



**SHORT COURSE FOR YOUNG
PARASITOLOGISTS
HAMBURG 2011**



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Dear Parasitologists,

Welcome to the 5th Short Course for Young Parasitologists being held in Hamburg!

The course is intended to bring together young Parasitologists from all disciplines and generate a forum for exchange of ideas and concepts. Your active participation is extremely important to make this course a successful one. Please present your project succinct and refer to the instructions for details. Most important, keep your presentation informal and engage your colleagues in a discussion.

We invited leading experts who will discuss their research topics with you in their comprehensive seminars. Take advantage of this opportunity and discuss your thoughts, projects and questions with them. All of them will be delighted to assist you in every possible manner.

What is not included in the program may well be an important part of the course, i.e. the dinners and pub visits at night. They'll be our reward for an intense program.

This course was accomplished with the generous help of the Deutsche Gesellschaft für Parasitologie. Without their support this course would not have been realized. Therefore, their contribution is particularly acknowledged.

We also wish to thank Prof. Dr. med. Tannich for hosting the course in the Bernhard-Nocht-Institute for Tropical Medicine.

Furthermore we would like to thank Institutional Money, Eurogentec, Nordea and ThermoFisher Scientific for their kind support and Professor Dr. Heinz Mehlhorn for the sponsorship of the Talk Prize.

Have a great stay and enjoy the course

Jude Przyborski and Katharina Dolata

P.S If you need to get hold of us urgently, here are our mobile phone numbers:

Jude: +49(0)151-40034065

Katharina: +49(0)178-6074235



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EXECUTIVE COMMITTEE OF THE DEUTSCHE GESELLSCHAFT FÜR PARASITOLOGIE

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PD Dr. rer. nat. Hartmann, Susanne, Berlin
PD Dr. habil. Przyborski, Jude, PhD, Marburg

General meeting of DGP Members:

During the next conference from 14.-17.03.2012 in Heidelberg

Homepage of the DGP: www.dgparasitologie.de

ORGANISATION OF THE SHORT COURSE FOR YOUNG PARASITOLOGISTS

PD Dr. habil. Przyborski, Jude, PhD

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Homepage of the course: www.pfalciparum.de

Contact: youngparasitologists@googlemail.com



ORAL PRESENTATIONS

The course language is English. The duration of an oral presentation is limited to 15 minutes plus 5 minutes discussion.

Our course room will be equipped with a projector and a computer. You are requested to bring your presentation on a memory stick. If possible, please try to save your presentation like this. If you have media which you must play from your own computer, please inform the chairperson of your session ahead of time. Please take your memory stick to your chairperson well in time, as the timing of the sessions is quite tight!

Presentations will, of course, be deleted from the course computer after the session.

TALK PRIZES

At the end of the course, we shall be awarding book prizes for the best 4 talks. Who wins the prizes will be decided democratically by voting. All participants get to vote for 3 talks. We'll be handing out voting forms after the last course, so please take notes during the course (you can use this book). In the case of a tie, we shall cast the deciding vote.

FOOD AND BEVERAGES

Although the hostel doesn't offer breakfast, we will still be able to have breakfast together at the hostel, as we (the organizers) will buy cereal, rolls, cheese, jam etc. If you have any special dietary requirements, we will TRY to comply with them.

During the coffee breaks coffee, tea and biscuits will be offered free of charge in the social room of the BNI.

For lunch or to have a snack you may choose from one of the many restaurants, coffee bars or bakeries near by.

Dinner will be provided only on Friday night. This doesn't mean that everyone has to do their own thing the other nights, just that we won't be able to pay for it!

SOCIAL PROGRAM

Wednesday, March 16, 2011, 9pm

Get together at a location to be announced
(Some free drinks included)

Friday, March 18, 2011, 7:30pm

Dinner at the Copper House
Davidstraße 37 (at the corner to Davidstrasse)
20359 Hamburg
(Dinner and free drinks included)



THE HOSTEL BUCH-EIN-BETT.DE

(see "A" on the map at next page)

Krantz Gastronomie und Freizeitanlagen Betriebs und Verwaltungs GmbH

Detlev-Bremer-Strasse 44, 20359 Hamburg

Phone: +49 (0)40 317 969 00

In case of urgency mobile phone: +49 (0)163 77 77 440

If you arrive at Hamburg Altona use either suburban railway line S1 direction Poppenbüttel or line S3 direction Harburg. The second stop ("Reeperbahn") is yours. Exit the station to the right and walk 500 m down the Reeperbahn street, then turn left into the Detlev-Bremer Straße.

If you arrive at Hamburg Central Station use either suburban railway line S1 direction Wedel or line S3 direction Pinneberg. Exit at the 4th stop "Reeperbahn". From here: see above.

Alternatively use subway line U3 direction Barmbek. Alight after 6 stops at "St. Pauli". Exit the station to the right and walk 250 m down the Reeperbahn street, then turn right into the Detlev-Bremer-Straße.

We will provide accommodation from 16 -19 March in shared rooms on dormitory bunks. Each room has its own bathroom with shower and WC. You should bring your own towels, soap, hairdryer (if needed) and padlock (necessary for the lockers).

COURSE VENUE: BERNHARD-NOCHT-INSITUTE

(see "arrow" on the map at next page)

