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› Münster Graduate School of Evolution

7th Annual Symposium

Münster, 21 - 22 March

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GRADUATE
SCHOOL OF
EVOLUTION

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MÜNSTER GRADUATE SCHOOL OF EVOLUTION

7th Annual MGSE Symposium

MÜNSTER GRADUATE SCHOOL OF EVOLUTION

ABSTRACT BOOK 2018

Edited by Jürgen Gadau, Vanessa Kloke and Joachim Kurtz

Institute for Evolution and Biodiversity

University of Münster

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Münster Graduate School of Evolution (MGSE)

MÜNSTER GRADUATE SCHOOL OF EVOLUTION

The MGSE is housed right next to the Institute for Evolution and Biodiversity, in close vicinity to Münster's City Palace (Hüfferstr. 1a).

Prof. Dr. Jürgen Gadau
Speaker of the MGSE

Dr. Vanessa Kloke
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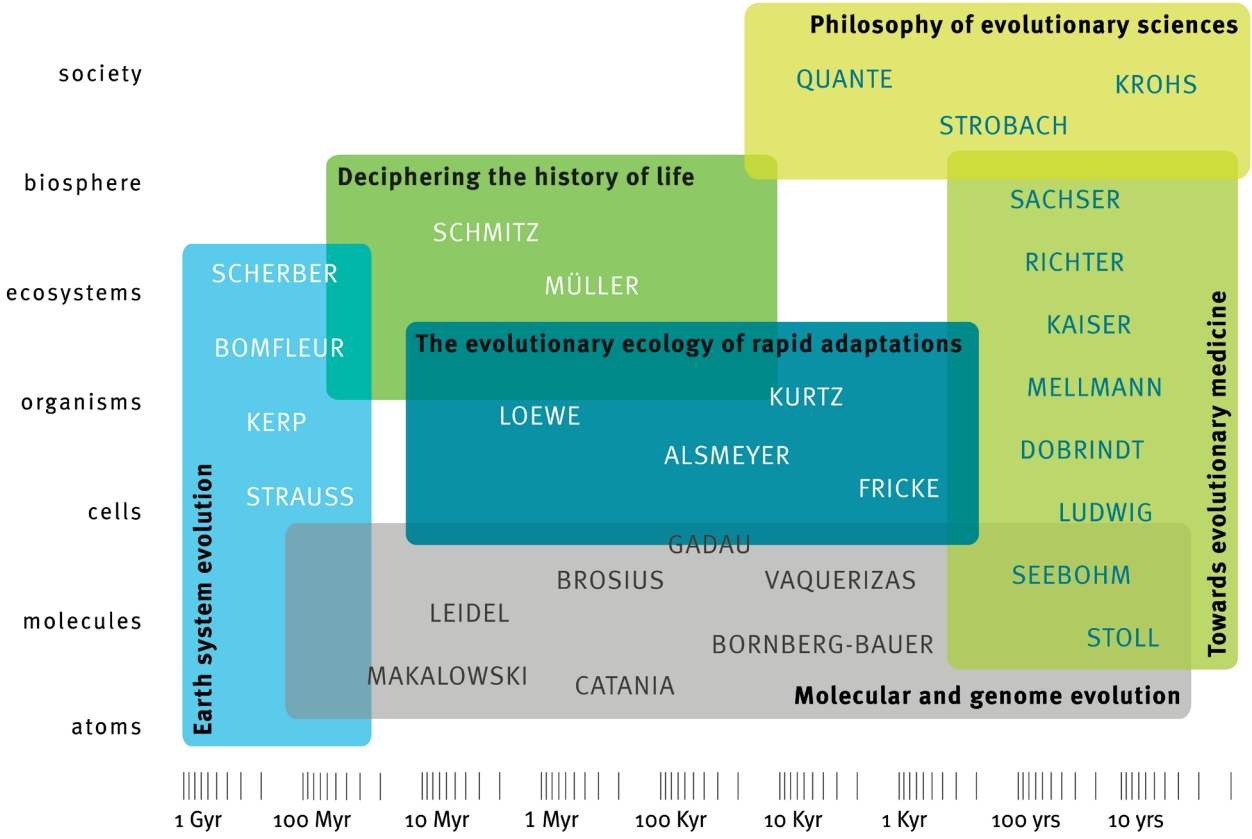
The Münster Graduate School of Evolution (MGSE) is an interdisciplinary association of researchers at the University of Münster bridging the Faculties of Biology, Medicine, Geosciences, Mathematics, and Philosophy.

The MGSE provides a structured study program for doctoral students of the different faculties in the general field of evolution. The doctoral students of the MGSE address a broad range of questions, from the evolution of earth to the evolution of evolutionary theory. MGSE students benefit from one another because similar general principles act across disciplines, thus allowing common theoretical approaches and experimental testing at different levels.

The 7th MGSE symposium will provide the doctoral students with the opportunity to present and discuss their ongoing PhD projects and to learn about studies taking place in other faculties and departments of the graduate school. Their posters and presentations are embedded into contributions from Principal Investigators of the MGSE as well as three keynote lectures by internationally outstanding guest speakers.


society
biosphere
ecosystems
organisms
cells
molecules
atoms
Philosophy of evolutionary sciences
QUANTE
KROHS
STROBACH
Deciphering the history of life
SCHMITZ
MÜLLER
SCHERBER
BOMFLEUR
KERP
STRAUSS
Earth system evolution
The evolutionary ecology of rapid adaptations
KURTZ
LOEWE
ALSMEYER
FRICKE
GADAU
BROSIUS
VAQUERIZAS
LEIDEL
BORNBERG-BAUER
MAKALOWSKI
CATANIA
Molecular and genome evolution
SACHSER
RICHTER
KAISER
MELLMANN
DOBRINDT
LUDWIG
SEEBOHM
STOLL
Towards evolutionary medicine
1 Gyr
100 Myr
10 Myr
1 Myr
100 Kyr
10 Kyr
1 Kyr
100 yrs
10 yrs

