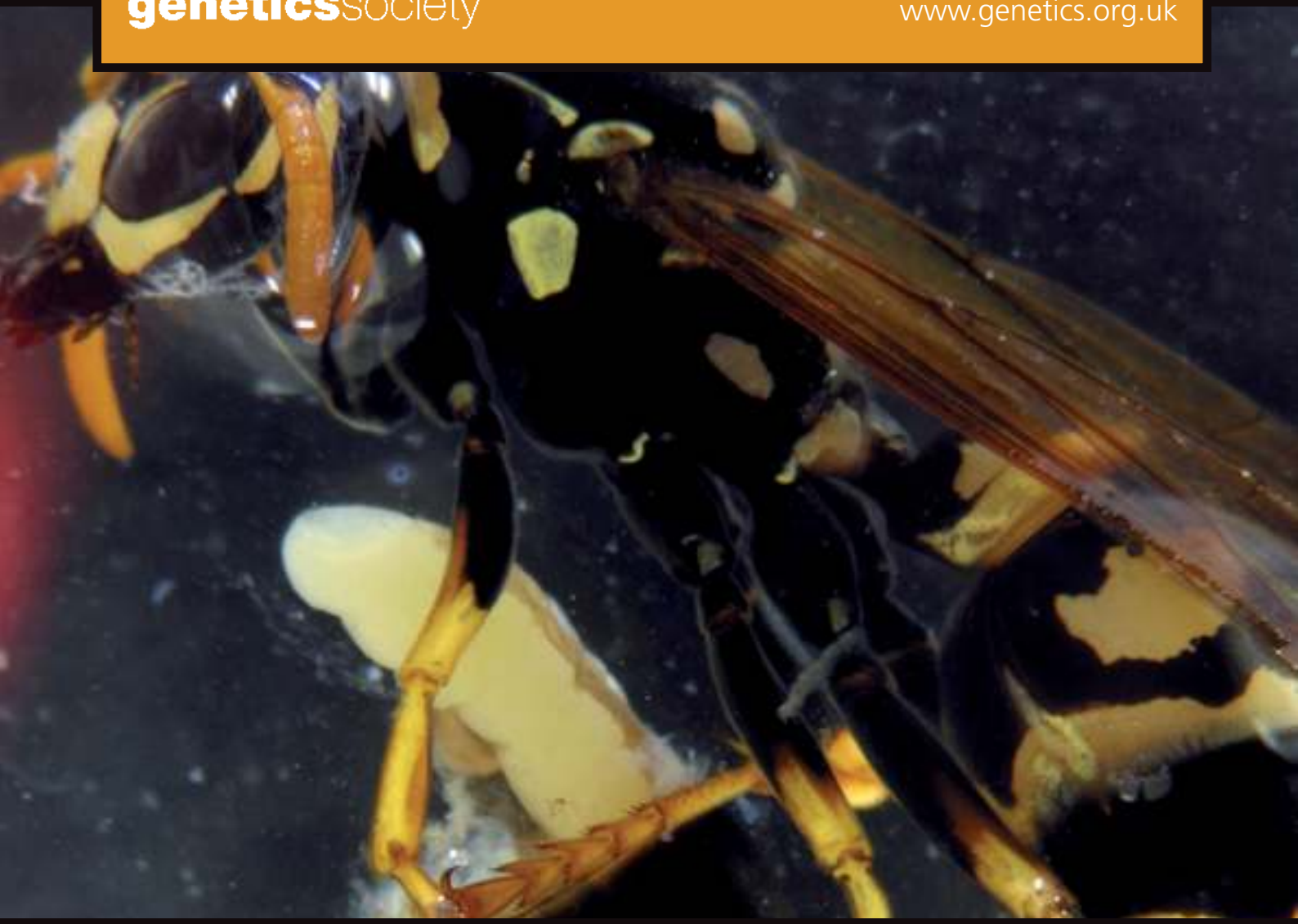
January 2009 . ISSUE 60

GENETICS SOCIETY NEWS

the geneticssociety

www.genetics.org.uk



IN THIS ISSUE

- Evolution Of Sexual And Asexual Reproduction Meeting
- A Guide To Undergraduate Summer Projects
- John Thoday: An Appreciation
- John Edwards: An Appreciation
- My Favourite Paper
- Student Travel and Fieldwork Reports

Genetics Society News is edited by Steve Russell. Items for future issues should be sent to Steve Russell, preferably by email to s.russell@gen.cam.ac.uk, or hard copy to Department of Genetics, University of Cambridge, Downing Street, Cambridge CB2 3EH. The Newsletter is published twice a year, with copy dates of 1st June and 26th November.

Two adult strepsipteran females (Family Xenidae) dissected from a Polistes paper wasp. From the fieldwork report by Dino McMahon on page 39 of this issue

A word from the editor

There is a focus on some more historical aspects of UK genetics in this issue with appreciations of both John Thoday and John Edwards, both of whom made important contributions to the development of genetics research and teaching in Britain. We also have a short report on a remarkable film of the 1948 International Genetics Congress, which was shown in Cambridge recently and contains the only known moving images of Ronald Fisher and JBS Haldane. By all accounts it was a fun event, with wizened members of the University shouting out the names of people they recognized.

As usual, we have reports from recent Genetics Society meetings including enjoyable one-day events on the evolution of sex and on the contributions model organisms can make to understanding human disease. We also have a crop of student reports on the meetings they attended with the aid of Genetics Society sponsorship, several more are available on the Society website. It's great to see the enthusiasm radiating from these reports and it's clear that the students, most of whom give poster or oral presentations, can get a great deal out of attending relevant conferences. As well as travel grants, the Society provides an increasing number of summer research studentships: we have recently increased the stipends and the lab expenses for these awards. Our Postgraduate student committee rep, Tom Nowakowski, has written a short piece aimed at budding young scientists who are considering embarking on a research career. I think it's useful for group leaders to consider some of the problems undergraduates face and try and encourage them by helping them find summer projects or funding. Tom's guide provides some pointers here.

By the time you receive this it will be 2009 and the 200th anniversary of Darwin's birth. There are a huge number of events around the UK marking this and the Darwin200 website is a useful guide to what's on (http://www.darwin200.org). The Genetics Society is sponsoring two events: a one-day Darwin and Development Symposium as part of the 2009 International Society for Developmental Biology Symposium in Edinburgh and a joint Genetics Society/Royal Society discussion meeting on Genetics and the Causes of Evolution over two days at the Royal Society. Unfortunately all may not be quite so rosy in the Darwin-fest garden: our Taxi Driver has some issues with the showbiz-style presentation of museum exhibitions.

As we reported in the last Genetics Society News, there were moves afoot to merge the Biosciences Federation and the Institute of Biology. Both organisations have voted on this and, as our VP for External Affairs John Brookfield reports, there are proposals to move forward and generate a single strong voice for UK bioscience.

As ever, anything you would like to write about in the general area of genetics, science policy or education, please get in touch.

Cheers

Steve Russell
University of Cambridge

Issue 60 . January 2009

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Printing
RPM Print & Design . Spur Road . Quarry Lane
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SPRING MEETING 2009

Common disease genetics: applying knowledge to health - what's next?

Friday 8 May 2009 The Royal Society, 6–9 Carlton House Terrace, London SW1Y 5AG

Genome-wide association studies (GWASs) are an important and exciting new chapter in human genetics research. We now know of well over 200 variants that influence susceptibility to a wide range of complex diseases. But where do we go from here? The aim of this meeting is to look back on the achievements of the past two years and reflect on how knowledge from this first generation of GWASs can be applied to improve human health in the most informed and ethical manner. Importantly, it will also discuss the best direction in which to take future research in this area.

Speakers:

Mark Walport (The Wellcome Trust, London, UK).
Complex disease genetics: progress and challenges.

David Clayton (Cambridge Institute for Medical Research, Cambridge, UK).
The role of genetic association studies in the study of common complex disease: achievements and limitations.

Inês Barroso (The Wellcome Trust Sanger Institute, Hinxton, UK). From GWAS hits to new biology – obesity as an example.

Nazneen Rahman (The Institute of Cancer Research, Sutton, UK). Clinical utility of breast cancer genes: current practice and future prospects.

Kari Stefansson (deCODE, Reykjavik, Iceland).
The impact of personalised genetic risk information.

Theresa Marteau (Kings College London, London, UK).
The risks of knowing genetic risks.

Nick Wareham (MRC Epidemiology Unit, Cambridge, UK).
Life after GWAS — what about the environment?

Scientific Organisers:

Tanita Casci (Nature Reviews Genetics, London, UK)
Peter Goodfellow (Department of Biosciences, University of Kent, Canterbury, UK)
John Todd (Cambridge Institute for Medical Research, Cambridge, UK)

Featuring:

The Genetics Society Balfour Lecture 2009 by **Matt Hurles** (The Wellcome Trust Sanger Institute, Hinxton, UK). Complex disease genetics: widening the focus to all classes of variation.

This meeting will include the Annual General Meeting of the Genetics Society and will conclude with a panel – audience discussion.

Registration:

Registration will be open shortly via the Genetics Society website **www.genetics.org.uk**

Registration fees
Members: £30. Non-members (academic): £80
Non-members (non-academic): £135
Undergraduate members may attend for free but must register in advance of the meeting.

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A Genetics Society Symposium in association with ISDB 2009

Darwin and Development

6th - 10th September 2009, Edinburgh International Conference Centre, Scotland.

SESSION 1 – VARIATION AND SELECTION

Variation Under Domestication
Elaine Ostrander, USA
(dog genetics, evolution and development)
John Doebley, USA
(domestication of maize from teosinte)

Variation Under Nature
Arhat Abzhanov, USA
(craniofacial development in Darwin's Finches)
Chris Kuhlemeier, Switzerland
(Petunia and pollinator-driven speciation)

SESSION 2 – HOMOLOGY AND ANCESTRY

Mutual Affinities Of Organic Beings
Michael Akam, UK

On The Imperfection Of The Geological Record
Phil Donoghue, UK

Genes Controlling Dentition and Its Evolution
Jukka Jernvall, Finland

Genomics And Developmental Genetics Of Acoel Flatworms: Clues To The Early Evolution Of Metazoa
Pedro Martinez, Spain

SCIENTIFIC ORGANISERS

Peter Holland, Oxford University and Phil Donoghue, University of Bristol.

The British Society for Developmental Biology will be hosting the International Development meeting in Edinburgh, September 6-10, 2009. As 2009 is the 150th anniversary of the publication of the Origin of Species, the Genetics Society will mark the occasion with a one-day symposium (two sessions) as part of this meeting.

for more information please visit **www.genetics.org.uk**

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The Genetics Society / Royal Society Discussion Meeting

Genetics And The Causes Of Evolution: 150 Years Of Progress Since Darwin

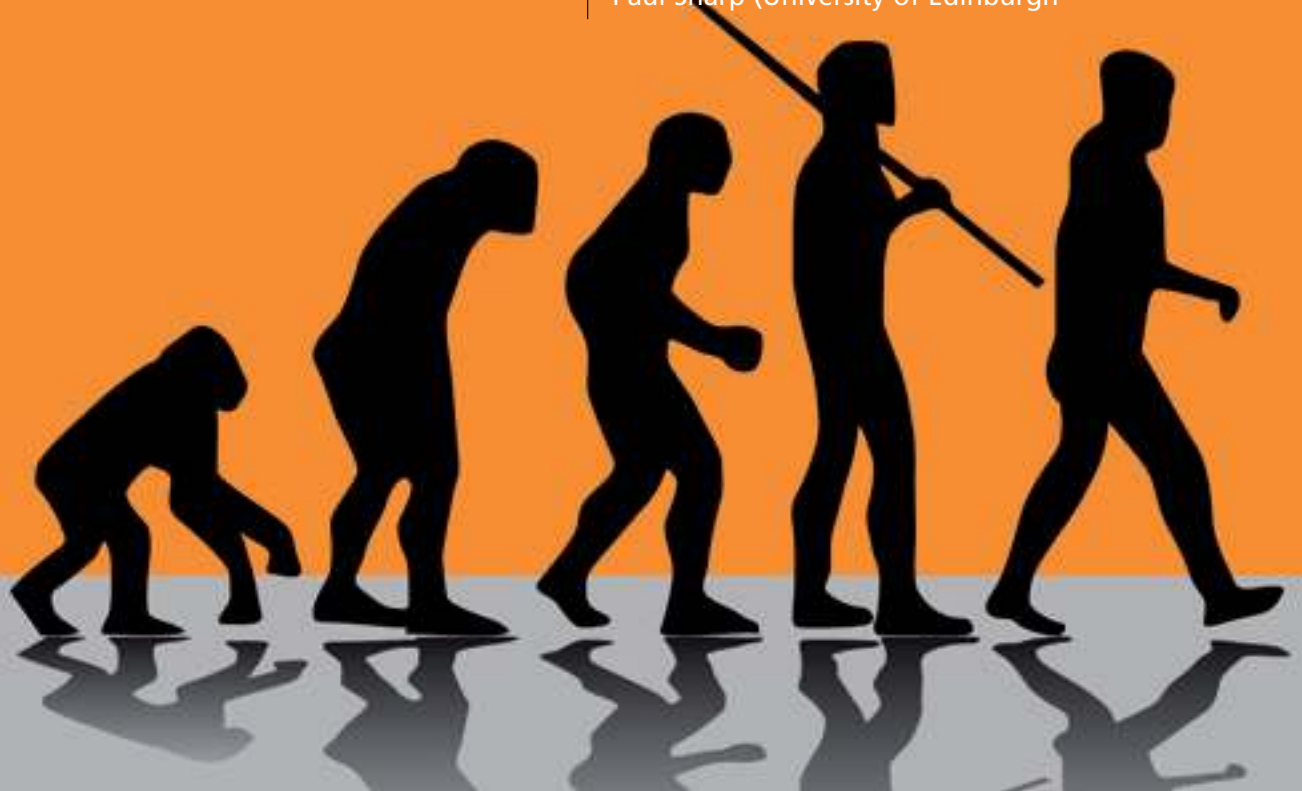
Thursday 12th and Friday 13th November 2009
The Royal Society, Carlton House Terrace, London SW1Y 5AG

Organisers: Mike Bonsall, University of Oxford and Brian Charlesworth, University of Edinburgh.

The Royal Society and the Genetics Society of the UK are organizing a two-day Discussion Meeting to celebrate the 200th anniversary of the birth of Charles Darwin, and the 150th anniversary of the publication of the Origin of Species. In Darwin's day, a lack of understanding of inheritance meant that the theory of evolution was incomplete. This crucial gap has been filled by over a century of research in genetics. The meeting features leading researchers in evolutionary biology and will illustrate how genetics has contributed to our understanding of the mechanisms of evolutionary change across a wide range of biological systems, from viruses to humans.

The meeting will include sessions on:
Natural Selection in Wild Populations
Natural Selection in Human Populations
Speciation
Experimental evolution
Evolution of Parasites and Hosts
Evolution of Domesticated Animals and Plants
Evolution of Animal and Plant Mating Systems
Evolution of the Genome

Speakers
Spencer Barrett (University of Toronto, Canada)
Nick Barton University of Edinburgh)
Dan Bradley (Trinity College Dublin)
Anthony Brown (CSIRO, Canberra)
Tracey Chapman (University of East Anglia)
Jerry Coyne (University of Chicago, USA)
Laurent Duret (University of Lyon, France)
Steven Frank (University of California, Irvine)
Rosemary Grant (Princeton University)
Hopi Hoekstra (Harvard University, USA)
Ben Kerr (University of Washington, USA)
Anna Di Rienzo (University of Chicago, USA)
Dolph Schluter (University of British Columbia, Canada)
Paul Sharp (University of Edinburgh



7 EXTERNAL MEETINGS DIARY

We will happily include any announcements for genetics-based meetings in this section. Please send any items to the editor. **s.russell@gen.cam.ac.uk**.

