

THE MESSENGER

Newsletter No. 9

October 2018 — 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 trick is growing up without growing old. A few months into Phase II, our NCCR is now in early adulthood – the right moment to devote an issue of The Messenger to how we have grown up over the last years and, importantly, to what the future will bring in terms of extra momentum. The center of the NCCR's activities and basis of its success lies in scientific excellence embedded in a collaborative climate. This newsletter provides two versions of this: first, a retrospect of accomplishments and, second, a personal account of our voyage so far, in the form of an interview with the co-directors. So what lies ahead of us? The best way to predict the future is to create it ourselves, and there are two distinct goals for which we are not yet tapping the NCCR's full potential. One of them is Knowledge and Technology Transfer (KTT). True – a delay in the progression of basic and applied sides is likely the natural course of projects. But probably we have also been (and here I am, regrettably, as guilty as anyone else!) oblivious to the potential for application lying idle within our scientific endeavors – let's seize these opportunities in Phase II. The second aim concerns communication. How can we be better at reaching out to the general public, to schools, to the medical community, and, yes, at creating that fascination for RNA research and its biomedical significance? The news here is that we are launching new activities together with a professional communication agency; I am excited to be involved in these efforts as the NCCR's new communication delegate. Writing

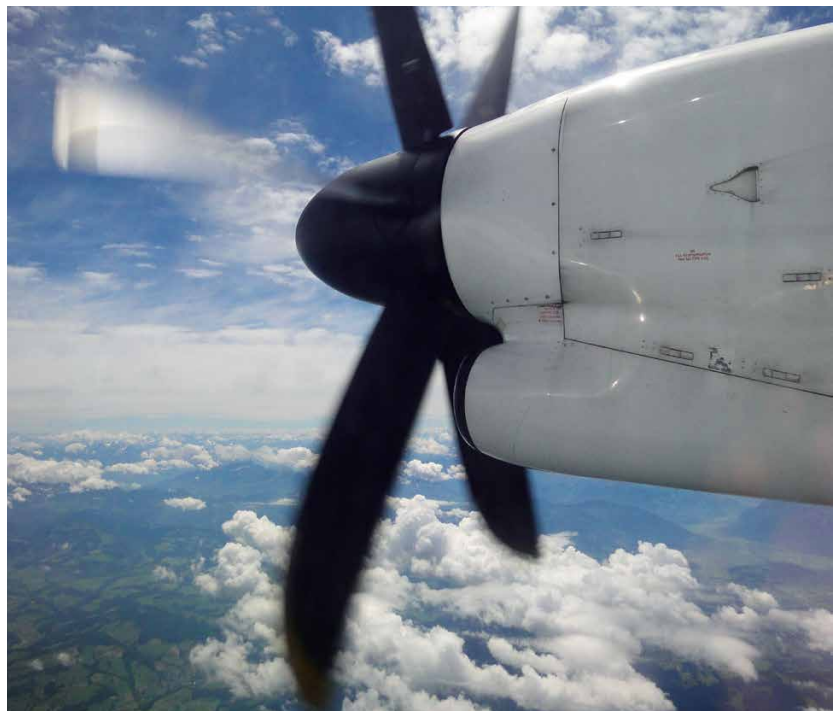
this editorial to The Messenger was my first task in that role.



David Gatfield
Communication Delegate &
Principal Investigator
NCCR RNA & Disease

Retrospect Phase 1

Propelling Swiss RNA Research Forward



The NCCR RNA & Disease started its second four-year phase this year: Time to look back at the first four years.

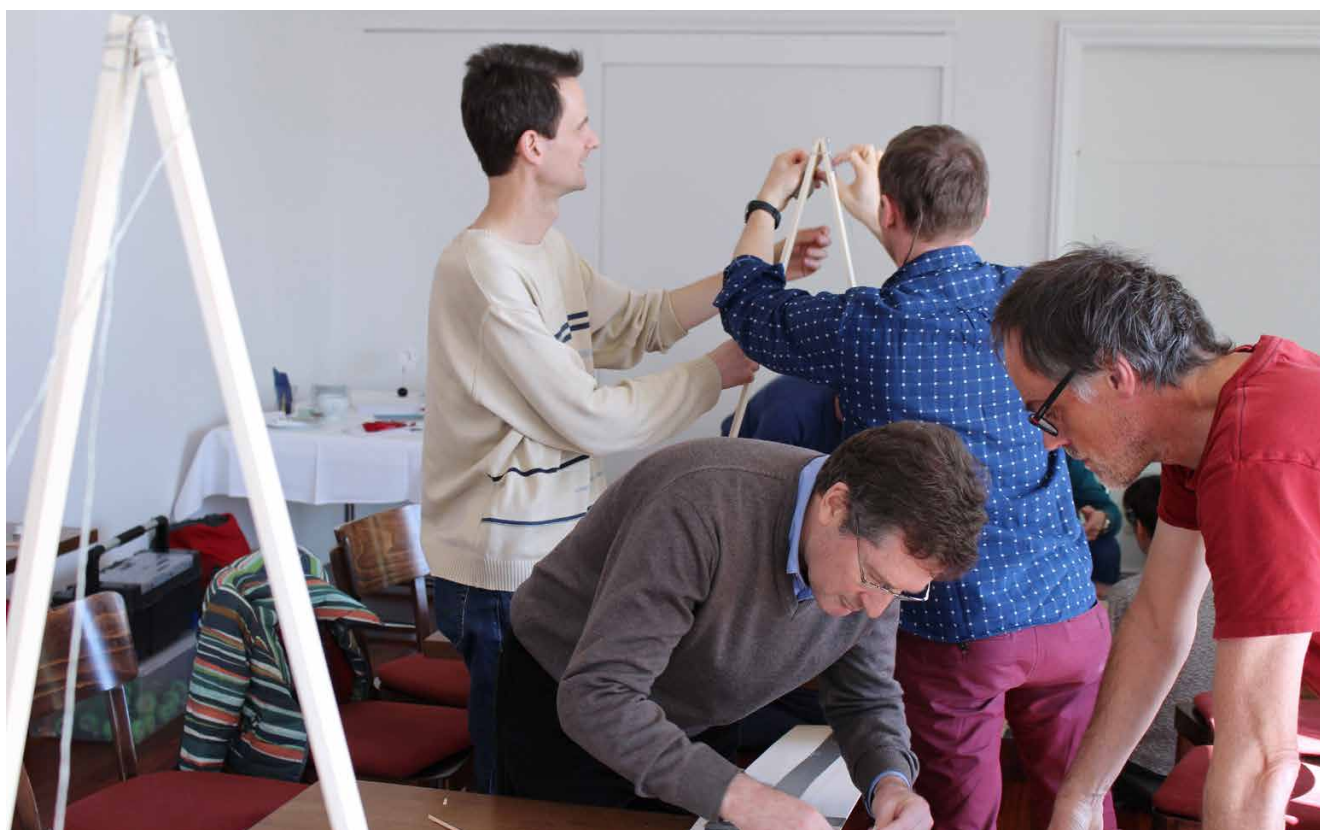
From its middle position within the central dogma of molecular biology, RNA has progressively moved center stage in the life sciences over the last decades. Several essential processes taking place at the RNA level have been discovered only quite recently – a development likely to continue in the near future. Due to the essential role of gene expression for life, these findings touch upon nearly every process in the body.