MÜNSTER GRADUATE SCHOOL OF EVOLUTION

MGSE Principal Investigators

Name, Title	Research Topic	MGSE Research Area
Alsmeyer, Prof. Dr. Gerold	Mathematical statistics	The evolutionary ecology of rapid adaptations
Bomfleur, Dr. Benjamin	Paleobotany	Earth system evolution
Bornberg-Bauer, Prof. Dr. Erich	Evolutionary bioinformatics	Molecular and genome evolution
Brosius, Prof. Dr. Jürgen	Experimental pathology	Molecular and genome evolution
Catania, Dr. Francesco	Evolutionary cell biology	Molecular and genome evolution
Dobrindt, Prof. Dr. Ulrich	Microbial genome plasticity	Towards evolutionary medicine
Fricke, Dr. Claudia	Evolution and sexual conflict	The evolutionary ecology of rapid adaptations
Gadau, Prof. Dr. Jürgen	Molecular evolution and sociobiology	Molecular and genome evolution
Kaiser, Prof. Dr. Sylvia	Behavioural biology	Towards evolutionary medicine
Kerp, Prof. Dr. Hans	Paleobotany	Earth system evolution
Krohs, Prof. Dr. Ulrich	Philosophy of science and of nature	Philosophy of evolutionary sciences
Kurtz, Prof. Dr. Joachim	Animal evolutionary ecology	The evolutionary ecology of rapid adaptations
Leidel, Dr. Sebastian	RNA biology	Molecular and genome evolution
Löwe, Prof. Dr. Matthias	Mathematical statistics	The evolutionary ecology of rapid adaptations
Ludwig, Prof. Dr. Stephan	Molecular virology	Towards evolutionary medicine
Makalowski, Prof. Dr. Wojciech	Bioinformatics	Molecular and genome evolution
Mellmann, Prof. Dr. Alexander	Hospital and environmental hygiene	Towards evolutionary medicine
Müller, Prof. Dr. Kai	Evolution of biodiversity of plants	Deciphering the history of life
Quante, Prof. Dr. Dr. h.c. Michael	Philosophy of ethics and practical philosophy	Philosophy of evolution and education research

Name, Title	Research Topic	MGSE Research Area
Sachser, Prof. Dr. Norbert	Behavioural biology	Towards evolutionary medicine
Scherber, Prof. Dr. Christoph	Animal ecology & multitrophic interactions	Earth system evolution
Schmitz, PD Dr. Jürgen	Experimental pathology	Deciphering the history of life
Seebohm, Prof. Dr. Guiscard	Myocellular electrophysiology	Towards evolutionary medicine
Stoll, Prof. Dr. Monika	Genetic epidemiology	Towards evolutionary medicine
Strauß, Prof. Dr. Harald	Historical and regional geology	Earth system evolution
Strobach, Prof. Dr. Niko	Philosophy of logic and language	Philosophy of evolution and education research
Vaquerizas, Dr. Juanma	Regulatory genomics	Molecular and genome evolution

MÜNSTER GRADUATE SCHOOL OF EVOLUTION

MGSE Doctoral Students

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Marco Chittò
Nicolle Demandt
Kevin Ferro
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Yeisson Gutiérrez López
Nadja Haarmann
Reza Halabian
Niklas Kästner
Matthias Kiel
Tabea Kischka
April Snøfrid Kleppe
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Aarón Lecanda Sánchez
Alexandra Mutwill
François Pellet
Susanne Sangenstedt
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Noble Selasi Gati
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Nina Kranke
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Amol Anandrao Patil
Binie Stieger
Valerio Vitali
Fengjun Zhang

MGSE Steering Committee

Jürgen Gadau (Speaker)
Hans Kerp
Tabea Kischka
Ulrich Krohs
Stephan Ludwig
Norbert Sachser
Susanne Sangenstedt

Ex Officio Members

Francesco Catania, Leader of the Evolution Think Tank
Vanessa Kloke, MGSE Coordinator
Joachim Kurtz, Spokesperson of the RTG EvoPAD
Maike Tietjens, Vice-Rector for Strategic Personnel Development

Ombudsperson

Hans-Dieter Görtz

Equal Opportunity Commissioner

Sylvia Kaiser

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The rapid evolution of pathogens and the rising rates of cardiovascular diseases and psychiatric disorders, pose major challenges to human health. Moreover, the distinction between health and disease may depend on individual life history and its interaction with the environment.

The Research Training Group EvoPAD (**Ev**olutionary **P**rocesses in **A**daptation and **D**isease) is an interdisciplinary PhD programme which integrates biological, medical, and philosophical research at the University of Münster with the core idea to use the theory of evolution to understand processes leading to adaptation and disease. It is funded by the German Research Foundation (DFG) and started in April 2017.

With its broad overarching scientific portfolio, the MGSE serves as umbrella organisation for EvoPAD, providing additional expertise and support. The PhD students of EvoPAD are associated to the MGSE.

RTG 2220
EvoPAD

Prof. Dr. Joachim Kurtz
Speaker of the RTG EvoPAD

Dr. Vanessa Kloke
Coordinator of the RTG EvoPAD

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Institute for Evolution and Biodiversity (IEB, Hüfferstr. 1)

In recent years, evolutionary research has become one of the focus areas of the WWU's research profile. Since its founding in 2005, the Institute for Evolution and Biodiversity (IEB) within the Faculty of Biology covers a diverse research spectrum in evolution and has attracted a number of new professors and junior groups from within and outside of Germany. The IEB currently integrates eight groups working on evolutionary ecology of animals, plants and microorganisms, phylogeny and evolution, aquatic ecology, biocomplexity, and evolutionary bioinformatics. The core question is how biodiversity and biocomplexity at all levels of the biological hierarchy arise through evolutionary processes.