2009 Young Physiologists' Symposium
6th – 7th April 2009
University of Sheffield
With the theme '*Physiological Signalling: From Genes to Function*' this meeting aims to attract a multidisciplinary audience and encourage the integration of different approaches on all functional levels in physiological research. The symposium will provide an excellent opportunity for young scientists from undergraduate to postdoctoral level to present their work and interact with other researchers at the same stage of their scientific career. Talks selected from submitted abstracts will be accompanied by plenary talks from Francis Ashcroft and Mike Hankins. The deadline for registration and abstracts is 1st February 2009.
www.bms.dept.shef.ac.uk/yps/

Gordon Research Conference: Mammalian DNA Repair
8th – 13th February 2009
Ventura, CA, USA
www.grc.org/programs.aspx?year=2009&program=mammdna

American Association for the Advancement of Science Annual Meeting
12th – 16th February 2009
Chicago, IL, USA
www.aaas.org/meetings/

Gordon Research Conference: Quantitative Genetics And Genomics
22nd – 27th February
Galveston, TX, USA
www.grc.org/programs.aspx?year=2009&program=quantgen

Keystone Symposium: Chromatin Dynamics and Higher Order Organisation
25th February – 2nd March 2009
Coeur d'Alene, Idaho, USA
www.keystonesymposia.org/Meetings/ViewMeetings.cfm?MeetingID=1000

Keystone Symposium: Genome Instability and DNA Repair
1st – 6th March 2009
Taos, New Mexico, USA
www.keystonesymposia.org/Meetings/ViewMeetings.cfm?MeetingID=979

Plant Genomes: Genes, Networks & Applications
4th – 7th March 2009
Cold Spring Harbor, USA
http://meetings.cshl.edu/meetings/plants09.shtml

50th Annual Drosophila Research Conference
4th – 8th March
Chicago, IL, USA
www.drosophila-conf.org/2009/ index.shtml

Annual Conference of the Association for General and Applied Microbiology
8th - 11th March 2009
Bochum, Germany
www.vaam2009.de/

Cold Spring Harbor Laboratory/Wellcome Trust conference: Genomic Disorders
9th – 11th March
Hinxton, UK
http://firstcontact.hinxton.wellcome.ac.uk/display_info.asp?id=115

51st Annual Maize Genetics Conference
12th – 15th March 2009
St. Charles, IL, USA
www.maizegdb.org/

The 25th Fungal Genetics Conference
17th – 22nd March
Pacific Grove, CA, USA
www.fgsc.net/25thFGC/FGC25.htm

Systems Biology: Networks
18th – 22nd March 2009
Cold Spring Harbor, USA
http://meetings.cshl.edu/meetings/network09.shtml

Genetics & Genomics of Infectious Diseases Symposium
21st – 24th March 2009
Singapore
www.nature.com/natureconferences/ggid2009/index.html

Synthetic Biology, Systems Biology and Bioinformatics Conference 2009
23rd – 25th March 2009
Cambridge, UK
http://conferences.theiet.org/biosysbio/

The Dynamic Cell
1st – 4th April 2009
Edinburgh, UK
www.jointbscbbs2009.org/

EMBO Conference on Chromatin and Epigenetics
13th - 17th May 2009
Heidelberg, Germany
www-db.embl.de/jss/EmblGroupsOrg/conf_112

European Society for Human Genetics Conference
23rd – 26th May 2009
Vienna, Austria
www.eshg.org/eshg2009/

74th CSHSQB - Evolution: The Molecular Landscape
27th May – 1st June 2009
Cold Spring Harbor, USA
http://meetings.cshl.edu/meetings/symp09.shtml

17th International C. elegans meeting
24th – 28th June 2009
Los Angeles, CA, USA
www.celegans.org/

SEB Annual Main Meeting 2009
28th June - 1st July 2009
Glasgow, United Kingdom
www.sebiology.org/meetings/Glasgow/glasgow.html

20th International Conference on Arabidopsis Research
30th June – 4th July 2009
Edinburgh, UK
http://arabidopsis2009.com/

Gordon Research Conference: Evolutionary & Ecological Functional Genomics
12th – 17th July 2009
Tilton, NH, USA
www.grc.org/programs.aspx?year=2009&program=evoeco

6th European Zebrafish, Genetics and Development Meeting
15th – 19th July 2009
Rome, Italy
http://zfin.org/zf_info/news/mtgs.html#rome

Gordon Research Conference: Human Genetics & Genomics
19th – 24th July 2009
Biddeford, ME, USA
www.grc.org/programs.aspx?year=2009&program=humangen

XXIV International Conference on Yeast Genetics and Molecular Biology
19th – 24th July 2009
Manchester, UK
www.yeastgenetics.org/

Society for Developmental Biology: 68th Annual Meeting
23rd – 27th July 2009
San Francisco, CA, USA
www.sdbonline.org/2009Mtg/Webpage.htm

Gordon Research Conference: Epigenetics
9th – 14th August 2009
Holderness, NH, USA
www.grc.org/programs.aspx?year=2009&program=epigen

European Society for Evolutionary Biology (ESEB) 2009
24th – 29th August 2009
Turin, Italy
www.eseb2009.it/uk/

9 SECTIONAL INTEREST GROUPS

The Genetics Society helps support several sectional interest groups by providing meeting sponsorship. We currently have 8 groups who organise sectional interest meetings with the organizers and dates of any forthcoming meetings are listed below. If you are interested in any of these areas, please contact the relevant organiser. Groups who wish to be considered for sectional interest group status should contact the Treasurer, Josephine Pemberton, in the first instance.

Arabidopsis
Organiser: Ruth Bastow (ruth@arabidopsis.info)
http://garnet.arabidopsis.info/

Archaea group
Organiser: Thorsten Allers
(thorsten.allers@nottingham.ac.uk)

British Yeast Group
Organiser: Alistair Goldman
(a.goldman@sheffield.ac.uk)

C. elegans
Organiser: Stephen Nurrish (s.nurrish@ucl.ac.uk)

Ecological Genetics Group
Organiser: Barbara Jones (b.jones@ccw.gov.uk)

Genetics Society Pombe Club
Organiser: Jacky Hayles (j.hayles@cancer.org.uk)

Mammalian Genetics & Development
Organisers: Elizabeth M. Fisher and Nick Greene
Contact: mgd.workshop@ich.ucl.ac.uk

POP group
Organiser: Deborah Charlesworth
(Deborah.Charlesworth@ed.ac.uk)

Drosophila
Organiser: David Ish-Horowicz
(david.horowicz@cancer.org.uk)
Monthly meetings are organised by:
Joe Bateman (joseph_matthew.bateman@kcl.ac.uk)

The Zebrafish Forum
Organiser: Rachel Ashworth (r.ashworth@ucl.ac.uk), Caroline Brennan (C.H.Brennan@qmul.ac.uk), Corinne Houart (corinne.houart@kcl.ac.uk).
There are meetings at 5:30pm-8.00pm on the first Thursday of every other month. Room G12, New Hunt's House, King's College - London SE1 1UL

Society Grants

The Society has increased the levels of support available for both the Heredity Fieldwork & Training Grants and the Genes & Development Summer Studentships.

Full details on these awards are available on pages 53 and 54.

Announcement

The Genetics Society Annual General Meeting

Friday 8th May 2009, The Royal Society, London

The 2009 Annual General Meeting of the Genetics Society will take place on Friday 8th May 2009, in the context of the Society's Spring Meeting on Genetics and Biotechnology held at the Royal Society, London. The business includes election of new members to the Society and of new members to four positions on the Society's Committee that fall vacant in May 2009.

Lists of new members proposed for election to the Society will be publicised via emails to members and on the web. Nominations for Committee vacancies proposed by the Society will also be publicised at a later date by emails to members and on the Society's website.

IMPORTANT NOTE

The 2009 AGM will allow advance voting by email for those unable to attend in person. Members will be notified by email of the motions to be voted on in this way and of the mechanisms for email voting. To ensure involvement in the AGM by this mechanism, please ensure that the Society has your correct email address. As a check, you should have received an email communication from the Society – sender "Christine Fender" christine.fender@genetics.org.uk - on 15 October 2008 inviting nominations for the Balfour and GS Medal competitions; if you did not receive this message, please contact mail@genetics.org.uk with an email address update.

Provisional AGENDA

1. Minutes of previous General Meeting (Saturday 10th May 2008); matters arising
2. President's Report
3. Honorary Treasurer's Report
4. Honorary Secretary's Report and Business for Transaction
    - (a) Balfour Lecturer 2010
    - (b) Genetics Society Medal 2010
    - (c) Applications for new membership
    - (d) Election of new committee members
5. AOB

The Sir Kenneth Mather Memorial Prize

This is an annual prize of £150 to reward a BSc, MSc or PhD student of any UK University or Research Institution who has shown outstanding performance in the areas of quantitative or population genetics.

Nominations should be made between July 1st and November 1st inclusive of each year through the local Head of Department or School of the nominee. Nominations should consist of no more than one page of A4, setting out the case for the nomination, including relevant comparison with other students where possible. Nominations should be sent to the Head of School, School of Biosciences, The University of Birmingham, Birmingham, B15 2TT, clearly labelled as a nomination for "The Sir Kenneth Mather Memorial Prize".

Nominations will be assessed by a panel of two people with experience in the area of quantitative/population genetics, one from the University of Birmingham and the other nominated by the UK Genetics Society. Decisions will be announced in December each year.

Sectional Interest Groups and One-Day Meetings

Sectional Interest Groups.
The Genetics Society would like to expand its portfolio of sectional interest groups. If your field would benefit from annual or biannual workshops or meetings with sponsorship of up to £2K per meeting, please read on.

Sectional Interest groups should: cover a coherent field of genetical research or activity, be held in the UK or Ireland, be open to all interested parties to attend and be organised by members of the Genetics Society. If you are interested in establishing a sectional interest group please submit a one-page proposal including the research topic, justification of requirement, proposed meeting frequency and proposed budget per meeting to the Honorary Treasurer, j.pemberton@ed.ac.uk

One-Day Meetings.
The Society funds several one-day meetings every year and we welcome suggestions from the membership for meeting topics. Meetings can be focused on any area of genetics. Suggestions, including the names of potential organisers (generally two people) and an indication of the likely range of talks, can be submitted to the Scientific Meetings Secretary a.ward@bath.ac.uk.

Postgraduate Representative

Dear undergraduate and postgraduate students, young scientists.

I hope your studies/research projects are going well. I would like to take this opportunity to remind you of the various funding opportunities you can apply for at the Genetics Society, which can help you along the way. The Society offers a number of Genes and Development summer scholarships for undergraduate students with no eligibility required except that the application has to be put through by a full member of the Society.

Postgraduates and post-docs can apply for various travel grants to assist attending scientific meetings as well as funding to help in organising symposia or conferences in a topic related to genetics. Below I present a short guide for undergraduate students on how to make their first steps on a scientific career. I hope it will be of help for those who would like to get some experience of working in the lab, but don't really know where to start.

Considering a future career science? Don't know where to start? We can help you with a concise guide for undergraduates interested in careers in science.

This guide is aimed at undergraduate and postgraduate students who are keen on science and would like to give a scientific career a try but don't know where to start. I produced this as a result of my personal experience when I was an undergraduate. I was very keen to become a scientist, but I had very little idea where to start since not many people in my family attended university. As an undergraduate, the majority of the teaching was lecture based with very little real one-to-one contact with the academic staff. It seemed almost impossible to imagine how one of your lecturers could become your supervisor. I was lucky, a few members of the academic staff were kind enough to explain to me some of the "machinery" behind taking an undergraduate student out of the lecture theatre and into the lab. Below are some questions I was asking on the way.

Well, here I am, never been in the lab except for a couple of practicals; I want to do science, where do I start?

This is probably the most important question you'll have to ask yourself at this stage, and it really takes a lot of self-reflection to be able to answer it. You have to decide what sort of science you want to do. Is it Neuroscience, Cell Biology, Genetics? They all have positions for good students, but which one interests you? Picking the right subject, even if you can't specify in very great detail what you want to do, will help you identify where you may want to apply. If you are not sure, read the next question.

Not sure, could be genetics, could be molecular biology, could be cell biology, don't know…

Welcome to the club, I also didn't know at first, but if you are either in your first or second year (second and third respectively in Scotland), you have plenty of time. Just pick the one that seems most fun, or if you had a good lecturer who introduced really interesting biology, that's probably a good place to start.

I want to be a molecular geneticist, sounds interesting, but what can I do, it seems like everything has already been discovered.

Very good point, up until the later stages of your undergraduate education you tend to be taught facts rather than theories or hypotheses you

could test during a PhD project. But you don't want to wait all that time. Instead, you can apply for a summer studentship or just work in somebody's lab for free if you are lucky enough to be able to afford to keep yourself. This is the second hard choice you have to make after deciding on a research area: You need to identify your future hero (or villain), a supervisor. Hopefully he or she will provide helpful advice with respect to any postgraduate career you are thinking about.

Sounds good so far, but where do I find a supervisor?

There are many places to look. Some students just talk to their favourite professor after a lecture, others look up some names on the websites of various departments, others look at departmental websites of other universities or even abroad. There are countless possibilities, but these often require quite a bit of time to fully explore. As soon as you know whom you want to work for, things become a lot easier. It is important to remember that group leaders are almost always on the look out for good, ambitious students and they may have some funding available to supply the research expenses necessary for helping train a promising student. Therefore, getting a placement is often a matter of confidence.

What about a project?

A good place to start is a summer project. Whether you work for 8 or more weeks this is a perfect gateway to the scientific world. First chance to be and work among real scientists, realise whether you really want to be a molecular geneticist, and why you cannot microwave food in the lab. Apart from having enough time to talk to some PhD students and post-docs in the building, you can finally find out if your supervisor is easy going or someone you will avoid for the rest of your career. At the same time, there is no real pressure on getting results. Your supervisor will probably build up your sky-high hopes for scientific success to the limits. But you probably won't get a noble prize. In most cases you'll be glad to have a nice graph that will be included in a paper. Still, great job! If you get nothing, there is one thing you really must remember: DO NOT GIVE UP. Just because you didn't get any results does not mean you are not suitable for science. It's only a summer project so don't be afraid to try, and chalk failures down as a learning experience.

Will I get paid?

Being a summer student means you probably will be living away from home and will need some pocket money for lunch. If your parents want you to find a summer work, you can kill two birds with one stone and apply for a summer studentship. In general you will get something in the region of £500 to £700 a month. Not a fortune, but it helps pay your rent, bills and buy some food. Where to apply? There are many organisations that you can apply to once you have your supervisor and potential project agreed. Wellcome Trust, Nuffield Foundation and scientific Societies often offer good deals for students to work in a designated lab for 6-8 weeks. This includes the Genetics Society sponsored Genes and Development summer studentships which can fund a stint in a lab for up to 10 weeks. Application deadlines vary from March to beginning of May, but the earlier you start looking for a project and talking to group leaders is of benefit, since it leaves you plenty of time to apply and search for different options.

Summer studentships are a great opportunity to have your first "go" in the lab. They always look great on your CV if you get external sponsorship and will put you in good standing when applying for PhD studentships afterwards. Good luck.

Some funding sources:

Genetics Society Genes & Development Summer Studentships

www.genetics.org.uk/genetics_society_summer_studentships

Nuffield Foundation Undergraduate Research Bursaries

www.nuffieldfoundation.org/go/grants/nsbur/page_412.html

Biochemical Society Summer Vacation Studentships

www.biochemistry.org/education/vacation.htm

Wellcome Trust Biomedical Vacation Scholarships

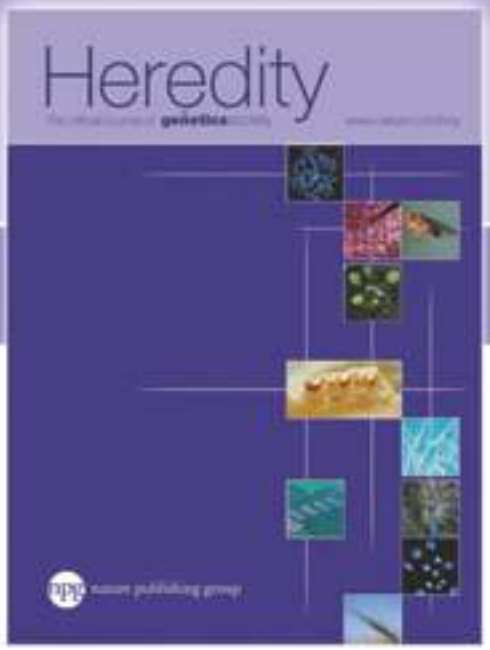
www.wellcome.ac.uk/News/2008/News/WTX052506.htm

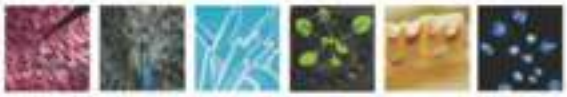
Many Universities have their own funding, for example for those interested in summer work at Cambridge, there is the recently established Amgen Scholars Programme (http://www.biomed.cam.ac.uk/amgenscholars/). Departments that have BBSRC Dotctoral Training Grants may have access to BBSRC Undergraduate Vacation Bursaries. Try a Google search for Summer Research Studentships or such like.

Tom Nowakowski
University of Edinburgh

Heredity

The official journal of the **Genetics** Society





cs, functional genomics and proteomics • population genetics (including human) • biometrical and statistical genetics • e
ics • ecological and evolutionary genetics • animal and plant breeding • cytogenetics • genomics, functional genomics and

Genetics Society members can access *Heredity* free of charge through **www.genetics.org.uk**

A commitment to authors and readers

To ensure the best possible service *Heredity* has recently launched:

- Online Article Submission – fast and easy submission via e-Journal Press
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Further benefits include:

Quality

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Visibility

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- Dedicated and targeted marketing, raising the profile of research published
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BIOSCIENCES FEDERATION INSTITUTE of BIOLOGY

News From The Biosciences Federation www.bsf.ac.uk

A New Voice for UK Biology

John Brookfield . Vice-President (External Affairs)

The United Kingdom has a Royal Society of Chemistry, and if government or others wish to hear what Chemists are thinking, the RSC would be their first port of call. For biologists, the situation is more complex, in that there are currently two groups that speak for biologists. On the one hand, there is the Biosciences Federation, an organisation whose members are the 45 member societies, including the Genetics Society. As Vice President for External Affairs, one part of my post is to liaise with the Biosciences Federation, and to represent the Society when the Biosciences Federation is called upon to express biologists' views on issues such as science and society, training in the biological sciences, and so on. However, in addition to the Biosciences Federation, there exists the Institute of Biology, which has a membership consisting primarily of individuals (although it also has 35 affiliated societies from the UK and overseas, but not including the Genetics Society). Many have felt that this splitting of the voice of biology into two groups, one representing societies and one, primarily, individuals, is inefficient, and that we should move to a more unitary and centralised organisation.

Thus, following a year or so of discussions between the Biosciences Federation and the Institute of Biology, in the first week of December, the Biosciences Federation and the Institute of Biology held votes on enabling motions which would set in train a process to establish a single organisation. So, on the 1st December, I found myself at an Extraordinary General Meeting of the Biosciences Federation, which voted for our enabling motion. Three days later the Institute of Biology passed their equivalent motion.

So now we are moving towards the goal of a single organisation, which will include both society and individual membership. The rather uninspiring working name of the new organisation is NewCo, but a more attractive name will be agreed on during 2009. What is now happening is the establishment of an Interim Council to bring NewCo into being. The members of the Interim Council are:

Mr. Ken Allen FIBiol, Dr Aileen Allsop,
Professor Julia Buckingham,
Professor David Coates FIBiol,
Professor Peter Downes OBE FIBiol,
Professor Anne Glover FRSE, Dr Pat Goodwin FIBiol, Professor Keith Gull FRS,
Professor Martin Humphries,
Ms Liz Lakin FIBiol, Dr William Marshall FIBiol,
Professor Dame Nancy Rothwell FRS FIBiol,
Professor Malcolm Press,
Professor Sir David Read FRS, Dr Lewis Smith.

