THE MESSENGER

Newsletter Nr. 2

February 2016 — 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

The NCCR has entered its second year of activity and has just had its first Annual Retreat in Kandersteg, which brought together ~100 RNA aficionados, among them 12 newly invited "associate" groups. The meeting was a great success. Together with the Swiss RNA Workshop, following a day later in Bern, it documented well the strength of Swiss RNA science and also the vitality of the field. RNA continues to surprise us with more and more secrets. Thousands of long non-coding RNAs, bacterial immunity CRISPR-Cas systems, and massive mRNA base modifications are just a few recent examples. These are complemented by development of sophisticated new technologies, such as single cell transcriptomics, ribosome foot-printing or cryo-EM, making it possible to study RNA-related phenomena at unprecedented depth and resolution. Finally, first drug candidates inspired by RNA research are in advanced clinical trials. What a treat for an RNA field dinosaur like myself!

What about the future? Clearly, the NCCR has to remain open and prepared to meet new challenges and opportunities. It should also be dynamic, offering access to new members at the cost of others, to comply best with its mission of "RNA and Disease". Should the network become more focused on selected important topics or rather welcome a large spectrum of RNA research, which meets excellence and the mission criteria? I am certainly in favor of the latter. Individual groups should remain focused in their research, but the network should remain broad, offering exchange of ideas and promoting even most unexpected collaborations.

Witold Filipowicz
NCCR RNA & Disease scientific advisory board member

First Annual RNA & Disease Retreat

Where NCCR scientists inspire each other

At the first annual RNA & Disease Retreat in Kandersteg NCCR scientists reported on their progress made in RNA research.

From January 19 to 21, more than 100 NCCR researchers, including scientists of NCCR laboratories and associate members from more than 30 different Swiss research groups, and invited guests gathered together amidst snow-covered mountains in Kandersteg at the beautiful Belle Epoque Hotel Victoria Ritter. The generous lobby, the plenary and dining hall with their parquet floors, stucco ceilings and chandeliers seemed well-suited to mingle with and learn from other network scientists, and to have exciting talks and lively discussions. Core of the NCCR retreat program were the eight plenary sessions of 15–20 minutes presentations of the progress in projects covering the three NCCR work packages "ncRNA functions", "RNA metabolism", and "Translation". An additional objective at this years' retreat was to introduce the associate members of the network with a personal presentation of their research.

The event also featured two guest speakers: Nicolas Favre, Head Patents and Licensing at the Friedrich Miescher Institute for Biomedical Research, who gave interesting insights into the do's and don'ts in intellectual property; and the NCCR SAB (scientific advisory board) member Jørgen Kjems from Aarhus University, Denmark, who talked about the roles of non-coding RNA in brain development and pathology. Generally – and this goes to all presenters – the high quality of the presentations was most striking: the talks were well prepared

First Annual RNA & Disease Retreat

Impressions of the 1st NCCR RNA & disease retreat in Kandersteg, January 19–21 2016

and the slide shows were visually engaged to capture the audience's attention. Lively discussions arose among the participants after the talks, and the four SAB members Witold Filipowicz, Jørgen Kjems, Adrian Krainer and Robert J. Schneider, who were present, gave valuable input.

The two poster sessions represented the second core objective of the retreat program and served as another platform for scientific discussions. There, scientists from all fields of the NCCR displayed and discussed their frontline research. The 43 posters presented demonstrated the diverse expertise in RNA research existing in the NCCR network covering the topics of the above-mentioned work packages and broad range of technologies.