Bernhard-Nocht-Strasse 74

20359 Hamburg

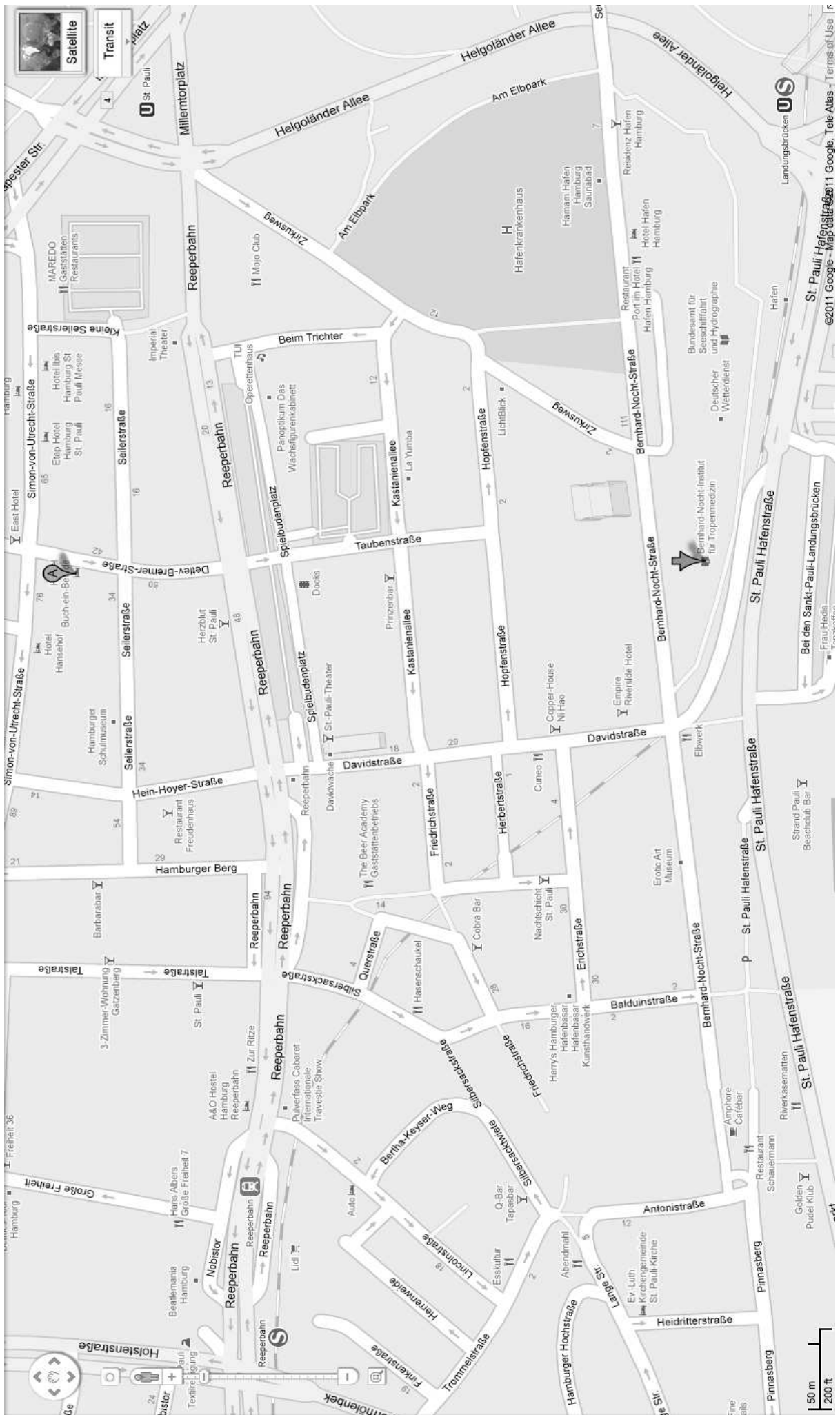
On the first day of our course we will pick you up at the hostel and we will walk together to the institute.

To go to the institute on your own way take the local trains S1, S2, S3 or the underground U3 to "Landungsbrücken".

Exit to the right following the hostel sign, go up the stairs (about 10000 steps) on your right hand side. When you get to the top, keep left and follow the path for 100 m. Then take the right hand path leading to a bridge. Turn left and go across the bridge, pass the Hotel "Hafen". You will find the BNI about 400 m further along on your left.

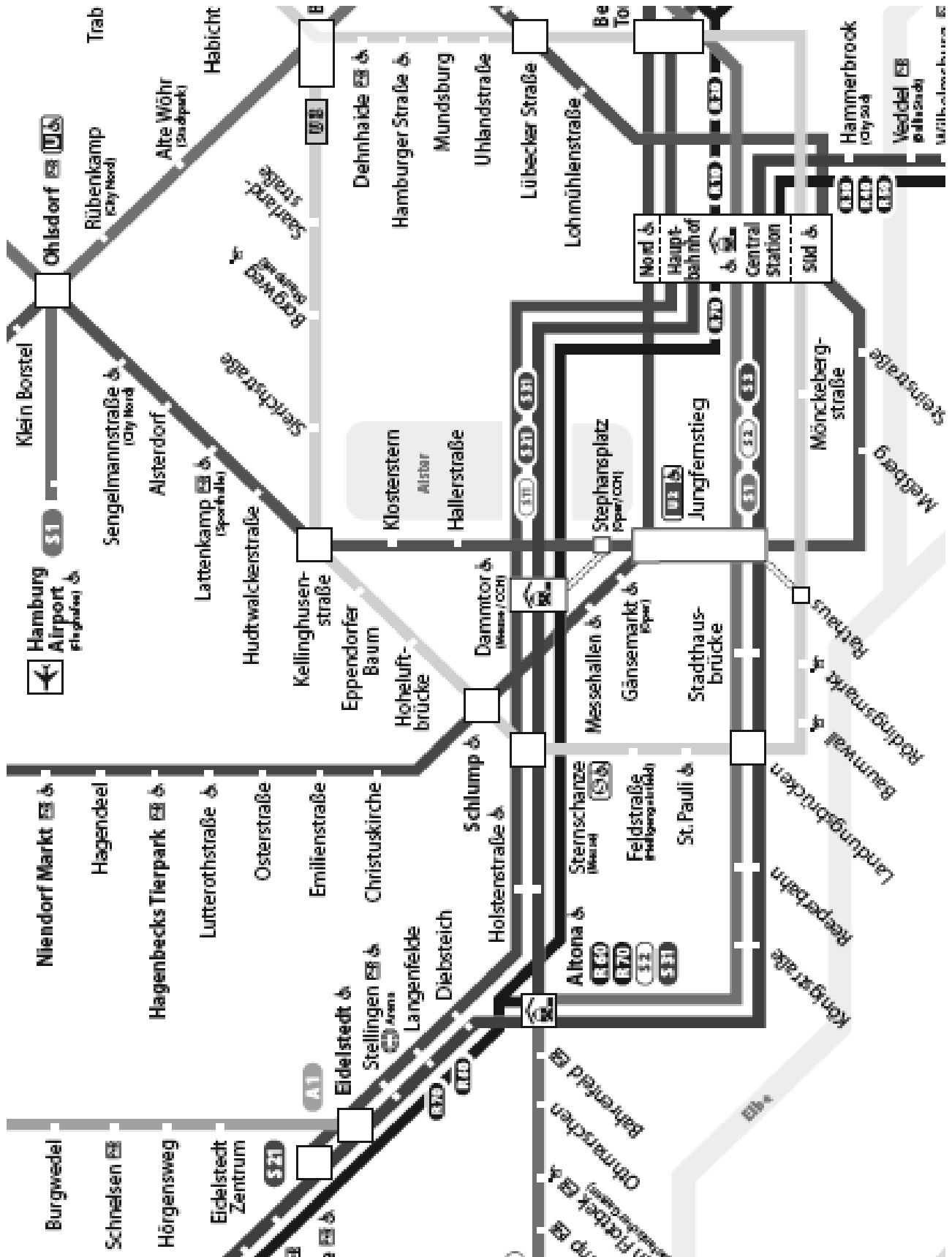


MAP



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PUBLIC TRANSPORT



COURSE SCHEDULE

	Wed 3/16	Thu 3/17	Fri 3/18	Sat 3/19
8 am		Breakfast @ Hostel 7:45am - 8:15am	Breakfast @ Hostel 7:45am - 8:15am	
9 am	Arrival at Hostel, Check in 9am - 12:45pm	Student Session I Metamonada 8:30am - 9:30am	Monica Hagedorn (Workshop 3) 8:30am - 12:15pm	Breakfast @ Hostel 8:30am - 9am
10 am		Jude Przyborski (Lecture) 9:45am - 11am		Student Session V Apicomplexa 1 9:15am - 10:15am
11 am		Student Session II Trypanosoma 11:15am - 12:15pm		Student Session VI Apicomplexa 2 10:30am - 11:30am
12 pm		Lunch break 12:15pm - 1:30pm	Lunch break 12:15pm - 1:30pm	Concluding remarks 11:30am - 12pm
1 pm	Welcome 1pm - 1:30pm			Lunch break 12pm - 1pm
2 pm	Egbert Tannich (Lecture) 1:30pm - 2:15pm	Tobi Spielmann (Workshop 2) 1:30pm - 6pm	Joachim Clos (Practical course 2) 1:30pm - 3:30pm	Course Event 1pm - 3pm
3 pm	Freddy Frischknecht (Workshop 1) 2:35pm - 6pm			Course Ends 3pm - 3pm
4 pm			Student Session IV "Worms" 3:45pm - 5:45pm	
5 pm				
6 pm				
7 pm	Joachim Clos (Practical course 1) 6:15pm - 8:15pm	Student Session III Vectors 6:15pm - 7:15pm		
8 pm			Dinner at the Copper House 7:30pm - 11pm	
9 pm	Meet and Greet drinks (Location TBA) 9pm - 11pm			
10 pm				
11 pm				