Not surprisingly, alterations in RNA metabolism therefore contribute to, or are even at the root of, the pathomechanisms of many diseases – with the number of such cases showing an upward trend.

These discoveries cannot only be applied for diagnostic purposes and outcome of disease predictions, but the involved molecular players represent potential drug targets as well. Moreover, several regulatory approvals over the last four years are proof-of-principle that RNA is druggable through oligonucleotides.

The NCCR RNA & Disease officially started its operations on May 1st, 2014, and its main funders are the Swiss National Science Foundation (SNSF) and its co-leading houses, the University of Bern and ETH Zurich. According to the [SNSF NCCR website](#), “NCCRs promote long-term research networks in areas of strategic importance for Swiss science, the Swiss economy and Swiss society”, with a number of main distinguishing features that include outstanding internationally visible research, knowl-

Retrospect Phase 1



Building Bridges

edge and technology transfer, education and the promotion of women. Furthermore, NCCRs should have a long-lasting positive effect on the field in Switzerland after they run out, i.e. after the potential maximum of twelve years of operations. Finally, NCCR research is expected to create significant added value through collaboration.

Besides direct collaborations between research groups, access to technologies can boost research interactions as well. NCCR researchers have access to eight technology platforms. Most of these platforms exist thanks to the NCCR RNA & Disease, while others are accessible to NCCR researchers at more favorable conditions. The NCCR RNA & Disease constantly revisits its technology platform portfolio to keep up with technological developments and the researchers' needs. A recent example is the new Structural Mass Spectrometry platform, which was implemented at the beginning of phase 2 and is advancing several NCCR projects already.

The propelling effect that the NCCR has had on Swiss RNA research manifests unmistakably at the level of publications. During phase 1 both the total number of publications acknowledging NCCR support and joint publications increased every year. Of note, more than half of all publications (and specifically of the collaborative ones) appeared during the last year of phase 1.

The increase in collaborative publications and projects – a number of them launched

bottom-up as a result of PhD students and postdocs meeting at NCCR events – attests to the high appreciation among the researchers for the added value that the NCCR RNA & Disease network provides. To foster exchange and joining forces between different labs (including non-NCCR and outside Switzerland), the NCCR awards [lab exchange grants](#), which complement the [SNSF mobility grants](#). More and more collaborative bridges are being built also with and among the [associate members](#), who otherwise do not receive core research funding. The high number (currently 22) of associate member groups thus clearly testifies to the network's attractiveness. Besides the associate members, also new member groups joined the network during phase 1.

Among the key members who joined during phase 1 are the junior principal investigators. Supporting junior PIs, who have the crucial career transition to a tenured position still ahead of them, is one measure contributing to the perennality of the field. Two of our junior principal investigators obtained tenured positions at their Swiss institutions during phase 1 and became now full members for phase 2. Also of note is that four out of nine junior PIs during phase 1 were female scientists.

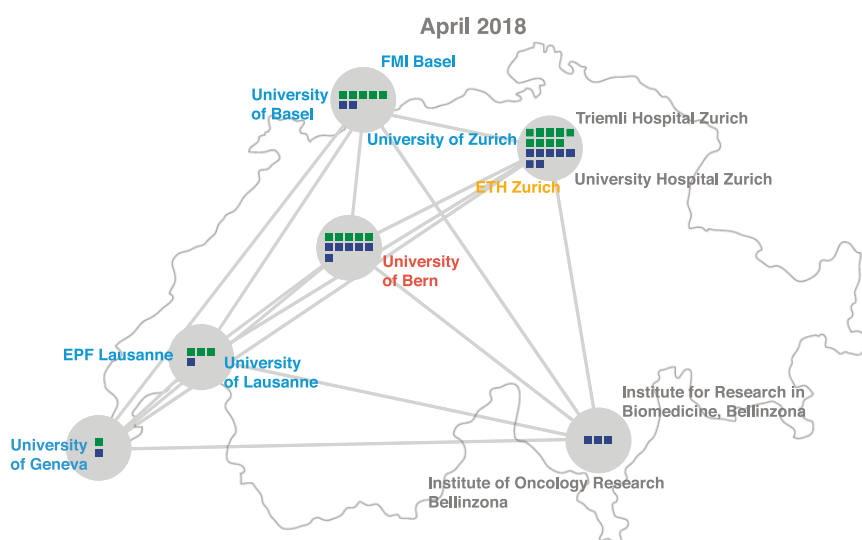
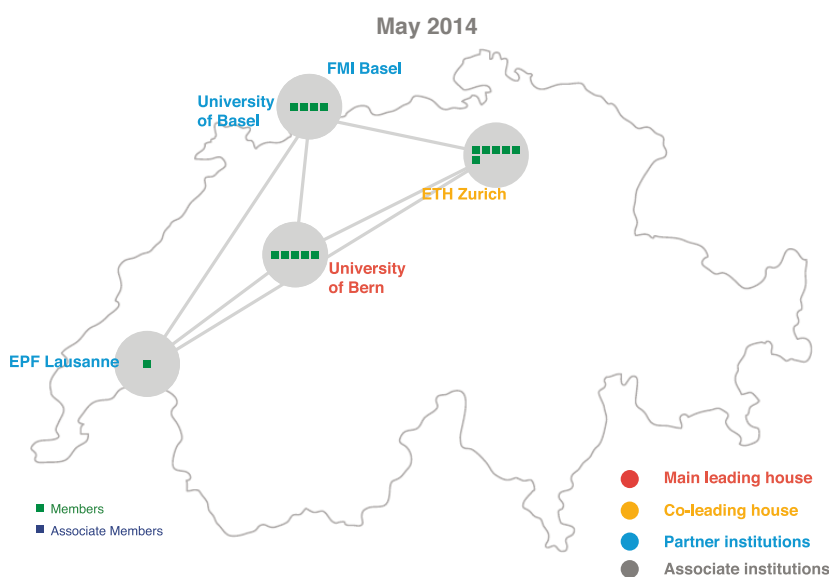
The NCCR RNA & Disease strives to help its male and female PhD students and postdocs to balance family duties and the advancement of their careers. The aim here