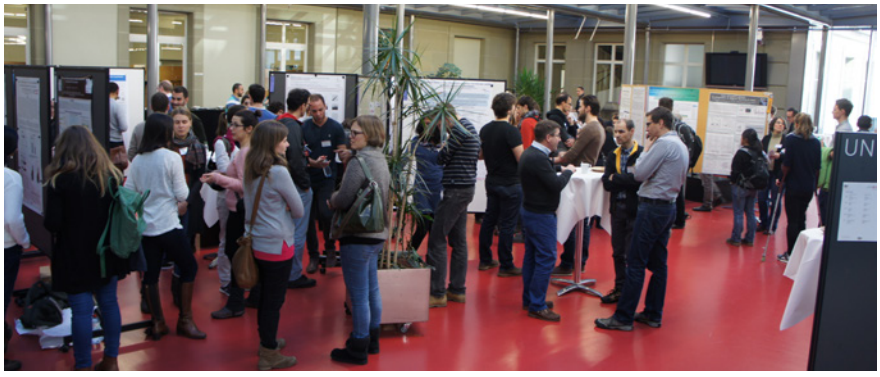
Besides the scientific sessions and the intellectual property rights excursion, Ana-Claudia Marques, the new NCCR delegate for equal opportunities as of May 2016 presented the results of the previously conducted survey on equal opportunities within the NCCR Network (results can be accessed here). Based on these results, the participants of the retreat were encouraged to discuss ideas concerning equal opportunities in groups of 5-8 people. The ideas were collected by the NCCR management and will be analyzed and considered for measures to achieve equal opportunities in the NCCR Network.

Leaving Kandersteg in full sun and fresh-fallen snow, most of the attendees did it with their head full of impressions. Some might need time to let the impression settle, others might have already clear ideas where to proceed after being stimulated by the retreat. In a year's time there will be the next retreat on RNA & Disease. For sure, the majority of this year's participants are already looking forward to attend again.

The Swiss RNA Workshop

Only one day after the NCCR retreat has ended, the Swiss RNA community held another meeting: The Swiss RNA Workshop. Already for the 17th time, scientsist from all over Switzerland and beyond met in Bern to learn about the newest results in Swiss RNA research. More than 200 researchers participated at this traditional event and attended the thirteen short talks and three keynote lectures held by Maria Carmo-Fonseca, Chris Norbury and Robert J. Schneider and visited the poster session with more than 50 posters.

NCCR Communications



The Swiss RNA Workshop January 22 2016

Erythropoietic Protoporphyria (EPP)

One of the projects presented in depth at this years' retreat concerns a collaboration on Erythropoietic Protoporphyria (EPP) between the groups of Jonathan Hall (ETHZ), Daniel Schümperli (University of Bern) and the new associated group of the Institute of Laboratory Medicine at Triemli Hospital Zürich (headed by Elisabeth I. Minder, Xiaoye Schneider-Yin and Jasmin Barman-Aksözen). EPP is a disorder of heme biosynthesis leaving patients highly sensitive to visible light. The disease arises from a deficiency in ferrochelatase that leads to an accumulation of protoporphyrin IX (PPIX) in the red blood cells and a leakage of PPIX into plasma, skin and bile. Accumulated PPIX absorbs energy from visible light leading to photodamage of the skin causing pain, burning and various other tissue damages. There is currently no cure for EPP. The predominant genotype underlying the disease in 95% of the patients is a splice modulating SNP combined with a loss of function mutation of the ferrochelatase gene. NCCR scientists follow the idea to correct the aberrant splicing and to restore functional levels of ferrochelatase in EPP-patients with two different approaches: the Schümperli group designs bifunctional U7 small nuclear RNAs, while the Hall group synthesizes splice-switching oligonucleotides (SSOs) that target the ferrochelatase pre-mRNA. To become effective, the therapeutic RNA constructs need to be delivered to the bone marrow, taken up by the erythroblasts and finally transported to their nuclei. This is where the Porphyria group of the Triemli Hospital comes into play: their humanized mouse disease model and patient cell lines allow to test delivery, uptake as well as efficacy of the RNA compounds.

Met in Kandersteg: Robert J. Schneider

«The deeper we dig, the more surprises we find»

Professor Robert J. Schneider, the newest member of the NCCRs' Scientific Advisory Board, shares his view on the NCCR RNA & Disease, the role of RNA in cancer, the rethink in drugging translation, and the value of scientific collaboration.