COURSE SCHEDULE

Wednesday, March 16th

- Until 12:45 pm Registration / check in at the hostel
- 12:50 pm Walk to the Bernhard-Nocht-Institute
- 1:10 pm Welcome
Dr. Jude Przyborski
- 1:30 pm Lecture: Introduction to Parasitology
Prof. Dr. med. Tannich, BNI Hamburg
- 2:15 pm Coffee break
- 2:35 pm Workshop 1: Digital image acquisition, analysis and presentation
Dr. Freddy Frischknecht, Heidelberg University Hospital
- 6:00 pm Coffee break
- 6:15 pm Practical Course Part 1: Stage differentiation of *Leishmania*
PD Dr. Joachim Clos, BNI Hamburg
- 9:00 pm Meet and Greet drinks (location TBA)



COURSE SCHEDULE

Thursday, March 17th

- 7:45 am Breakfast at the hostel
- 8:30 am Student Session I: Metamonada
Chairs: Sabrina Heiny, Philipp Stahl
Speakers: Eva Martincova, Jan Pyrih, Katerina Pilarova
- 9:30 am Coffee break
- 9:45 am Lecture: Protein trafficking in the human malaria parasite
PD Dr. Jude Przyborski, Philipps University Marburg
- 11:00 am Coffee break
- 11:15 am Student Session II: Trypanosoma
Chairs: Matthew Daley, Nicole Kilian
Speakers: Philipp Stahl, Stefan Mogk, Claudia Frey
- 12:15 pm Lunch break
- 1:30 pm Workshop 2: Interaction of the malaria parasite with its host cell
Dr. Tobias Spielmann, BNI Hamburg
- 6:00 pm Coffee break
- 6:15 pm Student Session III: Vectors
Chairs: Leila Bouazzi and Claudia Frey
Speakers: Francesca Jarero, Matthew Daley, Jan Drahota, Alison Howell



COURSE SCHEDULE

Friday, March 18th

- 7:45 am Breakfast at the hostel
- 8:30 am Workshop 3: Investigation of host-pathogen interactions
Dr. Monica Hagedorn, BNI Hamburg
- 12:15 pm Lunch break
- 1:30 pm Practical Course Part 2: Stage differentiation of *Leishmania*
Prof. Dr. Joachim Clos
- 3:30 pm Coffee break
- 3:45 pm Student Session IV: Worms
Chairs: Luis Barniol, Sascha Hanig, Alison Howell
Speakers: Juliet Davies, Nadia Ben Nouir, Sarah Hemer, Leila Bouazzi,
Tatiana Küster
- 7:30 pm Dinner at the Copper House



COURSE SCHEDULE

Saturday, March 19th

- 8:30 am Breakfast at the hostel
- 9:15 am Student Session V: Apicomplexa 1
 Chairs: Eva Martincova and Nadia Ben Nouir
 Speakers: Thierry Monney, Sascha Hanig, Stefanie Wiedmer
- 10:15 am Coffee break
- 10:30 am Student Session VI: Apicomplexa 2
 Chairs: Francesca Jarero and Sarah Hemer
 Speakers: Sabrina Heiny, Luis Barniol, Nicole Kilian
- 11:30 am Concluding remarks
 Talk Prize
- 12:00 pm Lunch break
- 1:00 pm Course Event: Harbour Trip
- 3:00 pm Course Ends



LECTURERS**PD. Dr. Joachim Clos**

Research Group Clos (Leishmaniasis I)

Bernhard Nocht Institute for Tropical Medicine, Bernhard-Nocht-Str. 74, 20359 Hamburg, Germany

The main focus of the group is the function and role of so-called heat shock proteins in the control of the *Leishmania* life cycle. The main signal for the conversion from the insect form (promastigote) to the mammalian stage (amastigote) is the increase of the ambient temperature and the acidic milieu of the host cells. An equivalent change *in vitro* is sufficient to trigger the parasite's conversion from the promastigote to the amastigote. The interplay of heat shock proteins with the signal transduction pathways is subject to our research efforts. Further activities are the identification and analysis of genetic factors that determine parasite virulence and drug resistance.

Dr. Monica Hagedorn

Research Group Hagedorn (Cell Biology)

Bernhard Nocht Institute for Tropical Medicine, Bernhard-Nocht-Str. 74, 20359 Hamburg, Germany

Intracellular pathogens, such as mycobacteria, are the cause of devastating diseases. In order to survive within host cells, they have evolved sophisticated strategies to evade cellular defense mechanisms. To develop potential vaccines or drugs it is essential to improve our understanding of these complex interactions between the host cell and the bacteria. Over the past decades the complexity of the host systems has hampered our advancements.

In our laboratory we use a model system to investigate the interactions between a host cell and intracellular pathogens. One focus of the group is to study mycobacteria virulence in the *Dictyostelium discoideum* - *Mycobacterium marinum* system, which recapitulates the events in mammalian systems. By combining the easy genetic and biochemical tractability of the *Dictyostelium* model system with biochemical, molecular- and cell biology approaches we aim to provide insights into fundamental mechanisms of host-pathogen interaction.

In order to establish an infection, pathogenic mycobacteria, such as *Mycobacterium tuberculosis*, manipulate and subvert cellular defense mechanisms of phagocytic cells. In addition to the establishment of infection, pathogen egress and cell-to-cell is essential to maintain an infection. Here, the group focuses mainly on investigating the exit of bacteria from the host cell.

Dr. Freddy Frischknecht

AG Frischknecht

Heidelberg University Hospital, Im Neuenheimer Feld 324, 69120 Heidelberg, Germany

Malaria is transmitted during the blood meal of the *Anopheles* mosquito, when *Plasmodium* sporozoites are injected into the skin. These sporozoites are highly motile using a unique type of motility system allowing them to move within the tissue in order to reach the blood circulation. This motility system is also important for the invasion of a variety of cell types at different stages of the *Plasmodium* life cycle thus being essential for the successful progression of the infectious cycle of malaria parasite.