is also to create long-lasting effects for the field and its researchers. Besides the flexibility grants (formerly called 120 % support grants) by the SNSF, [the NCCR devised a complementary scheme](#) called the "Pregnancy and Maternity Leave Compensation", which pays for a technician's salary during the last three months of pregnancy, who should then be able to advance the project with minimal guidance during the maternity leave absence. Moreover, NCCR researchers can apply for reimbursement of Emergency Childcare funding. In order for young researchers to interact with role models in the field, so far nearly every seminar speaker participated in lunches with PhD students and postdocs.

The [seminar series](#) is one of the key [educational activities](#) of the NCCR RNA & Disease. Others include the [RNA Biology PhD Program](#) and the [Predoc Program RNA & Disease Switzerland](#), as well as [two semester-long lectures](#) at the University of Bern and at ETH Zurich, and biannual [summer schools](#). Of note, the seminar series also provides international visibility via the speakers, who are international leaders in the field, which will hopefully attract RNA researchers from abroad.

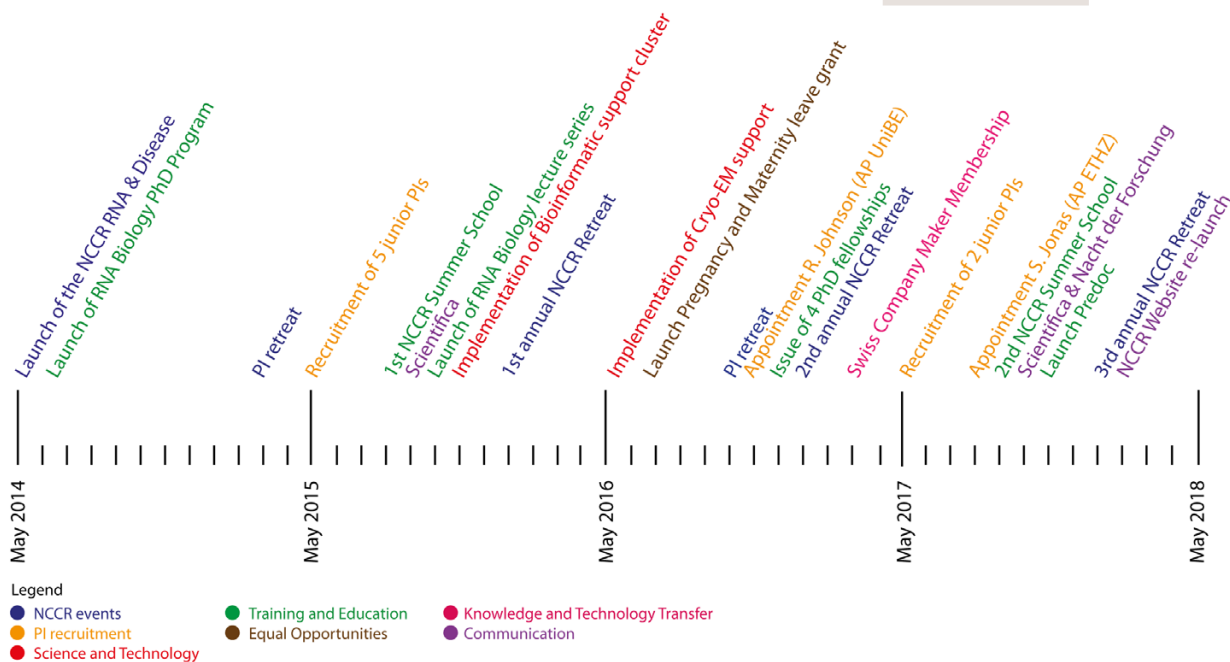
Increasing the visibility and raising awareness are two of the goals of the communication activities of the NCCR. Here, the aim is not only to promote the NCCR as a network, but also the importance of our research topic at large, ideally in a collaborative effort to-

Retrospect Phase 1



Network Evolution

NCCR Communications



Timeline Phase 1

gether with specific target audiences. While the internal communication is judged to be overall rather satisfactory, through meetings, email lists, the [Newsletter](#) and the [NCCR's website](#), new activities towards other audiences (e.g. the general public, the medical community, the pharmaceutical industry) will be further developed during Phase 2. Highlights of the communication activities towards the general public were the three participations to the Night of Research in Bern (2017) and the Zurich Science Days Scientifica (2015 & 2017), which each attracted thousands of visitors. The main lesson learned at these occasions was that with an appropriate format and communication instruments, even the rather complex research topics typically covered within the NCCR RNA & Disease can be successfully communicated to a lay audience. The communication efforts towards the medical community and the pharmaceutical industry are intertwined with the NCCR's Knowledge and Technology Transfer (KTT) activities.

During the first phase, numerous contacts between NCCR researchers and representatives of the pharmaceutical industry took place. Several patents were filed by NCCR researchers and for some projects, financial or substantial resource contributions were received from the companies involved in these projects. To boost translational activities, two new grant schemes are in the process of development and will be announced in the near future.

In phase 2 the NCCR RNA & Disease will continue to propel Swiss RNA research forward – hopefully at an even faster rate.

