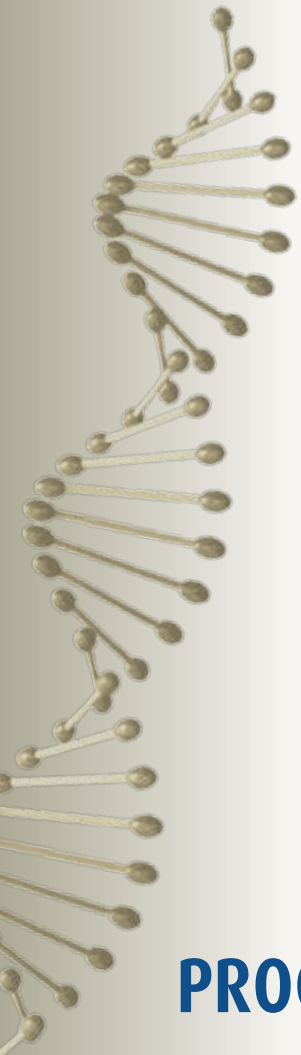
Annual Conference of the German Genetics Society (GfG)

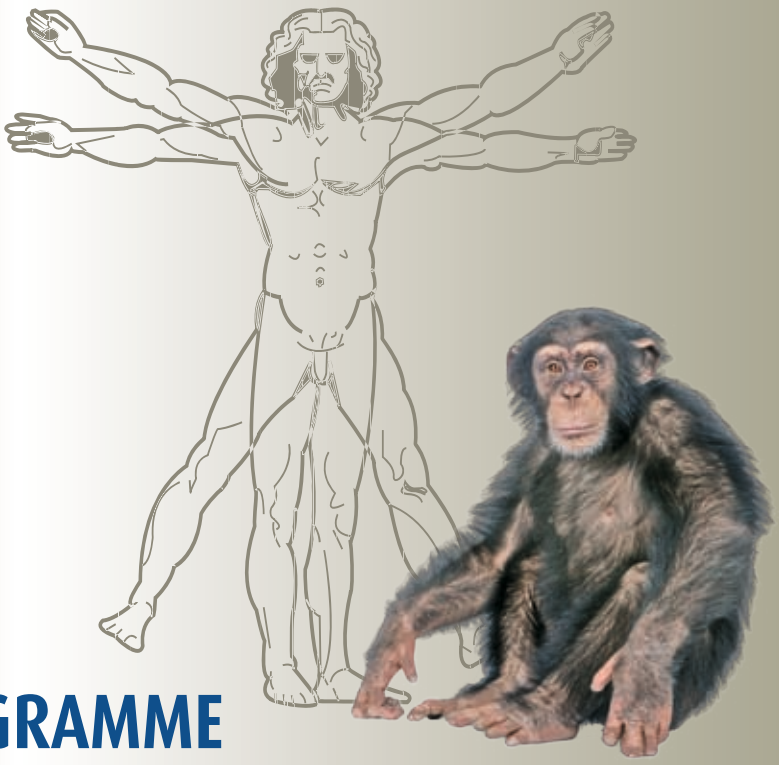
Evolution of Primates

16–18 September

2010

Jena • Germany





PROGRAMME

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Organisation and imprint

Venue and date
Friedrich-Schiller-University Jena
Rosensäle
Fürstengraben 27 • 07743 Jena (DE)

16–18 September 2010

Conference website
www.conventus.de/genetics2010

Organiser
German Genetics Society (GfG)
www.gfgenetik.de

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Institute of Human Genetics

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Dear colleagues and friends,

On behalf of the board of the German Genetics Society we kindly invite you to our Annual Conference to Jena, 16–18 September 2010. Our meeting will cover the topic "Evolution of Primates" with special emphasis on behavioral evolution, evolution of genome organisation, karyotype evolution, population genetics & principles of evolution and sequence evolution.

This topic fits perfectly in the frame of cultural Jena, home of Ernst Haeckel and its well-known Phylogenetic Museum dedicated to illustrate the development of life.

You are cordially invited to bring your scientific expertise to this conference and to make it, with your personal contribution, an outstanding scientific event.

It will be a great pleasure to welcome you to Jena in September 2010!

Yours sincerely,

Dr. Anja Weise
Friedrich-Schiller-University Jena
Institute of Human Genetics

Prof. Dr. Aria Baniahmad
Friedrich-Schiller-University Jena
Institute of Human Genetics

The abstracts can be found at the end of the programme brochure. The abstract numbers are stated before the presentation title.

14^{00}–14^{35}	**Welcome notes**
14^{35}–15^{35} Chair	**Population Genetics & Principles of evolution I** Christoph Englert (Jena/DE)
14^{35} **1**	**Sex and molecular evolution** Brian Charlesworth (Edinburgh/GB)
15^{05} **2**	**An ancient evolutionary origin of genes associated with human genetic diseases and cancer** Diethard Tautz (Plön/DE)
15^{35}–16^{00}	Coffee break & Visit of industrial exhibition
16^{00}–17^{30} Chair	**Population Genetics & Principles of evolution II** Wim Damen (Jena/DE)
16^{00} **3**	**The Neandertal Genome: insights into human origins** Janet Kelso (Leipzig/DE)
16^{30} **4**	**Recent positive selection of a human AR/EDA2R haplotype and its relationship to male pattern baldness** Axel Hillmer (Singapore/SG)
17^{00} **5**	**Primate-specific alternative splice site in CRYGA forms a truncated A-crystalline** Jochen Graw (Oberschleißheim/DE)
from 17^{30}	**Public lecture/öffentlicher Vortrag** **Bewegung und ihre Bedeutung für die Evolution der Primaten*** Manuela Schmidt (Jena/DE)
18^{45}	Get together in the Botanical Garden Jena (see page 18)

*This lecture will be held in German language.