We have previously shown that *Plasmodium* sporozoites are moving at very low speed in the salivary glands of infected mosquitoes. However, they are moving at high speed (2 $\mu\text{m/s}$) upon transmission into the vertebrate host. 'In vivo' microscopy showed that sporozoites can move extensively within the dermis and associate with blood and lymph vessels which they can both invade. We were able to follow single parasites in the various tissues visited by the sporozoite until it finally invades into a liver cell. We will deepen our understanding of these processes using state-of-the-art microscopes available at the department of parasitology and the BioQuant institute. Using simple *in vitro* imaging approaches in combination with common drugs we discovered some intriguing features of sporozoite motility. Some common themes with other motile cells suggest that we can use the *Plasmodium* sporozoite as a model system for cell motility. In addition we hope to uncover unique aspects of sporozoite gliding, which could lead to new ways of interfering with the parasite development in the mosquito vector or the mammalian host.



PD. Dr. habil. Jude Przyborski, PhD

AG Parasitologie

Philipps University Marburg, FB 17, Karl von Frisch Strasse, 35043 Marburg, Germany

My work concentrates on various topics related to protein trafficking mechanisms in the human malarial parasite *P. falciparum*. Techniques used include cell fractionation, proteomics, bioinformatics as well as recombinant DNA technology and transfection of blood stage *P. falciparum*. Our eventual aim is to characterise the trafficking pathways, and machinery of this intracellular parasite, with a view to identifying targets for intervention strategies.

Dr. Tobians Spielman

Laborgruppe Spielmann (in the Research Group Gilberger)

Bernhard Nocht Institute for Tropical Medicine, Bernhard-Nocht-Str. 74, 20359 Hamburg, Germany

We work with the parasite causing the severest form of human malaria, the protozoan *Plasmodium falciparum*. The development of *Plasmodium* parasites inside of human red blood cells is responsible for the symptoms of malaria in infected people. In this life cycle phase the parasite enters a red blood cell, where it grows and multiplies until it fills out most of the host cell. Thereafter the host cell ruptures, releasing up to 32 new invasive parasites that enter new red blood cells to continue the cycle.

The interaction of the parasite with its host cell is our key interest. *Plasmodium* parasites modify the red blood cell to generate an optimal niche for growth and at the same time ingest most of the host cell cytosol. We visualise these processes in live parasites using microscopy techniques including 3D imaging over time and use a range of molecular and cellular biology techniques to identify the parasite's molecular toolbox involved.

With our work we hope to provide a better understanding of processes central to malaria biology and to identify aspects not found in human biology that may therefore be suitable drug targets.

Prof. Egbert Tannich, MD

Head of Department of Molecular Parasitology

Bernhard Nocht Institute for Tropical Medicine, Bernhard-Nocht-Str. 74, 20359 Hamburg, Germany

The Department of Molecular Parasitology focuses its research on molecular aspects of amoebiasis and malaria, two parasitic diseases with high impact on morbidity and mortality in tropical countries. In particular, questions concerning variability, immune evasion and pathogenicity of *Entamoeba histolytica* and *Plasmodium falciparum* are investigated. In addition, the Department is involved in parasite diagnostics and in the development of new tools to improve detection of parasite infections in clinical samples. In collaboration with the Institute for Standardisation and Documentation for Medical Laboratories (INSTAND) the Department regularly performs external quality assessments of more than 300 laboratories in the diagnostics of faecal and blood parasites.



STUDENT SESSIONS

Thursday, 03.17.2011

I METAMONADA

Chairs: Sabrina Heiny and Philipp Stahl

Identification of transport channel in mitosome of *Giardia intestinalis*

Martincova E, Fixova I, Dolezal P p. 33

Unknown mitochondrial protein of *Giardia intestinalis* and its possible role in controversial apoptosis

Pyrih J, Rada P, Hrdy I, Tachezy J p. 37

Iron-sulfur flavoproteins from *Trichomonas vaginalis* hydrogenosomes

Pilarova K, Hrdy I p. 36

II TRYPANOSOMA

Chairs: Matthew Daley and Nicole Kilian

Genes of *Trypanosoma cruzi* GPI-biosynthesis enzymes and the immunological impact

Stahl P, Debierre-Grockiego F, Kimmel J, Schwarz R T p. 38

The role of Endonuclease G during apoptosis in *Trypanosoma brucei*

Mogk S, Duszenko M p. 34

The role of Endonuclease G during apoptosis in *Trypanosoma brucei*

Frey C, Duszenko M p. 25

III VECTORS

Chairs: Leila Bouazzi and Claudia Frey

Molecular aspects of early mosquito development

Jarero F, Krzywinski J p. 30

What is the prevalence of malaria in mosquitoes and children under 5 in the south west region of Burkina Faso?

Daley M, Ranson H p. 22

Specificity of mice immune response on sand fly bites

Drahota J, Rohoušová I p. 24

Is *Fasciola hepatica* present in Uganda?

Howell A, Stothard R, La Course J p. 29



STUDENT SESSIONS

Friday, 03.18.2011

IV “WORMS”

Chairs: Luis Barniol, Sascha Hanig and Alison Howell

Prevalence of *Fasciola* in Mount Elgon Region, Uganda

Davies J, Stothard R

p. 23

Vaccination with Heat Shock Protein 60 (HSP60) against experimental *Strongyloides ratti* infection

Ben Nouir N, Eschbach M L, Kingsley M T, Brattig N, Fleischer N, Breloer M

p. 20

Molecular characterization of evolutionary conserved signalling systems of *Echinococcus multilocularis* and development of novel drugs against Echinococcosis

Hemer S, Brehm K

p. 28

Molecular and functional characterization of dual specific phosphatase (DUSP) in term of its inhibitory effect on *Echinococcus* growth

Bouazzi L, Spiliotis M, Gottstein B

p. 21

Mefloquine – a novel treatment for Alveolar Echinococcosis

Küster T, Hemphill A

p. 32



STUDENT SESSIONS

Saturday, 03.19.2011

V APICOMPLEXA 1

Chairs: Eva Martincova and Nadia Ben Nouir

Vaccination with recombinant chimeric antigen confers protection against *Neospora caninum* cerebral infection in experimentally infected mice

Monney T, Rütli D, Schorer M, Hemphill A

p. 35

Study on Gamogony, Oocyst-wall formation and Sporogony in *Eimeria nieschulzi* a Parasite-Host-Model in the rat

Hanig S, Entzeroth R

p. 26

Host adaption of the *Eimeria* oocyst wall

Wiedmer S, Entzeroth R

p. 39

VI APICOMPLEXA 2

Chairs: Francesca Jarero and Sarah Hemer

Understanding the biological function of *P. falciparum* extended N-terminal ER-type signal peptides

Barniol L, Lingelbach, Przyborski J M

p. 19

Protein trafficking to the apicoplast of *P. falciparum*. Signals, pathways and molecules

Heiny S, Przyborski J M

p. 27

Induced protein export in *Plasmodium falciparum*-infected erythrocytes

Kilian N, Lanzer M

p. 31



Luis Barniol**Understanding the biological function of *P. falciparum* extended N-terminal ER-type signal peptides**

Barniol L, Lingelbach K, Przyborski J M
Department of Parasitology, University of Marburg, Germany

The initial stages of the *P. falciparum* secretory pathway appear to be similar to that in higher eukaryotes, but with some important differences. It has been noted that, although many *P. falciparum* proteins contain so-called “canonical” signal peptides (with a central hydrophobic region, a hydrophilic N-region and a C-terminal region), a number of secreted proteins contain an unusual N-terminal region prior to the hydrophobic signal peptide (recessed signal peptides). These N-terminal sequences appear to target proteins to the ER, but how exactly this occurs, and the function of the extension remains unknown. For reasons that are currently not well understood, recessed signal sequences are found mostly attached to proteins predicted to be transported to the cytosol of the host erythrocyte. These proteins underlie the pathology of malaria infection, as they confer novel properties to the infected host cell. In my PhD thesis, we aim to dissect such unusual secretory signal peptides, with a view to understanding their function. We shall use several *P. falciparum* N-terminal regions to this end, including classical signal peptides, as well as proteins containing recessed hydrophobic domains.