Interview Mühlemann/Allain

Face to face with the co-directors

Roland Fischer

The co-directors Oliver Mühlemann (OM) and Frédéric Allain (FA) give in this interview their personal account and opinions on the origins, state and future directions of the NCCR RNA & Disease

Let's start with a look back to the origins of the NCCR. How did you come up with the idea to start a big research network around RNA topics?

OM: Well, the story stretches back quite a bit. Fred and I, together with two other group leaders, obtained a Sinergia grant in the year 2011 to elucidate the role of the RNA-binding protein FUS in neurodegeneration. Through this collaboration, we met regularly. Furthermore, we both were part of an earlier NCCR application in 2008, which was called "Ribonet" and coordinated by Christian Leumann.

Which was not successful, right?

OM: No. But it was close. We made it to the last round, but when the final proposals were discussed by the SERI on a political level, after the SNSF had finished its scientific evaluations, we were not among the chosen projects.

Do you have an idea why things did not work out on the first go?

OM: Yes, we have a suspicion. For the second try four years later, we made sure the title of the project was catchier, having at least one word in the title non-scientists would understand.

FA: Which is a bit silly, because the disease aspect was already central in the "Ribonet" project, it was just not showing up in the title. Including it in the title obviously helped selling the idea at the political level.

Well done, one might say: sex – aka disease – sells. But the disease aspect is much more than just a good selling point, right?

FA: Absolutely. The goal of an NCCR is to create research that didn't already exist, so we were always very sure about this: We wanted to push the eminent basic research about RNA towards elucidating disease mechanisms and potentially finding actual cures.

Which brings us to the art of actually running an NCCR. How can you as directors

make sure the research is going in the 'right' direction?

OM: We never had a top down approach to this, the project ideas need to come bottom up from the labs. We as directors are not di-

"The goal of an NCCR is to create research that didn't already exist."

recting the research, we can only set criteria on choosing the most interesting projects.

FA: Which will actually become much more important now, starting the second phase of the NCCR. This second phase should lead to tangible results, as in phase 3 there will be a lot less money, which will be dedicated to projects that are close to potential applications. So we need to make sure that already phase 2 focuses more on actual clinically relevant solutions.

OM: That was the goal, right from the start. We knew that we have a group of people that is very strong in basic research, and we wanted to lead them towards thinking about making that knowledge useful in the clinic.

So how does that work, strategically? How do you set incentives? In other words: How do you make sure your project leaders are not just using the NCCR as a cash cow and continue to follow their own research interests? And rather connect and identify with the 'mission' of the NCCR?

FA: Well, frankly: You cannot force somebody to be bound to a network.

OM: Exactly. So far, we have given our Principal Investigators a lot of freedom. All you can do basically is sending out invitations to collaborate, to connect and make use of the network.

And these invitations have been accepted well?

OM: Very well, by and large. We are pretty happy about how most of the PIs do see themselves as part of a research network, and how new collaborations come out of the NCCR.

FA: Of course, some take these opportunities, some don't. Which is fine, as long as it is a majority that actually supports and nurtures the network. But whether they are actively "networking" or not, we get the same feedback from everyone: they all say the network is the most important part of the NCCR.



The co-directors of the NCCR RNA & Disease Oliver Mühlemann and Frédéric Allain.

Interview Mühlemann/Allain

I'm a bit surprised to hear you stressing this aspect that much. I would expect especially young researchers to come up with collaborative projects anyway, to connect with colleagues and enlarge their toolbox?

FA: No, in the biosciences it is still very common to publish papers with very few authors. That might be different in other fields like particle physics, but we still have to work on that. A typical career path is certainly not encouraging collaborations.

Which measures have proved especially useful to strengthen the network?

FA: I think most important are the retreats. The NCCR is hosting a yearly retreat for its researchers which is very popular.

OM: And now we are doing a dedicated one for PhD students as well, as a bi-annual summer school. These are great opportunities to meet other people, to learn about different perspectives on our field. Of course, we are all into RNA as a general topic, but method-wise there can be huge differences. The retreats allow you to open your scope and to consider new approaches.

"They all say the network is the most important part of the NCCR."

Anything else you want to mention as a 'network enhancer'?

OM: The technology platforms are a crucial aspect as well. They are kind of a toolbox, open for everybody. The idea is to share techniques as well as expertise within the whole NCCR network so that they can easily be used by anybody else as well.

So that's for the networking and the fostering of innovative ideas. Besides that, the SNSF stresses other aspects as key features of an NCCR, such as "education" and "promotion of women". How about that?

FA: We have installed a Predoc program, which in its second year is working out very well, after a bit of a difficult start. It consists of a one year fellowship for young students, something of a try-out: They rotate in three different groups in different cities to learn about all kinds of RNA research.

OM: It is a pioneer initiative, and it seems to develop into a nice success – I guess that is the kind of extra efforts the SNSF wants to see.

And how about the promotion of women? What have you achieved there?

OM: We are not doing too bad, I would say. The start was poor, honestly, with only 2 women amongst more 16 PIs. Now the ratio is 6 out of 24. And we keep on working on this.

FA: For instance, we are developing programs to keep women in science. Pregnancy and motherhood is a big issue here – this is still a reason for a lot of excellent female researchers to halt a promising career. Therefore, we have come up with a support program that pays for a lab technician to be instructed before maternity leave who can then carry on with the project for some months. This way, the research project does not just lay still in absence of the young mother. And she can just take over the project running on full steam after the maternity leave.

OM: Another successful initiative we have started: women only lunches. Whenever we have a female speaker in our NCCR seminar series, we organize a lunch during which our female researchers can meet their prominent colleague. I hear they are very popular and have quite an impact, on a confidence level. Sometimes all you need is a good role model, showing you that it can be done, that there is nothing special about being a woman and a successful researcher at the same time.

How do you feel about all these extra responsibilities that come with your job as NCCR directors? Do you fear that they absorb too much of your time? Or do you feel issues like gender equality are essential for your work as well?

OM: Well, yes, it is a lot of work – I probably wouldn't have put that many hours into these kind of topics, even if I am very aware of gender issues myself. But you are co-delegate for equal opportunities, Fred, you have to answer that question.

FA: I actually see it as another opportunity to network. There is a whole week conference coming up about promotion of women in research networks, and I'm very much looking forward to attending it. This is time very well spent.