09^{00}–10^{30} **Karyotype evolution I**
Chair Stephan Diekmann (Jena/DE)

09^{00} **Chromosomal evolution and heterochromatin in primate genomes**
6 Yuri Yurov (Moscow/RU)

09^{30} **Inter- and intraspecific Y chromosome variations in human and great apes**
7 Werner Schempp (Freiburg i. Br./DE)

10^{00} **An ancient addition to the ancestral part of the mammalian X chromosome was already enriched for brain specific genes**
8 Horst Hameister (Neu-Ulm/DE)

10^{30}–11^{00} Coffee break & Visit of industrial exhibition

11^{00}–12^{45} **Karyotype evolution II**
Chair Günter Theißen (Jena/DE)

11^{00} **Comparative cytogenetics technologies**
9 Thomas Liehr (Jena/DE)

11^{30} **Evolutionary breakpoints and fragile sites**
10 Kristin Mrasek (Jena/DE)

12^{00} **Outgroup cytogenetics**
11 Vladimir Trifonov (Jena/DE)

12^{30} **Aneuploidization of the brain is evolutionary conserved in vertebrates, while protective "antianeuploidization" is more effective in hominids**
12 Yuri Yurov (Moscow/RU)

12^{45}–15^{30} Lunch break & Visit of industrial exhibition

14^{00}–15^{30} **Member assembly-meeting**

15^{30}–16^{00} Coffee break & Visit of industrial exhibition

16^{00}–16^{30} **Gateff-Prize award**

16^{30}–18^{00}	**Evolution of genome organisation**
Chair	Mathias Platzer (Jena/DE)
16^{30}	**A population genomic approach to map recent positive selection**
13	Wolfgang Stephan (Planegg-Martinsried/DE)
17^{00}	**Evolution of novel gene functions by molecular domestication of**
14	**transposable elements**
	Zoltan Ivics (Berlin/DE)
17^{30}	**Mechanics of Chromosome and Karyotype Evolution – a Magnifying Lens**
15	**for Palaeogenomics**
	Ingo Schubert (Gatersleben/DE)

The next Annual Conference of the German Genetics Society will take place in September 2011 in Würzburg.

For further information please refer to www.conventus.de/genetics2011

08^{30}–10^{30} **Behavioral evolution**
Chair Ingo Kurth (Jena/DE)

08^{30} **Mice, chimpanzees and the molecular basis of speech**
16 Wolfgang Enard (Leipzig/DE)

09^{00} **Behavioral genetics: from primates to humans**
17 Klaus-Peter Lesch (Würzburg/DE)

09^{30} **Genetic methods in studies of primate sexual selection**
18 Antje Engelhardt (Göttingen/DE)

10^{00} **Population structure reveals male philopatry in Guinea baboons**
19 **(Papio papio) in the Niokolo-Koba National Park, Senegal**
Gisela Fickenscher (Göttingen/DE)

10^{30}–11^{00} Coffee break & Visit of industrial exhibition

11^{00}–12^{30} **Sequence evolution**
Chair Aria Baniahmad (Jena/DE)

11^{00} **Alternative splicing of RABL2 paralogs in human and chimpanzee**
20 Matthias Platzer (Jena/DE)
Klaus Huse (Jena/DE)

11^{30} **The role of RNA in gen(om)e evolution**
21 Jürgen Brosius (Münster/DE)

12^{00} **Demographic inferences from genome-wide SNP data: estimating**
22 **population size changes and time of admixture events**
Mark Stoneking (Leipzig/DE)

12^{30}–12^{50} **Identification of transcribed human endogenous retroviruses in health**
23 **and disease, and potential application to repetitive elements in non-human species**
Jens Mayer (Homburg/DE)

12^{50}–13^{20} **Summary note**

13^{20} Snack

Exhibitors, sponsors and media partners

We are grateful to the following institutions and companies for their generous support:

Exhibitors
Analytik Jena AG (Jena/DE)
Bioline GmbH (Luckenwalde/DE)
Beckman Coulter GmbH (Krefeld/DE)
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Sponsor for congress bags, pens, notepaper, name badges and lanyards
New Carl Zeiss MicroImaging GmbH (Jena/DE)

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BMJ Group (London/GB)
Journal of Medical Genetics

Lippincott Williams & Wilkins (London/GB)
Psychiatric Genetics
Pharmacogenetics and genomics

Nature Publishing Group (New York, NY/US)
Journal of Human Genetics
European Journal of Human Genetics

Oxford University Press (Cary, NC/US)
Mutagenesis

Polish Academy of Sciences (Poznan/PL)
Journal of Applied Genetics

S. Karger AG (Basel/CH)
Cytogenetic and Genome Research
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Springer (Dordrecht/NL)
Genetica

Springer (London/GB)
Cell and Tissue Research

Springer (Tokyo/JP)
International Journal of Clinical Oncology

Wiley-Blackwell (Oxford/GB)
Clinical Genetics

General information

Venue
Friedrich-Schiller-University Jena • Rosensäle, Fürstengraben 27 • 07743 Jena/Germany

Date
16–18 September 2010

Homepage
For latest information please visit www.conventus.de/genetics2010.

Registration
Please register at the registration desk onsite.

Registration Fees

GfG member*	0 EUR
Non GfG member	260 EUR
Student**, GfG member*	0 EUR
Student**, non GfG member	130 EUR
Dayticket non GfG members	130 EUR

* Please register as well.
** Please provide confirmation.

Congress registration fee includes:
For members, non members and students
Participation to all scientific sessions, admission to the exhibition, all congress documentation and coffee breaks.

For non GfG members
You have the possibility to become a member of the GfG. Please use the form on page 30.

Confirmation of payment
The confirmation about your registration is also valid as invoice for submission at the tax and revenue office. All fees are due after receipt of your invoice/confirmation form. Transfer payments must include the name of the participant and the invoice number otherwise they will not be accepted. Payments are also accepted by all major credit cards.

Catering
Catering will be provided during the official breaks within the industrial exhibition premises.

Check-in
Please find the check-in in the first floor.

Cloakroom
Please find the cloak room in the lecture hall "Großer Rosensaal".

Abstracts
The abstracts of the Annual Conference are published at the end of this programme brochure.

Credit cards
Major credit cards (Mastercard, Visa and American Express) are widely used in almost all the hotels, restaurants, shopping malls and every kind of stores.

Industrial exhibition
As part of the conference, an industrial exhibition will take place on the premises of the conference venue.

Insurance
The organiser assumes no responsibility for accidents or damage to the private property of participants. Please make your own arrangements for health insurance and any other necessary insurances.

Language
The official conference language is English.

Media check-in
Please find the Media check-in in the lecture hall "Großer Rosensaal".

Name badges
At the registration time, each registered participant receives a name badge. The badge must be clearly displayed in order to access the congress area during all scientific and social events.

