific Officer of the NCCR RNA & Disease. She did her Master thesis, PhD studies and postdoc in the field of RNA Biology. We welcome her on board of the management team!

Equal Opportunities Delegate

Stefanie Jonas agreed to succeed Ana Claudia Marques as the Delegate for Equal Opportunities. Her election took place at the last General Assembly of the NCCR. We thank Stefanie Jonas for taking over this important role for the network!

Postdoc Representative

Rajani Gudipatti, who is a postdoc in the Grosshans lab, became the new Postdoc Representative of the NCCR RNA & Disease succeeding Dritan Lika, who left the lab of Michael Hall.

PhD fellowships for associate member labs

The NCCR RNA & Disease launched a second call for NCCR associate members labs to submit PhD project proposals, which involve collaborations with NCCR member groups. The proposals submitted by Francesco Bertoni, Isabelle Mansuy, Vikram Panse and Gerhard Schratt were selected for funding.

New Bioinformatician in Bern

As of the beginning of June, Dr. Puneet Sharma started working for the NCCR as bioinformatician based in Bern.

Visit the NCCR's technology platforms website for contact details.

Support grants

Please visit our webpage for more information on the lab exchange program, mobility grant and measures in equal opportunities.

Upcoming events organized or supported by the NCCR RNA & Disease

> NCCR/SIB Summer School in Computational RNA Biology, August 23–28, 2020, Schwarzenberg (Luzern)
> Due to the SARS-CoV2 pandemic, no NCCR RNA & Disease seminars have been organized so far for the autumn semester 2020.
> 22nd Swiss RNA Workshop, January 29, 2021, Bern.

NCCR RNA & Disease Internal Events

> 6th Annual Retreat, February 1–3, 2021, Kandersteg

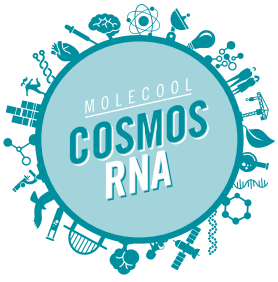
Jobs

PhD program in RNA Biology
The next application deadline is July 1, 2020.
Find out more on the PhD program website.

Predoc program in RNA & Disease
This year's application deadline was postponed to December 1, 2020.
Find out more on the Predoc program website.

Check the jobs's section of the NCCR RNA & Disease webpage for other openings.

Visit Molecool:
The NCCR's public outreach website.



IMPRINT

The National Centres of Competence in Research (NCCR) are a research instrument of the Swiss National Science Foundation

NCCR RNA & Disease
Phone: +41 31 631 38 12
office@nccr-rna-and-disease.ch
www.nccr-rna-and-disease.ch

Office Bern
University of Bern
Departement of Chemistry and Biochemistry
Freiestrasse 3, CH-3012 Bern

Office Zürich
ETH Zürich
Institute of Biochemistry, Biochemie II
HPP L14, Hönggerbergring 64
CH-8093 Zürich

UNIVERSITÄT BERN

ETH zürich

FNSNF
SWISS NATIONAL SCIENCE FOUNDATION