Once NewCo is up and running, its future will be determined by a new council which will be elected in 2009. For the Genetics Society, we will have to decide whether we wish to continue to be part of the new, fused group, and I hope that we do. Members of the Society who wish to find out more about the plans can obtain information on the Biosciences Foundation and the Institute of Biology's websites, at http://www.bsf.ac.uk/default.htm and http://www.iob.org/ respectively.

You may let me know your views or concerns at john.brookfield@nottingham.ac.uk

These are exciting times for biology in the UK and it could be that, as a community, our power and influence will be considerably enhanced by these developments.

A One-Day Genetics Society Meeting

Evolution Of Sexual And Asexual Reproduction

University of Bath, 5th September 2008

Organised by Laurence Hurst (Bath) and Roger Butlin (Sheffield)

Karel Janko . Institute of animal physiology and genetics . Czech Republic

Once biologists realised that sex is not necessary for reproduction, they started to ask: what is sex for? This question became even more of a paradox for evolutionary biology after Weissmann realised that asexuality has a strong reproductive advantage over sex. All else being equal, a parthenogenetic population should rapidly drive a competing sexual population to extinction since it does not waste reproductive effort on males, which are themselves unable to invest in the next generation. This was known at the end of the 19th century and, despite decades of intensive research and hundreds of scientific publications, we still do not know the detailed answer to the question of why we have sex or why, as a man, I am here at all!

The approaches adopted in the quest for understanding of the evolution of sex are diverse. One important class of studies incorporates purely theoretical efforts to formalise hypotheses explaining the persistence of sex in nature. Currently, we have about twenty mutually non-exclusive and falsifiable theories for the maintenance of sex. One may classify them into those concerning intrinsic factors (such as rates of deleterious mutation or recombination) and extrinsic factors (e.g. the invasion of parasites, role of the changing environment), or into classes assuming long-term versus immediate advantages of sex, or by other criteria. Hypotheses explaining the dominance of sex among Metazoans by the effect of deleterious mutation accumulation in asexuals or by the fast adaptation of parasites to clonal genotypes are generally preferred, although recently-published work considering the role of complex biotic interactions in heterogeneous environments showed that, in some parts of parameter space, sex may win even in the absence of these mechanisms.

Progress in modelling has lead to better definition of the parameter combinations required for sex to persist and so underpins empirical studies. Critical parameters include the per-genome deleterious mutation rate, the fitness effects of mutations as well as the type of epistatic interactions, population structure, virulence of parasites, etc.

Another type of research is devoted to the direct estimation of the parameter values in real organisms, which may then be compared to model predictions. Laboratory experiments are being carried out on organisms with short generation times, such as manipulative experiments aimed at comparing the adaptability of sexual and asexual strains. Some, but much less effort is being invested into empirical research dealing with the role of environmental complexity on the maintenance of sex. Phylogenetic and population genetic tools also provide ways to address the rate at which synonymous and nonsynonymous mutations accumulate in sexual and asexual lineages, as well as estimating the long-term fitness effects of clonality. Finally, a significant research effort is being devoted to seeking new sexual and asexual forms and to describing the evolutionary history of known forms. This may be either via direct observations and manipulation experiments or by determination of phylogenetic relationships of extant asexual and sexual lineages.

The talks at this meeting represented the diversity of approaches currently applied to understanding the evolution and origins of sexual and asexual reproduction with their strengths and limitations.

Thomas Lenormard (Montpellier, France) questioned the role of spatial structure in selecting for or against recombination and hence for or against sex. His conclusions were striking, especially given the traditionally minor role attributed to population structure

hypotheses in the evolution of sex. Contrary to previous beliefs, Lenormard showed that population structure influences the evolution of sex. Environmental heterogeneity causes local adaptation with formation of linkage disequilibria (LD). In such cases, migration of individuals out of their optimal environment may easily overwhelm other sources of LD, increase the frequency of suboptimal genotypes and, therefore, favour recombination even in infinite populations with no epistasis. Local drift further results in loss of polymorphism. Migration tends to restore it and recombination may be favoured by creation of novel allelic combinations. Surprisingly, this effect seems plausible even in weakly structured populations where the number of migrants per generation, Nm > 1. Finally, non-random mating was shown to alter the traditional outcomes from models of panmictic populations.

Peter Keightley (Edinburgh, UK) also adopted a modelling approach to test whether selection against new deleterious mutations explains the persistence of sex.

He first reviewed current data allowing estimation of the per-generation deleterious mutations rates in various organisms, leading to the conclusion that this parameter generally exceeds 0.1 among Metazoans. He subsequently simulated the evolution of a population segregating for up to 100 loci, acquiring both beneficial and deleterious mutations and with a recombination modifier gene. Modifiers increasing the recombination rate were particularly favoured in systems with initially zero recombination. Their advantage increased with increasing population size, because the number of polymorphic genes increases in such cases.

In large populations, drift becomes weak while hitchhiking effects may significantly influence the ratio of effective population size to census population size and recombination may free alleles from their background. This contrasts with previous analytical solutions based on two loci. Selection favouring recombination is surprisingly insensitive to epistasis in these models.

Two further talks dealt with issues surrounding epistasis and its role in the maintenance of sexual reproduction. Ryszard Korona (Krakow, Poland) has conducted an impressive series of experiments in yeast combining hundreds of pairs of deletions and testing for epistasis. Overall, there is little tendency for either positive or negative epistatic effects and this remains true when the tests are conducted in stressful environments. Christina Burch (North Carolina, USA) considered the evolution of robustness in model metabolic networks, whether this generates

The President congratulates the 2008 Balfour Lecturer, Daven Presgraves, University of Rochester.

epistasis among loci determining network components and the consequences of introducing recombination. Because recombination brings different combinations of components together, it selects for both robustness and negative epistasis. Negative epistasis can, in turn, favour recombination.

In his Balfour Lecture, Daven Presgraves (Rochester, New York) focused on the evolutionary consequences of restricted recombination in Drosophila, caused by inversions that are present on segregation distorter (SD) chromosomes. Presgraves studied the pattern of polymorphism at different genes with varying level of linkage to the genes responsible for SD. This enabled him to make inferences about the type and strength of selection at these loci. He found that restricted recombination increases the accumulation of deleterious mutations and reduces the fitness of SD chromosomes, preventing their spread.

Mike Lynch (Indiana, USA) presented work on the

Society President, Brian Charlesworth, presents the 2008 Mendel Medal to Matthew Meselson of Harvard University.

extraordinarily powerful Daphnia waterflea system, where clones repeatedly emerge from sexual ancestors, some of them being quite old. There is evidence that the non-synonymous substitution rate is higher in asexual lineages compared to sexual ones, putatively putting the asexual lineages at increasing risk of extinction as they age. The asexuality clearly has a contagious origin and one of the highlights of the meeting was to see the identification of the gene responsible for this process! The Rec8 gene, whose product is involved in pairing of homologues during meiosis, is disabled in clones by insertion of a transposable element, causing meiosis to stop at the equational division.

Jukka Jokela (Zurich, Switzerland) summarized an unprecedented series of studies on asexual New Zealand snails, which focused on the host-parasite coevolution of castrating trematodes and asexual snails, *Potamopyrgus antipodarum*. During about 20 years of research, Jokela and colleagues collected thousands of snails in New Zealand lakes, comparing the prevalence of parasites among sexual and asexual forms as well as among individual clonal lineages.

They documented repeated frequency cycles with the most frequent clone at a given time being replaced by currently rare ones in the next step of the cycle. The commonest clones usually displayed the highest prevalence of parasites and, most importantly, manipulation experiments suggested that the commonest clones are also the most vulnerable to local parasites (but not to allochthonous ones), while rare clones were resistant. This is exactly the pattern predicted by the Red Queen hypothesis, since one would expect parasites to adapt to the commonest local clone, which, therefore, would appear as the most sensitive while rare clones should mostly be resistant (except for those which were recently common but have declined in frequency).

Stefan Scheu (Darmstadt, Germany) presented surprising insights into the mysterious world of small creatures living in the soil. According to molecular clocks, oribatid mites lost sex almost 300 MY ago and may, therefore, be considered as the oldest known asexual animals. Yet, contrary to expectation for ancient asexuals, they comprise a diverse array of species with worldwide distribution. Phylogenetic data even suggest the possibility of the re-evolution of sex in the oribatid family Crotoniidae. This talk represented a direction of research that is currently under-appreciated: directly testing hypotheses about the role of environmental complexity in the evolution of sex. Using a theoretical model, Scheu predicted that asexuality should dominate in environments with a high risk of extinction and high resource supply but low resource complexity. A manipulation experiment in an Amazonian forest to test these predictions did not produce significant results but should inspire others to tackle such difficult but important work.

Non-marine ostracods are another group well known for possessing many asexual lineages. Dunja Lamatsch (Mondsee, Austria) described extraordinary diversity of both sexual and asexual lineages with a single morphospecies. Asexual lineages have been generated repeatedly both by loss of sex within populations and by hybridisation, leading to triploid clones, but it remains difficult to determine the ages of clones. There is a strong pattern of geographical parthenogenesis associated with an excess of triploid clones in northern Europe, which is surprising given that they can only be generated where sexual and asexual lineages co-exist around the Mediterranean.

The presentation by Matthew Meselson (Harvard, USA), winner of the 2008 Mendel Medal, provided another fascinating page in the long research story of the bdelloid

rotifers. After many fruitful years of research motivated by the question of how bdelloids could survive so long without sex, the answer seems to be here: they incorporate fragments of DNA from the environment into their genomes, which may result in the acquisition of novel evolutionary potential as it does in the case of bacterial transformation.

We learned that bdelloids have an extreme ability to survive desiccation (anhydrobiosis) during which their DNA becomes degraded. With their enormous potential to reconstruct their genomic DNA from such fragments, they may occasionally incorporate novel genes from dead organisms (even non Metazoans) in their environment, particularly near the telomeres. This was documented by experiments on the ability of bdelloids to repair DNA after irradiation and by the identification of non-Metazoan genes in their genomes. As Meselson said, this strategy could be called "necrozoophilia".

Apparently, research into the evolution of sex and asexuality is a dynamic and integrative field of evolutionary biology applying cutting-edge methods from theoretical biology, ecology and molecular biology. For me, however, one important type of analysis remains underexplored. Every model that is used to measure a parameter, or that is tested using estimated parameter values, is just a simplification of reality. We saw during this meeting that predicted thresholds when sex should win over asexuality often do not hold under more complex models. Thus, I believe we also need to ask how our assumptions about the consequences of asexual reproduction actually relate to real clonal organisms found in nature. Do they really suffer higher extinction rates, shorter life-spans and selection against older clones as predicted? Molecular phylogenetics and population genetics made enormous progress in recent years allowing us to estimate parameters such as extinction and speciation rates as well as their temporal shifts, to detect selective sweeps, measure population structure and so on, yet their application to data on asexuals has mostly been restricted to intuitive interpretations based on simple phylogenetic trees. The effects on phylogenies of predicted age-dependent extinction rates have not yet been evaluated.

The meeting also highlighted an important lack of communication between plant and animal biologists concerned with the evolution of sex and asexuality, which is a pity given the progress that plant biologists have made recently, for example in the identification of genes responsible for apomixis.

A Joint Genetics Society and British Society for Human Genetics Meeting

Human Genetic Disease: From Model Organism to the Clinic.

The Royal Society, 28th November 2008

Frances Wiseman . University College London . Institute of Neurology

The Genetics Society Autumn meeting was held jointly with the British Society for Human Genetics and covered the theme of genetic disease in clinical practise and model organisms.

The presentations covered a wide range of diseases from cancer to neurodegeneration, providing a welcome opportunity to hear about cutting-edge research from a variety of fascinating disciplines.

The first talk of the meeting was presented by Wolfgang Driever (University of Freiburg) and discussed the development of the dopaminergic neuronal system in Zebrafish. Many aspects of the dopaminergic circuits of Zebrafish are similar to those in mammals. ENU-induced Zebrafish mutants that have disrupted development of dopaminergic neurons were used to identify genes that are necessary for the formation of dopamingeric circuits in the Zebrafish. The functional importance of these genes was then studied using a range of behavioural tests.

In particular, null mutants of the orthopedia gene mimicked the specific loss of dopaminergic

neurons observed in Restless leg Syndrome in humans.

Like Zebrafish, Drosophila also offers a powerful model system in which to study neurological disease. Juan Botas (Baylor College of Medicine) described several fly models of human neurodegenerative disease and his group's high-throughput genome wide screening strategies to identify modifiers of these diseases. This unbiased approach permits the rapid identification and validation of modifiers, which then can be studied further in vertebrate models. In addition, comparing data from screens for modifiers of different neurodegenerative diseases can be used to identify genes that effect neurodegeneration in general and those that are disease-specific. For example, a comparison of the modifiers of fly models of the CAG repeat disorders, type 1 spinocerebellar ataxia and Huntington's Disease, revealed a number of genes that modify both the diseases, including a number of RNA-binding proteins, suggesting a common mechanism of pathogenesis.

The power of identifying features common to multiple diseases was clearly demonstrated by the presentation of clinical geneticist Han Brunner (Nijmegen Centre for Molecular Life Sciences). Professor Brunner discussed the grouping together of genetic diseases into a "cloud of similar phenotypes" illustrated by his groups work on Stickler Syndrome, and ciliopathies that include some forms Retinitis Pigmentosa and Joubert Syndrome. His group have used this approach to mathematically map similar diseases in the OMIM database, and demonstrate that this map correlates with sequence similarities of the causative genes and known protein-protein interactions.

The theme of ciliopathies was continued by Kathryn Anderson (Sloan-Kettering Institute), who presented her group's work on the importance of cilia in developmental patterning via Sonic hedgehog (Shh) signalling. Most mammalian cells have a single primary cilia, a complex organelle with a unique intraflagellar transport system. Cilia morphology defects have been linked to mutations in components of this specific transport system, for example the wimple mutant lacks primary cilia. This mutant develops early embryonic patterning defects because of the key role cilia play in Sonic hedgehog (Shh) signalling.

Cheryll Tickle (University of Bath) presented the final talk of the morning session. She introduced the chicken as an important model system for vertebrate limb development, in part because of the relative accessibility of the embryo during development. In addition, a number of specific chicken mutants have been identified, such as the talpid3 mutation, which is polydactylous and develops an enlarged wing-bud. Talpid3

Nick Hastie captivated the audience with an animated talk on the roles of WT1.

mutants lack primary cilia on the notochord, which affects Shh signalling despite normal expression of the protein. The work of Professors Tickle and Anderson illustrate the profound role intracellular transport systems play in cellular signalling.

Jenny Morton (University of Cambridge) presented data on the effect of CAG repeat length on the R6/2 mouse model of PolyQ Disease. Doctor Morton introduced a number of cognitive tests for mice developed by her group, including a two-choice discrimination touch screen test for mice (Cambridge Mouse Touch Screen Project, CamToP) a voluntary task that is highly sensitive to cognitive function.

The test was used to investigate the effect of CAG repeat length on cognitive function in the R6/2 mouse model. Unexpectedly, expansion of the CAG repeats to lengths greater than 300 repeats lead to an extension of the mouse models life-span and the formation of extranuclear inclusions, which are also observed in humans with Huntington Disease. However, when disease did occur in the animals with the longer repeat length the clinical presentation was indistinguishable from that observed in the standard 150 CAG repeat model. These data provide mechanistic insight into the impact of somatic CAG repeat expansion on Huntington Disease.

Neal Copeland (Institute of Molecular and Cell Biology, Singapore) described his and Nancy Jenkins' work on the Sleeping Beauty transposable element as a gene-trap cancer model. When this mobile genetic element, which contains a strong promoter sequence, was introduced into the mouse genome a high frequency of haematopoietic cancer developed. These cancers were studied to identify the genes disrupted by the inserted element; a number of genes were more commonly affected. However, no single cancer gate-keeper was observed, rather a gene member of a number of key pathways had to be disrupted to permit the development of cancer.

By restricting the activation of the Sleeping Beauty element to specific tissues this technology was also able to induce gastro-intestinal epithelia cancers. Interestingly, very few of the genes most commonly disrupted in the sleeping beauty induced haematopoietic and gastrointestinal cancers were the same, suggesting that the pathway to cancer may differ greatly between tissues.

The presentation of Nadia Rosenthal, who is the director of the EMBL Monterotondo Laboratory near Rome, also illustrated the power of mouse models in tackling human disease. Professor Rosenthal's presentation focused on the balance between inflammation and regeneration in relation to loss of cardiac muscle after heart attack, illustrated by her IGF-1 mouse model. This work highlighted the importance of the recruitment of the anti-inflammatory M2 type of macrophage to the site of muscle damage mediated by IGF-1. This "healing" cell type accelerates the resolution of inflammation permitting rapid tissue recovery.

Robin Ali (University College London) presented his group's work on the development of treatments for inherited retinal degeneration. By working on the mouse and spontaneous Briard dog model of type 2 Leber Congenital Amaurosis, Professors Ali's group developed somatic gene-therapy for this loss-of-function disease. Their targeted gene-therapy approach restored sight in both animal models, and a small, but functionally significant, improvement in vision occurred in one patient treated in the preliminary Phase I/II trial. Moreover, no immune response to the AAV vector used in this study was detected in any of the treated patients, suggesting this is not only an effective but also a safe form of treatment. This work illustrated the vital role the study of animal models of human disease plays in the development of new clinical practise.