"We seem to agree at least 95 percent of the time."

So, on the other hand: are there parts of your director's job you don't enjoy particularly?

FA: I would say the only part I really do not like is the annual review. The rest is not a burden of any kind.

OM: Yes, the annual reviewing process is definitely an overkill. I think one could easily do this every second year. Or, even every four years, like they do in Germany with their SFBs.

FA: Then again, it has to be said that the processes have been useful as well, even if they are a great expenditure, we've got a lot of valuable feedback. All in all, things are definitely less bureaucratic here than in other countries, so we should not complain too much.

Let's close with a little résumé after the first 4 years. Can you give us each a personal highlight?

OM: I would choose the summer schools and the retreats. They have proven to be full of enriching experiences, scientifically as well as personally.

"There are plenty of interesting stories... We just need to tell them."

FA: For me, it is the networking aspect.

OM: Yes, absolutely, I would agree on that. What we have achieved in terms of networking.

And how was the experience of working as co-directors?

FA: I must say, working with Oliver has been a great joy – we seem to agree at least 95 percent of the time. This is really valuable.

OM: Same here.

But everything can't be sweetness and light, right? Do you see weak spots as well, with potential for improvement?

OM: Well, there is another aspect in the NCCR requirements that we certainly have to put more effort into: knowledge and technology transfer. In particular, the SNSF appears to have high expectations when it comes to patents and foundations of start-ups, that is a tough one. But Jonathan Hall, our knowledge & technology transfer delegate is doing a great job, so I hope we will be seeing positive results there in phase 2 and 3.

FA: And we are investing more into outreach, also to the general public – we are building up a new communication strategy, actually, together with a professional communication agency. There are plenty of interesting stories when it comes to RNA and the body and diseases. We just need to tell them.

Research highlights

Research highlights from NCCR laboratories

Roland Fischer

Disrupting Telomerase

How to stop cancer cells from proliferating unlimitedly? That is one of the central questions in cancer research, as these cells have found several ways to circumvent normal cell aging. One of the key players in this is telomerase, which is highly active in cancer cells, keeping their telomeres intact and making the cells virtually immortal. Telomerase counteracts telomere shortening and cellular senescence in germ, stem, and cancer cells by adding repetitive DNA sequences to the ends of chromosomes. In normal cells telomeres are susceptible to damage by reactive oxygen species (ROS), but the exact consequences of oxidation of telomeres on telomere length as well as the mechanisms that protect from ROS-mediated telomere damage are not well understood.

Wareed Ahmed and Joachim Lingner from the Swiss Institute for Experimental Cancer Research at the Ecole Polytechnique Fédérale de Lausanne have recently unveiled a new detail in the mechanism of the protection of telomeres. 8-oxoguanine nucleotides at 3' ends of telomeric substrates efficiently inhibit telomerase in vitro, whereas, at internal positions, they suppress G-quadruplex formation and were therefore proposed to promote telomerase activity. As Ahmed and Lingner show in a recent *Genes & Development* paper, two cooperating enzymes, peroxiredoxin 1 (PRDX1) and 7,8-dihydro-8-oxoguanine triphosphatase (MTH1) prevent accumulation of oxidized guanine in the genome. Disrupting the peroxiredoxin 1 (PRDX1) and 7,8-dihydro-8-oxoguanine triphosphatase (MTH1)

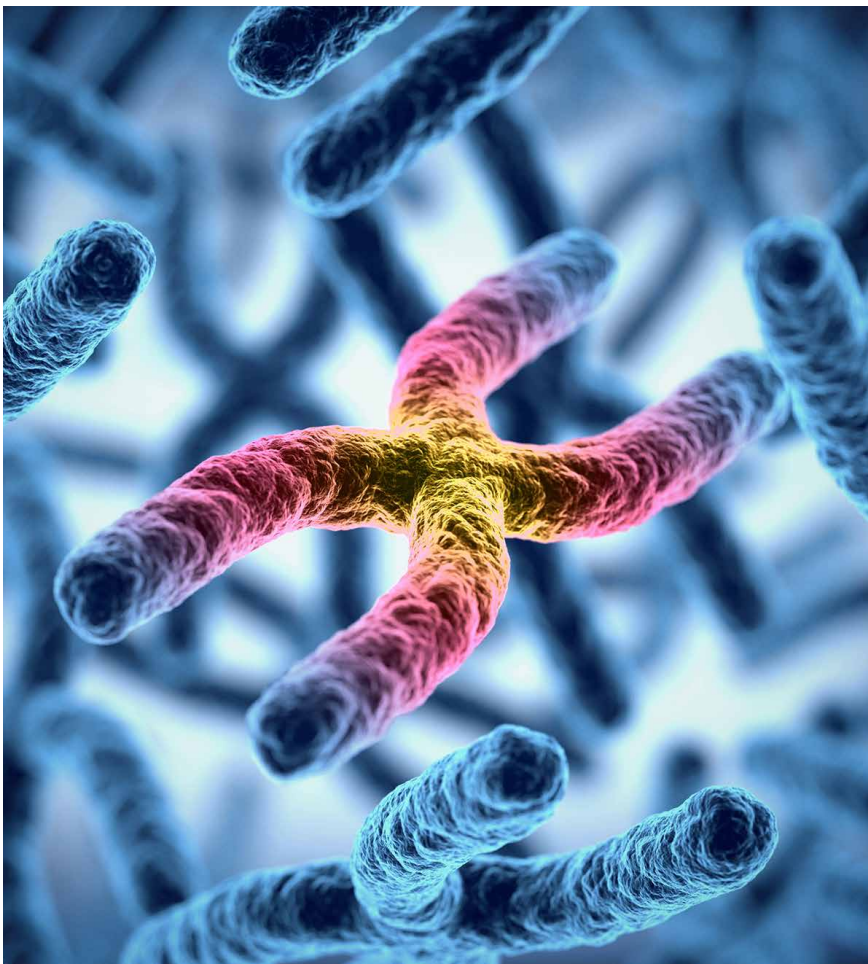
genes in cancer cells led to telomeres being susceptible to oxidative stress and damage. As a consequence, the cells' telomeres shrank with every round of cell division, eventually disappearing altogether.