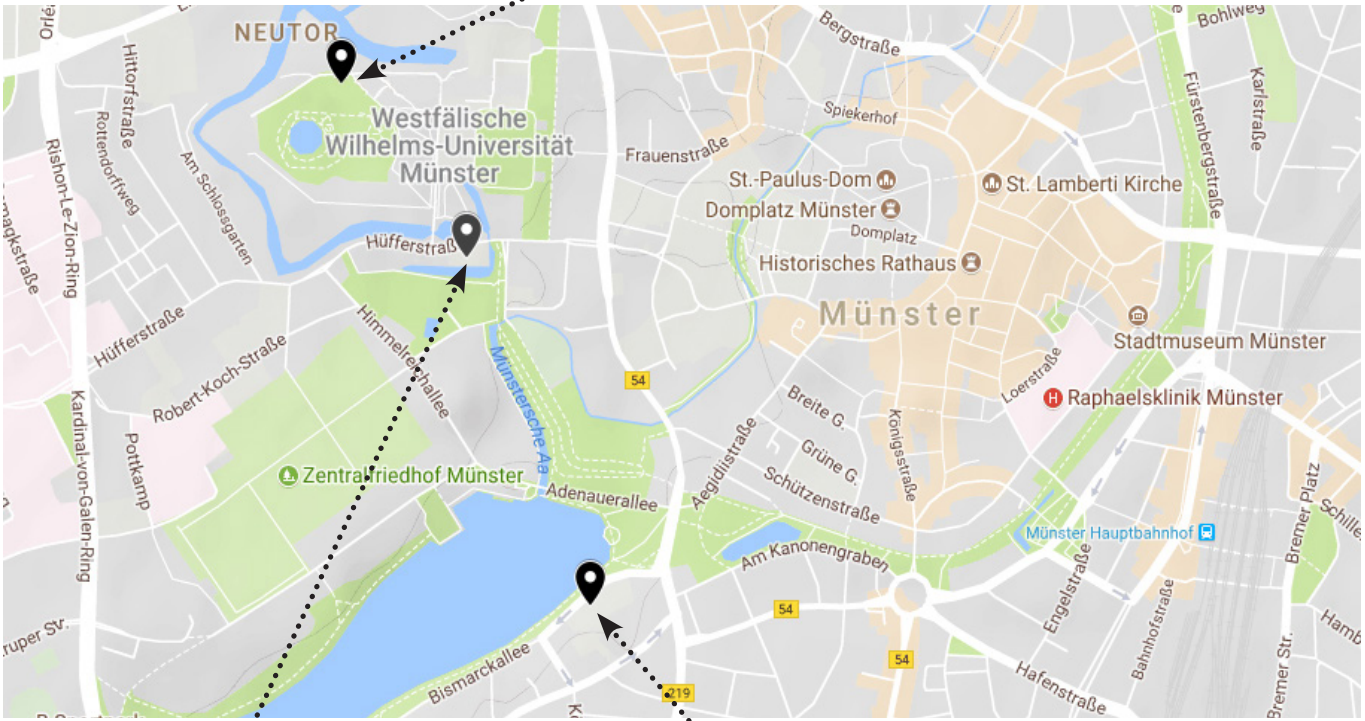
Schlossgarten Café (Schlossgarten 4)

The conference dinner will take place in the Schlossgarten Café. The restaurant is located in 5 minutes walking distance from the IEB, in the heart of Münster's most beautiful park complex and next to the Botanical Garden. The buffet opens at 19.00.

On Thursday, we offer all participants the possibility to have lunch at the canteen **"Mensa am Aasee"**. You'll find a food voucher for a meal at your choice including a non-alcoholic drink behind your name tag.

© Schloßgarten Café

Schlossgarten Café
Schlosgarten 4



Institute for Evolution and Biodiversity
Lecture Hall HHü, Hüfferstr. 1

Mensa am Aasee
Bismarckallee 11-11b

Wednesday, 21 March 2018

13.15 - 13.45 Introduction
Lecture hall HHü, Hüfferstr. 1

13.15 Welcome address by the Speaker of the MGSE, Jürgen Gadau

13.30 Welcome address by the Speaker of the RTG EvoPAD, Joachim Kurtz

13.45 - 15.45 Public lectures - EvoPAD session
Lecture hall HHü, Hüfferstr. 1

Chair: Joachim Kurtz

13.45 Michael Lynch
Arizona State University
Mutation, drift, and the origin of subcellular features

14.45 Francesco Catania
Institute for Evolution and Biodiversity
Are genetic and epigenetic inheritance linked? Lessons from *Paramecium*

15.15 Erich Bornberg-Bauer
Institute for Evolution and Biodiversity
Emergence of de novo protein coding genes from 'dark genomic matter' - fact or fiction?

15.45 - 16.15 Coffee break
Foyer, Hüfferstr. 1

16.15 - 17.00 Talks by PhD students
Lecture hall HHü, Hüfferstr. 1

Chair: Ana Sofia Lindeza

16.15 April Snøfrid Kleppe
Institute for Evolution and Biodiversity
Translational readthrough in yeast

16.30 Valerio Vitali
Institute for Evolution and Biodiversity
Sub-optimal growth temperatures impact the fidelity of Programmed DNA elimination in *Paramecium tetraurelia*

16.45 Florian Wünnemann
CHU Ste Justine Research Center, Montreal
Single cell landscape of mammalian heart maturation

17.00 - 18.15 Poster session
First floor, Hüfferstr. 1

18.30 - open end Dinner buffet
Schloßgarten Café (Schlossgarten 4)

Thursday, 22 March 2018

09.15 - 11.15 Public lectures
Lecture hall HHü, Hüfferstr. 1

Chair: Jürgen Gadau

09.15 Leo Beukeboom
University of Groningen
Evolution of insect sex determination systems

10.15 Claudia Fricke
Institute for Evolution and Biodiversity
Male reproductive ageing is accompanied by a decline in male ability to produce adequate ejaculates

10.45 Jürgen Brosius
Institute of Experimental Pathology
Exaptation: From Darwin to genomes

11.15 - 11.45 Coffee break
Foyer, Hüfferstr. 1

11.45 - 12.30 Talks by PhD students
Lecture hall HHü, Hüfferstr. 1

Chair: Matthias Kiel

11.45 Nadja Haarmann
Institute of Hygiene
In search for the factors contributing to *Escherichia coli* O104:H4 exceptional pathogenicity