Nadia Ben Nour**Vaccination with Heat Shock Protein 60 (HSP60) against experimental *Strongyloides ratti* infection**

Ben Nour N, Eschbach M L, Kingsley M T, Brattig N, Fleischer N, Breloer M
Bernhard-Nocht-Institut für Tropen Medizin, Hamburg, Germany

Strongyloides ssp. are gastrointestinal nematodes that affect approximately 30-100 million mainly in tropic and subtropic regions. Heat shock protein 60 (srHSP60) was characterized as an abundant constituent of excretory/ secretory products (E/S) of *S. ratti*. Here we analyze the outcome of HSP60 vaccination during the experimental murine *S. ratti* infection.

A primary *S. ratti* infection with 1000 third stage infective larvae (iL3) that is resolved spontaneously induced partial protection to a second infection. The vaccination with *S. ratti* lysate also showed significant lower number of adults in the gut but unchanged larval output in the feces compared to naïve mice. In contrast, srHSP60- vaccinated mice showed even higher number of adults in the gut and higher larval output compared to naïve mice.

S. ratti-specific antibodies composed out of only IgM and IgG1 were only induced in the *S. ratti* lysate- vaccinated group and the positive control group (iL3). srHSP60- specific IgG1, IgM, but also IgG2b and IgG2c isotypes were induced selectively in srHSP60- vaccinated mice.

in vitro stimulation with *S. ratti* lysate of lymphocytes derived from spleen of the different studied groups showed that IL-13 as an indicator of Th-2 response was produced in spleen cells derived from both, naïve and lysate vaccinated- group but not in srHSP60- vaccinated group, whereas the Th-1 associated cytokine IFN- γ was only produced in srHSP60- vaccinated mice. Moreover, *in vitro* stimulation with srHSP60 of lymphocytes derived from spleen of different studied groups showed also that IFN- γ was mainly produced by spleens from srHSP60- vaccinated mice. However, IL-13 was not detected in any studied group of mice.

The vaccination with srHSP60 worsened the outcome of infection and shifted the mice immune system towards Th-1 response which is deleterious for the host. We suggest that the secretion of srHSP60 by *S. ratti* during natural infection may weaken the protective Th-2 response and thus may contribute to the evasion of the host immune defense.



Leila Bouazzi**Molecular and functional characterization of dual specific phosphatase (DUSP) in term of its inhibitory effect on *Echinococcus* growth**

Bouazzi L, Spiliotis M, Gottstein B
Institute of Parasitology, University of Bern, Switzerland

Echinococcosis caused by larval stage of *E. multilocularis* (metacestode) of the cestode *Echinococcus multilocularis* (alveolar echinococcosis or alveolar hydatid disease; AE) represents a major public health problem associated with significant socio-economic losses in many countries world-wide. Prevention and treatment of AE is a major issue of public health authorities. In order to improve such initiatives, we need to acquire sufficient basic knowledge on the disease, including the understanding of the molecular mechanisms triggering successful parasite proliferation even in the presence of a host immune response.

As part of this work, we will elucidate basic molecular events controlling the expression of dual specific phosphatases (DUSP). These molecules have long been associated with cell growth, cell proliferation, and cell survival. DUSP appear to be selective for dephosphorylating phosphothreonine and phosphotyrosine residues within kinases.

Molecular and functional analysis of the DUSP molecules will allow us to understand in greater details the molecular mechanisms controlling growth of the parasite.

In the frame of this study, twelve DUSP orthologues were identified so far by blast analysis of the *E. multilocularis* genome. These sequences have been/will be expressed in *E. coli* to perform both functional assays and produce antibodies which can be used for immunodetection and pull-down assays. For deeper characterization, a yeast two hybrid approach is planned to identify the specific binding partners. Finally, RNA interference strategies are planned to assess the importance of the DUSP on the growth and development of *E. multilocularis* metacestodes and to examine the enzyme regarding a putative drug target function.



Matthew Daley

What is the prevalence of malaria in mosquitoes and children under 5 in the south west region of Burkina Faso?

Daley M, Ranson H
Liverpool School of Tropical Medicine, United Kingdom

This project aims to determine the prevalence of malaria in humans and mosquitoes in the south west region of Burkina Faso. This will be done by sampling at four sites; two rural sites and two sites close to health centres. 100 children from each site will have finger prick blood samples taken and rapid diagnostic kits will be used to test this for malaria. If the result is positive the children will be treated, if negative paracetamol will be provided and they will be advised to seek further medical help if they are symptomatic. The GPS location of all the samples taken will be recorded.

The mosquitoes will be collected using exit traps on 8 houses per village. Some dissections will be performed in the field, and the majority of mosquito infections will be determined by PCR on return to the LSTM. The prevalence of malaria will be compared with the distance from breeding sites and the distance from health centres.

This project will be run at the same time as one looking at the prevalence of Lymphatic Filariasis. It will be able to assess the level of co-infection in mosquitoes, and whether the M or S forms of the *Anopheles gambiae* complex are more likely to transmit either LF or malaria. Also, the clinical signs and symptoms will be recorded before the rapid diagnostic test, allowing the appraisal of the diagnostic criteria the proportion of children clinically diagnosed with malaria vs. actual number.



Juliet Davies

Prevalence of *Fasciola* in Mount Elgon Region, Uganda

Davies J, Stothard R
Liverpool School of Tropical Medicine, United Kingdom

Fasciolosis is a zoonotic disease that has become a serious public health problem in some parts of the world. It is caused by the liver fluke species *Fasciola hepatica* and *F. gigantica* which are transmitted by lymnaeid snail species. Current worldwide estimates of human infection are 2.4 to 17 million people infected with 91.1 million at risk. With reports of substantial infection of *Fasciola* within livestock reported, a study of its prevalence within the human population is required. A sample of the population in the Mount Elgon Region in Uganda will be tested for fasciolosis, along with collection of the intermediate host species of *Fasciola* spp.



Jan Drahota

Specificity of mice immune response on sand fly bites

Drahota J, Rohoušová I, Volf P
Charles University, Prague, Czech Republic

Sand flies saliva is known to have immunological effects on the host and moreover it is able to enhance success of the transmission of *Leishmania*. On the other hand immunisation of experimental models with the saliva gives them a partial protection from the Leishmaniasis.