Dr. Robert J. Schneider

NYU School of Medicine, USA

Dr. Robert J. Schneider is the Albert Sabin Professor of Molecular Pathogenesis and Professor of Radiation Oncology at the NYU School of Medicine, and Associate Dean for the Office of Therapeutics and Industry Alliances. His research focuses on the molecular basis of metastatic breast and ovarian cancers and the development of new therapeutics. His work also includes investigation of cancer stem cells and adult stem cells, and interconnections with the inflammatory response. He is the author of more than 150 peer-reviewed papers and has received numerous awards and prizes in recognition of his achievements. He is a co-founding scientist of six biotech companies focused on translating oncology research to the clinic. Since 2015 Robert Schneider is a member of the NCCR RNA & Disease Scientific Advisory Board.

Dr. Robert J. Schneider online.

How did you become a member of the Scientific Advisory Board of the NCCR?

About a year ago, I was speaking at a conference where some of the NCCR members were present. My work tends to be much more translational, directed much more to human medicine, but it does range from basic research to clinical trials. The NCCR members were intrigued by the possibility of bringing an advisor on who helps translate more basic research into much more clinical understanding. That's how it occurred.

What will be your task at this advisory position?

This is my first round and we will meet on Friday [at the Swiss RNA Workshop]. So I don't have experience as an advisory member. However, I think that what the NCCR has done is really extraordinary – bringing together this diverse group of biologists, all working on RNA with an understanding that it is at the basis of human disease. I cannot think of any other program like it in the world. This immense density of RNA-based research you have to capitalize on. It is a brilliant thing to do.

Which of the three NCCR work packages represents most of your own work?

I actually work in most of those areas. My work has traditionally been very much involved in the translational control of gene expression, particularly in human cancer and cancer stem cells, but I also work on the control of mRNA stability. Now we are showing that the control of mRNA stability is a master regulator of adult stem cell fate as well. The deeper we dig, the more surprises we find. And some of our most basic understanding is being challenged the more we investigate. For example, proteins, I like to call them "dumb" mRNA binding proteins, which simply were known as hnRNP proteins, turn out to have enormous biological activities. Their entire function is based on whom they interact with, so they can control major physiological pathways in cells without possessing any enzymatic activity. And now we understand that they are master regulators of the adult stem cells, and that mutations in some of these mRNA binding proteins are key players in many human diseases. Thus, it makes a great deal of sense to focus on these three areas of RNA biology.

What do we know about translational control in cancer?

A number of years ago when my lab and others began trying to understand the role of mRNA translation in human cancer, most people believed that the translational control of mRNA was simply a secondary effect. We and others have now demonstrated that it actually is a driver of human cancer, particularly in cancer stem cells, which are the cells we know cause cancer recurrences, metastases and in many cases resistances to chemotherapies. So it is important to understand the biology of cancer with respect to translation. But we also know that translation in cancer cells is druggable, and there in lies the real promise. My work is very much involved in drugging the translation apparatus in human cancer. We had an observation – it was a paper in Molecular Cell – that in four years we will manage to develop a drug and we will bring it all the way through phase II clinical trial to block the translation of VEGF, which is one of the major factors that promote angiogenesis in tumor cells. All this work can be translated into new drug discoveries.

You did your PhD in biophysical chemistry. When did you become interested in molecular pathology of cancer?

I wanted to start my career by getting the most rigorous training in the understanding of macromolecules and their interactions. Biophysical chemistry gave me that. But at the same time I found it too far removed from where I wanted to be, namely to bring science into the clinic. So while it was an excellent training I felt very happy to move into biology. We now come full circle. When I first trained, structural biology was just a dream. Being able to do the kinds of studies we now routinely do, such as to look at the kinetics of interaction of macromolecules, that was not possible then. Now it is possible, and the training I received 35 years ago for my PhD we now use all the time.

Did you ever consider a medical training?

In fact, I did. I was in a program doing much of the first two years of medical school while we worked on our PhDs at the same time. There I received considerable training, at least in terms of book knowledge. Later I

was able to learn how to conduct clinical trials. That is something you just learn – courses don't teach you how to develop clinical trials. So, I am a PhD but I am also a leader of the breast cancer program, and I co-direct a number of clinical trials. There is no reason that with a PhD you can't do all of that.

When did you get sparked to focus on investigating the role of RNA in human diseases?