The Genetics Society Medal winner Professor Nicholas Hastie, Director of the MRC Human Genetics Unit, Edinburgh, presented the last talk of the meeting. Professor Hastie talked about the Wilms' tumour gene, WT1. Mutations

President-elect, Veronica van Heyningen, presents the 2008 Genetics Society Medal to Nick Hastie, Director of the MRC Human Genetics Unit, Edinburgh.

in WT1 are found in around 20% of Wilms' tumours, a form of paediatric kidney cancer. WT1 has a well established role in kidney development and functions to control the transition between epithelial and mesenchymal cell fate. WT1 is expressed in many tissues but often in only a limited number of cells. In the heart epicardium, expression of WT1 is required for cardiac mesoderm formation, absence of the gene leads to a thin myocardium and bleeding, suggesting that this gene may have an important role in cardiac repair. Interestingly the effect of WT1 expression on the regulation of down-stream factors, such as Wnt4 and E-cadherin, is cell type specific. Extraordinarily, globally knocking-out WT1 in the adult mouse has a dramatic effect on maintenance of several tissues, suggesting that this developmental/cancer-associated gene has an ongoing essential role in the adult. Professor Hastie's exciting talk was followed by the presentation of the Genetic Society Medal by the President-Elect, Professor Veronica Van Heyningen, in recognition of Nick's great contribution to genetics research throughout his career.

In conclusion, this was an exceptional meeting with a broad range of talks of the highest quality. The topics covered were diverse, spanning the study of model organisms to clinical trials. The meeting provided all attendees with a clear view of the future study of genetic disease and how to translate research from bench to bedside.

2nd Mammalian Genetics, Development and Disease Meeting

4th July 2008, School of Biosciences, Cardiff University

Rosalind John . School of Biosciences, Cardiff University

The 2nd Mammalian Genetics, Development and Disease Meeting, sponsored by The Genetics Society, The Company of Biologists, Cardiff School of Biosciences and Wales Gene Park was held on American Independence Day in Cardiff. On this occasion, we were fortunate to have Professor Lawrence Wilkinson as our Keynote speaker. Professor Wilkinson holds a Cardiff University Link Chair between Medicine and Psychology and is notable for his recent work in the rapidly evolving area of behavioural genetics/epigenetics, with an emphasis on cognition. He spoke about the interaction between genomic imprinting and brain development and function. Imprinted genes, which represent less than 1% of the total number of genes in the mammalian genome, are those that are expressed from only one parental allele. It has been clear for many years that the appropriate expression of these imprinted genes is vital for normal embryonic development. What has become apparent more recently is the role that imprinted genes play in cognition. Professor Wilkinson discussed several neuropsychiatric and neurological disorders that show parent-of-origin effects including attention deficit hyperactivity disorder, autism, bipolar disorder and schizophrenia. He also spoke about the intriguing data from mouse transgenic studies suggesing that genomic imprinting may play a role in the coadaptation between females and their offspring and other adult mammalian-specific behaviours. A future goal will be to ascertain whether these altered behaviours are set during embryogenesis or as a consequence of loss of the adult function of the imprinted gene. A table of imprinted genes and their roles in cognition is available at http://www.bgg.cardiff.ac.uk/imprinted_tables/index.html.

There were several excellent short presentations from early career stage researchers. The imprinting theme was continued with two talks on *Grb10*. Dr Al Garfield (Medicine, Cardiff) discussed a potential role for this gene in controlling social dominance while Mike Cowley (CRM, Bath) described his work defining the molecular mechanisms regulating the gene's intriguing allele-spatio-temporal expression profile. Dr Mike Storm (CRM, Bath) spoke about his work investigating stem cell renewal. Using microarrays, the group were able to identify genes whose expression was altered by an inhibitor of the (PI3K) signalling to further investigate their regulation. Dr James Matthews (Genetics, Cardiff) talk focused on Stat3, a gene with a vital role in maintaining the integrity of the stem cell compartment in the small intestine. Dr Daniel Eberhard (CRM, Bath) described a population of cells in the embryonic bile duct with characteristics of pancreatic endocrine cells that could provide a source of insulin-producing cells for therapeutic transplantation. Dr Ilyas Khan, (CTBL, Cardiff) described a telomere length analysis of aging adult articular progenitor cells cultured in vitro. The role of GITL, a member of the TNF superfamily, and its ligand GITRL in regulating neuronal branching and length was presented by Dr Gerard O'Keeffe (Neuroscience, Cardiff) while Dr Alysia Battersby (Genetics, Cardiff) described an elegant project combining in vitro analysis of targeted ES cells with in vivo functional characterisation of transgenic mice to reveal a role for Fgf15 in modulating insulin signalling. A large scale screen to identify regulators of the Wnt pathway was presented by Jamie Freeman (MCB, Cardiff) which highlighted the importance of synergistic interactions and computational analysis while Dr Araxia Urrutia, a Royal Society Dorothy Hodgkin Research Fellow and L'Oreal UK Women in Science Fellow (Bath University), described her work using a bioinformatics approach to understand more about the functional significance of gene location.

Additional talks focused on using transgenic technologies to understand disease processes. Dr Marcela Votruba (Optometry, Cardiff) presented her work on the autosomal optic atrophies (OPA) making use of the various ENU Mouse Mutagenesis Projects to identify mice carrying mutations in two of the OPA genes which are now undergoing functional phenotyping to assess vision acuity and aging. Cleo Bonnet (Medicine, Cardiff) described the Tuberous Sclerosis models and her PhD work identifying a cell polarity

defect that may underlie renal cystic disease. Dr Emma Rennel (MRL, Bristol) described the potential therapeutic options of an alternatively spliced VEGF mRNA in neovascular eye disease and cancer. Owen Peters was the youngest contributor of the day in the first year of his PhD with Professor Vladimir Buchman (Genetics, Cardiff). Owen described his preliminary work characterising the phenotype linked to over expression of one member of the synuclein family, which induces a progressive loss of motor skills modelling Parkinson's disease.

The meeting elegantly showcased the depth and breadth of expertise in virtually all areas of biomedicine - from basic research to the development of new therapies. A major theme to this meeting was the use of stem cell and transgenic technologies to understand both normal and abnormal development in mammals. These technologies are based on the pioneering work by many individuals and we were delighted that Professor Sir Martin Evans, FRS (Genetics, Cardiff) and his two US-based colleagues, Mario Capecchi and Oliver Smithies, were acknowledged for their contribution to this field of research with 2007 Nobel Prize for Medicine. Prizes of £50 were awarded to Mike Storm, Cleo Bonnett and Al Garfield.

4th London Fly Meeting 19th Sept 2008, King's College London

Özge Özkaya . Leicester University

The 4th London Fly Meeting organised by the London Fly Club with the support of the Genetics Society, took place in the Greenwood Theatre in King's College London. The one-day symposium was packed with a very interesting list of seminars and brought together scientists from the USA as well as Europe and Great Britain. The meeting opened with the Keynote address of Professor Mark Krasnow followed by Professor Andrea Brand who described the work undertaken in her laboratory focusing on the role of Prospero, a transcription factor that acts as a switch between proliferation and differentiation. Dr Barry Dickson gave an overview of Drosophila reproductive behaviour and the role of Sex Peptide in the nervous system to conclude the Neurobiology and Behaviour session.

The second part focused on Intracellular Signalling and featured speakers describing different signalling pathways during development. Dr Arno Müller presented several time-lapse images investigating the role of FGF signalling during cell migration, whereas Professor Jessica Treisman focused on the role of EGF signalling in eye development. Professor Richard Mann explored the role of the Hox protein Ubx in wing versus haltere development.

Finally, the 3rd part of the meeting included three speakers taking an evolutionary approach to development. Professor Michael Akam reviewed segmentation mechanisms in arthropods, focusing on myriapods and chelicerates. He compared the segmentation process in these lineages to that of vertebrates and discussed how the *Drosophila* segmentation process may have evolved. Dr Virginie Orgogozo

Delegates at the fly meeting. Image © Özge Özkaya

on the other hand presented examples of evolutionary changes in 2 *Drosophila* species, *D. pachea* and *D. sechellia*, and addressed how understanding the genetic basis of evolution requires a synthesis between developmental biology and population genetics. The day was concluded with the seminar given by Professor Michael Levine on early development of the *Drosophila* embryo.

Overall this symposium was an excellent opportunity for me to meet with other members of the *Drosophila* research community, including some old colleagues and new acquaintances. I would like to thank the Genetics Society for awarding me a Junior Scientist Travel grant to attend this meeting.

Film of the 1948 International Genetics Congress

A.W.F. Edwards . Gonville & Caius College, Cambridge

A private film of the 1948 International Genetics Congress, Stockholm, has been found and re-edited by Professor Bengt O. Bengtsson, of the University of Lund. Taken by one of the participants, Nils Nybom, it received its first showing on 27th November in Cambridge at the invitation of Professor A.W.F. Edwards with the support of the Sir Ronald Fisher Memorial Trust and the Department of Genetics (to both of whom, thanks).

The film is in black-and-white, without sound, and shows scenes from the pre-Congress demonstrations in plant breeding in the south of Sweden as well as informal gatherings of participants outside the Congress buildings in Stockholm and on the conference tours. There are no interior shots.

There were over one hundred participants from Britain, some of whom, such as R.A. Fisher, J.B.S. Haldane, G. Pontecorvo, C.D. Darlington, K. Mather and Charlotte Auerbach, have attracted name labels in the newly-edited version. Others, such as F. Yates, E.B. Ford and D.J. Finney, have also been identified, but some well-known people still await discovery in the film – for example, D.G. Catcheside, L.L. Cavalli, D.S. Falconer, H. Harris, H. Kalmus, P.B. Medawar, Ursula Mittwoch, R.R. Race, Ruth Sanger, A. Robertson, J.M. Thoday, C.H. Waddington and Mary Lyon. Happily some of these are still with us and may be able to direct us to their youthful images! There are of course many famous names from elsewhere to be seen, including Muller, Dobzhansky, Goldschmidt, Demerec and even the elderly Tschermak. The film offers thirty minutes of great interest, for which Professor Bengtsson is to be warmly congratulated. In due course no doubt the film will be made more widely available, but for the moment any enquiry should be directed to Professor Bengtsson and the Mendelian Society of Lund at Bengt_Olle.Bengtsson@cob.lu.se.

A private film of the 1948 International Genetics Congress, Stockholm, has been found and re-edited by Professor Bengt O. Bengtsson, of the University of Lund.

John Marion Thoday, FRS

1916 – 2008

Donald McDonald and Michael Ashburner, FRS . Department of Genetics, University of Cambridge

Professor John Thoday, FRS, who held the post of Arthur Balfour Professor of Genetics, University of Cambridge from 1959 to 1983 and was President of the Genetical Society from 1975 to 1978, died on August 25, 2008, aged 91.

John Thoday at his desk in Cambridge in the '60s.

John Thoday, one of the most influential geneticists of the second half of the 20th century, was born in Derbyshire, the third son of Professor David Thoday, FRS, and Mary Gladys Thoday. Both his parents were botanists, and when he was 6, his father was appointed to the chair of botany in the University of North Wales in Bangor. Thoday himself went on to read botany at Bangor, graduating in 1939.

During this time he developed an interest in the genetic basis for heritable variation, stimulated by the revolutionary work on plant chromosomes then being done by C.D. Darlington. On graduation, he began a PhD in the Botany School in Cambridge in September 1939 under the supervision of David Catcheside.

1939 was however, hardly the best time to begin a PhD, and his studies were interrupted by war service in aerial photographic intelligence for the RAF, which took him to postings in Cairo and Algiers, as part of the war in the Western desert.

In later years he would relish recounting his experiences of the heat, the dust and the flies, and the chaos and confusion of war, but also of the camaraderie experienced by many of his generation who served in WWII. Demobbed with a commission in 1945, he resumed his academic studies, taking a job as a radiobiologist at Mount Vernon Hospital. This was just after the use of the atomic bomb on Hiroshima and Nagasaki, and the realization that science urgently needed to know more about the effects of radiation on biological tissue.

While there, he made the significant discovery that X-rays break and damage chromosomes more effectively in the presence of oxygen, a finding which was later to lead to a much better understanding

John made the significant discovery that X-rays break and damage chromosomes more effectively in the presence of oxygen, a finding which was later to lead to a much better understanding of the role of radiation in inducing mutation and genetic variation.

of the role of radiation in inducing mutation and genetic variation.

His next move was to a lectureship in the University of Sheffield, again in the department of Botany, where over the next decade he not only set up a Department of Genetics, but also began the research into the basis and evolutionary significance of differences between individuals which established his reputation. In 1953 he published an important paper on the genetic components of fitness, and initiated a research programme using *Drosophila*, which was aimed at locating those genetic factors that contributed to the continuous variation seen in characters like height or weight or crop yields. Research by Thoday and his students in the decade from the mid-1950s overturned the then widely held view that such characters could not be studied using conventional genetic methods. Working with such seemingly esoteric characters as fly bristle numbers, Thoday and colleagues developed genetic methods to locate such "polygenes", as they were termed, on the genetic map. Despite their esoteric nature, the principles and concepts developed in this work were seminal to developments, twenty and thirty years later, in human genetics, and are now fundamental to our understanding of the genetic basis of many complex human diseases.

Thoday moved to Cambridge in 1959, to the Arthur Balfour professorship with the task of developing the study of genetics in Cambridge. A Professorship of Genetics had been founded in Cambridge in 1912, but the first incumbent, Punnett, appears to have concentrated largely on research and tennis, and been less interested in the teaching of students. Punnett's successor, R.A. Fisher, although hugely distinguished as a statistician and geneticist, was also focused on research rather than teaching undergraduates. As a result the department in 1960 was small and made little contribution to undergraduate teaching: Thoday's brief was to build up the department and its role in the teaching of biology. His appointment came at an opportune time. Change was in the air, in particular a feeling that there was a need for reform in the teaching of biology. Thoday, with his experience of jointly teaching botanists and zoologists in Sheffield, immediately threw his energies into these reforms. Despite opposition from many of his academic colleagues to his championship of what to them was a new and upstart scientific discipline, he was brilliantly successful at this. For the first time the teaching of genetics took its rightful place alongside the established subjects of zoology, botany and biochemistry in the first two years of the Cambridge Natural Sciences Tripos.

Thoday served as president of the Genetical Society (as it was then called) from 1975 to 1978. By the time he retired from his chair in 1983, he had the satisfaction of presiding over a thriving research department, which was also a core contributor to the teaching of biology in Cambridge. He played a significant part both in University administration, chairing major university committees involved with resource allocation, and as a fellow of Emmanuel College, where he is still remembered as a lively member of the high table and as a keen bowls player.

Perhaps John Thoday's biggest contribution was in highlighting the critical importance of genetic differences between individuals

in understanding how biology and evolution actually operate. From the 1960s onwards until fairly recently, geneticists tended to divide into two camps. In one camp were molecular biologists, who wanted to understand the molecules that drove heredity, the structure of DNA and how the information in DNA was expressed in cells and organisms. In the other camp were population biologists who were primarily interested in genetic differences and how these drove the processes of evolution and natural selection. John Thoday's approach to the teaching of genetics reflected his breadth of interest in the subject and he maintained that although a student might chose to specialise in either the molecular aspects or the whole organism aspects of the subject, they should always be able to understand what the other camp were talking about. He established a strong tradition that the department, unlike so many others in the UK, should provide a very broad education in genetics, from molecular biology to population genetics, from cytology to human genetics.

The peak of his research output occurred at a time of great productivity in genetics. It was a period of great innovation in the technology and methods applied to research in genetics and allied disciplines. Thoday and a few others stood out because they were not merely brilliant experimentalists but also philosophers of the subject. He, more than most of his contemporaries, was able to fit new discoveries in genetics into their broader biological contexts. In particular, he was deeply concerned with the genetics of human society and with the genetic consequences both of the stratification of society and of its corollary, social mobility. In the classic nature versus nurture debate, his understanding of the complex interactions between genetics and environment made him despair equally of genetic determinism and of those who contended that environment lay at the root of society's problems. Few at that time took such a broad and holistic view of genetics.

In 2004, John opened the newly refurbished *Drosophila* lab in the Cambridge Genetics Department, which is named the John Thoday Laboratory in his honour. Here, John is pictured chatting to Michael Ashburner.

John Thoday had a penetrating and incisive intelligence, and relished the cut and thrust of debate, whether about science or politics or indeed any other topic. He described himself as an old fashioned liberal, who believed in democracy despite its defects, and he had a lifelong contempt of and scepticism for dogmatic views, whether scientific or political. He liked to challenge his colleagues and students, delighted in making provocative remarks to stimulate a thorough-going argument, and he had a fund of stories, some remarkably scurrilous, about the public and private lives of some of the major figures in 20th century genetics.

As Professor and head of department, he toured the department regularly, talking to students and staff at the bench to find out what they were doing. At a time when many University professors were aloof and distant figures in their departments, he could regularly be found in the bar of the local pub playing darts (and competing aggressively) with his staff and research students. A cheerful, ebullient and convivial man, he left an indelible mark on genetics and on the teaching of biology in the University of Cambridge, and will always be remembered with affection and respect by his colleagues and students.