In the presented study we show that the effect of the immunisation is species specific for these types of immune response: proliferation of T-lymphocytes and production of IgG antibodies (by ELISA tests and immunoblots) both at hosts infected or uninfected with *Leishmania*.

My PhD thesis is about creating a recombinant protein from sand fly saliva for further immunological research.



Claudia Frey**The role of Endonuclease G during apoptosis in *Trypanosoma brucei***

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Apoptosis is a significant contributor to the morphologic and functional development of multicellular organisms, however the advantages of this process for unicellular organisms are much less evident. In protozoa programmed cell death (PCD) contributes to regulate the size of parasite population. Apoptosis has been found in many different single-cell eukaryotes including the species of *Trypanosoma brucei*. In this work we have evaluated the effects of Staurosporine and PGD₂ on Endonuclease G translocation in *T. brucei*. PGD₂ is known to induce apoptosis in these parasites. The distinctive signs of apoptosis such as phosphatidylserine exposure, loss of mitochondrial membrane potential, membrane blebbing and DNA degradation can be observed after treatment with PGD₂. Staurosporin has no proved apoptotic effect but has shown a significant anti-parasitic activity against *T. brucei*. Endo G is a mitochondrial protein which is associated with DNA fragmentation after cell death is triggered. We have investigated the role of Endo G in PCD by using the bloodstream form of *T. brucei*. Our experiments show that PGD₂ and Staurosporin can induce translocation of Endo G to the nucleus.



Sascha Hanig**Study on Gamogony, Oocyst-wall formation and Sporogony in *Eimeria nieschulzi* a Parasite-Host-Model in the rat**

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Oocysts are crucial stages of the parasites to survive in the environment until they can infect a new specific host to maintain their life cycle. Oocysts are highly resistant to mechanical and chemical stress and consist of two layers, formed by preformed wall-forming bodies of type I and type II synthesised during macrogametogony. Transfection of sporozoites from *Eimeria nieschulzi* with vectors, containing promoter sequences and genes coding for heterologous Gam-proteins (from the sequenced avian parasite *Eimeria tenella*) and reporter genes (YFP, tomato) will allow studying the dynamics of oocyst wall formation. Deletion of different parts of the Gam coding genes in various reporter gene vectors will produce results about the cellular signals which are involved in the wall forming process and in the transport and pathway of Gam proteins.



Sabrina Heiny**Protein trafficking to the apicoplast of *P. falciparum*. Signals, pathways and molecules**

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Plasmodium falciparum, like most other apicomplexan parasites, harbours a unique organelle, the apicoplast, that is indispensable for parasite survival within its host cells. The majority of apicoplast-resident proteins, however, are nucleus-encoded and therefore must be transported to the complex plastid posttranslationally, a process that is complicated by the four membranes surrounding this organelle. The signals responsible for trafficking most proteins to the apicoplast are well described and comprise an N-terminal bipartite leader sequence consisting of a hydrophobic signal peptide and a plastid-targeting transit peptide, whereas it is still a matter of debate when and where in the course of the secretory pathway sorting of apicoplast resident proteins occurs.

Our research interests focus on both pre-protein transport to and subsequent translocation across the four membranes surrounding the complex plastid of *Plasmodium falciparum*. It has been postulated that sorting of apicoplast proteins follows a Golgi-independent route. However, recent data from our laboratory suggest that sorting might take place via the cis-Golgi. To further elucidate this, we aim to create and study transgenic parasites expressing bipartite leader sequences fused to reporter genes tagged with C-terminal ER-retrieval sequences, partially in combination with common inhibitors of the secretory pathway.



Sarah Hemer**Molecular characterization of evolutionary conserved signalling systems of
Echinococcus multilocularis and development of novel drugs against
Echinococcosis**

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As a model organism for flatworm parasites, we study the fox-tapeworm *Echinococcus multilocularis* whose larval stage causes alveolar echinococcosis (AE), one of the most dangerous parasitoses world-wide. AE is characterized by an infiltrative, tumor-like growth of the parasitic metacestode within the intermediate hosts organs, involving totipotent stem cells which crucially contribute to parasite tissue renewal and metastasis formation. Chemotherapeutic anti-AE treatment options are currently very limited and rely on benzimidazoles (BZ) which target parasite β -tubulin. However, due to an affinity of the drugs to host β -tubulin, BZ treatment is often associated with severe side-effects and is merely parasitostatic in vivo. My work focuses on flatworm signalling pathways which display structural and functional similarities with cancer-associated signalling systems of mammals and are crucially involved in metacestode development to establish potential targets for novel therapeutic approaches. Those pathways are often targeted in mammals due to their role in cancer development. The biochemistry of these components is thus well studied and many inhibitors exist that modify their activity. In my work I am specifically interested in components of the insulin/IGF-I mediated signalling pathway. Previous work of our group, using in vitro cultivation systems for larvae and cells of the fox-tapeworm *Echinococcus multilocularis*, indicated that host-derived insulin significantly stimulates metacestode regeneration from primary germinal cells and sustains metacestode growth. Two receptor tyrosine kinases, named EmlR1 and EmlR2 were identified, which showed significant homologies to tyrosine kinases of the insulin/IGF-I receptor family. Furthermore homologues of the PI3 and akt kinases could be identified along with other components of their signalling cascade. The activation of these proteins by host insulin and their potential role as inhibitor targets are currently investigated. Combination therapies are widely used in the treatment in various diseases and offer great potential for the approach of AE. The results show that inhibitors can exhibit additive potential when used in combinations. The modeling of parasite specific inhibitors on the basis of the already existing and the increased effectivity through combinations could improve AE treatment and decrease side effects of the therapy.



Alison Howell**Is *Fasciola hepatica* present in Uganda?**

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Fasciola hepatica is a parasite of great economic importance in cattle and sheep worldwide. It is also a zoonosis. It is known to be present in Uganda's neighbouring countries including Tanzania and Kenya.

In this study we will use various methods to determine whether *F. hepatica* is present in highland areas of Uganda:

1. Snail collection from water bodies near livestock grazing areas looking for *Lymnaea truncatula*.
2. Look for flukes and eggs in livers of cattle from abattoirs in Uganda, use faecal concentration techniques to look for fluke eggs, and take serum from these cattle to validate an ELISA antibody test on serum and possibly milk
3. Following validation of the ELISA, faecal and blood samples will be taken from live cattle in the area and tested for fluke
4. Fluke and snail samples will be speciated using PCR in the UK to differentiate from *Fasciola gigantica* and *Lymnaea natalensis*.
5. Collection of GPS and water chemistry data from the snail sampling sites will enable us to map the likely locations of snails and thus the possible transmission areas of *F. hepatica*.

A similar study will look at whether *F. hepatica* is infecting people in the area.



Francesca Jarero

Molecular aspects of early mosquito development

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Anopheles gambiae is an important and effective vector of parasites, most notably malaria. Little is known about gene expression in embryos of the mosquito. Embryo transcriptome data gathered by next generation sequencing will be experimentally validated. RT-PCR experiments will be performed to verify the expression of selected sequences and to confirm the existence of novel genes and novel alternative splice variants. The elucidation of these transcripts will hopefully lead to the development of novel control methods and/or to the identification of targets for novel insecticides that could be incorporated into malaria control programmes.