12.00 Marco Chittò
Institute of Hygiene
Regulation of P4-type integrase expression in uropathogenic *Escherichia coli*

12.15 François Pellet
Department of Philosophy
What is disease?

12.30 - 14.00 Lunch break
Canteen at the Aasee

14.00 - 14.45 Talks by PhD students
Lecture hall HHü, Hüfferstr. 1

Chair: Sergio Ávila Calero

14.00 Nicolle Demandt
Institute for Evolution and Biodiversity
Experimentally parasite infected sticklebacks increase risk-taking behaviour of uninfected shoal members

14.15 Alexandra Mutwill
Department of Behavioural Biology
Shaping & reshaping of biobehavioural profiles beyond adolescence – A study in guinea pigs

14.30 Niklas Kästner
Department of Behavioural Biology
A brain without serotonin - effects of complete brain serotonin deficiency on social behaviour in female mice

14.45 - 15.15 Coffee break
Foyer, Hüfferstr. 1

15.15 - 16.15 Public lecture
Lecture hall HHü, Hüfferstr. 1

Chair: Claudia Fricke

15.15 Paula Stockley
University of Liverpool
Cryptic diversity explained by sexual selection

16.15 Farewell
Lecture hall HHü, Hüfferstr. 1

Mutation, drift, and the origin of subcellular features

MÜNSTER GRADUATE SCHOOL OF EVOLUTION

KEYNOTE LECTURE

organised in cooperation with

RTG 2220 EvoPAD

Although natural selection may be the most powerful force in the biological world, it is not all powerful. As a consequence, many aspects of evolution of the molecular level can only be explained by the inability of natural selection to operate. This general principle explains a lot about the diversity of genome architectures across species, and also appears to extend to numerous higher-level features of cells: the evolution of the ~1000-fold range in mutation rates that exists among species; greatly elevated rates of transcription error; the divergence of the multimeric states of proteins; and the phylogenetic drift of gene-regulatory vocabulary.

An attempt will be made to describe how these biological observations can be explained at the theoretical level, in some cases using methods derived from statistical mechanics. A fundamental principle is that although natural selection relentlessly pushes traits to the highest possible level of refinement, the limits to perfection are dictated by the power of random genetic drift rather than by intrinsic molecular limitations on repair mechanisms or by selection for an optimum mutation rate. The implications of this drift-barrier hypothesis are that the population-genetic environment imposes a fundamental constraint on the paths that are open vs. closed for evolutionary exploration in various phylogenetic lineages, hence defining the patterns of adaptation seen at the molecular and cellular level. Additional examples may be drawn from recent observations on the bioenergetic costs of maintaining and expressing genes.

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KEYNOTE LECTURE

Leo W. Beukeboom

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University of Groningen

Sex determination is a universal developmental process resulting in two sexes, female and male. The genetic mechanisms of sex determination are very diverse and evolutionary labile. Insects exhibit a variety of mechanisms at the chromosomal and gene level. Many groups possess sex chromosomes that carry sex-specific sex determining genes. Male heterogamety (females XX, males XY or XO) is most abundant, but female heterogamety (females ZW or ZO, males ZZ) and haplodiploidy (females diploid, males haploid) occurs in some groups. Insect sex determination pathways consist of series of genes that regulate each other in a hierarchical fashion. The chromosomal constitution functions as a primary signal. Sex chromosomes can carry a dominant male (e.g. Y chromosome) or female (e.g. W chromosome) determining gene or their number is being counted (e.g. XX/XO system). In most insects, transformer processes the primary signal and regulates the sex-specific splicing of the binary switch gene doublesex at the bottom of the cascade. Sex determination mechanisms can evolve by gene duplication, changes in hierarchical position of genes, novel molecular function of genes, and alterations in transcriptional or translational regulation. Examples of each of these processes will be provided.

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Cryptic diversity explained by sexual selection

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Darwin's original insights on sexual selection were influenced by examples of conspicuous male contest competition and ornamentation. More recent studies offer a broader perspective, driven by investigation of less conspicuous but equally fascinating adaptations linked to post-copulatory sexual selection. Using mammalian examples, I will illustrate how this broader perspective affords important new insights for understanding the function of diverse and enigmatic male reproductive traits such as penis bones, mating plugs and sexual accessory gland secretions.

KEYNOTE LECTURE

Paula Stockley

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Are genetic and epigenetic inheritance linked? Lessons from Paramecium

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The core tenets of the Modern Evolutionary Synthesis are that mutations occur at random and that heritable genetic variation fuels adaptation through natural selection. These tenets neglect the impact that the external environment may have in triggering the emergence of non-random changes, a possibility that supporters of epigenetic inheritance vigorously maintain. Here, I will present some of our recent findings on experimentally-evolved lines of the single-celled ciliate *Paramecium*. Our observations clearly show environmentally-induced variation and indicate a viable path towards the synthesis of models of evolution based on genetic and epigenetic inheritance.

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Rebecca Hagen
Valerio Vitali

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Emergence of de novo protein coding genes from 'dark genomic matter' - fact or fiction?

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Proteins are the workhorses of the cell and, over billions of years, they have evolved an amazing plethora of extremely diverse and versatile structures with equally diverse functions echoing the evolution of all forms of life. Recent computational developments and availability of thousands of genomes make it possible to reconstruct the course of evolution, often at amazing accuracy. Previously, it was assumed that all proteins result from old ones but recent advances in comparative genomics have repeatedly called some 10%-30% "orphan" genes which contain ORFs, are expressed and translated but lack a homolog in closely related outgroups. Since some map to inter-genic regions, thy have emerged "de novo" from previously non-coding 'dark genomic matter'. While novel proteins in general tend to be disordered, fast evolving, weakly expressed but also rapidly assuming novel and physiologically important functions, *de novo* transcripts already show clear signs of structural maturity. We investigated the dense and data-rich insect phylogeny genomes which features many major innovations such as sociality, viviparity etc. First, we find that recently split lineages undergo accelerated genomic reorganisation, including the rapid gain of several hundred novel genes. Second, novel genes are particularly abundant in social insects. Third, novel genes in ants are scattered uniformly across genome and between established genes. Finally, our results indicate that the genetic mechanisms creating orphan genes - such as gene duplication, frame-shift fixation, creation of overlapping genes, horizontal gene transfer, and exaptation of transposable elements - act at different rates in insects when compared to other animals.