Opening hours	Thursday	Friday	Saturday
Check-in/media check-in	12^{00}–19^{00}	08^{00}–18^{00}	08^{00}–14^{00}
Industrial exhibition	12^{00}–19^{00}	08^{00}–18^{00}	08^{00}–14^{00}

Restrooms
Please follow the signage or ask at the check-in.

Information to oral lectures
Speakers are asked to submit their lectures at the Media check-in (at least two hours before your lecture). Please follow the signage on site! To ensure the smooth running of all lectures, the speakers are asked to keep the allocated speaking time.

Only a few years after the political revolution in the Eastern part of Germany took place, Jena has emerged as a city of economic, scientific, cultural and social prestige.

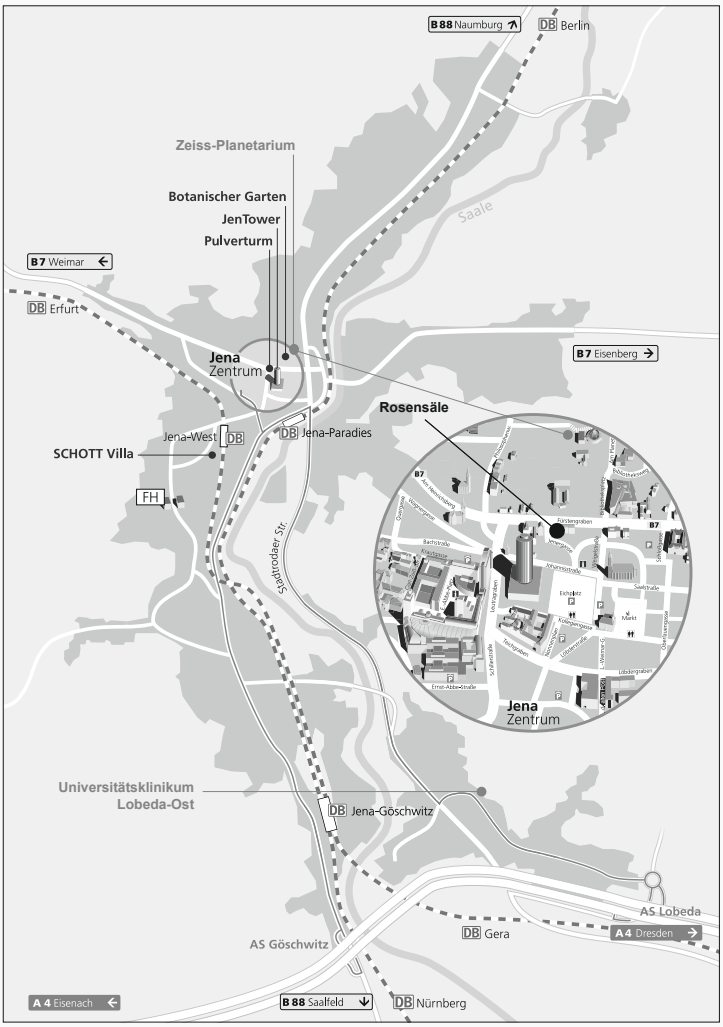
It is home to world-known institutions like the Fraunhofer Institute and three Max-Planck-Institutes. It looks back on a impressive history in the field of optical works and research strongly associated with names like Carl Zeiss and Ernst Abbe and it is also famous for its glass manufactory founded by Otto Schott.

Nearly 25000 students decisively contribute to Jena's image as Thuringia's intellectual center, which is mainly based on philosophers of international prominence like Schiller, Goethe and Hegel. The intellectual development of Jena's University was signaled with its foundation in the Collegium Jenense in 1548, when the formerly Dominican monastery became the site of training of protestant clergy and teachers.

This institution by then known as "Hohe Schule" of Jena under the sovereign of Johann Friedrich I, did not receive imperial privilege as university until 1558 and was later named after its member Friedrich Schiller in the 1930s. Apart from its tradition as center of philosophy and science, Jena is also considered a place where to enjoy a coffee in an enchanting atmosphere while reading Schiller's Wallenstein.

Jena's Wagnergasse is famous for its variety of homely cafés inviting for a little rest after a broad sightseeing tour including crucial sites such as Schiller's Garden House, the medieval city wall with the Tower of John or the city church.

City map of Jena



Discover Jena on your own! The tourist information will help you to find an individual programme for your stay.

Jena Tourist-Information
Markt 16
07743 Jena (DE)
Phone +49 (0)3641 49 80 50
www.jena.de

We will also provide city maps of Jena at the check-in desk.

Get together

Welcome to the Annual Conference of the German Genetics Society 2010! Recall the first day and socialise over beverages and snacks in the Botanical Garden of Jena.

Date	Thursday, 16 September 2010
Hour	18^{45}
Venue	Botanical Garden Jena Fürstengraben 26 07743 Jena (DE)
Registration	required
Fee	included

© photo: Thomas Bopp

1: Sex and molecular evolution

Charlesworth B.
Institute of Evolutionary Biology, School of Biological Sciences, University of Edinburgh, Great Britain

The evolutionary consequences and advantages of sexual reproduction and genetic recombination are classic problems of evolutionary biology. Recent work on variation and evolution at the DNA sequence level has shown that regions of the genome with low levels of genetic recombination exhibit reduced genetic diversity, and a reduced efficacy of natural selection. These observations are consistent with the results of theoretical models of the effects of selection on variation and evolution at genetically linked sites. These models also imply that selection may have significant effects on linked sites even in freely recombining regions of the genome. The consequences of a complete lack of genetic recombination are discussed in relation to the evolution of the Y chromosome, showing that an effectively asexual mode of reproduction can lead to a large decline in fitness over evolutionary time.