Nicole Kilian**Induced protein export in *Plasmodium falciparum*-infected erythrocytes**

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Department of Infectious Diseases, Heidelberg University, Germany

Malaria tropica is with over 250 mio. clinical cases per year the most threatening parasitemia worldwide putting a huge selective pressure on the people living in the endemic areas. This selective pressure is resulting in a strong parasite-host co-evolution which further leads to the favouritism of several host polymorphisms to avoid the severe complications followed by an infection with *Plasmodium falciparum*.

It has been known for a long time, that several erythrocyte polymorphisms, for example hemoglobin C (HbC) and hemoglobin S (HbS) can protect against severe malaria. One reason for this effect is the decreased cytoadherence of the parasitized erythrocytes in the microvasculature which is caused by an altered display of parasitic adhesion proteins on the erythrocyte surface. The adhesion proteins are trafficked to the surface of the erythrocyte via an golgi-like compartment called the Maurer's clefts. The Maurer's clefts are of parasitic origin and play a key role in parasite-host interactions since they receive and harbour over 200 different parasitic proteins. During my PhD thesis I am examining the export of two proteins of the Maurer's clefts: PfSBP1 and STEVOR in HbAA, HbCC and HbSC erythrocytes. For the examination of the protein movement in the different erythrocytes I am using a conditional export system which allows a specific induction of PfSBP- and STEVOR-export.



Tatiana Küster**Mefloquine – a novel treatment for Alveolar Echinococcosis**

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Alveolar echinococcosis is caused by the metacestode stage of the fox tapeworm *Echinococcus multilocularis* and causes severe disease in the human liver and occasionally other organs, which is fatal if treatment is unsuccessful. The present chemotherapy is based on benzimidazoles and it has been found to be parasitostatic rather than parasitocidal usually consisting of lifelong uptake of big doses of drug. New treatment options are urgently needed but seldom developed due to the rare occurrence of the disease. A screening of drugs currently used for the treatment of other parasitic diseases was performed. The selected candidate, mefloquine, applied in the prevention and treatment of malaria showed promising results *in vitro*, but was never tested *in vivo* against the disease. The present study describes the treatment of echinococcosis in Balb/c mice with mefloquine administered orally or intraperitoneally for a period of eight weeks. A possible synergy with albendazole was also investigated. Treatment with mefloquine administered intraperitoneally presented a reduction in total parasite weight similar to albendazole oral treatment. Whether the action of the drug is indeed parasitocidal remains to be investigated. The mechanism of action of mefloquine against *Plasmodium falciparum* is not yet clear. Mefloquine is believed to prevent the heme polymerization in the host red blood cells containing the plasmodium parasite, but this mechanism was not yet proved. Such a mechanism would also not explain the action of the drug in *Echinococcus multilocularis* metacestodes, as this is not an intracellular parasite. Affinity chromatography with a mefloquine-bound matrix followed by mass spectrometry of binding proteins resulted in the possible identification of the *Echinococcus multilocularis* ferritin. Ferritins are well conserved intracellular proteins responsible for the storage of iron in a non-toxic form, as well as controlled iron transport. Free iron is toxic to cells as it acts as a catalyst in the formation of free radicals from reactive oxygen species. The binding of ferritin by mefloquine might induce changes in the intracellular iron metabolism of the parasite, causing its death. This mechanism has still to be confirmed.



Eva Martincova**Identification of transport channel in mitosome of *Giardia intestinalis***

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Giardia intestinalis is not only an important human pathogen but also an excellent model organism for studying mitochondrial evolution. Due to its anaerobic lifestyle it has reduced mitochondrion (mitosome). Mitosomes contain very few proteins and lost all its genome. There has been only single mitosomal function proposed so far – FeS cluster biogenesis. However, import pathway to the mitosomes remains elusive.

While Tom40 homologue is present in outer mitosomal membrane, co-chaperones Pam18 and Pam16, are present in inner membrane. In classical mitochondria, these chaperones (together with HSp70) constitute a motor complex for the inner membrane translocase Tim23. However, no homologue of Tim23 has been found in *G. intestinalis* genome, neither in proteome of isolated mitosomes. Hence, *G. intestinalis* might employ unique translocase adapted to import of extremely small set of proteins (14 known so far) and lack of measurable membrane potential. To characterise this translocase and other components of the import apparatus in *G. intestinalis* we developed two systems for isolation of such channel.

One system is based on DHFR properties, which we used to introduce a "molecular plug" into the channel. Second one is based on antibodies against Pam16 and Pam18, which should be in physical contact with the translocase.



Stefan Mogk**The role of Endonuclease G during apoptosis in *Trypanosoma brucei***

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Endonuclease G is specifically activated by apoptotic stimuli and is able to induce nucleosomal fragmentation of DNA in *Trypanosoma brucei*. This nuclease is mitochondrion-specific and translocates to the nucleus where it cleaves DNA. It represents a caspase-independent apoptotic pathway initiated from the mitochondria. Part of the Endo G gene was cloned into p2T7, which allows to transcribe dsRNA for RNA interference. Monomorphic single marker bloodstream form cells (SMB) were stably transfected to yield a heritable and inducible knockdown of Endo G. Two clones were selected and characterized by growth experiments, apoptosis induction and FACS analysis. To verify knockdown efficiency we hybridized Northern probes against the dsRNA transcripts. We found a significant increase of dsRNA upon induction, suggesting that the cell lines are an appropriate means to investigate Endo G deficiency. The elucidation of molecular mechanisms leading to PCD in *Kinetoplastidae* could help to find new targets for chemotherapeutic drug development.



Thierry Monney**Vaccination with recombinant chimeric antigen confers protection against *Neospora caninum* cerebral infection in experimentally infected mice**

Monney T, Rütli D, Schorer M, Hemphill A
Institute of Parasitology, University of Berne, Switzerland

Neospora caninum (Apicomplexa: Eimeriina: Sarcocystidae) is reported as the leading cause of bovine abortion, thus the disease represents an important veterinary health problem and is of high economical significance (Hemphill and Gottstein 2000; Dubey et al. 2007). Currently, only one vaccine against bovine neosporosis is available on the market (Bovilis Neoguard®, Intervet). This vaccine is based on tachyzoite protein extract but confers only partial protection against the disease (Romero et al. 2004; Innes and Vermeulen 2006). There is therefore an urgent need for an efficient vaccine against bovine neosporosis.