By identifying these two enzymes that apparently protect chromosomes from oxidative damage and shortening, the EPFL scientists have uncovered a potential new anticancer strategy for stopping telomerase, the enzyme that immortalizes tumors. So far, attempts to efficiently block telomerase in cancer have not been fruitful in the clinic. The discovery of the cooperating enzymes opens up a new opportunity to indirectly block telomerase. "Instead of inhibiting the enzyme itself, we target its substrate – the chromosome end – making it un-extendable by telomerase," says Lingner.

To determine the roles of MTH1 and PRDX1 in suppressing oxidation of guanine in the genome, the team stained nuclei of HCT116 wild-type, PRDX1 knockout, MTH1 knockout, and PRDX1/MTH1 double-knockout cells with antibodies recognizing 8-oxoguanine (8-oxo G). It could be shown that 8-oxo G was incorporated into the genome in a oxygen concentration-dependent manner. The staining for 8-oxo G was enhanced in MTH1 knockout cells and further pronounced in the PRDX1/MTH1 double-knockout cells.

The work opens novel avenues to target telomeres and telomerase in cancer cells. Notably, recent data show that cancer cells to be more vulnerable than non-cancer cells to ROS. Thus, increasing ROS may preferentially target cancer cells, and several chemotherapeutic cancer drugs as well as ionizing radiation either induce ROS or reduce the cellular antioxidant capacity. The authors ask the intriguing question whether these drugs may be used to target telomeres and whether they can be boosted for telomerase inhibition by combing them with inhibitors for MTH1 and PRDX1.

[Ahmed W. and Lingner J. \(2018\). *Genes & Development* 32\(9–10\). 658-669](#)



Research highlights

miRNAs are not just “on” or “off”

Small noncoding RNAs, known as microRNAs, regulate many biology processes by silencing target mRNAs. Studying the *let-7* miRNA in *C. elegans*, Giovanna Brancati and Helge Großhans at the Friedrich Miescher Institute for Biomedical Research and University of Basel have now described mechanisms of target specialization of miRNAs, shedding new light on miRNA function.

let-7, highly conserved across animals and an important developmental regulator and tumor suppressor gene, is part of a family of sequence-related miRNAs. The members of a specific miRNA family, or ‘sisters’, share an identical 5’ terminal ‘seed’ sequence. This part of the miRNA, comprising nucleotides two through eight, is considered the main determinant for target identification. Usually, target mRNAs contain ‘seed matches’, i.e. heptamers that base pair with perfect Watson-Crick complementarity to the miRNA seed. These were found to be necessary and sufficient for silencing in studies using ectopic miRNA expression. Hence, miRNA family

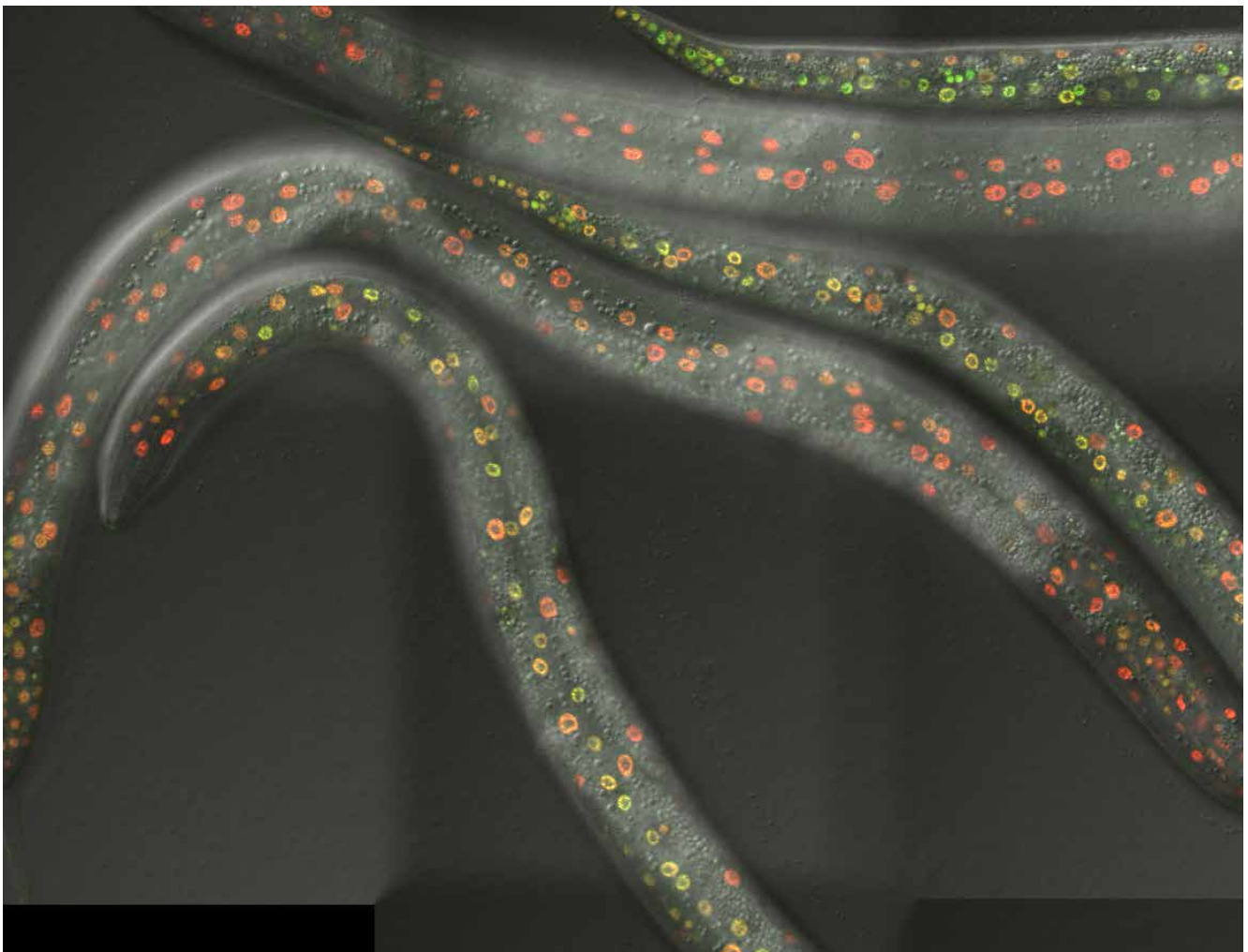
members are thought to act redundantly on targets with perfect seed matches.