It started actually in my post-doctoral research where I was working on adenovirus, which is a prototypic virus for cancer. In humans this virus just causes common colds, but in rodents adenovirus causes tumors. It was one of the first approaches to begin to understand the development of cancer. I became a RNA biologist when we investigated what were called the virus-associated RNAs and how they regulated interferon signaling and ultimately translational control. I still remember my great disappointment because I wanted to work on RNA splicing, which was incredible hot at that time. I was very disappointed until I published my discovery that these small RNAs were regulators of translation in a paper in Cell. Then, of course, I decided this is a great new field to enter. This work brought me to translational control when very few people were thinking about it at that time.

One of your biggest research efforts is in the field of advanced breast cancers for which there is no cure at the moment. Are new treatment options on the horizon?

We have made no progress when breast cancer is metastatic and we still cannot speak of cures. Once a woman has late stage 3 and stage 4 breast cancer we no longer speak of cures. That's a real problem. In fact, there are certain forms of breast cancer that begin as metastatic disease – one of which I have dedicated quite a bit of my research effort to – known as inflammatory breast cancer. While that is only about five percent of breast cancer it is up to 15 to 20% of annual mortality. The field has made absolutely no progress on inflammatory breast cancer survival, which in a third of the cases begins as a metastatic disease and roughly 30% is pregnancy associated, with a mean survival of 2.5 years.

It might start to change now that we are beginning to see small effects of the immune checkpoint inhibitors. There is a concentrated research effort solely on metastatic disease now. It is very hard to work on metastasis because the animal models are not good, and by the time metastases have occurred in patients there are a lot of mutations as well. We have to change our view of the way of treating cancer because we can't any longer think about treating cancer one mutation at the time. We need to bring into the clinic drugs that act on immune checkpoint changes – and that is also where translational control comes to bear. If we drug translation then we are able to block entire hubs or centers of translation for specific types of mRNAs, such as survival mRNAs, DNA damage, DNA repair mRNAs in tumors and mRNAs that are involved in development of T regulatory cells that suppress the anti-tumor immune response. The mRNAs all have specific requirements. So drugging protein synthesis offers us the ability to downregulate entire constellations of genes at the translational level. Genes that are required for the survival and metastasis of tumor cells and other specific functions can also be drugged selectively at the mRNA level. That's why I focused on this area. And we had some successes here.

You co-founded several biotech companies, such as PTC Therapeutics. How does the development of PTC299 an inhibitor of VEGF mRNA translation go along?

We took it all the way through phase II clinical trial. That was actually a remarkable experience. From the time I had published a paper on the mechanism of tumor-specific translation under hypoxia in 2009, we began work with PTC Therapeutics. It was four years from the time of discovery to the time we had a drug that PTC filed for an IND [Investigational New Drug Application] with the FDA. With PTC we received a grant of 2.5 million dollars to actually take the drug into the clinic. We saw remarkably good responses in phase II clinical trial in metastatic estrogen receptor positive breast cancer. We saw excellent results in pediatric glioma as well. The problem with the drug was its hepatotoxicity, which was not that severe and was related to the drug, not its mechanism of action. The company decided to put the drug on a shelf, so they are not developing it any more. But we do have back-up drugs that don't show this hepatotoxicity. The discussion now is whether it makes sense to bring those drugs back into the clinic. But it was the first time that anybody had drugged translation by creating a small molecule that actually selectively blocks translation at the mRNA level.

What should be considered when developing RNA-based cancer drugs?

If we want to drug translation in cancer we – the pharmaceutical industry and other major players – need to think differently. When drugging translation, we should not do it at a level that does not cause a large decrease in overall protein synthesis. We only need to decrease protein synthesis by about 20 percent. It isn't necessary to block protein synthesis severely because selectively decreasing the translation of specific mRNAs, those that provide cancer cell survival, drug resistance and proliferation of the cancer cell, have increased requirements for translation and are more readily inhibited. The problem in cancer drug studies is that drugs are used at what is called the maximum tolerated dose [MTD]. All that we do by drugging cancer cells at the MTD level when targeting protein synthesis is to increase toxicity. Instead, we need to drug cancer cells at the level that achieves synergy with established anti-cancer agents such as genotoxic DNA damage agents – many of the common chemotherapeutics. Translation inhibiting drugs on their own should have little or no impact but when combined with existing chemotherapy can actually be quite useful. This different way of thinking about going after cancer requires a conceptual change in clinic trials.