2: An ancient evolutionary origin of genes associated with human genetic diseases and cancer

Domazet-Lošo T. [1, 2], Tautz D. [1]
[1] Max-Planck Institut für Evolutionsbiologie, Plön, Germany
[2] Laboratory of Evolutionary Genetics, Division of Molecular Biology, Ruđer Bošković Institute, Zagreb, Croatia

Several thousand genes in the human genome have been linked to a heritable genetic disease. The majority of these appear to be non-essential genes (i.e. are not embryonically lethal when inactivated) and one could therefore speculate that they are late additions in the evolutionary lineage towards humans. Using a phylostratigraphic approach (1), we have studied the evolutionary emergence of such genes and find that human genetic disease genes have a long evolutionary history. They are significantly over-represented among the genes that have emerged during the early evolution of the metazoa (2). Conversely, genes specific to the mammalian lineage are highly underrepresented among them. Hence, genes involved in genetic diseases are not simply a random subset of all genes in the genome, but are highly biased towards ancient genes.
Similar patterns can also be seen for genes involved in cancer (3). Cancer should be tightly connected to multicellular life since it can be viewed as a malfunction of interaction between cells in a multicellular organism. We found that there are indeed two strong peaks of emergence of cancer related genes, one at the time of the origin of the first cell and the other around the time of the evolution of the multicellular metazoan organisms. These peaks correlate with two major classes of cancer genes, the "caretakers", which are involved in general functions that support genome stability and the "gatekeepers", which are involved in cellular signaling and growth processes. Interestingly, this phylogenetic succession mirrors the ontogenetic succession of tumor progression, where mutations in caretakers are thought to precede mutations in gatekeepers.
1. Domazet-Lošo T, Brajković J, Tautz D: A phylostratigraphy approach to uncover the genomic history of major adaptations in metazoan lineages. Trends Genet 2007, 23:533–539.
2. Domazet-Lošo T, Tautz D: An ancient evolutionary origin of genes associated with human genetic diseases. Mol Biol Evol 2008, 25:2699-2707.
3. Domazet-Lošo T, Tautz D: Phylostratigraphic tracking of cancer genes suggests a link to the emergence of multicellularity in metazoa. BMC Biol. 2010, 8:66.

3: The neandertal genome: insights into human origins

Kelso J.
MPI Evolutionary Anthropology, Department of Evolutionary Genetics, Leipzig, Germany

The Neandertals are the closest evolutionary relatives of present-day humans. Comparison of the Neandertal genome to that of present-day humans therefore offers a unique opportunity to identify genetic changes specific to anatomically fully modern humans. We have produced a draft sequence of the Neandertal genome composed of over 4 billion nucleotides from three individuals. Through comparisons of the Neandertal genome to the genomes of five present-day humans from different parts of the world we identify a number of genomic regions that may have been affected by positive selection in ancestral modern humans. These include genes involved in metabolism, cognitive and skeletal development. We also present a catalog of genomic changes that have become fixed, or have risen to high frequency, in modern humans during the last few hundred thousand years. Finally, we analyze the relatedness of Neandertals to present-day humans in different parts of the world. We show that Neandertals shared more genetic variants with present-day humans in Eurasia than with present-day humans in sub-Saharan Africa, suggesting that gene flow from Neandertals into the ancestors of non-Africans occurred before the divergence of Eurasian groups from each other.

4: Recent positive selection of a human *AR/EDA2R* haplotype and its relationship to male pattern baldness

Hillmer A.M.[1,2], Freudenberg J.[3], Myles S.[4], Herms S.[1], Tang K.[5], Hughes D.A.[5], Brockschmidt F. F.[1], Ruan Y.[2], Stoneking M.[5], Nöthen M.M.[1, 6]
[1]Department of Genomics, University of Bonn, Bonn, Germany
*[2]Genome Institute of Singapore, A*STAR, Singapore*
[3]Center for Genomics and Human Genetics, Feinstein Institute for Medical Research, Manhasset, NY, USA
[4]Institute for Genomic Diversity, Cornell University, Ithaca, NY, USA
[5]Department of Evolutionary Genetics, Max Planck Institute for Evolutionary Anthropology, Leipzig, Germany
[6]Institute of Human Genetics, University of Bonn, Bonn, Germany

Genetic variants at the human androgen receptor *(AR)*/ectodysplasin A2 receptor *(EDA2R)* gene locus on chromosome X are associated with male pattern baldness (androgenetic alopecia, AGA) in Europeans. Previous observations of long-range linkage disequilibrium at the *AR/EDA2R* locus are consistent with the hypothesis of recent positive selection. We further investigated this signature and its relationship to the AGA risk haplotype. The haplotype homozygosity suggested that the AGA risk haplotype was driven to high frequency by positive selection in Europeans although a low meiotic recombination rate contributed to the high haplotype homozygosity. Further, we found high levels of population differentiation as measured by F_{ST} and a series of fixed derived alleles along an extended region centromeric to *AR* in the Asian HapMap sample. The predominant AGA risk haplotype also carries the putatively functional variant 57K in *EDA2R*. It is therefore probable that the AGA risk haplotype rose to high frequency in combination with this *EDA2R* variant, possibly by hitchhiking on a positively selected 57K haplotype. Although 57K is a good candidate as a causative variant for AGA, this hypothesis seems less likely as this allele is fixed in East Asia whereby the prevalence of AGA is lower in this population compared to Europeans.

5: Primate-specific alternative splice site in *CRYGA* forms a truncated γA-crystallin

Graw J.[1], Schmidt W.[2]
[1]Helmholtz Zentrum München, Institut für Entwicklungsgenetik, Neuherberg, Germany
[2]Zentrum für Augenheilkunde, University Giessen, Giessen, Germany

Mutation analysis for hereditary cataracts in human families is actually dependent mainly on genomic DNA from blood and the detection

of splice site mutations is rather limited. Therefore, we tested the possibility to use material from cataract surgery for mutation analysis at first in *CRYG* genes having a high probability for cataract-causing mutations.
Lens material from surgeries was collected and immediately frozen to -80°C. mRNA was isolated and cDNA synthesized according to standard procedures; genomic DNA was prepared from blood of the probands. PCR-amplified DNA fragments were characterized by sequencing.Analysis of lens-derived cDNA from three unrelated boys and one girl suffering from congenital cataracts gave good results for cDNA quality and DNA sequencing. In particular, we observed for all expressed *CRYG* genes (*CRYGA*->*CRYGD*, encoding γ-crystallins) the expected sizes and the wild-type sequence. Additionally, all *CRYGA*-amplification products of the four children showed an additional band in the agarose gels representing a splice site variation. At the 3'-end of the 2nd exon, 71 bp are lost creating an altered open-reading frame, and after 12 new amino acids a premature stop codon follows. Species comparison revealed that this alternative splicing is due to a primate-specific C->A exchange in exon 2 (cDNA position 180). The experimental finding is supported by two previous entries of EST clones in public databases demonstrating the same feature.
This finding demonstrates an additional case of an extinction of a γ-crystallin encoding gene in primates, since the *CRYGE* and *CRYGF* genes are well known as pseudogenes in humans, but required for lens transparency in rodents.

