In this issue

- Medal awarded
- Meetings
- Student and Travel Reports

The Genetics Society News is edited by Manuela Marescotti and items for future issues can be sent to the editor, by email to m.marescotti@brainwave-discovery.com. The Newsletter is published twice a year, with copy dates of July and January.
A W O R D  F R O M  T H E  E D I T O R

A word from the editor

Welcome to ISSUE 74.

2016 has just begun and the ideal way to ease into the New Year is to take a break from the bench and to leaf through the new issue of the Genetics Society newsletter with a cup of coffee!

In particular, you will find a very topical interview granted to Kat Arney by Professor Alison Woollard, upon being awarded the JBS Haldane lecture, because of her great commitment and ability to communicate genetics. Professor Woollard and Kat went together through the “genetic revolutions” mentioned by Alison during her JBS Haldane lecture. I am not going to spoil the surprise, but I will say that one of these revolutions, in Professor Woollard’s opinion, is the controversial topic of “genome editing”. In fact, interestingly, this issue is also addressed by another article, authored by a novice scientist, but a promising one. David Walker (the author and a veterinary student) imagines the consequences of the “genetic revolutions” on human evolution and, thus, our future.

Besides these two exciting articles you will find a number of reports written by the scientists supported by the Genetics Society to organise or to attend a genetics-related event. I would like to highlight that these articles are no less interesting than the two mentioned above. In fact, these articles clearly convey the enthusiasm burning in you after a conference, where you had the opportunity to bring together scientists of the same field to network and build up new collaborations that will benefit their research; or, that of a young scientist, having presented their work to a highly specialised audience that gave useful inputs for their study.

To conclude, I think that this enthusiasm is a great fuel and inspiration to go back to work, after the holidays, and plan the next experiments…and, hopefully, the next holidays!

Read on and enjoy.
Best wishes,
Manuela Marescotti

David Walker (the author and a veterinary student) imagines the consequences of the “genetic revolutions” on human evolution and, thus, our future.
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www.genetics.org.uk
We are awash with whole genome sequencing data from normal tissues and cells from a very wide variety of organisms from bacteria to humans. In addition, there are equally large sets of data derived from human clinical samples. We have learnt that sequence variation between individuals may be associated with differences in gene expression which in turn can lead to changes in phenotype and to disease. However, most of this variation is not currently interpretable because, apart from changes affecting the tiny fraction of the genome that codes for proteins, we do not understand the functional significance of most genome variation.

Our challenge is to distinguish functional from non-functional variants, and to understand how they cause changes in phenotype between individuals and throughout evolution. This meeting brings together scientists using genetics, genomics, computational, cell and developmental biology to discuss how to identify functional elements in the non protein-coding portion (99%) of the genome and to determine how they affect gene expression. Such elements include distal regulatory elements driving spatial and temporal gene expression and non-coding RNAs. Speakers at the meeting will be chosen to draw on examples from multiple plant and animal species.

Confirmed Speakers
To be announced.
You can keep up to date by visiting www.genetics.org.uk

Scientific Organisers
Wendy Bickmore, University of Edinburgh, UK
Doug Higgs, University of Oxford, UK
Chris Ponting, University of Edinburgh, UK
Martin Taylor, University of Edinburgh, UK
Richard Flavell, Ceres Inc, USA

Award Speakers
Felicity Jones, Max Planck Institute, Germany (Balfour 2016)
Duncan Odom, Cancer Research UK (Mary Lyon 2016)
Ben Lehner, Center for Genomic Regulation, Spain (Balfour 2015)
We will happily include any announcements for genetics-based meetings in this section. Please send any items to the editor.