John Hilton Edwards, FRS

1928 - 2007

Prof. Malcolm Ferguson-Smith, FRS . Department of Veterinary Medicine, University of Cambridge

John Edwards was born in 1928, the elder son of a distinguished London surgeon. Disruption caused by World War II may have interfered with his early schooling before he went to Uppingham in 1942. He left there with a distinction in Physics at HSC level and a passion for gliding obtained during his time in the OTC Air Squadron. The Gliding Club featured prominently during his three years preclinical course at Trinity Hall, Cambridge and this, with some uninspiring lecture courses, he regarded as the probable reasons for graduating with a IIIrd in the Natural Science Tripos. However, he greatly enjoyed his time in the Zoology Department and this influenced his later career. He went on to clinical studies at the Middlesex and Central Middlesex Hospitals, during which he joined the Territorials, and graduated in medicine in 1952. Instead of moving on to pre-registration House jobs, he joined as medical officer with an interest in Zoology, on board the Research Ship "John Biscoe" of the Falklands Islands Dependencies Survey and spent nine months in Port Stanley and the South Atlantic. On his return he sought a Junior House Officer post and married Felicity Toussaint, a fellow medical student at the Middlesex. His first hospital post was in Neurology (with Douglas McAlpine) at the Middlesex, but a routine medical examination for National Service in the Army revealed a tuberculous lesion in one lung. Following six months' treatment, employed partly in reading up on statistical methods, he took a second house job in gastroenterology (with Avery Jones) and then six months as a Senior House Officer in Psychiatry at Knowle County Asylum. He became interested in brain pathology and this led to an SHO job in Pathology at the Central Middlesex. In 1956, using his statistical knowledge, he successfully applied for a Lectureship in Epidemiology at Birmingham University to work with Thomas McKeown and Lancelot Hogben in the Department of Social Medicine. He was greatly influenced by Hogben and learned to apply statistics to information in the malformation register, namely to the epidemiology of dislocation of the hip and of neural tube defects. John pursued his clinical interests by going on ward rounds at the Children's Hospital once a week and this led him to an interest in genetics. McKeown suggested that he join the new MRC Unit in Population Genetics at Oxford directed by Alan Stevenson, an expert in congenital malformations. John was at the Unit from 1958 to 1960, but kept regular contact with the Children's Hospital in Birmingham. He resigned from the MRC Unit in 1960 to take a year's sabbatical at the Children's Hospital in Philadelphia where he consolidated his cytogenetics interests, interacting with Peter Nowell and David Hungerford. In 1961 he returned to the vacant Lectureship at the Department of Social Medicine, this time with a half-time connection with Douglas Hubble's Department at the Nuffield Institute of Child Health. John set up there a small cytogenetics laboratory to study Down syndrome and to provide a chromosome diagnostic service. Promotion to Senior Lecturer followed in

In 1979 John was offered and accepted the Professorship of Human Genetics at Oxford University following Walter Bodmer's resignation.

1965, and to Reader in Human Genetics in 1966. At this point he took a year's sabbatical at Cornell Medical Centre and the New York Blood Centre with James German. On his return he was awarded a personal Professorship in Human Genetics and moved the cytogenetics service and genetics clinic to the Women's Hospital. John was elected to the Royal Society in 1979 for "contributions to human cytogenetics and genetic epidemiology including elucidation of the threshold model for multifactorial traits and pedigree linkage analysis". In the same year he was offered and accepted the Professorship of Human Genetics at Oxford University following Walter Bodmer's resignation to become Director of Research at the ICRF in London. He retired from his Oxford appointments in 1995. John died of prostate cancer on 11th October, 2007. He is survived by Felicity and his four children.

At the time John Edwards entered academic medicine as Lecturer in 1956, genetics played virtually no part in the practice of medicine. The molecular structure of DNA had been solved only three years earlier, and the discovery that humans had 46 chromosomes and not 48 was made the same year that John was appointed Lecturer. Today, genetics is at the heart of medicine and DNA is the basis of diagnostic pathology. John was one of the pioneers that helped to make this remarkable transformation possible. The late Jerome Lejeune was another pioneer, and his contribution was to discover in 1959 the extra chromosome 21 responsible for Down syndrome. One year later John, working with David Harnden, discovered the next chromosome disorder due to an extra chromosome 18. This condition became known as Edwards Syndrome. The discovery was no chance observation. It was due to sound clinical intuition. He appreciated that the pattern of multiple minor malformations and mental handicap in Down syndrome could be the clues that could lead to other chromosomal syndromes. Other less astute clinicians were looking at the chromosomes of Mendelian disorders and major malformations and finding nothing. Chromosome analysis in those days depended on bone marrow or testis samples, both requiring invasive and traumatic procedures. John developed a "painless" skin biopsy method instead, which involved pinching a tiny fold of skin with forceps and slicing the exposed part with a sharp scalpel blade. He practised this on himself, producing multiple tiny scars on his knees. The method proved completely acceptable to his young patients.

John Edwards in 1996. Image ©Ross Shipman

In the years that followed John continued his interest in chromosome abnormalities and described patients with mosaic trisomy, triploidy and various translocations. He established a small laboratory for this work in the Nuffield Institute of Child Health in Birmingham. In one important study he personally analysed the chromosomes of 128 patients with Down syndrome born to young mothers in order to determine to what extent

He appreciated that the pattern of multiple minor malformations and mental handicap in Down syndrome could be the clues that could lead to other chromosomal syndromes.

inherited translocations and maternal mosaicism contributed to the frequency of the condition. Only one inherited translocation was found and he rightly concluded that routine analysis of affected children caused unnecessary distress and was unwarranted. When prenatal diagnosis was introduced later in 1970, the recurrence of Down syndrome in women with an affected child was found to be less than half a percent. The option of prenatal diagnosis, however, gave couples the reassurance necessary to contemplate further pregnancies. John's laboratory was one of those that were early in the field in providing prenatal diagnosis. Indeed, as early as 1956, he had written to the Lancet drawing attention to its possible use in the diagnosis of foetal genetic disorders.

John's contribution to the development of diagnostic cytogenetics was very important, but he also made outstanding contributions to other aspects of human genetics including linkage mapping, genetic susceptibility to common disease and comparative genomics. He attended all eleven Human Gene Mapping Workshops from 1973 to 1991. These workshops provided the chromosome maps that led eventually to the human genome project and the complete DNA sequence of the human genome in 2001. John's contribution was to provide novel computing methods for assigning and ordering genes onto their specific chromosomes. He was an expert in statistics and mathematical genetics and wrote his own computer programmes. He insisted that his colleagues make their primary data freely available and was an early advocate for openness in human genetics. At the time when few human genes were mapped to chromosomes, John developed the novel idea of exclusion mapping using negative linkage which determined the chromosomal regions that could be excluded by the data.

From the beginning of his career John was interested in how to determine genetic susceptibility to common diseases such as diabetes and heart disease. His aim was to distinguish the effects of single genes of low penetrance from the combined effects of a multiplicity of genes. His ideas are encapsulated in a paper entitled "The simulation of mendelism" published in 1960. This paper is considered one of his best. He returned to this theme on many occasions up to the last year of his life with many critical papers on the proper use of sib-pair analysis, on haplotype mapping, and on various aspects of allelic association. He was an undoubted leader in this complex field.

John's interest in the conservation of linkage groups between species led in the 1980s to a series of papers with Mary Lyon, Tony Searle and other mouse geneticists at Harwell, comparing the chromosomal homologies of mouse and human. He designed a graphical representation of homologies based on comparative mapping that became known as the Oxford grid. The grid revealed in simple form the large blocks of chromosome in which groups of genes are conserved between the two species. With the help of Frank Nicholas, a database has now been created in which the genomes of many species, including farm animals has been created. These grids are very valuable for evolutionary studies, as well as being a demonstration of the extraordinary conservation of chromosome structure within the animal kingdom.

Among clinical geneticists, John was an outstanding diagnostician and his clinical experience led to notable papers on the characterisation of X-linked hydrocephalus due to stenosis of the aqueduct of Sylvius, and on the delineation of the Cornelia De Lange and Peutz-Jeghers syndromes. He ran genetic counselling clinics throughout his professional life and I know from several sources that he had an excellent rapport with his patients. They greatly admired him and appreciated the time and care he devoted to them. He was always extremely helpful to his clinical and scientific staff and was a kind and generous supervisor of graduate students. He had a particular talent for inspiring his students and staff and it is agreed that this was one of his most important legacies of his time as Professor and Head of

Department in Birmingham and, from 1979, in Oxford. Other appointments included Consultant to the University of Iceland from 1967, where he helped to establish record linkage of all Icelandic births from 1840. He was also Visiting Professor of Human Genetics at the Memorial University of Newfoundland from 1977, Consultant in Human Genetics to the World Health Organisation from 1972, and Visiting Professor at the University of Sydney in relation to his work on comparative mapping. John acknowledged how he was greatly influenced in his career by mathematical geneticists including Lancelot Hogben, Lionel Penrose, Cedric Smith and Jim Renwick.

It was always great fun to be with John. His conversation was full of amusing anecdotes and he always had an apt analogy to emphasise a particular point. For example, he suggested that "reduced penetrance allows a mutant gene to advance like a wolf in sheep's clothing". On the decline of breast feeding, and the possible selection against human milk production, he noted "If the breast now influences selection by shape rather than function, this has no long term genetic hazards." On another occasion, when discussing with someone the pros and cons of prenatal diagnosis, he asked "Madam, if you were a kangaroo would you look in the pouch?"

I have collected a few memories of John from those who knew him well. Ian Craig, his colleague in Oxford, remembers "The genius of the man came in thinking laterally, diagonally and all other ways except in a vertical direction. His clinical knowledge and insight, combined with a deep understanding of genetic principles, provided a robust backdrop to our mapping work. My memories are of kindness combined with eccentricity." Oliver Mayo writes "…no one else in my scientific world combined insight, keen humour and capacity to confuse and illuminate simultaneously." Walter Bodmer notes: "He had a fine feel for human genetics, including a historical perspective, and always an original way of looking at problems and presenting them." Eleida Freire Maia, his student in the 1970s, remarks that: "to study under John's supervision was a special gift. He taught ways of solving problems not easily found." Ed Southern comments, "He had a brilliant mind; lesser intellects had difficulty following his reasoning and, in my case, it would often take months for the penny to drop. But what pennies!" Tom Roderick recalls, "He had a marvellous brain that short-circuited so many esoteric concepts with each other. A vivid memory I have of him is his arched back bending over the computer with face close to the keyboard."

"The genius of the man came in thinking laterally, diagonally and all other ways except in a vertical direction. His clinical knowledge and insight, combined with a deep understanding of genetic principles, provided a robust backdrop to our mapping work. My memories are of kindness combined with eccentricity."

I share with Sue Povey, Andrew Read, Dian Donnai and many others, fond memories of those extraordinary annual seminars, organised by John in the last week of January and held in the old Genetics Department Library in the Oxford biochemistry building. A small group of colleagues were invited to contribute on a subject that John felt was ripe for discussion. Those who chose to use 35mm slides competed with inadequate blackout curtains and temperamental projector, while others lounged on ancient sofas and decaying armchairs. But the output was some original and highly productive discussion and we always returned home refreshed with new ideas. This is just one example of John's great ability to make us think constructively and for this, and for many of his other gifts, we will remember him with the greatest affection.

My Favourite Paper(s)

Ian J. Jackson . MRC Human Genetics Unit, Western General Hospital, Edinburgh

Molecular Characterization of the Mouse Agouti Locus (1992)

Bultman et al. *Cell* 71:1195-1204.

Cloning of the mouse agouti gene predicts a secreted protein ubiquitously expressed in mice carrying the lethal yellow mutation (1993)

Miller et al. *Genes and Development* 7:454-467.

These two papers are among my favourites for a number of reasons. They hark back to the days when finding the gene affected by mutations was hard, well before genome sequences made things much easier. The gene they identify, encoded at the mouse agouti locus, had a terrific genetic history. Predictions had been made by a few, based on genetics, about how it might interact with other genes to regulate hair pigmentation, but these predictions had seemed too simple. Once the gene was cloned the simplicity of the interactions was apparent, and the basis of some of the most interesting mutations were revealed. Finally the mechanism of pleiotropism of one of the oldest alleles led into an area of behavioural genetics and saw substantial investments by major drug companies. Pretty much all of this is there in these two papers.

So first, the history: "wild type" mouse hair is coloured by two pigments, black eumelanin at the tip and base and yellow phaeomelanin in the middle, called the agouti pattern. Strictly speaking a true wild type mouse has a pale, phaeomelanic, belly (called white-bellied agouti) but many lab agouti strains have lost the pale ventrum. These patterns are determined by two alleles of the *agouti*, or *A*, gene. Another allele, *nonagouti*, is recessive to agouti, and also dates back to when mice were first kept in the lab. It results in mice being uniformly black (this allele is present in C57BL/6 mice for example). A fourth allele also dates back to the beginning of mouse genetics. This one, *dominant yellow*, or A^{y}, has a dominant effect that makes the hairs uniformly yellow, as the name suggests, but it was also shown by Cuenot in 1905 to be homozygous embryonic lethal. This was the first embryonic lethal mutation described in the mouse. A^{y} had added interest to mouse biologists as mice with the mutation became obese, and had an increased risk of cancer. Through the 90 years leading up to these papers many more alleles of *agouti* were identified and they could be arranged in a dominance series, in which any yellowing properties were dominant to those that increased black pigment.

Much later a second locus was characterised; the "*extension*" locus at which recessive mutations, when homozygous, extended the yellow pigment portion so that it covered the whole hair. Dominant alleles of this gene had the opposite effect of reducing the yellow band and darkening the hair. So there were two loci, both affecting the balance of black to yellow pigment, but recessive mutations at one had the phenotype of dominant mutations at the other, and vice versa. Surely the gene products had to interact? Furthermore, grafting experiments showed that the *extension* gene acted in the melanocyte whilst the *agouti*

gene acted in the surrounding skin: a clear indication of a receptor and a ligand. Making that leap, however, was harder than you might think as the receptor that was the candidate for regulating the yellow/black pigment switch gave black pigment when stimulated, the opposite action of that predicted for *agouti*.

This candidate receptor, the melanocyte-stimulating hormone (MSH) receptor, now called MC1R, was cloned earlier in 1992, and sure enough it was the product of the *extension* locus. Recessive yellow was a loss of function mutation and the dominant darkening alleles were missense mutations which increased activity, or gave MSH-independent activity (1). With part of the system pinned down, now it was possible to postulate a 3-component system responsible for normal hair colour; MC1R, its agonist MSH and an inhibitor of signalling, perhaps encoded by *agouti*.

Both these papers started from the same point; a fragment of DNA cloned by Rick Woychik's group at Oak Ridge National Labs, from a chromosomal inversion which gave a *nonagouti* phenotype and thus probably disrupted the *agouti* gene (2). Both groups, Woychik's and Greg Barsh's at Stanford used this probe to identify an mRNA in the region. Sequencing showed that it encoded a small protein that was likely to be secreted, and Northern blots showed it was expressed exclusively in the skin. Bultman et al showed that it was still present in skin which lacked melanocytes, as the grafting experiments said *agouti* should be. Both groups found that the gene was not expressed at the beginning or the end of hair growth, but only in the middle when the band of yellow pigment was made. Bingo! Exactly the characteristics of a secreted protein which directed melanocytes to make yellow pigment. This observation alone, showing transcription tightly coupled the hair growth cycle, would make these papers among my favourites.

Both went on to look at the gross structure and expression of the gene in the *agouti* alleles. It fit the bill perfectly. White bellied *agouti* mice expressed the gene on their ventrum all the time, whilst *agouti* animals showed the pulse of expression during hair growth. Another allele, *black and tan*, showed constitutive expression in the tan belly but no expression on the black back. Likewise no expression could be seen in the skin of black, *nonagouti*, mice. Southern blotting detected what looked like DNA insertions 5' of the coding region in *black and tan* and in *nonagouti* DNA. DNA rearrangements were also seen in *dominant yellow* mice; the result of which was overexpression of the *agouti* mRNA. The mRNA from the Ay allele was not only seen in skin but also in all tissues examined. The jump to the basis of the pleiotropic obesity seen with this mutation was not difficult to make; it must be acting on another protein, probably a receptor which regulated body weight.

That was as far as these papers went, but the findings effectively set up a lot of further work. As predicted, the *agouti* gene product, ASP, was later shown to bind to MC1R and to inhibit its activity (3,4). The structural basis for this is still far from being understood. The gene has two promoters: one is active throughout the whole growth of the hair, but is only expressed in ventral skin. This promoter has been lost in *agouti* mice. The other promoter is expressed in skin everywhere, but is synchronised with hair growth so that it is only activated during a narrow time window (5). What DNA elements and transcription factors regulate the spatial and temporal activity of the promoters is still unknown.

The DNA insertion in *nonagouti* mice reported in these papers essentially inactivates both promoters (except, strangely, in the skin behind the ears and around the genitals). The insertion is actually a double integration of a retrotransposon into a second retrotransposon. Reversions of the *nonagouti* mutation are relatively common through loss of the retrotransposons by recombination of the LTRs. If the outer element recombines,

then both retrotransposons are removed and the gene reverts to the wild type, white bellied agouti form. If the inner element only is lost, leaving one element behind, the mouse becomes *black-and-tan*; the repression of the ventral promoter is removed, but the hair-cycle specific promoter remains blocked so the mouse has a black back and a tan belly (6).

The upregulation and ectopic activation of *agouti* transcription in A^{y} is due to a chromosomal deletion which removes the *agouti* promoter and puts its coding exons downstream of the promoter and 5' noncoding exons of a ubiquitously expressed gene, *Raly*. The homozygous embryonic lethality of A^{y} is due to the absence of the coding region of *Raly* and another gene, lying between *Raly* and *agouti*, *Eif2s2* (7). Interestingly, numerous other dominant yellow agouti alleles have been characterised, but none of them are recessive lethal. Almost all of these alleles turn out to be caused by the integration of a transposable IAP element upstream of *agouti*, and promoter activity from the IAP drives ectopic and unregulated expression of *agouti* and hence the yellow coat (8,9). All the IAP-induced dominant yellows have variegation of expression and have dark patches of fur where the *agouti* gene is not expressed. This is associated with methylation of the IAP element, and inactivation of its promoter. Interestingly the degree of methylation and promoter inactivation is epigenetically inherited, so that mothers with large patches of dark fur tend to give rise to progeny also with large patches (9,10).

Finally, the effect of ectopic *agouti* expression on obesity has spawned a large research enterprise. The notion that *Agouti* protein was acting on another receptor and causing obesity was confirmed. The related receptor, MC4R, is expressed in the brain, and Agouti protein will prevent its activation (11). Knockout mice for MC4R have an obese phenotype, as do humans with mutations in the same gene (12,13). *Agouti* is not normally expressed in the brain, but a related gene, *AGRP*, is, and it is part of a complex set of signalling molecules that regulate feeding behaviour and weight homeostasis (14).

Agouti coat colours, from Jackson Laboratory website. http://www.informatics.jax.org/greenbook/figures/figure21-1.shtml

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A Taxi Driver Writes...