6: Chromosomal evolution and heterochromatin in primate genomes

Yurov Y.B., Vorsanova S.G., Iourov I.Y.
Mental Health Research Center, Russian Academy of Medical Sciences, Moscow; Institute of Pediatrics and Children Surgery, Rosmedtechologii, Moscow, Russia

Significant portions of the primate genome are heterochromatic and made up largely of non-coding repetitious (satellite) DNA sequences. Heterochromatin is a very rapidly evolving segment of the genome demonstrating expressive variations on molecular and chromosomal levels between and within species. Human constitutive heterochromatin composed of alpha and "classical" satellite DNA, cross-hybridizing to chromosomes of all primates, including orangutan, gorilla, and chimpanzee. Satellite DNAs are not distributed uniformly between species due to genomic reposition and sequence divergence during evolution and are characterized by various species-specific differences (suprachnomosomal families, relative chromosome-specificity, co-localization with ribosomal genes, and the presence of transcribed genes within the heterochromatin). The persisting of heterochromatin throughout primate's evolution is in agreement with it key role in diverse biological processes, including centromere function, chromosome replication, gene silencing and nuclear organization. Recent findings illuminate molecular genetic and epigenetic mechanisms underlying these processes and highlight the importance of heterochromatin and repeated DNAs in maintaining the integrity of chromosomes and genomes. The repetitiveness of heterochromatin provides the unsolved technical problems for the complete sequencing and analyzing of a primate genome. However, analysis of available sequences of great ape-specific and human-specific alpha satellite DNA have identified the genome-wide waves of expansions of new satellite variants during the human evolution (Shepelev et al., 2009). Each wave of expansions covered many chromosomes and corresponds to a new primate taxon. The possibility to reveal and date extinct ancestors will provide a unique tool for the reconstruction of primate phylogeny by the analysis of satellite DNA layers within the heterochromatin.

7: Inter- and intraspecific Y chromosome variation in human and great apes

Schempp W.
University Freiburg, Institute of Human Genetics Freiburg, Germany

Comparative FISH using probes from human Y-specific ampliconic fertility genes *DAZ* (deleted in azoospermia) and *CDY* (chromodomain

protein Y) disclosed a so far never described variation of a species' Y chromosome in the common chimpanzee (*Pan troglodytes*). In marked contrast, no variation was seen among the bonobo (*P. paniscus*) Y chromosomes. Although chimpanzee and bonobo both show polyandrous mating behaviour with potentially high levels of sperm competition, the contrasting patterns of Y-chromosomal variation in these closely related species might have an explanation in the context of their markedly different social and mating behaviour. In chimpanzees, multiple males copulate with a receptive female during a short period of visible anogenital swelling, and this may place significant selection on fertility genes. In bonobos, however, female mate choice may make sperm competition redundant, since ovulation in this species is concealed by the prolonged anogenital swelling, and because female bonobos can occupy high-ranking positions in the group and are thus able to determine mate choice more freely. It is interesting that FISH studies in gorillas and orangutans similarly failed to detect intra-species variation in spermatogenesis genes. It is apparent that monoandrous mating behaviour in gorillas, as well as the preference of female mate choice in orangutans, similarly diminishes sperm competition thus mirroring the situation in bonobo.

8: An ancient addition to the ancestral part of the mammalian X chromosome was already enriched for brain specific genes

Hameister H.
genetikum, Neu-Ulm, Germany

Previous analyses of human disease-related genes revealed an enrichment of genes responsible for sex and reproduction and for brain functions on the X chromosome. Here we examine the evolutionary history of this X chromosomal enrichment and investigate the potential role of adaptive changes in its origin. Such analysis is possible since the mammalian X chromosome can be traced back to three ancestral chromosomal segments ("building blocks") in the chicken genome. The modern mammalian X chromosome arose from two consecutive translocations to the most ancestral part of the chromosome. The first segment was translocated before separation of the monotremes more than 148 million years ago (MYA), while the second segment was translocated after separation of the marsupials less than 130 MYA. Using microarrays, we surveyed patterns of tissue-specific gene expression (testis, brain, liver, and heart) in the chicken. This allowed us to analyze the chromosomal distribution of tissue-specific genes and examine their annotated functions (GO terms). We found that the chromosomal segment that translocated to the X chromosome more than 148 MYA was already enriched for genes expressed specifically in the brain. Now just these chromosomal segments show a high concentration of X-linked mental retardation genes on the human X chromosome. The enrichment of genes involved in sex and reproduction appears to be the result of adaptive changes, whereas the brain functions are highly conserved. A comparative transcriptome analysis between chicken and mouse indicates that the expression pattern of brain, heart, and liver genes is well conserved between birds and mammals. Remarkably the highest transcriptome conservation was found for brain genes. In contrast, testis genes show much less conservation. Different scenarios for the evolution of sex chromosomes in birds and mammals are discussed.

9: Comparative cytogenetics technologies

Liehr T., Mrasek K., Kosyakova N., Weise A.
Institute of Human Genetics, Jena University Hospital, Jena, Germany

The comparison of genomes derived from species within one, or several more closely related clades is a basic tool of evolution biology. This comparison can be performed on different levels of resolution. The closest look is for sure possible by DNA-sequencing, leading to an enormous amount of data including information on repetitive elements where still is not clear if the latter are some kind of "junk-

DNA* or highly important for gene-regulation and expression. A more informative, and maybe also for the specification more important approach, is to study genomes by comparing their karyotypes. This is the field of comparative cytogenetics. Progress was achieved here, as in cytogenetics in general, always coupled closely to technical achievements in chromosome preparation, staining and new possibilities for chromosome (breakpoint) characterization. Here we give an overview on these steps of technical developments, including pure chromosome staining, cytogenetic routine banding, molecular cytogenetics (M-FISH, SKY, FISH-banding), and array-based approaches. Examples are given, where these approaches were and are still used and what are their advantages and shortcomings. Supported in parts by DLR/BMBF RUS 09/008.