Male reproductive ageing is accompanied by a decline in male ability to produce adequate ejaculates

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Senescence, the deterioration of physiological function and increased risk of death with advancing age, is accompanied by a loss of reproductive functions. We used the fruit fly *Drosophila melanogaster* to document reproductive ageing in males by studying known determinants of male reproductive success. Male reproductive ageing to date has been mainly explained by a reduction in sperm quality. However, in addition to sperm males also transfer seminal fluid proteins (Sfps) in their ejaculates, which cause profound changes in females upon receipt at mating and are vital for male reproductive success. We here allowed males to age up to seven weeks and then tested their ability to reproduce successfully. We particularly measured aging male ability to induce female post-mating changes known to be elicited by Sfps. As we found male ability to induce these female post-mating changes to decrease with age we then investigated age-dependent gene expression changes for five representative Sfp genes. We found a decrease in male Sfp gene expression with age that matched our findings of reduce phenotypic responses. Hence, male reproductive ageing is accompanied by a decline in functionality of the male accessory gland, the main tissue producing the majority of the male Sfps. Together our findings indicate that with advancing age male flies transfer less effective ejaculates with significant effects for male reproductive success.

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Mareike Koppik
Hanna Ruhmann

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Jürgen Brosius

Institute of Experimental Pathology
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Darwin was among the first to notice that (mostly disused) organs or anatomical structures can acquire new functions. About 80 years ago, scientists began to realize that the duplication of genes was a necessary prerequisite for the generation of novelty at the molecular level. Apart from proposing the term "exaptation" instead of the teleologically burdened "preadaptation", Vrba andGould suggested that hitherto neutrally evolving nonaptations could be a source for exaptations as well. This concept was perfectly borne out by discoveries at the genetic level, e.g., that neutrally evolving transposed elements (mostly parts thereof) were exapted as various gene modules acting as diverse regulatory elements and even novel protein coding exons. Examples from phylogenetic studies on TE exaptation underscored that, although this process is not rare, persistence is less frequent. Exaptation can occur at any time after TE insertion proving that potentially any randomized neutral sequence (nonaptations) can serve as raw material for exaptations and, thus, the extra junk of our genomes can be considered a store or resource for potential aptations. Naturally less frequent than exaptation of novel modules modifying existing genes, complete *de novo* arisen protein coding genes have been reported. Due to the pervasive transcription of genomes, neutral transcripts also can be exapted as functional non-protein coding RNAs (npcRNAs). For example, a recent study revealed that promoter activity of protein coding RefSeq genes are frequently (co)regulated via the act of transcription from a nearby overlapping locus by inhibiting binding of transcription factors or RNA polymerase. The resulting transcripts whose *sequences* (apart from the promoter elements on the DNA they are traversing) are not under purifying selection, yet the transcripts have the potential to be exapted as npcRNAs.

Translational readthrough in yeast

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During protein synthesis the genetic instructions are passed from DNA via mRNA to the ribosome to assemble a protein chain. Occasionally the ribosome ignores stop codons in the mRNA and translation continues into the untranslated region (3'-UTR). This process, called translational readthrough (TR), yields a peptide chain that becomes longer than one would predict from the DNA sequence alone.
Peptide sequences vary in propensity for translational errors, which may yield evolutionarily constraint by limiting evolutionary paths. What opportunities exist for proteins to evolve and for novel traits to come about? The effects of erroneous translation on protein evolution is little understood. Here we investigated TR in *Saccharomyces cerevisiae* by analysing ribosome profiling data. We clustered proteins as either prone or non-prone to TR, and conducted comparative analyses. We find a relatively high frequency of TR across a broad range of genes, including ribosomal subunit proteins. Our main finding is that proteins undergoing TR are highly expressed and have intrinsically disordered C-termini. We suggest that highly expressed proteins compensate for the deleterious effects of TR by having intrinsically disordered C-termini.

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Sub-optimal growth temperatures impact the fidelity of Programmed DNA elimination in Paramecium tetraurelia

Valerio Vitali
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Although ciliates Internal Eliminated Sequences (IESs) have decayed from transposons into non-autonomous genetic elements, the extent to which they continue to play an active role in shaping genomes structure is not known. At each event of sexual generation, IESs must be precisely eliminated from the germline so that a functional somatic genome may be reconstituted via Programmed DNA elimination (PDE). Although organismal survival requires the high fidelity of PDE, evidence suggests that IES excision is not foolproof. Moreover, PDE-mediated somatic variability may be inherited across sexual generations as a consequence of epigenetic mechanisms, making heritable somatic mutations eligible for environmental evaluation. Furthermore, the extent to which the efficiency of PDE is influenced by different environmental conditions is yet to be established. To address this gap, we allowed clonal *Paramecium tetraurelia* lines to undergo autogamy at suboptimal growth temperatures and subjected the resulting somatic genomes to ultra-deep Illumina-sequencing. We find that departures from the optimal growth temperature during autogamy can significantly affect the efficiency and specificity of PDE. Even further, we show that most of the significantly retained IESs in response to sub-optimal growth temperatures lie within exons and have elevated retention scores (IRS); thus, an environmental change can substantially alter IES excision in hundreds of genes.