Darwin Big Idea Big Exhibition

Josephine Pemberton

What is it with the modern museum exhibition designers? How come they can't let visitors absorb facts at their own pace from quiet contemplation of exhibits laid before them? These were my thoughts as I left the Darwin Big Idea Exhibition after my first visit to the Natural History Museum for about 20 years.

The exhibition follows Darwin's life, achievements and legacy in marvellous detail with many choice and wonderful exhibits. The exhibition is a joint one, organised by no less than five great museums: the American Museum of Natural History, New York; the Museum of Science, Boston; the Field Museum, Chicago; the Royal Ontario Museum, Toronto; and the Natural History Museum, London.

The exhibition has been to all of these other venues before London, and this, perhaps, provides a clue to some of the things I found so distracting about it.

For £9 one enters a large, high-ceilinged gallery where one is instantly and simultaneously assailed by noise and plunged into darkness.

Inside this single room are two small open plan cinemas showing movies with loud commentaries, one loudspeaker commentating on a specific part of the exhibit, one TV screen with biologists commenting on 'what is a theory?', one screen that talks if anyone presses buttons on it and several speakers playing bird sounds (presumably from South America or the Galapagos). Oh, and there was also the guy on his mobile phone even though the ticket asks for these to be switched off. With all this aural assault,

Darwin may have been confused by some of the exhibits used to illustrate evolution!

I found it next to impossible to concentrate on the text of the notices I was trying to read. Perhaps I have got too precious on the matter, but I have got used to reading in the quiet, and now I cannot do it in the noise.

The low lighting level was a further distraction. There is something really weird about having sounds and exhibits trying to create the atmosphere of a sun-baked tropical shore while one is actually in the semi-darkness. I understand that precious exhibits need low light, but is *this* low really needed on a couple of giant tortoises? Couldn't there have been a *bit* more light on the vermilion flycatcher, the subject of a specific notice, but so dim up in its tree that it could have been anything red including an escaped Christmas robin. Some of the notices were so dark it was a real strain to read them.

'What is a theory?' is a continually playing video screen that appears partly motivated by the need to combat the Intelligent Design movement. One could question whether we need this in the UK, but if we do, while it's nice to have North American biologists acknowledging our hero, could we not have had some Brits too? Of the five speakers on the loop (which can be viewed at http://www.amnh.org/exhibitions/darwin/evolution/theory.php) I only recognised Francis Collins and Niles Eldrige; I regret that I had no knowledge of who Kenneth Miller, Georgia Dunston or Eugenie Scott were until I looked them up on the internet. The average British visitor will surely be even more in the dark.

Was there really no room for any British evolutionary biologists, including perhaps some of our fine selection of Genetics Society presidents and maybe some of those that might be recognised by the public, such as Richard Dawkins or Steve Jones, when the show reached the UK?

And don't get me started on the Ladybug Game. Oops! I've got myself started. Ladybugs are what the Americans call Ladybirds, and as we all know they are not bugs but beetles i.e. amongst Darwin's favourite taxa. In the Ladybug Game the cartoon beetles have a green-orange colour polymorphism and the background foliage on which they live can be manipulated by the user between green and the rather improbable orange. During the game, birds appear on the screen and eat the beetle morphs in frequencies that vary with their crypsis against the background. New beetles appear, presumably in morph frequencies determined by the survival of the previous generation. In this way one can demonstrate the principle of natural selection to oneself. Lovely, but given Darwin's proclivities for facts, is it not shocking the exhibition should choose ladybirds for this game? Ladybirds are distasteful aposematically-coloured insects that are rarely predated by birds and rarely occur in green (I asked Mike Majerus and he should know).

I am not strong on Darwin history but in my imagination, all those years between the voyage of the Beagle and the publication of the Origin of Species would have contained hour upon hour of quiet contemplation of the facts before him, arising from detailed examination of the many specimens and experiments he conducted. Surely he would have been appalled at the distracting cacophony that is this exhibition?

This is a serious exhibition trying to put over a serious bit of history. Although the Natural History Museum invites school groups to come and see it, I would say this exhibition is not really appropriate for children; it is for interested adults who have paid to get in, and for this, I suggest, the atmosphere of an art gallery rather than the circus would be more appropriate.

'What is a theory?' is a continually playing video screen that appears partly motivated by the need to combat the Intelligent Design movement.

Early house mice (Mus musculus musculus) in the New World?

Eleanor Jones . University of York

House mice were originally native to the northern part of the Indian sub-continent, and have spread to occupy a near-global distribution mostly through transport by humans. Their close association with humans makes them an intriguing species to study, as their phylogeography should reflect the pattern of human movement and colonization that led to their spread. A number of studies have investigated the phylogeography of the house mouse with this in mind, including a recent paper by Searle et al. (2008) that examined house mice throughout the United Kingdom and Ireland, and found a mitochondrial DNA (D-loop) lineage that matched the areas of activity of the Norse Vikings. In my PhD research, I have increased the number of samples available from the British Isles and extended the sampling of house mice to other regions that were heavily influenced by the Norse Vikings, namely Norway, the Faeroe Islands, Iceland and Northern France. My central hypothesis has been that the Norse Vikings carried a specific D-loop lineage of house mice with them as they settled new areas and that these arriving mice were able to establish themselves and persist to the present day. However, the Vikings occupied a kingdom that spread across the Atlantic as far as Greenland and North America, and they certainly carried the house mice with them as far as Greenland as attested by archaeological remains from excavated Viking farmsteads (McGovern et al. 1987). Could they have carried the house mice as far as Newfoundland? I was awarded a Heredity Field Grant, which allowed me to extend my sampling to see whether this was the case.

The Viking site at L'Anse aux Meadows in Newfoundland. © E Jones

Having obtained the necessary permits, in September 2008 I went to Newfoundland to sample mice, starting with a trip to the archaeological site at L'Anse aux Meadows. Suitable house mouse habitats (i.e. barns with livestock and feed-hoppers) are quite thin on the ground in that part of

Newfoundland, but I obtained 50 samples from five different locations in the west of the province. The majority of sites where I found mice were farms although, appropriately enough, an agricultural research station was also fertile territory. There were some unexpected visitors to the Longworth live traps (see picture). I have typed the samples for genetic markers that discriminate between the different *M. musculus* sub-species; morphologically, the mice resemble M. *m. domesticus*, but elsewhere I have found a surprising number of hybrids, so it was important to check. In the case of the Newfoundland mice, they appear to be 'pure' M. *m. domesticus*, the same subspecies as found in the area occupied by Norse Vikings, but also elsewhere in western Europe. I will sequence mitochondrial D-loops from some of the samples, and compare these to existing sequences to see if the mice are older immigrants from Norway, or more recent arrivals from Britain or Canada. If these results are promising, I will screen the samples for a panel of microsatellite markers to get a clearer picture of where the mice arrived from.

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The author with a chipmunk recently evicted from a Longworth trap. © E Jones

Ford, *Maniola* & The Isles of Scilly

David J Hosken . Centre for Ecology & Conservation, School of Biosciences, The University of Exeter.

The great ecological geneticist EB Ford conducted many studies of the meadow brown butterfly, *Maniola jurtina*, a species with a wing spot polymorphism on the underside of the hind wing. Individual females have between 0 and 5 spots, typically with a mode of two (Ford 1975). One of Ford's most striking findings came from work on the Isles of Scilly, an island chain off the Cornish coast. These islands are all found in close proximity to each other, being maximally separated by about 9 miles, and fall into two distinct size classes; small islands of about 40 acres or less and large of about 300 acres or more. In work that spanned 14 years (from 1946-1959) Ford and co-workers found that female spot-distributions on each island were constant, with a couple of exceptions discussed below. However, while the patterns of spottiness on the larger islands were very similar, having more or less identical proportions of 0, 1 & 2 spot females, they differed greatly on the smaller islands, with each small island home to populations that are highly individual in the spot distribution patterns (Ford 1975).

The author with a female *Maniola jurtina* © DJ Hosken

Ford interpreted these facts as evidence for very strong

The view from Tresco on the Isles of Scilly. © DJ Hosken

selection one each island because for the most part the population sizes are too great for these differences to be attributed to drift, migration between islands was thought to be negligible and the constancy of the patterns over time seems inconsistent with either (Ford 1975). Furthermore, ecological disturbance resulted in changing spot frequencies. For example, removal of a cattle herd from the island of Tean resulted in vegetational changes and a new spot distribution pattern that subsequently remained constant, and similar results were seen on White Island after a storm (Ford 1975). All of which led Ford to suggest local selection was the culprit.

More recent work in the 1970's documented spot patterns similar to those reported by Ford and additionally, discontinuities in spot patterns were mirrored by discontinuities in the frequencies of allozyme (esterase) morphs (Handford 1973). However, there has been no comprehensive population genetic study to assess potential gene flow between these populations and no comprehensive survey has been conducted on the islands subsequent to the 1970's to see if patterns of spottiness have remained constant.

In August of this year, I visited the Isles of Scilly with four helpers to resample some of Ford's study sites. We were very lucky as our arrival coincided with the arrival of some hot and sunny weather, and the first things that were noticeable were the enormous numbers of *M. jurtina* present at some of these sites and how close some island were, even though their historical patterns of spottiness were extremely different. (In all honesty, these were probably the second things we noticed – the first being how beautiful the Scillys are). This close proximity of some islands makes it difficult to believe that there isn't substantial migration between some areas, but time will tell. At present we have a particularly good sample from St Mary's and preliminary analysis indicates that the patterns there are as reported by Ford. How other areas compare is not known yet, and with term just commencing, we'll have to wait a bit for these results. The system and site has tremendous potential. Not only are the animals very numerous, the small size of some islands offers the potential to document selection *in situ*. By combining some common garden rearing, mark-release-recapture and population genetics, we ultimately hope to be able to thoroughly test Ford's central thesis, that difference across the islands are due to strong local selection. Additional funding is being looked for to fund some of this, but the funds from the Genetics Society have already been helpful in kick-starting our return to this iconic system.

I would very much like to thank the Genetics Society for their help in funding the fieldtrip, the Scillies Wildlife Trust for their permission to collect on the Scillies and Annette Stucki, Simon Baxter, Tom Tregenza & Nina Wedell for their help with the collections.

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Catching butterflies requires great agility (as displayed by Tom Tregenza) © DJ Hosken

Silverfish, solitary bees and *Strepsiptera*: encounters with peculiar parasites in the Carpathian basin

Dino McMahon . Department of Zoology, University of Oxford

Abandoned warehouses from soviet days greeted us along the meandering, rainy July-night drive through empty streets into Budapest. An inauspicious start to our fortnight's search for an elusive group of parasitic insects quickly faded, however, upon our arrival into town: discussions over hot goulash soup in the company of our well established Hungarian link (whose generous hospitality and broad knowledge as an all-star naturalist were indispensable) overturned our fortunes at once.

The goal here in the centre of the Carpathian basin was simple: to survey and sample the region's "twisted-wing parasites" or strepsipterans (Insecta; Order Strepsiptera) for short. Strepsipterans are by all accounts quite unusual creatures. They are obligate parasites of other insects, including silverfish (Thysanura); cockroaches and mantids (Dictyoptera); crickets and grasshoppers (Orthoptera); bugs (Hemiptera); wasps, ants and bees (Hymenoptera) and flies (Diptera). Excluding a putatively basal lineage, all strepsipteran females are neotenous (retaining larval characteristics). They remain within the host indefinitely and can be viewed simply as egg-making machines. Males, on the other hand, pupate within hosts; developing wings and a usual suite of adult insect characters (such as antennae, mouthparts and compound eyes), leave the host immediately to search for a mate (Kinzelback 1978; Kathirithamby 1989; Kathirithamby 1991). Males deliver sperm to the eggs through an opening in the head (cephalothorax) of the partially extruded female who is still resident in the host. She may subsequently produce many hundreds of thousands of active 1st instar larvae, all of whom are obliged to crawl out through the same opening in order to reach the outside world.

Why were we here? Southeastern Europe is believed to be an important glacial refugium (Willis *et al*.1998; Stewart & Lister 2001), and combined with the avoidance of severe deforestation and industrialization the region has preserved a diverse biotic environment, containing an

Typical Transylvanian habitats. © A. Hayward

abundance of rare taxonomic groups. An opportunity to collect and record some of the more unusual European species of Strepsiptera therefore seemed to present itself. Newly sampled specimens would further our taxonomic understanding of an obscure parasitic insect group and its hosts' distribution, but they would also serve as important additions to a parallel study for my DPhil investigating the molecular phylogenetics of Strepsiptera, with a focus on exploring the timing and origin of a unique insect radiation. Our first stop was Transylvania. From Hungary, we crossed the Romanian border near Szeged to the South East, and entered a zone famous for vampires and gypsies. Although the closest thing to a vampire was a toy bat hanging from the rearview mirror of our hire car, a number of interactions with

Romanian gypsy culture did colour our journey. Spare money earned from illicit trade on the black market has led to the development of a ubiquitous but really quite unusual phenomenon, particularly near border towns, whereby enormous imitation baroque mansions are half-constructed, commonly with garishly reflective aluminium roofs displaying absurdly elaborate eave and gable metal workings, and left as uninhabited public displays of wealth. Half a day's drive through such towns brought us to the foothills of the mountains. Comprising a sizeable portion of the Carpathian range, Transylvania has preserved swathes of pristine subalpine forest; and despite the ceaseless march of progress (Romania's booming economy has led to significant deforestation in the western territory) we were lucky enough to witness some of it. A very wet day permitted a trek into Apuseni mountain reserve (Pádis, central Carpathians), a zone of deep ravines shrouded in deciduous subalpine forest. On a ledge of one of the deepest drops we stumbled across a large *Vipera* nestled below a decaying wooden rampart. After a scramble down a secure path to the base we noted a sharp drop in temperature and the faint sound of rushing water, to the side a large cavern led diagonally downwards. Minutes later we arrived at a deep subterranean river swelled with rain; in the afternoon we were able to find the overland source on the other side of the mountain. After three days of collection in this and nearby areas, we returned to Hungary and headed west of Budapest, towards Lake Balaton for a week's collection in a much warmer and drier habitat located in the western basin. Hot days were spent exploring the area, collecting as many insect groups in as many different locations as possible.

Collecting methods throughout the expedition included black and white light traps, malaise traps (in long term fixed locations), yellow pans, sweep netting and hand collecting (a surprisingly effective method for specific insect targets). For hosts and their resident females and/or immature males active methods were most effective, whereas collection of free flying Strepsiptera (males by default) was more readily achieved using passive traps. In this way, underrepresented genera in the phylogeny were specifically targeted by searching for hosts, although passive methods turned out to be just as useful.

Among the specimens collected, the most intriguing were free flying males caught in malaise traps belonging to the family Halictophagidae (a probable new species); a female found in a solitary andrenid bee, probably pertaining to the family Stylopidae, and a number of individuals belonging to the genus *Xenos* found in individuals from several nests of the paper wasp *Polistes*. All of which will have important contributions to my DPhil's research. These specimens and the links established (including the long term malaise trap material continually being collected in Romania) would not have been possible without help from the Genetics Society, to whom I am very grateful. Thanks must also be extended to Dr Alexander Hayward (University of Oxford) and Zoltan Ács for scientific and logistical assistance throughout the expedition.

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A free-flying strepsipteran male collected in Romania (family Halictophagidae) © A. Hayward

Plant Biology 2008

Joint Annual Meeting of the American Society of Plant Biologists & Sociedad Mexicana De Bioquimica Rama: Bioquimica y Biologia Molecular de Plantas

26th June – 1st July 2008, Merida, Mexico

Armando Bravo Garcia . University of Oxford

With almost 1,000 scientists from 40 different countries, mainly Mexico and the USA, this year's Joint Annual Meeting between the American Society of Plant Biologists and the Mexican Biochemical Society took place in the gorgeous city of Merida, in south-east Mexico. With wonderful weather and hospitable Mexicans, all the presentations were given in a new convention centre in the north of the city. The five-day meeting included talks from a wide range of fields in plant molecular biology, from environmental physiology, tropical agriculture and root biology, to genome evolution, plant systems biology and epigenetics. Almost every field encompassed in plant molecular biology was included, with a fair amount of scientists presenting posters or talks.

The opening symposium included three talks from three outstanding researchers: Samuel Zeeman, Sarah Hake and Luis Herrera-Estrella. The latter, presented a slightly controversial talk about transgenic plants and their release into the field. Each day, one or two sessions with symposium talks were followed by four different mini-symposia that ran simultaneously. Each mini-symposium included four talks about key aspects in their respective subjects.

The only frustration was the fact that so many interesting talks were taking place simultaneously, and so it was impossible to attend all of them. Of particular interest to me were the symposia about photosynthesis and organelle biology, because they related to my work on plastid development. Also, the talks on gene regulation and genome evolution were very interesting, covering different aspects of their respective fields. The variety of systems used by the different research groups was impressive, in particular the fact that researchers were looking beyond model organisms such as Arabidopsis thaliana. Talks were presented on tropical trees, crop species like maize, tomato and rice, and even more exotic species like cassava (Manihot esculenta), giving the audience the opportunity to diversify their knowledge.

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During lunchtime, every day, the poster sessions provided me with the chance to find out about people outside the UK working in the same field as I

am. This allowed me to directly approach them and have interesting conversations about our research, to network with different people and to make contacts for future work prospects (i.e. post-doc positions). Again, the variety of the posters was so wide, that decisions had to be made in order to have enough time to visit the most relevant ones, and, if lucky, to find the person responsible for the research. This showed me that there are more people working in the same field (or a closely related one) than I thought, and allowed me a good exchange of useful information.

In addition, I presented my doctoral work in a poster entitled "Chloroplast development in two distantly related plant species". It is quite comforting to meet scientists interested in one's own work, and also to get feedback on it. This in itself was a highlight for me.