10: Evolutionary conserved breakpoints and fragile sites

Mrasek K., Liehr T., Weise A.
Institute of Human Genetics, Jena University Hospital, Jena, Germany

Fragile sites (FS) are considered as normal structural features of mammalian chromosomes. It is hypothesized that FS and a major part of evolutionary appearing and conserved breakpoints are identical on cytogenetic and maybe even on molecular level. However, this assumption was based up to recently almost exclusively on GTG banding studies. We checked, on DNA level, the hypothesis that fragile sites maybe conserved as evolutionary stable breakpoints in primates, i.e. in *Gorilla gorilla, Pongo pygmaeus, Pan troglotydes* and *Hylobates lar.* Here fore we first exactly mapped 21 Aphidicolin (Aph) common FS (cFS) by means of by Fluorescence *in situ* Hybridisiation (FISH) using bacterial artificial chromosomes (BACs). Thus, together with the cFS described in the literature now 42 cFS are mapped on a molecular base. In a second step we compared known evolutionary conserved breakpoints, i.e. 9 macro- and 27 microrearrangements with the molecular proved localization of corresponding cFS. Thus, in our study for the first time a molecular cytogenetic co-localization of approximately 75% of human cFS and evolutionary breakpoints could be proven.
Supported in parts by Evangelische Studienwerk e.V. Villigst, IZKF Jena (Promotionsstipendium to CS and KW, and Start-up S16 to AW), IZKF together with the TMWFK (TP 3.7 and B307-04004), Ernst-Abbe-Stiftung, DFG (LI 820/15-1) and Deutsche Fanconi-Anämie-Hilfe e.V.

11: Outgroup cytogenetics

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Understanding of modes and rates of karyotype evolution in primates is impossible without comparing it with karyotypes of species from other mammalian and vertebrate taxa. The use of outgroup helps to understand if particular traits are ancestral or derived and thus to reconstruct the changes occurred during the evolution of particular species or any other taxon. Chromosomal characters are quite convenient to use for phylogenetic reconstructions due to the rare homoplasic events and polymorphisms. Particularly the chromosomal characters are important in cases when all the molecular and morphological data give obscure results. Here we show that the study of outgroup species helps to recognize the ancestral chromosomal traits of primates and highlights the extremely low rates of karyotype evolution within the group. Although mammals include clades with both very rapid and rather slow karyotype evolutionary rates, but generally they are characterized by an increased rate of karyotype evolution in comparison to reptiles, birds, amphibians and fishes. Here we discuss the reconstructed primate evolutionary tree with all large karyotypic changes occurred in different branches. Although the reasons for the shifts of evolutionary rates from slow to enhanced remain unclear, we discuss what might possibly influence these changes.

12: Aneuploidization of the brain is evolutionary conserved in vertebrates, while protective "antianeuploidization" is more effective in hominids

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The genetic basis of the human brain evolution is incompletely understood. One striking feature of the brain revealed during the last decade is referred to the presence of mosaic aneuploidy in the developing and adult mammalian brain. It noteworthy that aneuploidy was also observed in lower vertebrates. Thus, molecular cytogenetic analyses have shown 84% of cells of the teleost fish brain to be aneuploid (Zupanc, 2008). The postnatal murine brain possesses 15-20% of aneuploid cells (Westra et al., 2008). The adult human brain has approximately 10% of aneuploid cells (Yurov et al., 2005; Iourov et al., 2006, 2009). Therefore, the rate of aneuploidy in the postnatal brain significantly decreases throughout vertebrate evolution correlating with increase of neuronal complexity. Mosaic aneuploidy of the adult mammalian brain is the result of progressive aneuploidization of the developing brain (Yurov et al., 2007). Interestingly, about 30% of aneuploid cells are observed as in the developing murine brain (Rehen et al., 2001) as in the developing human brain (Yurov et al., 2007). Recently, a hypothesis suggesting the existence of a protective process arbitrarily called "antianeuploidization", which is responsible for decrease of aneuploidy rates after birth via apoptotic cell death or mitotic catastrophe cascade, was proposed (Yurov et al., 2007; Iourov et al., 2008). Here, taking into account that aneuploidy rates are almost equal in the developing brain, but are different in the adult brain, we have proposed an evolutionary hypothesis of brain aneuploidization. It is assumed, that developmental chromosome instability mediated by aneuploidization is an integral component of the early brain development. Further, as neuronal complexity increases, the brain should be more and more significantly negatively affected if the overwhelming majority of cells are aneuploid. Therefore, to grant higher functioning of the central nervous system, more effective protective "antianeuploidization" mechanisms has to be generated. The latter explains why the amount of aneuploid cells is less in hominids than in other vertebrates. Together, one can conclude that somatic genome variations mediated by aneuploidization, being evolutionary conserved in the vertebrate brain, are differently processed throughout ontogeny in different species. It is to note, that these aspects of genomic evolution have not been ever addressed. Finally, to support out hypothesis, molecular neurocytogenetic studies of primates other than humans are strongly required, because of possessing potential to highlight shared pathways of brain aneuploidization and "antianeuploidization" and their evolution in vertebrates.

13: A population genomic approach to map recent positive selection

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In several model organisms (whose genome has been sequenced) scans of DNA sequence variation have been carried out to elucidate the recent demographic and adaptive histories of these species. Using selective sweeps, it is possible to identify adaptive events in the genome. I will briefly describe these mapping methods and discuss some examples, in which the target of selection could be determined down to the gene level. For instance, in the case of the tandemly duplicated *polyhomeotic* (*ph*) genes of Drosophila a sweep was localized to a large intron of one of the duplicates. Apart from this intron, the *ph* copies are very similar at the sequence level, but have begun to functionally diverge. Our results suggest that strong positive selection acting on the transciption factor binding sites of the large intron of one of the *ph* genes drives neofunctionalization despite the presence of gene conversion (*i.e.* concerted evolution between the duplicates).