Your lab also focuses on the regulation of the inflammatory response and its intersection with cancer development, i.e. the control of degradation of short-lived inflammatory cytokine and proto-oncogene mRNAs. How does your work progress within basic research?

What we have done is to knock out some of these key proteins that control mRNA stability. Because much of my work is also translational and my eye is almost always on the clinic, we have been able to take those findings and directly connect them to a number of human disorders. We have a paper going out the door showing that some of these mRNA binding proteins that control mRNA stability go to the heart of human skin disorders such as psoriasis, and that they are involved in the control of stem cells in the epidermis. In another paper to be submitted soon, we also show that mice develop a form of muscular dystrophy through the loss of control of mRNA stability within adult muscle stem cells. If you keep your eyes open and you move it from the cell to the animal, it is not that hard to make the connection to human disease. So, basic research can be easily translated.

Where do you see progress in disease areas that make you feel optimistic?

In many areas. In particular, now that I am Associate Dean for Drug Discovery, which is something, I am very excited about. A tremendous amount of my effort goes into that. I am excited and I am really hopeful. I would say that I give the NYU administration enormous credit for two things: one, giving me this large budget to be able to

Met in Kandersteg: Robert J. Schneider

translate research – not my own but everybody's research – into both drug discovery and new clinical observations. And secondly, enabling me to have the biggest thrill and the biggest satisfaction, which is in helping my colleagues translate their basic research into drug discovery efforts that will impact on human diseases. When you think about it, at the NYU as at so many academic medical centers, we have well over a hundred world-class laboratories. To be able to help them translate their research into new drug discovery is extraordinary. It is bigger than any pharmaceutical company. The institution is allowing me, with a staff of 20 people, to invest in this and conduct what is basically a virtual biotech within academia. To do new drug discovery is extraordinary and we have a lot to show for that in just a few years time.

And what about areas where progress is slow no matter the effort?

Let me tell you why I am optimistic. I am optimistic because the old model of the pharmaceutical industry has failed and they now understand that it is failed. So here is what happened: the majority of the failure that we have had in the last 20 years or so in making new inroads in cancer and other human diseases is largely because there was this enormous separation between pharmaceutical industry drug discovery efforts and academic laboratories. Pharma now understands that and has come back to embrace academic research, not just in terms of funding, but in terms of direct collaboration. The amount of discovery that takes place in academic institutions is extraordinary. Almost none of this enormous research effort in academia has been captured in the past, other than industry reading the literature but not gaining the expertise and insight of real experts on a day-to-day basis. Now that they are working closer together with academia, and now that you can start your own virtual biotechs because you can outsource much of what you needed to do, things are changing rapidly compared to the past. And so many more people are starting their own companies and stay in academia at the same time, so I am really very hopeful.

You have several academic appointments and many responsibilities. How do you manage to keep the overview over ongoing research relevant to you?

You can't. You have to work as a team and you have to rely on your team members and collaborators. That's why I think what the NCCR is doing is really brilliant. You have to be able to work collaboratively and collegially, because nobody can keep up anymore.

What would you advise scientists who want to work collaboratively? Where does collaboration end and where does competition start?

My view is that the easiest way for people to become comfortable collaborating with each other is first to protect your discoveries by filing for intellectual property patent applications. Because a patent application is not a secret but a disclosure where you are protecting your discovery. I think worldwide academics need to become much more sophisticated about protecting their discoveries. Once you file the patent application there is no reason not to collaborate. In fact, you can have mutual intellectual properties. So I think much of the competition and secrecy come from people not protecting their own inventions.