In total, 6 key symposia, 28 mini-symposia and 740 posters divided into 52 categories were presented in Plant Biology 2008 meeting. Additionally, a few small workshops were provided for anyone interested. Two of them, one of which I attended, dealt with career advice. The workshop I went to was entitled "Getting the most out of the Postdoc experience", and was indeed useful. Prominent investigators covering different experiences of research (like academic research, teaching, industry, government and funding bodies) were invited to an informal dinner and talk with students like me. It was very helpful to hear about different ways to do research or be involved in research, especially now that I am close to the end of my PhD and have to make decisions about the future. In summary, the Plant Biology 2008 meeting was very well organized, a great success, and an exciting opportunity for people like me to learn and network within the scientific community. I am very grateful to the Genetics Society for funding my participation in it.

14th European Meeting of PhD Students in Evolutionary Biology,

8th - 13th September, Einsiedeln, Switzerland.

Ruth McCole . Department of Medical & Molecular Genetics, King's College London

The 14th European Meeting of PhD Students in Evolutionary Biology (EMPSEB) took place in the idyllic town of Einsiedeln, Switzerland. Surrounded by rolling hills and mountains, an ancient monastery and perfect lake, the location was breathtaking. Organised by and for PhD students, the meeting of just over 80 participants had a relaxed and intimate feel. Free time was divided between enjoying meals together, swimming in the lake, hiking in the mountains or relaxing and socialising in the venue's large basement. The four full days were also packed with student talks, as every participant had the opportunity to present their work, and the nine guest speakers each provided an hour-long exploration of their current research. Discussion sessions with the invited speakers were also held over beers in the evening, some groups settled on the terrace to watch dusk fall over the mountains and discuss science.

Throughout the conference, the idea of 'cheaters' in a social system was evoked many times. David Queller, of Rice University, Texas, USA, discussed the organisation of the social amoeba *Dictyostelium discoideum*. These single-celled organisms come together upon starvation to form fruiting bodies which produce spores that can disperse to a better location, supported by a stalk in which all the cells will die. It has been possible to identify 'cheater' mutants which when mixed with wildtype *dictyostelium* cells enter the fruiting bodies in higher than normal proportions. In this way, the 'cheaters' are able to benefit from the production of the stalk, without suffering the cost of cell death by being included. One such cheater mutant is the *csaA* gene, which encodes a cell adhesion protein. Before dispersal, amoebae form a 'slug' and move together to a location where they form the fruiting bodies. Cells mutant for *csaA* slide to the back of the slug end up predominantly in the fruiting body. However, under the more difficult conditions of the wild, *csaA* mutants are not as successful, as they cannot aggregate into a slug on soil's uneven surface. This highlights another theme of the meeting – the importance of examining effects of the laboratory environment and how it can differ from the natural world.

Cheaters were also mentioned by Tom Wenseleers of Katholic University Leuven, Belgium. In the context of social insects, it is puzzling why more queens do not develop in hives in an attempt to 'cheat the system' and produce young themselves, instead of becoming sterile workers. Some of this problem is resolved by the complex patterns of genetic relatedness between different members of the hive. However it was found that in many different species, caste is actually enforced by 'policing' i.e. killing of excess queen larvae, either by the queen or by other workers.

Ashleigh Griffin from the University of Edinburgh, UK spoke about cheaters in bacteria in her plenary talk. Bacteria often engage in behaviour that can be described as a 'public good', where each member of the group does something, such as manufacturing a signalling molecule, which benefits the group as a whole. An example of this is in quorum sensing, where bacteria signal their presence to one another so that the group as a whole can tell the population density at any time. In this way, the group is able to decide when enough members are present (group is quorate) for engaging in new behaviours to be worthwhile. Cheaters may not produce the signalling molecules necessary for quorum sensing, but still benefit from the receipt of those signals and the group behaviours they elicit. These ideas have applications for medicine, as Ashleigh's experiments showed that mice infected with quorum sensing deficient bacteria (cheats) are less likely to die from the infection. This is because the cheats can't engage in all the group behaviours that cause bacterial infections to be so effective and virulent. When considering infections which have been present in the host for a long period of time, such as in the lungs of cystic fibrosis sufferers, it may be important to consider the evolution of the bacteria in the situation, including the possible emergence of cheats which lack quorum sensing, which might be treated in different ways.

In a fascinating contribution to the student talks, Peter Meintjes of the University of Auckland, New Zealand, discussed cheaters in an altogether more positive light. His model for the evolution of multi-cellularity begins with a single-celled organism that after cell division does not separate.

In a fascinating contribution to the student talks, Peter Meintjes of the University of Auckland, New Zealand, discussed cheaters in an altogether more positive light. His model for the evolution of multicellularity begins with a single-celled organism that

after cell division does not separate.

Many of these cells could form a group that exists together. However, the group needs to be able to reproduce new groups in different locations, via a primordial germ-line in a manner analogous to multicellular organisms. In his model, cheater cells that arise could benefit from the actions of the group but are not obligate members; they can leave and found new groups, allowing reproduction. Peter is now attempting to use the bacterium *Pseudomonas fluorescens* to evolve such a system of group formation and foundation of new groups in the lab.

Other highlights from the student talks included Rudy Jonker, Wageningen University, Netherlands, with the most amusingly named talk, "How to lose a kid in 10 months". The Barnacle goose, *Branta leucopsis*, exists in several European populations with different migration patterns. Rudy is studying the length of parental care in these different populations, as these geese have a culturally transmitted migration strategy and so are expected to exercise different levels of parental care in the different populations. Rudy has also studied the changes in timing of the start of migration over some decades and how this corresponds with the point at which adult geese abandon their young.

Decided by the organising committee, the prize for the best student talk was awarded to Aniek Ivens of the University of Groningen, Netherlands. Her talk on the evolution of mutualisms between different species explained the theoretical and practical aspects of her project with clarity and style.

Monastery: the Benedictine Einsiedeln Abbey. © R McCole

In all, the 14th EMPSEB was a huge success, due to the tremendous efforts of the organising committee from Switzerland, headed by Ralph Dobler of the University of Basel. Traditionally the organisers for the next year are chosen at the end of the conference and so the 15th EMPSEB conference will be organised by the Dutch participants and held in the Netherlands next year. My thanks to the organisers and participants for making the 14th EMPSEB fascinating and great fun.

20th International Pig Veterinary Society Congress (IPVS),

22nd – 26th June 2008, Durban, South Africa

Craig R G Lewis . The Roslin Institute and R(D)SVS, University of Edinburgh

June of 2008 was a month full of experience for me. Thanks to the Genetics Society I was able to attend the 20th International Pig Veterinary Congress (IPVS) that was, for the first time, hosted on the African continent. The congress was held in the "leading convention centre in Africa" (award winner for the last six years) in sunny Durban on the coast of Indian Ocean and in the heart of the homeland of the Zulu people.

The five-day congress was an outstanding experience. To illustrate the overwhelming nature of the congress I will draw on a few statistics: 2199 registered delegates, 8 keynote lectures, 295 oral presentations and 623 poster presentations. Indeed within my field of Porcine Reproductive and Respiratory Syndrome research there were 83 abstracts to keep me busy.

Of course with so many abstracts and such a high calibre of presentation it would be impossible to mention all of the great talks but stand out speakers did include: Dr. Dale Polson (Boehringer Ingelheim) on PRRS (porcine reproductive and respiratory syndrome) eradication on-farm utilising vaccination and risk assessment software, Prof. Michael Murtaugh (University of Minnesota) who talked about tools for PRRS elimination and recommendations for future vaccine research and Dr. Lucina Galina-Pantoja (PIC) who presented work showing alleles favourable to litter size in sows affected by PRRSV. The new perspectives that all of the speakers shared will doubtlessly be utilised in papers and the overall discussion in my thesis.

The conference scientific committee also allowed me to present my work entitled "Genetic parameters for commercially important traits on a farm infected with porcine reproductive and respiratory syndrome (PRRS) virus: Can we use selection to help solve the PRRS problem?" as an oral presentation. My talk went really well and I was asked many insightful questions both directly after the talk and at tea afterwards.

It goes without saying that at a congress of this size there are many networking opportunities and I made the most of my opportunity to speak with field leading scientists.

Safari Jeep, somewhere inland. © CRG Lewis

After the congress I also had the chance to see some of South Africa. Thanks in part to the junior scientists travel grant I received from the Genetics Society I travelled inland to see a pig processing plant and talk with industry people in South Africa about pig production, diseases, business and breeding, this was a once in a lifetime opportunity I was glad not to miss. I also had the opportunity to go on safari and to see the animals I only usually see in Edinburgh zoo in their natural environment. This was a truly humbling and breathtaking experience. I also realised a childhood dream to see the mighty warthog boar fighting, through the dense African undergrowth. I now return to Roslin rested, wiser, and full of enthusiasm for the remainder of my PhD.

Human Genome Variation Meeting 2008

15th – 17th October 2008, Toronto Canada

Susan Walker . Institute of Genetics, University of Nottingham

From its first meeting of a handful of like-minded individuals gathered to discuss the prospects for studying human variation, the Human Genome Variation meeting has come along way. For its tenth anniversary, this year's meeting saw over 250 delegates from 70 different countries gather at the beautiful setting of the Old Mill on the bank of the Humber River in Toronto.

In recent years, we have witnessed massive developments in the technologies available for studying human variation, which has lead to an explosion in our knowledge of both single nucleotide variants and much larger structural and copy number changes. As these variants are uncovered, we are beginning to appreciate their contributions to phenotypic differences. To accommodate the fast moving pace of the field, the meeting was structured to give ample opportunity for discussion and speakers had been asked not to submit titles to encourage presentation of their most recent findings.

Not surprisingly, given the fantastic power of both array platforms and new sequencing technologies to investigate whole genomes in a single experiment, the majority of talks were concerned with investigations on a genome wide scale. There were sessions focused on investigating the extent to which individual genomes differ, particularly in copy number variants and other structural changes, and others discussing patterns of population differentiation in the variants observed. On numerous occasions were we reminded of the vast number of Genome Wide Association Studies (GWAS) now identifying correlations between variants and phenotypes and recent data was presented from Type II diabetes, height and obesity studies. This prompted significant discussion of challenges we now face, firstly in differentiating true associations from false positive results and secondly on how to discover causal variants. The massive amount of data being generated by both investigative and association studies highlighted the requirement for new databases to store the data in ways which can be used by individuals worldwide. To serve this growing need, multiple fledgling databases were presented: some help catalogue the sheer volume of diversity discovered and others aim to help in establishing links between variants and phenotypes.

Rapid development with GWAS also stimulated extensive debate of a more ethical nature (which continued at an off-site symposium), primarily discussing the prospects for personalised medicine, the importance of not making too much of our findings and losing public trust. There was also deliberation on aspects of data protection and how much data from GWAS should be made publicly available, risking identification of individuals from cohorts.

In addition, there was a keynote presentation from Svante Pääbo. This came in the form of a refreshing digression into the challenges faced in studying Neanderthal Genomics and the progress that has been made in investigating the divergence of the modern human, Neanderthal and Chimpanzee.

Overall, it was a hugely interesting meeting from which the general feeling was of excitement at the immense quantity of data that is being generated and the discoveries being made. The excitement was tempered with some uncertainty as to where and how to take these findings forward. However, there was a hugely unifying sentiment that by sharing information through openly available databases and establishing collaborations throughout the world, these are now issues that need not be tackled by single individuals but by an ever growing community of individuals with common goals.

The 7th Annual Meeting of the Complex Trait Consortium

31st May – 3rd June 2008, Montréal, Canada

Alex Lam . Roslin Institute and University of Edinburgh

After having been stranded at Charles de Gaulle airport because of an engine problem and subsequently stayed overnight at an airport hotel nearby, I arrived at Montreal some 30 hours since I stepped out of my front door in Edinburgh. Rain was pouring down and it was much colder than I expected for Montreal in summertime. In fact it felt just like a normal day in Edinburgh, except I was really quite sleepy. I turned up just in time for the registration at McGill University. My determination to stay awake was rewarded by a warm welcome at the reception and some fruity Quebecois beer.

The Complex Trait Consortium (CTC) was first set up to manage an ambitious project of creating 1000 lines of laboratory mice, collectively called the Collaborative Cross (CC), which will have fine mosaic genomes of eight commonly used mouse strains for genetic mapping. The CTC meets every year to report on breeding progress as well as to provide a platform for scientists who are interested in areas such as quantitative trait loci (QTL) analysis and systems genetics to present their work. The meeting began on Saturday afternoon with an update on CC by Rob Williams of University of Tennessee and Gary Churchill of the Jackson Laboratory. As expected, after a series of brother-sister mating, the lines are becoming more and more inbred. This has some impact on fertility and survival in some of the lines. Differences in behaviour between lines are also becoming apparent, illustrated by video footage. The CTC anticipate that they will be on course to complete the breeding program in two years time.

Non-CC related talks began on Sunday morning, with Philippe Gros of McGill University giving his keynote speech on host-pathogen interactions in malaria infection in mice. The second keynote speaker was Steve Scherer from the Sick Kids Hospital in Toronto. His talk on Tuesday was quite a breath of fresh air for the heavily mouse-centric conference as it covered the latest, most trendy topic in genetics - copy number variants (CNVs), in relation to human studies. Steve began with a

McGill University campus with downtown Montreal in the background. © A Lam.

first-hand account on how prevalent CNVs are in the human genome and the difficulties with current technology in detecting CNVs. In the second half Steve entertained us with some of the success stories he and his colleagues have experienced recently in understanding the genetics of autism. CNVs were shown to be associated with certain defects in mental developments in some cases where previous rounds of screening using single nucleotide polymorphisms (SNPs) alone had failed to account for the clinical observation in case families.

Over the three days, there were many 20 minute talks as well as

As expected, after a series of brother-sister mating, the lines are becoming more and more inbred. This has some impact on fertility and survival in some of the lines.

two excellent poster sessions over a cup of coffee and one or five yummy, but probably rather unhealthy, doughnuts. Luckily, my talk was scheduled in the morning session on Sunday, so I could actually relax and enjoy listening to the talks for the rest of the conference. As I do not work on mice and I am a solely desk-based geneticist, it was a real eye opener to learn about the work by others combining forward and reverse genetics to advance our understanding in the molecular mechanisms underlying complex traits. Although most of the participants were mouse geneticists, there were also some contributions from people working with fruit flies, rats and dogs. Most relevant to my PhD studies were talks on the methodological aspects of gene expression QTL analysis, but some of the talks I found most interesting were those accounts where QTL mapping has been an absolute success. For example, in studying inter-individual variability in pain sensitivity, Jeffrey Mogil's group characterized a coding variant in the gene encoding for the beta 3 subunit of the sodium potassium ion pump as a novel determinant of pain sensitivity. The experiment began with a QTL mapping exercise using an intercross between a resistant and a sensitive inbred mouse strain. Had the QTL experiment not been carried out, they admitted that nobody would have thought about selecting the ion pump as a candidate gene for pain sensitivity. For me as a statistical geneticist, it is particularly pleasing to hear about how results from "number crunching" exercises can lead to hypotheses being tested on the bench in animal models and taken all the way to patients in the clinics.

Most relevant to my PhD studies were talks on the methodological aspects of gene expression QTL analysis, but some of the talks I found most interesting were those accounts where QTL mapping has been an absolute success.

I also very much enjoyed the talk by Inga Murawski from McGill University about Vesico-Ureteric Reflux (VUR), a human congenital urinary tract defect in which urine in the bladder flows back up the ureters into the kidneys, causing kidney damage. In humans the disease shows a high degree of clinical heterogeneity, hence successful replication of disease susceptibility loci from human association studies of VUR has been elusive. Inga, however, has managed to show some promising results in QTL mapping in mice as more direct phenotyping can be carried out on studying the ureteric bud formation as well as the length of the intravesical ureters in inbred crosses. Despite that human genome-wide association studies have been grabbing much of the attention in high impact journals in more recent times, Inga's work demonstrated that the use of animal models remains an indispensible tool in combating multifactorial inherited diseases.

All in all, the meeting was absolutely fantastic. There was a very friendly atmosphere, and plenty of interaction with all the participants, from eminent geneticists to budding young scientists. During the several days I spent in Montreal, I made a lot of new friends and contacts whom I look forward to meeting again in the future. Next year, the conference will be in Manchester, so it should take a lot less than 30 hours to get there. I would like to thank the Genetics Society for the making it possible for me to attend the meeting.

DNA Replication and Genome Integrity

18th – 21st July 2008, Salk Institute for Biological Studies, La Jolla, USA

Michelle Hawkins . The University of Nottingham

The fifth DNA Replication and Genome Integrity meeting took place at the Salk Institute, which overlooks the Pacific Ocean just north of San Diego. Organised jointly by Salk, Caltech and USC, a packed schedule of 64 talks in just over three days meant a huge range of topics could be covered through talks from researchers across three continents.

Rodney Rothstein got things underway with the keynote lecture describing his groups work characterising novel genes that affect rad52 foci formation in budding yeast. He also discussed lineage-specific asymmetric division in budding yeast. Components of the kinetochore and centromere segregate asymmetrically and he speculated that this might be involved with non-random segregation of centromeric sequences leading to establishment of different cell lineages. Perhaps budding yeast will eventually serve as a model organism for stem cell lineage specificity. Later in the conference Thomas Rando presented results in a similar vein which showed that there is asymmetric segregation of sister chromatids in muscle cells. This asymmetry is in accordance with the immortal strand hypothesis proposed by Cairns in 1975, where the differentiated cell inherits newly replicated DNA while the stem cell retains the old DNA strands. It was pleasing to see new data supporting an old controversial theory and there are many more exciting questions to be addressed on this topic.