Recently, this dogma has proved “shaky” as sequences outside the seed, the ‘seed-distal’ parts where sisters differ from each other, were reported to promote silencing by individual miRNAs. As described in a Nucleic Acids Research Breakthrough Article, Brancati and Großhans now show, through in vivo studies in *C. elegans*, that the extent of specificity gained through the seed-distal pairing is real but modest. However, if target sites additionally contain imperfect seed matches, the seed-distal pairing can provide clear discrimination among miRNA sisters. In the case of *let-7*, this is indeed required to support robust temporal control of *C. elegans* development, by preventing inappropriate activity of *let-7* sisters on a key target of *let-7*. In addition, the authors further reveal that different target site architectures require different miRNA concentrations for silencing.

Altogether, their results challenge a model of ‘one size fits all’, where at a given con-

centration, a miRNA is globally either ‘on’ or ‘off’ in a cell, silencing all of its targets at sufficiently high concentrations and none at low ones. Variable, target site-dependent activity was already speculated to be a miRNA feature in the early days of the miRNA field: miRNAs were likened to rheostats, whose activity is gradually adjusted by two features, namely the extent of target site complementarity to the miRNA and miRNA abundance. However, a lack of explicit experimental testing of such context-dependent function and the rising popularity of the ‘seed-match only’ model caused this hypothesis to fade from view. Brancati and Großhans propose that it is time to revisit the idea of miRNAs functioning as rheostats and subject it to further testing.

[Brancati G. and Grosshans H. \(2018\) Nucleic Acids Research 46\(7\), 3259-3269 \(open access\)](#)



Picture kindly provided by Giovanna Brancati.

Research highlights

RNA impact maps

The group of Mihaela Zavolan from the Biozentrum of the University of Basel has recently presented two new computational tools for studying three prime untranslated regions of mRNA, especially the poly(A) site: PAQR, a robust computational method for inferring relative poly(A) site use in terminal exons from RNA sequencing data called, and KAPAC, an approach to infer sequence motifs that are associated with the processing of poly(A) sites in specific samples.

In a recent *Genome Biology* paper they demonstrate that these methods help uncover regulators of polyadenylation in cancers and also shed light on their mechanism of action. As an important side result, the researchers also stress the importance of assessing the quality of samples used for high-throughput analyses, as this can have substantial impact on the estimates of gene expression.

Despite our current understanding of 3'-UTRs, they are still relative mysteries. Since mRNAs usually contain overlapping control elements, it is often difficult to specify their identity and function, let alone the regulatory factors that may bind at these sites. There are a number of methods though to study the

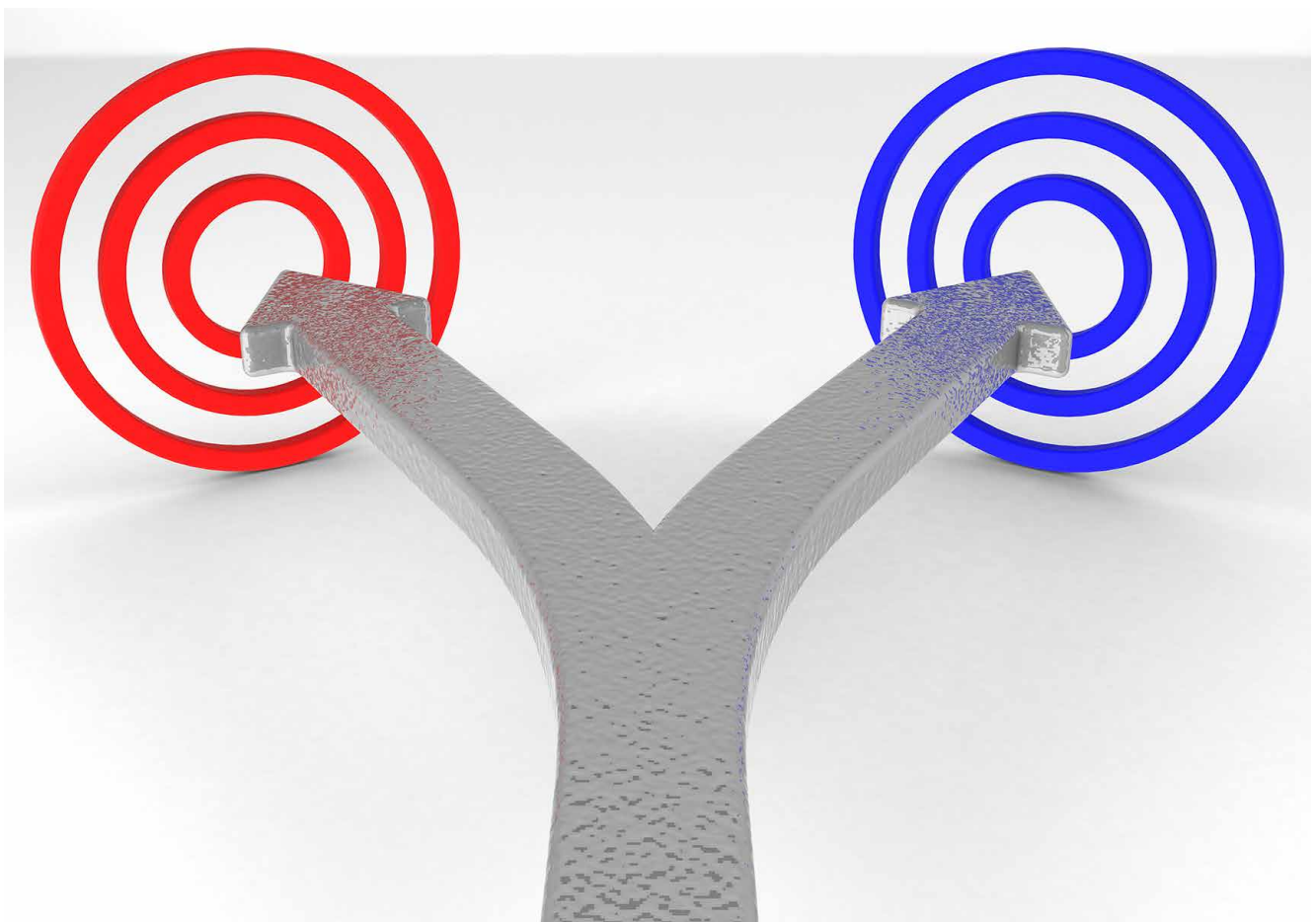
complex structures and functions of the 3' UTR. Computational approaches, primarily by sequence analysis, have shown the presence of one or more miRNA target sites in as many as 60% or more of human 3'-UTRs. Software can rapidly compare millions of sequences at once to find similarities between various 3' UTRs within the genome.