Interview: Thomas Schnyder

Research highlights

Pioneering work from NCCR laboratories

To the outsider, it pretty much looks like a scientist's nightmare. A class of small new components of the cell machinery is discovered and the more we find out about them, the more complex the picture gets, every answer leading to ten new questions. For the inspired scientist though, it must feel like a dream, an incessant expanding frontier, a true research Eldorado. Two examples of pioneering work from NCCR labs.

by Roland Fischer

miRNAs caught in the act

This must be a bit like colonizing a new land, for the molecular biologist. But pioneers need maps. They need to know how things are connected. They need to know which paths lead where.

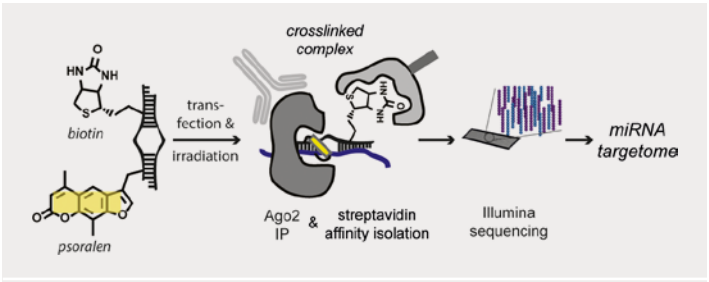
A multidisciplinary initiative from the ETH Zürich and the Biozentrum at the University of Basel, amongst others, may help finding the way. Most biological processes in mammals are regulated by miRNAs, and identification of miRNA targets therefore is one of the big challenges of molecular biology. Complex target site prediction algorithms have been very successful in matching the 6- to 8-nucleotide "seed" region of a miRNA to potential targets, but they do not provide enough information to reliably predict unorthodox miRNA-RNA interactions. Now Imig and Brunschweiger et al. figured out a way to capture miRNA targets in cells biochemically. Their approach, termed miRNA crosslinking and immunoprecipitation (miR-CLIP), uses pre-miRNAs doubly modified with photoreactive psoralen and biotin groups.

MiR-CLIP isolates miRNA targets using a multistep process. After delivery of the synthetic miRNA, RNA-protein crosslinks are induced within the silencing complex, and then the psoralen is activated, forming crosslinks between the miRNA and target RNA. The crosslinked complex, containing synthetic miRNA, target RNA and protein components of the silencing machinery, is then isolated in a two-step process by immunoprecipitation against Ago2, a silencing complex component and then streptavidin affinity purification.

miR-CLIP was established in HeLa cells, seeking targets of miR-106a. Many of the identified targets corresponded to canonical targets predicted by predictive tools. Furthermore miR-CLIP identified numerous new targets, though the validity of these putative targets is not yet clear. "miRNAs will usually interact with hundreds or even thousands of sites in the cell", the group leader Jonathan Hall from the ETH Zürich explains. "To be able to take a snapshot and to capture these interactions may very well open up new avenues of research."

Indeed, one unexpected and interesting find was that H19, a large intergenic noncoding RNA (lincRNA), was identified as a target of miR-106a. LincRNAs are a diverse and mysterious class of long noncoding transcripts important for gene regulation.

Together, these studies suggest that there are yet-undiscovered mechanisms of post-transcriptional regulation through miRNAs. And, as the authors conclude: "They also highlight a need for new experimental methods to complement and expand existing computational target prediction methods."



The miR-CLIP method

Original article in Nature Chemical Biology

The mysterious vault RNA

What is the role of the vault complex? The cell component was first described in 1986 but to date remains something of a scientific mystery. The gigantic complex is by far the largest cellular RNP identified to date; several functions have been suggested for it. These include roles in nucleocytoplasmic transport, intracellular detoxification processes and hence in multidrug resistance of cancer cells, signalling, apoptosis resistance, innate immune response, DNA damage repair and recently also in nuclear pore complex formation.

And, to make things still more obscure, what is the role of the vault RNA, so-called because it was first found associated to the vault complex? Today we know that a significant portion of vtRNA is not associated with the vault particle and the vtRNA does not seem to have a structural role, as its digestion does not alter the particle structure. This suggests that its role is functional and its association with the vault particle may be part of that function.