14: Evolution of novel gene functions by molecular domestication of transposable elements

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Transposable elements have been commonly viewed as molecular parasites producing mainly neutral or deleterious effects in host genomes through their ability to move. However, during the past two decades, major interest has been focusing on the positive contribution of these elements in the evolution of gene regulation and in the creation of diverse structural host genes. Indeed, DNA transposons carry an attractive and elaborate enzymatic machinery as well as DNA components that have been co-opted in several cases by the host genome via an evolutionary process referred to as molecular domestication. One possible explanation for the recurrent use of transposase domains for the assembly of new genes is the ability of these enzymes to bind DNA sequences dispersed at many chromosomal sites in genomes. To test this model, we study SETMAR, a gene that originated in the anthropoid lineage from the fusion of a histone methyltransferase SET domain to a *mariner* transposase, and whose function remains enigmatic. The core DNA-binding specificity of SETMAR for a is characterized by a 19-bp motif located in *Hsmar1*/MADE1 transposons, which are present in high copy number in the human genome. Based on this information and on comparative sequence analysis across 11 anthropoid primates, we detected a significant excess of conserved SETMAR binding sites, suggesting the action of purifying selection to maintain a network of such sites. Furthermore, a subset of *Hsmar1*-derived miniature inverted-repeat transposable elements (MITEs) comprises the palindrome regions of the hsa-mir-548 family of microRNA (miRNA) genes in anthropoid primates. These miRNA genes may also serve as specific sites for SETMAR binding, which could modify the expression of the regulatory miRNAs. To provide evidence for the sequence-specific binding of SETMAR to a subset of the conserved putative binding sites and microRNA genes, ChIP experiments were performed using hemagglutinin-tagged SETMAR protein transiently expressed in human cells. Our results show that SETMAR binds a subset of these conserved sites and miRNA genes *in vivo*, suggesting that this protein functions as a sequence-specific DNA-binding factor in a primate-specific gene regulatory network.

15: Mechanics of chromosome and karyotype evolution – a magnifying Lens for palaeogenomics

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Nuclear genomes are contained within chromosomes representing genetic linkage groups. Number, size and shape of chromosomes which constitute the karyotype of an organism may vary considerably between groups of eukaryotes. Comparative genetics, genomics and cytogenetics provide tools to trace the evolutionary history of extant genomes. The genetic terminology (chromosome fusion, nonreciprocal translocation etc.) applied to define genomic differences between organisms does not always precisely reflect mechanics of chromosome rearrangements underlying the evolutionary karyotype alterations. Our aim is to bridge genomic and cytogenetic insights interpreting evolutionary genome alterations in a parsimonious way and in accordance with known cytogenetic constraints. Most findings of comparative (palaeo) genomics and comparative chromosome painting can be explained based on chromosome rearrangements which are inducible by genotoxin treatment and can be observed microscopically in post-treatment mitoses and after passing meiosis.

16: Mice, chimpanzees and the molecular basis of speech

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Identifying the genetic changes responsible for the phenotypic differences between humans and their close primate relatives is important from an evolutionary, medical and cultural perspective. The primary challenge facing researchers today, after analyzing the genomic data, is experimentally testing hypotheses concerning the genetic basis for human-specific traits. One of the more prominent hypotheses of this kind states that two amino acid changes in the transcription factor FOXP2 have been fixed in humans by positive selection due to some effect on speech and language. We have introduced these substitutions into the endogenous Foxp2 gene of mice. Although these mice are generally healthy, they have qualitatively different ultrasonic vocalizations, decreased exploratory behavior and decreased dopamine concentrations in the brain suggesting that the humanized Foxp2 allele affects basal ganglia. In the striatum, a part of the basal ganglia affected in humans with a speech deficit due to a non-functional FOXP2 allele, we find that medium spiny neurons have increased dendrite lengths and increased synaptic plasticity. Since mice carrying one non-functional Foxp2 allele show opposite effects, this suggests that alterations in cortico-basal ganglia circuits might have been important for the evolution of speech and language in humans.

17: Behavioural genetics: from primates to humans

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Adverse childhood experiences are associated with increased risk for psychiatric diseases later in life, especially anxiety disorders and depression. Several studies indicate that whether a individual develops disorders of emotion regulation following early life stress is influenced by variation of the serotonin transporter gene (*5-HTT*). Multimodal fMRI in humans suggested that life stress interacts with the *5-HTT* genotype to influence amygdala and hippocampal resting activation. There are also compelling data from nonhuman primates. In rhesus monkeys (Macaca mulatta), maternal separation during the first months of life results in deficient social adaptation and peer interaction. These deficiencies are related to brain serotonin system function, based on testing for interactions between early life stress and *5-HTT*: in addition to main effects of *5-HTT* genotype and early stress to variation in serotonergic function in later life, *5-HTT* also interacts with deleterious early rearing experience to influence attentional and emotional resources, stress reactivity, and alcohol preference and dependence. However, the molecular mechanisms by which stress increases disease risk in adulthood is not known, but may include epigenetic programming of gene expression. Various gene-by-environment interaction (GxE) paradigms in the mouse allow to study the molecular mechanisms underlying epigenetic programming by early adverse environment in an animal model amenable to genetic manipulation. Using these GxE paradigms it was shown that prenatal stress or dominant/subordinate social interaction on anxiety-related behavior is modulated by inactivation of *5-HTT*. These findings suggest that the molecular mechanisms involved in these GxE models are relevant to the etiology of disease in humans.

18: Genetic methods in studies of primate sexual selection

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Since the development of methods suitable for the analysis of very low and/or degraded amounts of DNA, molecular techniques have

become increasingly attractive to primatologists, particularly those studying animals under natural conditions. I will present current applications of genetic methods to topics of primate sexual selection such as male reproductive skew, hybridization, sexual signals and parental investment using studies on wild macaques as examples.

19: Population structure reveals male philopatry in Guinea baboons (*Papio papio*) in the Niokolo-Koba National Park, Senegal

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Dispersal is a life history trait, which has major implications for both the dynamics and the genetic makeup of populations. Understanding its specific pattern can contribute to our knowledge about a species` evolution and ecology. Baboons have served as an important model for investigating the evolution of primate social systems. However, in contrast to most of their congenerics, Guinea baboons (*Papio papio*) have barely been studied yet, and our knowledge about their social system and behavioural ecology is still very limited. Based on anecdotal observations that male Guinea baboons show a peculiar high degree of tolerance towards each other which could be due to kinship, we hypothesized that this species exhibits male philopatry and female dispersal. Using non-invasive samples we genotyped more than 150 Guinea baboon individuals from five localities in the Niokolo-Koba National Park, Senegal, at 14 autosomal microsatellite loci. Despite the fact that there is no obvious geographic barrier to gene flow, the population shows significant structuring suggesting isolation by distance. Furthermore, there is evidence for higher structuring in males than in females, as expected if males are the more philopatric sex and females disperse. Relatedness between male and female dyads, respectively, differs significantly between the sexes among troops, with males being on average less related among troops than females, also supporting the hypothesis of male philopatry. Comparison of relatedness within troops, however, did not yield any significant results. This could be a result of the large size of the troops and low reproductive skew. This study is the first to investigate population genetics in Guinea baboons and gain evidence for male philopatry in this species. While the causes of this exceptional pattern remain unclear, this is a first step in understanding the so far largely unknown social organization of Guinea baboons. It reinforces the view that the social system of this species deviates significantly from other baboon taxa. To test hypotheses about possible ultimate causes for this system, broader ecological, behavioural and genetic research is needed to investigate this pattern in more detail.