The first full day saw talks on subjects ranging from replication initiation and forks to origin regulation and cancer therapeutics. Highlights included David MacAlpine and Joyce Hamlin talking about global characterisation and identification of replication origins in *Drosophila* and mammals respectively. Replication origins are a long-standing interest of mine so these talks were fascinating. A three hour poster session enabled more detailed discussion between attendees and the 40 posters presented were varied and complemented many of the talks given over the course of the meeting. I

Highlights included David MacAlpine and Joyce Hamlin talking about global characterisation and identification of replication origins in Drosophila and mammals respectively.

presented a poster describing our work isolating and characterising the replication origins in the archaeon *Haloferax volcanii*. I enjoyed showing what can be done technically in a "non-traditional" model organism and highlighting how our findings show similarities and differences from the eukaryotic origins that were the focus of the conference.

As the topic moved from my primary interest of DNA replication to DNA repair and epigenetics I became less familiar with the content and learnt a lot of acronyms in a short space of time! Replication timing, telomeres and centromere were topics that came up repeatedly and it was a refreshing chance to catch up on the research in these areas. Personally I was pleased to see a talk given by Tomoki Yokochi who was a colleague a few years ago, it was nice to see what my old group has been working on lately. Steve Kowalczykowski's elegant method of visualising recombinational DNA repair at the single-molecule level was also a treat; you can't get bored watching real-time examples of foci formation! The final day focused on genome stability and epigenetics and included the one *E.coli* talk of the meeting by Susan Lovett. She described her group's model for recA-independent template switch fork repair and their search for the key proteins involved.

Overall this conference was well worth attending. The excellent organisation and talks on all aspects of my favourite topic ensured I got a lot out of it. "The awesome power of …insert your system here…genetics" was almost the meeting slogan and exemplified the point that there are many ways to get at the same question. For my part, the "we don't know how this happens" type of comments inspired me. This conference reminded me that there is still so much to do in this field and I look forward to making a contribution. I would like to thank The Genetics Society for partially funding my conference expenses.

The architect Louis Kahn created a welcoming and inspiring environment for scientific research at the Salk Institute.

Auxin 2008 meeting

4th – 9th October 2008, Marrakech, Morocco

Michalis Barkoulas . Department of Plant Sciences, University of Oxford

If you find an international meeting whose main topic coincides with the main interest of your research, this is of course really nice. But if the above meeting takes place somewhere you always wanted to go, then you are really lucky! This is exactly how I felt after reading the official announcement for the auxin 2008 meeting that was organised last October in Morocco.

Morocco is a destination I have always wanted to visit and, in retrospect, all my expectations were fulfilled. The amazing aspect about Morocco is that is so near to Europe, and yet it is so different. Although Marrakech is a very touristic Moroccan city, it maintains a strong local identity, making it relatively easy for the visitor to get a good flavour of the local lifestyle. Picturesque alleyway-type markets, colourful architecture and beautiful weather all compose Marrakech's unique character. The main attraction of the city is the central square, Djemaa el Fna, which is located within the old fortified city and is thought to be the largest square in Africa. In the morning, the square is full of dancers, musicians, story-tellers, snake-charmers and orange juice sellers. By night, the square turns into an open air restaurant with tables and benches set up to accommodate visitors, mostly locals, who want to try traditional Moroccan dishes like couscous and tajines, together with a cup of the traditional mint tea.

But what about the meeting itself? The auxin meetings are organised once every four years and, as the name suggests, they are focused on the current advances in the field of auxin biology. Auxin is a small, non-peptide plant hormone, which takes its name from the Greek word "afxano" meaning "to grow". Although auxin meetings are themed on a single plant hormone, they are not overly specialised. This is mainly because auxin is involved in virtually all aspects of plant development; therefore there is always considerable variety in the talks. This year's meeting was very successful, gathering more than 150 participants who have been at the forefront of the top scientific discoveries in the field. The meeting was conveniently organised with two morning and two evening sessions per day, covering a variety of subjects such as auxin biosynthesis and auxin signalling in relation to plant growth and development. The meeting also benefited from daily poster sessions that allowed sufficient interaction time between poster presenters and all meeting participants.

There were three keynote speakers: Ben Scheres (University of Utrecht, The Netherlands) who also gave an eloquent opening lecture of the meeting, Ning Zheng (University of Washington,

Although Marrakech is a very touristic Moroccan city, it maintains a strong local identity, making it relatively easy for the visitor to get a good flavour of the local lifestyle.

USA) and Thomas Laux (University of Freiburg, Germany) who gave the final seminar just before the departure. Over the last decade, Ben Scheres has been a leader in the root development field and has published a number of outstanding papers on how auxin gradients, together with the *PLETHORA* (*PLT*) transcription factors, regulate the positioning of stem cells in the root meristem. However, to everybody's surprise, in this meeting Ben Scheres presented some data on shoot and not root development. His group has recently discovered that plt loss-of-function mutants also show abnormal positioning of floral organs, putting forward the idea that an auxin-PLT feedback loop may operate not only in root patterning, but also in the shoot architecture. One of the major advances in the auxin field was the recent identification of the F-box protein TRANSPORT INHIBITOR RESPONSE1 (TIR1) as an auxin receptor. Auxin binding to TIR1 results in the proteasome-dependent degradation of its substrates, some of which are known inhibitors of the auxin signalling. To obtain a detailed understanding of the interaction between auxin and its receptor, Ning Zheng and his colleagues obtained X-ray crystal structures of the auxin receptor TIR1 alone and in complex with auxin and substrate polypeptides. Their results excitingly revealed a novel role for the plant hormone in enhancing substrate recruitment by TIRI, and Ning Zheng discussed the implications of these findings on human drug discovery. Finally, Thomas Laux presented the latest data from his group on the role of the WUSHEL related homeobox genes in the early stages of plant embryogenesis and the subsequent formation of the plant body axis.

Marrakech by night; the Djemaa el Fna

Most of the talks were focused on the model plant species *Arabidopsis thaliana*, but there were a few exceptions. For example, Paula McSteen (Penn State University, USA) presented her latest data on auxin and inflorescence development in maize, and Cris Kuhlemeier (University of Bern, Switzerland) presented some new transport-based models for phyllotaxis and vein formation in tomato. In addition, Miltos Tsiantis (University of Oxford, UK) discussed how *Cardamine hirsuta*, a compound leaf relative of *A. thaliana*, can be used to understand the auxin-dependent evolution of leaf shape in crucifer plants, and Luiz Irina Calderon-Villalobos (Indiana University, USA) presented data on the evolution of auxin signalling with experiments performed in moss.

Overall, it was a very interactive meeting with lively, informal discussions following every talk. I am very thankful to the Genetics society for funding my trip and thus allowing me to contribute to what proved to be a week of very exciting and fully enjoyable scientific activity.

Genetics Society one-day meetings

Graduate students may apply for travel costs to attend these meetings. The cheapest form of travel should be used if possible and student railcards used if travel is by train. Airfares will only be refunded in exceptional circumstances. Grants for overnight accommodation are not available. Applications for travel grants should be made using the registration form, before the final deadline for the meeting.

Meetings with Genetics Society Sponsorship

These include the annual *Arabidopsis, C. elegans, S. pombe* and Pop Group meetings. Graduate Students may apply for travel grants to attend these meetings. Applications should be sent to the Genetics Society, at least one month before the meeting. The cheapest form of travel should be used if possible and student railcards used if travel is by train. Airfares will only be refunded in exceptional circumstances.

Genetics Society Travel Grants for Junior Scientists

PhD students and postdocs (within two years of viva) who have been members of the Genetics Society for at least one year may apply for grants of up to £300 to attend conferences in the area of Genetics that are not sponsored by the Genetics Society (Please note a maximum of one grant every three years will be awarded to any junior scientist).

Applications should be submitted by email at least one month before the meeting, to the GenSoc Office (mail@genetics.org.uk) using message subject "TGJS application" and your surname. Applications should include a brief outline of the value of the meeting to the applicant, an outline of any presentation to be made at the meeting and estimated costs. Please ask your supervisor to send a very brief email in support. Recipients of travel grants will be asked to write a short report that may be included in the newsletter.

Heredity **Fieldwork and Training Grants** supporting field-based genetic research and training

Purpose: To provide grants from £1,000 to £1,500 to cover the travel and accommodation costs associated with pursuing a field-based genetic research project or in visiting another laboratory for training (i.e. to learn a new technique). The scheme is not intended to cover the costs of salaries for those engaged in fieldwork or training, or to fund attendance at conferences. The work should include a strong genetical component.

Eligibility: The scheme is open to any member of the Genetics Society who has been a member for at least one year. The research field should be one from which results would typically be suitable for publication in the Society's journal Heredity. Only one application from any research group will be admissible in any one year. Applications should be made using the form available on the Genetics Society's web page. The application form requests a short summary of the research project for which funds are sought. This should explain the role of the proposed field research in the overall project, and indicate how the grant will be used to facilitate the field research. A detailed budget for the fieldwork will be required, as well as an outline of other possible sources of funding. Applications from PhD students or post-docs should be accompanied by a letter (or e-mail) of support from your supervisor or lab head.

Closing date: There is one closing date of 31st January each year. Awards will be announced within two months of the closing date to allow time for fieldwork preparation. At the end of the grant a short report will be requested from the grant holder. This should be in a format that is suitable for publication in the Genetics Society newsletter. A maximum of one grant per individual every three years will be awarded.

Genes and Development summer studentships

supporting field-based genetic research and training

Purpose: To provide financial support for undergraduate students interested in gaining research experience in any area of genetics by carrying out a research project over the long vacation, usually prior to their final year.

Eligibility: Studentships will only be awarded to students who have yet to complete their first degree i.e. those who will still be undergraduates during the long vacation when the studentship is undertaken. There are no restrictions concerning the nationality or membership status of the student, and the student does not have to attend a UK university. A maximum of 40 studentships will be awarded. The studentship will consist of an award of £225 per week for up to 10 weeks to the student plus a grant of up to £750 to cover expenses incurred by the host laboratory. Both elements of cost must be justified. The award will be made to the host institution.

Applications are invited from members of the Genetics Society who have been members on or before the deadline of March 31st, and who run a research group within a University or Research Institute or a commercial research facility. Applications must be for a named student and must include the student's CV together with a reference from their tutor (or equivalent). Undergraduate students are encouraged to seek a sponsor and to develop a project application with the sponsor.

A panel of members of the Genetics Society committee will review applications. Feedback on unsuccessful applications will not be provided. The successful applicants will be required to submit a short report from the students within two months of completion of the project.

Full details and on-line application form are available at the Genetics Society website

Sir Kenneth Mather Memorial Prize

This is an annual prize of £150 to reward a BSc, MSc or PhD student of any UK University or Research Institution who has shown outstanding performance in the areas of quantitative or population genetics.

Nominations should be made between July 1st and November 1st inclusive of each year through the local Head of Department or School of the nominee. Nominations should consist of no more than one page of A4, setting out the case for the nomination, including relevant comparison with other students where possible. Nominations should be sent to the Head of School, School of Biosciences, The University of Birmingham, Birmingham, B15 2TT, clearly labelled as a nomination for "The Sir Kenneth Mather Memorial Prize".

Nominations will be assessed by a panel of two people with experience in the area of quantitative/population genetics, one from the University of Birmingham and the other nominated by the UK Genetics Society. Decisions will be announced in December each year.

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Order Form 2009

Genes & Development

Please return this from to: The Genetics Society, Wallace Building, Roslin BioCentre, Roslin, Midlothian, EH25 9PP

The new personal subscription rate for Genes and Development for 2009 is £128, inclusive of airmail delivery. The subscription runs on a yearly basis from January 1st. The full subscription will be charged and back issues supplied when applications are made after January of each year.

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the Genetics Society

AIMS

The Genetics Society was founded in 1919 and is one of the world's first societies devoted to the study of the mechanisms of inheritance. Famous founder members included William Bateson, JBS Haldane and AW Sutton. Membership is open to anyone with an interest in genetical research or teaching, or in the practical breeding of plants and animals.

MEETINGS

The main annual event of the Society is the Spring Meeting. This has at least one major symposium theme with invited speakers, and a number of contributed papers and/or poster sessions.

One day mini-symposia are held during the year in different regions so that members from different catchment areas and specialist groups within the society can be informed about subjects of topical, local and specialist interest. Like the spring symposia these include papers both from local members and from invited speakers. One of these meetings always takes place in London in November.

YOUNG GENETICISTS' MEETINGS

Currently there are three meetings devoted to talks and posters by students and junior postdocs. Promega UK is sponsoring travel to these meetings and prizes for the best contributions, plus costs for the three winners to attend the following Spring Meeting and national finals.

INVITED LECTURES

The Mendel Lecture, in honour of the founder of modern genetics, is given usually on alternate years at a London Meeting by an internationally distin-guished geneticist.

To encourage younger geneticists, the Balfour Lectureship (Named after our Founder President) recognises the contribution to genetics of an outstanding young investigator, who must normally have less than ten years postdoctoral research experience at the time of the lecture. The winner gives the lecture at the Spring Meeting.

INTERNATIONAL LINKS

The Society has many overseas members and maintains links with genetics societies in other countries through the Inter-national Genetics Federation, the Federation of European Genetics Societies and through the International Union of Microbiological Societies.

PUBLICATIONS

The Society publishes two major international scientific journals: *Heredity*, concerned with cytogenetics, with ecological, evolutionary and bio-metrical genetics and also with plant and animal breeding; and *Genes and Development*, which is jointly owned with Cold Spring Harbor Laboratories and which is concerned with molecular and developmental aspects of genetics.

The Genetics Society was founded in 1919 and is one of the world's first societies devoted to the study of the mechanisms of inheritance.

Full and student members are entitled to reduced subscriptions both to these journals and also to *Genetical Research*, published by Cambridge University Press, to *Trends in Genetics*, a monthly journal published by Elsevier with review articles of topical interest aimed at the general reader, *Nature Genetics*, published by Nature Publishing company (MacMillan Magazines Limited), *Current Biology* journals, *BioEssays* and *Chromosome Research*.

A newsletter is sent out twice a year to inform members about meetings, symposia and other items of interest.

SPECIALIST INTERESTS

Six specialist interest areas are covered by elected Committee Members: Gene Structure, Function and Regulation; Genomics; Cell & Developmental Genetics; Applied and Quantitative Genetics; Evolutionary, Ecological and Population Genetics; Corporate Genetics and Biotechnology. The Committee Members are responsible for ensuring that the various local and national meetings cover all organisms within the broad spectrum of our members' interests.

the geneticssociety

membership form

Membership includes free online subscription to Heredity

Please complete this form and return it, along with your payment to, The Genetics Society, Wallace Building, Roslin BioCentre, Roslin, Midlothian, EH25 9PP. Complete this section carefully. The information you provide will help us to correspond with you efficiently and ensure that your details are accurately held on our membership database.

1. IDENTIFICATION (as data controllers we adhere to the Data Protection Act 1998)

Title: Prof. ☐ Dr. ☐ Mr. ☐ Miss. ☐ Mrs. ☐ Ms. ☐

Last Name:	First Name:
Institution:	
Institution Address:	
Postcode:	Country:
Telephone:	Fax:
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Your home address should only be given when there is no alternative **Please ensure that you have included your email address**

2. AREAS OF INTERESTS (tick as appropriate)

Gene Structure, Function and Regulation	☐	Genomics	☐
Cell and Developmental Genetics	☐	Applied and Quantitative Genetics	☐
Evolutionary, Ecological & Population Genetics	☐	Corporate Genetics and Biotechnology	☐

3. STUDENT MEMBERSHIP (if this section is not applicable please go to section 5)

As a student member of the Society you are eligible to apply for a grant to defray the cost of attendance at meetings organised by the Society. Full details regarding grants is available on registration. In addition, after one year full membership you can apply for a grant of up to £300 for overseas travel to international meetings held outwith the Society.

If you are applying for an undergraduate membership please state year of graduation:

If you are applying for a postgraduate membership please state year of starting research:

Signature of Head of Department/Supervisor

Please note: After four years' postgraduate membership you will be required to pay the full subscription fee.

mail@genetics.org.uk

4. MEMBERSHIP FEES

Membership entitles you to reduced rate entry to meetings, discounts on journals, free Society newsletters plus free online access to *Heredity*. The annual subscription charges are as follows (please tick applicable box):

☐ Full Member: ***£25.00** ☐ Postgraduate Member: ***£15.00** ☐ Undergraduate Member: **£5.00**

* There is a reduction of £5.00 for full and postgraduate members paying by Direct Debit

5. PAYMENT

Option 1: **Direct Debit (UK Bank Accounts only)**

Complete this membership form and send it to the address below. On receipt you will be sent a DIRECT DEBIT MANDATE to complete and return with instructions enclosed.

I wish to pay by direct debit (tick box if applicable) ☐ **Paying by Direct Debit saves Full members and Postgraduates £5**

Direct Debit Membership subscriptions are renewed on an annual basis running from 01 June – 31 May or 01 December - 30 November depending on date of application

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I enclose a cheque for the sum of £ _____ payable to **'The Genetics Society'**

Option 3: **Card Transaction**

Please note that ☐ Visa ☐ MasterCard * (handling charges apply raising membership fees by 3.6%)

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Card No	Start Date:	Expiry Date:	Issue No. (if applicable)

Cardholder Name:	Signature of Cardholder:

6. MEMBERSHIP NOMINATION

Your application for membership of the Genetics Society will not be accepted without the signature of a FULL MEMBER nominating you for membership. In instances were no full member is available you must submit a copy of your CV along with a short Academic Reference. Your application will then be considered by the Committee. Alternatively, you may contact the Society by email for a list of Society Reps in your area.

Signature of nominating FULL MEMBER
(please print name in block capitals after signature _____

I enclose a copy of my CV along with an Academic Reference for consideration by the Committee (Tick box if applicable) ☐

Please return your membership application form along with any attachments to: The Genetics Society, Wallace Building, Roslin BioCentre, Roslin, Midlothian, EH25 9PP marking your envelope MEMBERSHIP APPLICATION.

Please note that the approval of new members is ratified at the Spring Meeting as part of our AGM

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Date Received	Date Processed	Membership No.
DD No.	**Nominal Code**	**Membership Pack Sent**

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