The overall goal of our investigations on *N. caninum* is to develop a vaccine that limits both the cerebral infection and the transplacental transmission. It is very unlikely that this could be achieved by a single antigen, but most likely by a mixture of parasite antigens or a vaccine that contains a number of relevant antigenic domains of different proteins. Since promising results were obtained with a combination of the recombinant forms of three secreted proteins, NcMIC1, NcMIC3 and NcROP2 in the reduction of cerebral infection and vertical transmission in infected mice (Debache et al. 2009), we focused on the use of these proteins for further vaccination strategies. In order to increase the immunogenic potential of these antigens, the production of four different chimeric proteins based on their putative antigenic domains was investigated. Balb/C mice were vaccinated with the different proteins and challenged with *N. caninum* tachyzoites. The survival rate and health of the mice was monitored and one of the chimeric protein conferred complete protection against clinical symptoms in the vaccinated mice. The parasite burden in the brain was assessed by conventional quantitative real time PCR. Total IgG as well as IgG1 and IgG2a titers in the serum were assessed by ELISA and IFN γ and IL4 levels in serum were quantified.



Katerina Pilarova**Iron-sulfur flavoproteins from *Trichomonas vaginalis* hydrogenosomes**

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Trichomonas vaginalis is an anaerobic, flagellated protozoan parasite which causes vaginitis in woman and urethritis in man. These are the most common nonviral sexually transmitted diseases in the world.

Trichomonas lacks typical mitochondria and instead possesses hydrogenosomes, organelles of anaerobic energy metabolism that produce hydrogen and synthesise ATP by substrate-level phosphorylation. Hydrogenosomes were discovered almost 40 years ago in trichomonads parasitizing cattle and only relatively recently were shown to share a common ancestor with mitochondria. The genome of *Trichomonas vaginalis* was annotated in 2007. Based on typical amino terminal extensions, many thus far unidentified proteins were predicted to reside in hydrogenosomes. Among these were iron-sulfur flavoproteins, coded by 7 genes. The function of iron-sulfur flavoproteins in trichomonads is unknown, but the studies of homologous proteins from archaebacterium *Methanosarcina thermophila* suggest that they could play a role in oxidative stress management and that they could reduce and thus detoxify oxygen and hydrogen peroxide to water. In *Methanosarcina*, these proteins are probably involved in electrontransporting pathways where ferredoxins function as electron carriers. *Trichomonas vaginalis* together with another pathogen *Entamoeba histolytica* are the only eukaryotes known to possess these proteins. Thanks to the characteristic prosthetic groups (iron-sulfur cluster and flavin mononucleotide), these proteins can serve as one electron / two electron switch in redox reactions and thus could play an important role in hydrogenosomal electrontransporting pathways. Our goal is to verify the hydrogenosomal localization and expression status of these proteins in *T. vaginalis* and to identify their physiological function. Based on qRT PCR data, some homologues are transcribed differently in wild-type trichomonads in comparison with those resistant to metronidazole, the only available drug against the disease caused by trichomonads. Thanks to their redox properties, it is possible that iron-sulfur flavoproteins, besides their likely role in oxygen and hydrogen peroxide detoxification, are involved in metronidazole reductive activation. Our results obtained with recombinant trichomonas proteins indeed indicate their activity with hydrogen peroxide. Experiments designed to identify other redox partners of iron-sulfur flavoproteins are under way.



Jan Pyrih**Unknown mitosomal protein of *Giardia intestinalis* and its possible role in controversial apoptosis**

Pyrih J, Rada P, Hrdy I, Tachezy J
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Tah18 is a recently described protein which is commonly present in eukaryotic cells. Two hypothesis have been postulated so far. First that Tah18 plays crucial role in cytosolic Fe-S cluster development and transfer, second that Tah18 plays an important role in apoptosis. Although it is unclear whether apoptosis pathway does exist in *G. intestinalis*, we describe here some interesting observations about giardia homolog of Tah18.

We have biochemically characterized tagged protein isolated from bacteria. It can reduce some artificial acceptors such as 2,6-dichlorophenolindophenol, Methyl viologen and cytochrome c in presence of NADPH. We have also found that it can reduce giardia cytochromes b5. These observations are in agreement with results published about Tah18 from *Saccharomyces*.

Giardia Tah18 seems to have dual localization in cell which depends on yet unknown conditions. In few cells this protein is present in mitosomes while in the others Tah18 has cytosolic distribution.

Whether Tah18 interacts with cytochromes b5 physiologically is the main goal of further research as well as determining nature of possible mechanism of protein relocation from cytosol into mitosomes.



Philipp Stahl**Genes of *Trypanosoma cruzi* GPI-biosynthesis enzymes and the immunological impact**

Stahl P, Debierre-Grockiego F, Kimmel J, Schwarz R T
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The haemoflagellate *Trypanosoma cruzi* is the causative agent of the protozoan zoonotic Chagas' disease, or American trypanosomiasis that affects approximately eight million people in Latin America. The parasite is transmitted by blood-sucking insects of the triatomine species, blood transfusion, organ transplantation or congenitally. Recent incidence rates show an increase in Chagas outbreaks and vector propagation in South America after the vector has been successfully eradicated in the nineties.

The acute phase of disease triggers an immune response that restricts the dissemination and proliferation of parasites. However, parasites are able to persist in different tissues for decades causing the characteristics of Chagas' disease, cardiomyopathy and megasyndromes of the gastrointestinal tract and can even lead to death. Infection with *T. cruzi*, elicits macrophages to produce high levels of pro-inflammatory cytokines. Even though parts of the host-parasite interactions have been elucidated, many interactions of the parasite with the host cell remain in many aspects undefined. In that context, glycosylphosphatidylinositols (GPIs), components of a dense surface coat of glycolipids expressed by *T. cruzi* play possible roles. GPIs serve ubiquitously as anchors for proteins, complex carbohydrates or mucins and are known to induce an inflammatory response in macrophages. My diploma thesis describes a new method of purifying amastigote forms of *T. cruzi* and deals with the potential capability of GPIs of amastigotes to induce an pro-inflammatory response in macrophages *in vitro*. Secretion of TNF and IL-12, as well as nitric oxide (NO), incubated with purified GPIs has been observed. Furthermore, the induction of apoptosis in rat cardiomyocytes was determined after incubation with amastigote parasites. These results suggest that cardiomyopathy observed in Chagas' disease might be in part due to local inflammation in response to the GPIs and to apoptosis of cardiomyocytes induced by *T. cruzi*.



Stefanie Wiedmer**Host adaption of the *Eimeria* oocyst wall**Wiedmer S, Entzeroth R
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Excystation processes in different *Eimeria* species and the particular composition of the oocyst walls are insufficiently understood. It is a widely held belief that *Eimeria* oocysts are generally protease resistant and the excystation of sporocysts proceeds mechanically. Indeed, there are no hints for protease sensitivity of avian *Eimeria* species, whereas the protease pepsin can be used for the excystation of several rodent *Eimeria* species. Observations by transmission electron and fluorescence microscopy showed degradation of the inner oocyst wall of sporulated *Eimeria nieschulzi* oocysts after incubation with pepsin. SEM further revealed two polar caps and one micropyle structure on oocysts. Based on these microscopic studies biochemical examinations of the oocyst wall of *E. nieschulzi* are necessary. New developed monoclonal antibodies directed against *E. nieschulzi* macrogametocytes identified proteins of wall-forming bodies by indirect immunofluorescence tests. Using immunofluorescence and immunoelectron microscopy the involvement of wall-forming bodies during the development of the oocyst wall of *E. nieschulzi* can be observed by these monoclonal antibodies.



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