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Single cell landscape of mammalian heart maturation

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The heart is the earliest organ to form in the developing mammalian embryo. A number of different cell types contribute to the complex development and growth of the heart. While a small set of canonical markers for the identification of different cardiac cell types have been identified, the molecular footprint of primary heart cells during development remains unknown. Novel advances in sequencing technologies now enable the parallel transcriptomic profiling of tens of thousand single-cells from complex tissues. Using a single-cell transcriptomics (scRNA-seq) approach known as Drop-seq, we analyzed single cells from mouse hearts during stages of heart growth and maturation. Single-cell transcriptomics enabled the identification of known cardiac cell types as well as novel sub populations and previously unknown marker genes in the heart. Cell type specific gene expression changes during stages post heart formation revealed critical switches in gene regulatory networks responsible for maturation of major heart structures. To unravel the evolutionary conservation of heart maturation in different species, we applied Drop-seq to human as well as rat hearts. Comparative analysis of single-cell transcriptomes between species highlighted conserved cell type signatures contributing to the maturation of the fully functional mammalian heart.

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In search for the factors contributing to Escherichia coli O104:H4 exceptional pathogenicity

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Ivan Kouzel
Helge Karch
Petya Berger
Alexander Mellmann

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Escherichia coli O104:H4 (*E.coli* O104:H4) was identified as the pathogen causing the largest outbreak of bloody diarrhea and haemolytic syndrome (HUS) in Germany in 2011. It is hypothesized that the exceptional pathogenicity of this strain derives from its hybrid character with virulence determinants originating from enterohemorrhagic *E. coli* (EHEC; Shiga toxin) and enteroaggregative *E. coli* (EAEC; aggregative adherence fimbriae I), respectively. Here, we address the question if *E. coli* O104:H4 displays virulence traits superior to the ones of regular EAEC strains, which could thus contribute to its exceptional pathogenicity. In this study, we compare the *E. coli* O104:H4 strain C227-11 ϕcu (cured of the Shiga toxin-encoding phage) with the prototypical EAEC strains 55989, 042, 17-2. The strains were characterized using adherence, biofilm, motility, acid resistance, ELISA IL-8 and cytotoxicity assays. The outbreak strain C227-11 ϕcu and the EAEC strain 17-2 adhere strongest to epithelial cells. EAEC 042 is the best Biofilm producer of the characterized strains, as well as displays the strongest motility and triggers the strongest IL-8 response. C227-11 ϕcu and EAEC 55989 behave similarly in most phenotypical assays, which is in agreement with their close genotypic relationship. Our phenotypical characterization lead us to the conclusion that *E. coli* O104:H4 displays strong but not exceptional virulence traits when compared to the analyzed EAEC strains. Thus our data indicate that the synergistic effect of EHEC and EAEC virulence factors rather than a superior EAEC phenotype contribute to the exceptional pathogenicity of the 2011 outbreak strain.

Regulation of P4-type integrase expression in uropathogenic Escherichia coli

MÜNSTER GRADUATE SCHOOL OF EVOLUTION

Horizontal acquisition and loss of genomic islands (GEIs) play key roles for bacterial evolution and the adaptation of pathogens during pathogen-host interaction. P4-type integrases are believed to play key roles in the chromosomal insertion and excision of GEIs. This type of integrase usually specifically recognizes the encoding GEI. However, not much is known about the regulation of integrase expression. We therefore investigate (i) environmental conditions which modulate expression of integrase-encoding genes in *E. coli*, and (ii) the impact of nucleoid associated proteins on expression of integrase genes. Reporter gene fusions were generated in the chromosome of uropathogenic *E. coli* strain 536 in order to monitor promoter strength and regulation of the P4 integrase-encoding genes of PAI I536-PAI VI536. Our data showed that the promoter of the integrase gene of PAI I536 is the most active one followed by the promoter of the integrase genes of PAI II536 and PAI III536, while the activity of the promoters of the integrase genes of PAI IV536 PAI V536 and PAI VI536 are very weak under the conditions tested. Overall the expression level of the integrase genes appears to correlate with the instability of their cognate GEIs. In order to screen for potential regulators of integrase expression, the genes *fis*, *hns*, *hupA*, *hupB*, *ihfA*, *ihfB*, *lrp*, *stpA*, *dps* coding for the major nucleoid-associated proteins (NAPs) were inactivated. The impact of different NAPs on the integrase promoter activities is currently under investigation.

Marco Chittò
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In the contemporary literature about the nature of disease, we can distinguish between two main groups of theories of disease, which may be labeled “axiologism about disease” and “dysfunctionalism about disease”. These two groups of theories of theories are based on two different intuitions that we have about what disease is.

Axiologism about disease accounts for our intuition that attributing to a certain part of an organism the property of being diseased is negatively evaluating this part of the organism, viz. attributing to it a certain negative value; saying “x is diseased” is a certain negative evaluative judgment. We would intuitively say that the specific negative value at issue is a certain lethal value (along death), by contradistinction with a vital value. Examples of vital values are the values of life and health.

Dysfunctionalism about disease accounts for the intuition that, when we judge that a certain part of an organism is diseased, we mean by this that this part of the organism incorrectly functions (malfunctions, or is dysfunctional). Saying that, e.g., x’s liver is diseased is saying that x’s liver incorrectly functions.

It is obvious that a definition of disease should account for, in a unified and coherent way, both intuitions at the basis of axiologism and dysfunctionalism about disease. The purpose of this talk is to suggest a definition of disease - that we may label “essentialism about disease” -, according to which disease is the destruction of the essence of a processual part of a good organism.

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Experimentally parasite infected sticklebacks increase risk-taking behaviour of uninfected shoal members

Trophically transmitted parasites frequently increase their host risk-taking behaviour, facilitating transmission to the next host. The consequences of such increased risk-taking for uninfected group members have hitherto received little attention. We hypothesized that increased host risk-taking behaviour can spill over to uninfected group members, thereby increasing the risk-taking of uninfected group members. We used the tapeworm *Schistocephalus solidus*, which increases the risk-taking behaviour of its three-spined stickleback host (*Gasterosteus aculeatus*), to facilitate transmission to its final host, a fish-eating bird. Groups of 6 sticklebacks with 0, 2, 4 or 6 experimentally infected individuals were confronted with a simulated bird attack, whereby we scored risk-taking of each individual pre- and post-attack. Prior to attack, we found no differences between infected and uninfected individuals. However, after the attack, individuals from groups consisting of only infected individuals showed higher risk-taking behaviour than individuals from groups with only uninfected individuals. More importantly, the risk-taking behaviour of infected individuals was unaffected by the number of uninfected group members. In contrast, uninfected individuals increased their risk-taking behaviour with increasing number of infected group members. Our results show that parasite infection not only increases risk-taking of infected hosts, but also of uninfected group members, shedding new light on the social dynamics involved in host-parasite interactions.