But what exactly is this function? NCCR researchers from the University of Bern have recently shed light onto the mystery. Looking at B-cells infected with the Epstein-Barr virus (EBV), Polacek et al. were able to show that expression of the vtRNA protects cells from undergoing apoptosis.

In previous work, they had shown that EBV infection of Burkitt lymphoma cells leads to an upregulation of different kinds of vtRNAs. Now they individually overexpressed most latent EBV-encoded proteins to check for vtRNA expression and this way identified the latent membrane protein 1 (LMP1) as trigger for vtRNA upregulation.

The upregulation renders the cells amenable to efficient EBV infection by protecting them from undergoing apoptosis. To make sure that it was really the vtRNA that is responsible for the anti-apoptotic effect, they conducted knockdown experiments of the major vault protein (MVP), the principal protein component of the vault complex – virus establishment rates remained high in the knockdown cell line. While the MVP has been previously suggested to inhibit apoptosis in senescent cells, their study is the first report demonstrating general apoptotic resistance upon vtRNA expression in malignant B cells.

Original article in Nature Communications

Announcements

People

Congratulations to Profs. Marc Bühler and Mariusz Nowacki who have been granted ERC consolidator grants!

Further, we would like to introduce Stefan Reber (laboratory of Prof. Oliver Mühlemann) as the PhD representative and Dr. Dritan Liko (laboratory of Prof. Michael Hall) as the postdoc representative of the NCCR RNA & Disease. Stefan and Dritan represent the PhD students of the RNA Biology PhD program and the NCCR postdocs, respectively, in the general assembly of the NCCR RNA & Disease.

Upcoming events organized or supported by the NCCR RNA & Disease

> NCCR RNA & Disease monthly seminar series at University of Bern and ETH Zürich

> Farewell Symposium for Daniel Schümperli: June 9–10 2016, Bern

NCCR RNA & Disease internal events:

> 2nd NCCR RNA & Disease Site Visit: March 22–23 2016, Bern

Passed events organized or supported by the NCCR RNA & Disease

> 1st NCCR RNA & Disease Retreat: January 19–21 2016, Kandersteg, Hotel Belle Epoque Victoria

> NCCR RNA & Disease general assembly: January 20 2016, Kandersteg, Hotel Belle Epoque Victoria

> The 17th Swiss RNA Workshop: January 22 2016, Bern, UniS

> 1st PhD Retreat of the RNA Biology PhD program: 22–23 January 2016, Bern (read more below)

> Symposium – assistant Professorship in RNA and Cancer at the Department for Medical Oncology, University Hospital Bern: February 24 2016

Jobs

PhD program in RNA biology
PhD position in Neuroepigenetics

Find out more on our website.

Support Grants

The NCCR RNA & Disease granted a lab exchange to Stefan Reber (Mühlemann lab) for a three weeks visit in the laboratory of Prof. Eva Hedlund at the Department of Neuroscience, Karolinska Institutet, Stockholm, Sweden.

Melanie Jambeau (Polymenidou lab) was issued a doctoral mobility grant for a 6 months stay in the laboratory of Prof. Clotilde Lagier-Tourenne, Massachusetts General Hospital and Harvard Medical School, Boston, USA.

Marina Cristodero (Polacek lab) was granted a 120% Support grant.

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

1st PhD Retreat of the RNA Biology PhD program:

After 4 days of science at the NCCR RNA & Disease Retreat and the Swiss RNA Workshop, the PhD students of the PhD program in RNA biology met for their first retreat. A dinner in Bern on Friday evening served as a perfect platform to digest the science of the past days. To get to know each other better, the students went for an interactive game called "Bavarian curling". Stefan Reber, PhD representative and organiser of the event states "the informal format created a relaxed atmosphere and facilitated lively discussions. Half of the PhD students participating in this PhD retreat were already present at the NCCR retreat in Kandersteg, and all of the students attended the Swiss RNA Workshop in Bern. Therefore, we decided already before the PhD retreat not to include an official science session this time. However, this did not hinder people to talk about their projects and their work in their laboratories."

Bavarian Curling – If not already before, the ice was definitely broken on the ice.

IMPRINT

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

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