20: Alternative splicing of RABL2 paralogs in human and chimpanzee

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The paralogs *RABL2A* and *RABL2B* are young gene duplicates with a short evolutionary history and high sequence similarity (>98%). Duplication of the ancestral gene on chromosome 22 took place during hominid evolution only recently. Orangutan (*Pongo pygmaeus)* and most likely gorilla (Gorilla gorilla) still represents the ancestral situation with a single *RABL2* gene in their haploid genome (as for mouse, rhesus monkey and African green monkey). Thus, the gene duplication must have occurred after divergence of gorilla but before the split of human (*Homo sapiens*) and chimpanzee (*Pan troglodytes*). Compared to the common ancestor of chimpanzee and humans, chimpanzee retained the situation after the duplication event with two paralogs in the subtelomeric region of chromosomes 2b and 22. In human lineage, the fusion of chromosomes 2a and 2b formed chromosome 2, finally placing *RABL2A* to its current peri-

centromeric position at 2q13 in the human genome. Relative expression of *RABL2A* and *RABL2B* is equal in chimpanzee but different in human. Here, *RABL2B*'s expression exceeds that of *RABL2A* 3–4fold.
All primates show alternative *RABL2* splicing by forming two major isoforms with respect to exons 5 and 6 in lymphoblastoid cell lines. These exons may be either mutually included or excluded. In all primates carrying single-copy genes the short isoform exhibits similar fraction of about 20 % while in chimpanzee and human a higher fraction of about 40 % is observed. Analysis of paralog-specific splicing in human and chimpanzee revealed that the short isoform is formed by just one paralog, but surprisingly not in the respective orthologous genes (*RABL2A* in human and *RABL2B* in chimpanzee). The corresponding other paralog is spliced constitutively to the long isoform. Although the reasons for the reversed imaged situation remain to be elucidated, our data provide evidence for a subfunctionalization on the level of splicing in accordance with the duplication/degeneration/complementation model for the retention of paralogs. Remarkably, core splice sites (at least ±5 nt) are identical among all human and chimpanzee *RABL2* genes.

21: The role of RNA in gen(om)e evolution

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Many multicellular organisms generate large amounts of superfluous DNA either by segmental duplications or, importantly, by the continuous conversion of RNA to DNA. One of the consequences is that almost every sequence in our genome, directly or indirectly, could be traced back to reverse transcription of an RNA molecule. Any cellular RNA from messenger RNA (mRNA) to non-protein coding RNA (npcRNA) to virus-related RNA can serve as template for retroposition. Most of these sequences are devoid of any function, evolve neutrally and after 100-200 million years, their origin is not discernible anymore. At any stage of decay, such extra sequences can be recruited (exapted) into a novel function, be it as regulatory region, npcRNA or as (part) of a protein coding region. This process often has a gradual component, involving additional events, such as point mutations and indels. In case of novel exon acquisition, for most part, an initial recruitment is slightly deleterious or neutral at best and often the event will not persist. Even if at one stage more or less beneficial, it could be lost again over time prior to subsequent speciations, or after speciations in certain lineages, or occasionally it could persist in all subsequent lineages. Examples will be given employing phylogenetic studies in mammals.

22: Demographic inferences from genome-wide SNP data: estimating population size changes and time of admixture events

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Genome-wide data for hundreds of thousands of SNPs can now be routinely obtained from any human population, and such data exists for thousands of individuals. However, the ascertainment bias inherent in the choice of SNPs included on genotyping platforms complicates demographic inferences from such data. In particular, traditional approaches based on the allele frequency spectrum are rendered useless. We have therefore been investigating an alternative approach for estimating population size changes by inferring a statistic called the SNP-based length of ancestral shared haplotypes (SPLASH). We show via simulations that the SPLASH statistic is robust to ascertainment bias and provides accurate estimates of past population size changes, and we apply the method to empirical data for an African and a European population. In addition, we have developed a novel method, based on wavelet transform analysis, to date admixture events based on

the number and size of ancestry blocks in the genomes of admixed individuals. This method outperforms existing methods and can be applied to genome-wide data (either SNPs or full sequence data) from any species.

23: Identification of transcribed human endogenous retroviruses in health and disease, and potential application to repetitive elements in non-human species

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About half of the human genome mass consists of repetitive elements, and the same is true for the genomes of many other species. It is well accepted that repetitive/mobile DNA has had—and continues to have—a profound impact on host genomes. About 8% of the human genome is comprised of human endogenous retroviruses (HERVs), remnants of ancient germ line infections by different exogenous retroviruses. HERVs contribute significantly to the human transcriptome because of intrinsic promoters and transcriptional regulators. Many HERV loci retained transcriptional activity, HERV transcripts are found in every human tissue, and HERV transcription appears tightly regulated. Some HERV families may be involved in human disease. We previously identified transcribed loci of HERV families of clinical interest by utilizing characteristic sequence differences between individual loci of a particular HERV family, that transcribe into HERV RNA and thus subsequently generated cDNA sequences. For example, we identified for the HERV-K(HML-2) family, that has been associated with germ cell tumors, 23 transcribed proviruses and distinct proviral expression patterns in GCT and other selected tissues. We identified various transcribed loci for the HERV-W family, that has been associated with Multiple Sclerosis, and our data suggest that previously reported sequences of a so-called Multiple Sclerosis-Associated Retrovirus (MSRV) actually are in vitro recombinants of endogenous HERV-W transcripts. However, despite of own and others' efforts there is no comprehensive picture of the contribution of HERVs to the human transcriptome and their potential roles in human diseases, yet. We therefore will now study in a specific and somewhat larger scale project transcription of selected HERV families in health and disease, and we will thus contribute to filling a crucial gap in ongoing initiatives for characterizing the human transcriptome. Our strategies for identifying transcribed HERV loci are, in principle, applicable to endogenous retroviruses and other repetitive elements in non-human species. Ongoing sequencing of genomes as well as transcriptomes of various species will generate significant information for further understanding the biological role of repetitive elements in those species.

Supported by grants from DFG and HOMFOR

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