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Phenotypic plasticity describes the ability of individuals to change phenotypic traits in response to the environment. After focusing on pre- and postnatal development as sensitive phases, recently adolescence has been identified as another time window for phenotypic adjustment in mammals. Using male guinea pigs as a model system, the aim of the present study was to investigate in which way biobehavioural profiles are shaped by different social environments and whether they are reshaped after transfer to another social condition in early adulthood. To test this, a low and a high individual number situation were explored. After weaning, males were either kept together with one female each in pairs (P) or in large mixed-sex colonies with a graduated age structure (C). From an age of 120 days on, individual cortisol and testosterone responsiveness to novelty was assessed on a monthly basis. At 240 days of age, males of both conditions were transferred to a P-situation with an unfamiliar female. One month later, hormonal responsiveness to novelty was assessed again. Before transfer, P-males were shaped for a high cortisol responsiveness and low testosterone levels, whereas C-males showed a low cortisol responsiveness and high testosterone levels. After transfer to a P-situation in early adulthood, biobehavioural profiles of C-males were reshaped towards low testosterone levels and a high cortisol responsiveness resembling the profile of P-males. P-males themselves maintained their original endocrine profile. The present study clearly showed that shaping and reshaping of biobehavioural profiles is possible beyond adolescence, suggesting that plasticity can be preserved also in early adulthood.

A BRAIN WITHOUT SEROTONIN - EFFECTS OF COMPLETE BRAIN SEROTONIN DEFICIENCY ON SOCIAL BEHAVIOUR IN FEMALE MICE

Serotonin (5-HT) is an evolutionary ancient molecule. As a neurotransmitter in the mammalian brain, it plays a key role in the development of the central nervous system as well as the fine-tuning of almost every behavioural system. Surprisingly, however, Tph2 knockout mice (KO) completely lacking brain 5-HT are viable and brain morphology is not different from that of their wildtype conspecifics (WT). This study aimed to elucidate the effects of 5-HT deficiency on social behaviour in female mice. For this purpose, female Tph2 wildtype (n = 40) and homozygous knockout mice (n = 40) were housed with a same-sex conspecific of either the same or the opposite genotype in large, enriched terraria. Mice were filmed for 2 hours during the dark phase using an infra-red camera system, and social behaviour was recorded. The main findings were: KO females displayed overall more social investigation behaviour and were much more aggressive than the expectably docile WT females. Most interestingly, when two KO females were housed together, one was highly aggressive, while the other displayed defensive behaviours. Thus, while female social behaviour is altered by 5-HT deficiency and seems to be shifted towards male-typical traits, it still lies within the normal range of this species' behaviour. It remains a major question, how the brain compensates the complete loss of this evolutionary conserved system. Shedding light on this might contribute to a better understanding of the important role of 5-HT in the modulation of mammalian behaviour.

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Evolution of new genes in primates

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Many genes are restricted to a single species or clade. Multiple mechanisms have been proposed to explain the evolution of novel genes. These include extreme divergence of duplicated genes, horizontal gene transfer from distant species, and *de novo* origins form ancestrally non-coding DNA. *De novo* genes are of particular interest as represent genes without homologs. While multiple examples of *de novo* originated genes have been found several open questions remain including how best to identify novel genes, their precise mechanism of origin, and the how they become integrated into biological processes. Using comparative bioinformatics approaches we identify candidate *de novo* genes in 11 primate species. Moreover, we compare the properties of putative *de novo* transcripts and protein-coding genes with conserved genes to shed light on the origins and evolution of novel genes and determine the transition from ancestrally non-coding genomic sequences to established protein-coding genes.

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Enzyme evolution: Balancing stability, folding and catalytic specificity assisted by ancestral reconstruction

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Evolutionary pathways by which proteins have evolved in Nature over billions of years have resulted in a diversity of structures that carry out many functions. Directed protein evolution in the test tube can emulate natural evolution, but is often limited by low hit rates and small improvements during evolutionary cycles. Furthermore, the combination of mutations that is needed for large improvements cannot always be reached by one-by-one mutational steps due to general loss-of-function or epistatic ratchets. The question then arises how evolutionary dead ends can be avoided. Important parameters that shape these fitness landscapes around proteins are e.g. stability and catalytic activity/specificity. We have reconstructed ancestral sequences inferred from phylogenetic relationships between members of the alkaline phosphatase superfamily1-3. Expression of these enzymes and mapping substrate specificity on the genetic relationships allowed the identification of the ancestral nodes between which transitions in primary function occur. The latter is one of the key processes in the evolution of new functions. The specificities of current enzymes suggest that the change in primary function is the result of a shift in substrate preference rather than a de novo evolutionary invention of a activity. The main aim of this project is to explore sequence space these ancestral and extant AP-superfamily enzymes using directed evolution approaches. Connecting the 'fitness parameters' i.e. the intricate interplay of activity/stability/epistasis, of the ancestral and extant enzymes to the shape of their local fitness landscape should provide quantitative insight into which properties are most important in determining the evolvability of enzymes.

Analysis of reassortment events of influenza A viruses from human and swine origin, in different host models

The two main mechanisms driving the evolution of IAV are: 1) accumulation of point mutations and 2) combination of genomic segments from different parental strains caused by co-infection. This reassortment process is responsible for the generation of most flu pandemics in the past, particularly the latest H1N1-2009 "Swine flu". The genome of this pandemic virus consists of RNA segments from human, avian and swine origin. Since its outbreak in 2009, this strain has fully replaced the previously circulating H1N1 lineage in the human population. Moreover, it has been re-introduced into the swine population, generating a variety of new reassortants in combination with the circulating swine IAVs. This high dynamic of reassortment and adaptive mutations is of public health concern, since it is currently unclear whether these variants may pose a risk to humans.