Promising attempts to construct "RNA maps" relating the position of cis-acting elements to the processing of individual exons, have shown the potential of such an approach by combining mapping of RNA-binding protein (RBP) binding sites with measurements of isoform expression. However, whether the impact of a regulator can be inferred solely from RNA sequencing data obtained from samples with different expression levels of various regulators is not known. To address this problem, the Zavolan group has developed KAPAC (for k-mer activity on polyadenylation site choice), a method that infers position-dependent activities of sequence motifs on 3' end processing from changes in poly(A) site usage between conditions. The group refers to the activities of individual motifs inferred by KAPAC as "impact maps", by analogy with RNA maps, and

to emphasize the fact that their approach does not use information about RBP binding to RNA targets.

With their method, the group was able to demonstrate that the modeling of poly(A) sites (PAS) usage in terms of motifs in the vicinity of PAS can reveal global regulators, while the reconstructed position-dependent activity of their corresponding motifs provides insights into their mechanisms. In their paper, they pay special attention to the fact that some of the uncovered proteins are splicing factors, which in their opinion underscores a general coupling between splicing and polyadenylation. The new methods already have proven relevant for medical research, as running PAQR and KAPAC on RNA sequencing data from normal and tumor tissue samples uncovered motifs that can explain changes in cleavage and polyadenylation in specific cancers. In particular, they suspect polypyrimidine tract binding protein 1 to be a regulator of poly(A) site choice in glioblastoma.

[Gruber A.J. et al. \(2018\) *Genome Biology* 19 \(1\), 44 \(open access\)](#)



Announcements

People

We would like to welcome Sebastian Leidel as a new principal investigator of the NCCR RNA & Disease. At the beginning of August Sebastian Leidel took up his position as Full Professor of RNA Biochemistry at the Department of Chemistry and Biochemistry at the University of Bern. He concomitantly joined the NCCR RNA & Disease as a principal investigator. His group researches the role of RNA modifications in health and disease.

We also welcome as a new associate member Jacob Corn, who started his new position as a Full Professor of Genome Biology at the Molecular Health Sciences Platform at ETH Zurich. His lab develops and uses the CRISPR-Cas genome editing technology as a tool for basic research and potential therapeutic applications.

Congratulations to Oliver Mühlemann, the director of the NCCR RNA & Disease, for his election as a new member of the SNSF research council. He started his new function at the SNSF in division 3 (Biology and Medicine) on October 1, 2018.

We would like to congratulate Joan A. Steitz (HHMI and Yale University, New Haven, USA) on being awarded the 2018 Lasker-Koshland Special Achievement Award in Medical Science for "Leadership in RNA biology and in scientific mentorship".

We also congratulate Adrian Krainer (CSHL) for receiving the 2019 Breakthrough Prize in Life Sciences for his contributions to the development of nusinersen, which was approved in 2016 for the treatment of SMA.

Technology Platforms

The NCCR RNA & Disease has a new bioinformatics support person Dr. Rene Dreos, who is based at the University of Lausanne.

Dr. Pascal Röthlisberger is the new responsible person for the RNA synthesis platform at ETH Zurich.

The bioinformatics support position in Zurich is currently vacant. A new person should be hired towards the end of the year.

[Visit the technology platform website of the NCCR RNA & Disease for more information and the contact details.](#)

Support Grants

Emil Dedic (Allain lab) and Agnese Pisano (Panse lab) and Yinjie Yang (Stoffel lab.) received NCCR RNA & Disease lab exchange grants.

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

Swiss RNA Workshop

The 20th edition of the Swiss RNA Workshop will take place on January 25, 2019, in Bern. Keynotes will be given by Eric Miska, (Gurdon Institute, University of Cambridge, United Kingdom) and Alena Shkumatava (Curie Institute, Paris, France). Registration and abstract submission deadline is December 14, 2018.

[Visit the workshop's website for more information and to register.](#)

Upcoming events organized or supported by the NCCR RNA & Disease

> [NCCR Seminar Series:](#)

Matthias Hentze (EMBL Heidelberg), November 12, University of Bern & November 13, 2018, ETH Zurich

Marina Rodnina (MPI for Biophysical Chemistry Göttingen) December 3, University of Bern & December 4, 2018, ETH Zurich

Jennifer Doudna (University of California, Berkeley, USA) March 3, University of Bern & March 4, 2019, ETH Zurich

Alexander Mankin (University of Illinois, Chicago, USA) March 18, University of Bern & March 19, 2019, ETH Zurich

Reinhard Lührmann (Max Planck Institute for Biophysical Chemistry, Göttingen, Germany) April 1, University of Bern & April 2, 2019, ETH Zurich

Michaela Frye (University of California, Berkeley, USA) May 13, University of Bern & May 14, 2019, ETH Zurich

NCCR RNA & Disease Internal Events

> Joint retreat with the with the Vienna RNA research community, January 30 – February 3, 2019, Fuschlsee, Austria

Jobs

PhD program in RNA Biology

The next application deadline is December 1, 2018.

[Find out more on the PhD program website](#)

Update links

[Bioinformatics position in RNA biology – Bühler and Grosshans labs, FMI Basel](#)

[PhD and Postdoc Positions - Gene Regulation by RNA modifications - Pillai Lab, University of Geneva](#)

[Check the jobs's section of the NCCR RNA & Disease webpage for other openings.](#)

IMPRINT

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NCCR RNA & Disease

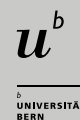
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