To assess whether this process is also happening during co-infection of cells in vitro, our aim is to study the reassortment between human H1N1pdm09 and swine IAV, which produced reassortant IAVs in nature in the past. For this purpose, we will perform co-infection of swine as well as human lung epithelial cells and then apply 7 successive passages to allow reassortment to occur. After each passage, we will analyse the genome composition of the viral progeny by deep sequencing. Our specific goals are: to evaluate the differences in the reassortment process in the two different hosts, and to analyse the viral fitness of the resultant reassortant viruses, considering the effects of mutations originated from adaptation during passaging.

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The field of *de novo* gene emergence has experienced rapid growth in recent years, and looks to characterize the appearance of new protein-coding genes from previously non-coding genetic material. Such a scenario is intuitively unlikely, given the vastness of sequence space and the apparent rarity of functional proteins within it. However, genes from a range of eukaryotic organisms have recently been found to have emerged from both intergenic and intronic regions. Comparative genomic approaches have further indicated the ubiquity of this mechanism and described the general sequence properties of *de novo* emerged genes. Furthermore, in Drosophila species, novel genes frequently show testis-biased expression and have been shown to play a crucial role in male-fertility – but to date no *de novo* protein has been fully characterized, leaving a gap in our understanding of their likely structural and functional properties.

We aim to functionally and structurally characterize a number of *de novo* proteins. This will hopefully shed some light on the early evolutionary steps taken by these newly born genes, as well as the properties of the 'random' sequences from which they emerge.

Additionally, a better understanding of this category of proteins may help inform future protein engineering strategies: recently emerged proteins may represent especially evolvable starting points for directed evolution efforts, opening the door to exploration of previously inaccessible functionalities by means of directed evolution.

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Tribolium castaneum and E. coli Nissle 1917 - a novel experimental system for studying probiotics and host-parasite interactions

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Experimental studies of the interactions of probiotic bacteria with their hosts and the resulting fitness effects can provide important insight into host-microbiome evolution. *Escherichia coli* Nissle 1917 (EcN) is a nonpathogenic *E. coli* strain, used as a probiotic for treatment of various intestinal disorders in humans. This strain does not express pathogenicity factors such as adhesins or toxins. It is not invasive and not uropathogenic, but rapidly killed by non-specific defense factors of blood serum and has antagonistic activities against other microorganisms. Several traits contribute to this strain's probiotic character, incl. its anti-inflammatory activity, inforcement of the intestinal epithelial barrier, promotion of colonic motility and induction of the gut immune response. For large-scale experimental approaches, the availability of a simple invertebrate model host would be ideal, but is currently not available. We here introduce the red flour beetle *Tribolium castaneum*, a well-established model organism for studies of evolution and infections, as a suitable experimental host for the probiotic strain EcN. Our preliminary experiments have shown that EcN colonizes the gut of the beetles when added to the diet. We will analyze effects of EcN on the fitness and life span of the beetles after successful colonization of their gut. This novel experimental system will also enable to further investigate pathogen evolution. Serial passage experiments of the pathogen in hosts with different immune status and with and without their natural microbiota will be conducted. Bacterial evolutionary processes will be studied by tracking changes of both the bacterial phenotype (e.g., virulence) and the genotype (e.g., re-sequencing of bacterial populations and representative clones)of EcN and give insight into its behavior in different host organisms.

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Phosphorylation of serine 205 of influenza A virus NS1 protein as determinant of adaptation and functional evolution

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Influenza A virus (IAV) infections are still a major burden of mankind. High evolution rates as well as the ability to infect a wide range of hosts leads to seasonal epidemics and occasional pandemics. IAV non-structural protein 1 (NS1) is a multi-functional protein that plays diverse roles during virus replication and has been linked to host adaptation. Currently circulating H1N1 viruses in humans show six amino acid changes compared to the swine origin pandemic (pH1N1) virus, which entered the human population in 2009 and eradicated previously circulating H1N1 viruses. In this regard, NS1 of pre-pandemic H1N1 showed high prevalence of serine (S) at position 205 while pH1N1 exhibits an asparagine (N) at this position. Over the course of adaptation in humans, pH1N1 re-acquired S205 suggesting an important role in host adaptation. Interestingly, we found NS1 S205 to be phosphorylated during pre-pandemic H1N1 virus infection. To analyze the importance of S205 phosphorylation in viral replication and host adaptation, we substituted S205 of a pre-pH1N1 to non-phosphorylatable glycine, aspartic acid to mimic constitutive phosphorylation as well as pH1N1 asparagine. Phosphorylation mutants replicated less efficiently compared to wild type. Furthermore, these mutants showed decreased amounts of viral proteins, which can be attributed to a diminished expression of viral mRNAs. So far, we hypothesize that tight temporal regulation of S205 phosphorylation is needed for efficient viral replication and might be re-gained as determinant of functional evolution during adaptation of pH1N1 to the human host, which will be focus of future studies.

The GBoL5 WebApp - A DNA barcode management and analysis system

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DNA barcoding allows to identify species using predefined target genes. It proves especially useful when samples are degraded, fragmented or consist of hard to identify parts, e. g. larval stages of insects or seeds and roots of plants. Multiple national and international groups and consortia work on creating a reference database of these marker gene sequences. The German Barcode of Life (GBoL) project started its first funding period in 2012, aiming to create such a database of all common and frequent species in Germany.
The responsibility of the GBoL5 sub-project is barcoding the approximately 4,800 land plant species native to Germany. Coordinating the efforts of the participating institutes from different parts of Germany creates the need for a shared information management system. The web application presented here provides users with tools such as automatic primer read pre-processing, automated read assembly to contigs, various query possibilities and more. These tools and the uploaded data, e. g. target species and marker sequences, are available at all times from various devices through an online interface. To increase its usefulness, new features are constantly developed and deployed. A future focus is on implementing analysis methods directly into the database-backed app to allow for statistical testing of biological hypothesis. Another important aspect is the integration of next generation sequencing data into the existing structure of the app.

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