**Fundamentals of Clinical Genomics**
13—15 January 2016
Wellcome Genome Campus, Hinxton, Cambridge, UK

**Genomic Practice for Genetic Counsellors**
3—4 February 2016
Wellcome Genome Campus, Hinxton, Cambridge
[https://registration.hinxton.wellcome.ac.uk/events/item.aspx?e=573](https://registration.hinxton.wellcome.ac.uk/events/item.aspx?e=573)

**Mouse Models of Disease**
9—11 February 2016
Wellcome Trust Genome Campus, Hinxton, Cambridge, UK
[https://registration.hinxton.wellcome.ac.uk/events/item.aspx?e=488](https://registration.hinxton.wellcome.ac.uk/events/item.aspx?e=488)

**Translational Bioinformatics Workshop**
22—26 February 2016
Guy’s Hospital, London, SE1 9RT
[www.guysandstthomasevents.co.uk/translational-bioinformatics-workshop-2016/](http://www.guysandstthomasevents.co.uk/translational-bioinformatics-workshop-2016/)

**Evolutionary Systems Biology: From Model Organisms to Human Disease**
2—4 March 2016
Wellcome Genome Campus, Hinxton, Cambridge UK
[https://registration.hinxton.wellcome.ac.uk/events/item.aspx?e=568](https://registration.hinxton.wellcome.ac.uk/events/item.aspx?e=568)

**Single Cell Biology**
8—10 March 2016
Wellcome Genome Campus, Hinxton, Cambridge, UK
[https://registration.hinxton.wellcome.ac.uk/events/item.aspx?e=571](https://registration.hinxton.wellcome.ac.uk/events/item.aspx?e=571)

**Genomics of Rare Disease: Beyond the Exome**
13—15 April 2016
Wellcome Trust Genome Campus, Hinxton, UK
[https://registration.hinxton.wellcome.ac.uk/events/item.aspx?e=575](https://registration.hinxton.wellcome.ac.uk/events/item.aspx?e=575)

**Genomics of Brain Disorders**
25—27 April 2016
Wellcome Genome Campus, Hinxton, Cambridge, UK
[https://registration.hinxton.wellcome.ac.uk/events/item.aspx?e=576](https://registration.hinxton.wellcome.ac.uk/events/item.aspx?e=576)

**NGS 2016 Glasgow Conference: Applications & Data Analysis**
27—28 April 2016
IET Glasgow: Teacher Building, 14 St Enoch Square, Glasgow G1 4DB
[https://biotexcel.com/event/ngs-2016-glasgow/](https://biotexcel.com/event/ngs-2016-glasgow/)

**16th International Xenopus Conference**
28 August—1 September 2016
The Genetics Society helps support several sectional interest groups by providing meeting sponsorship. We currently have 11 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 see the Society website for further details.

**Arabidopsis**  
Organiser: Ruth Bastow  
(ruth@garnetcommunity.org.uk)  
www.garnetcommunity.org.uk

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

**British Yeast Group**  
Organiser: Jane Usher  
(j.usher@exeter.ac.uk)

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

**Ecological Genetics**  
Organiser: Paul Ashton  
(Genetics@BritishEcologicalSociety.org)

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

**London Fly meetings**  
Organisers: Manolis Fanto and Nic Tapon  
(manolis.fanto@kcl.ac.uk) and  
(nic.tapon@cancer.org.uk)

**Mammalian Genetics and Development**  
Organisers: Nick Greene, Andrew Copp,  
Andrew Ward  
(ich.mgdwshop@ucl.ac.uk)

**Mammalian Genes, Development and Disease**  
Organisers: Rosalind M John and David Tosh  
(JohnRM@cf.ac.uk)

**Meiosis group**  
Organisers: Hiro Ohkura  
(h.okhura.ed.ac.uk)

**Population Genetics Group**  
Organiser: Barbara Mable  
(pgg@populationgeneticsgroup.org)

**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
## Honorary Secretary’s Notices

**Tanya Whitfield**. Honorary Secretary, University of Sheffield

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### The Genetics Society Annual General Meeting

The 2016 Annual General Meeting of the Genetics Society will take place during the Society’s Autumn Scientific Meeting in November 2016, as there is no Spring Meeting this year. Details will be announced in the July Newsletter. Business at the AGM will include the announcement of our awards for 2017, and the election of new members to the Society.

Minutes of the April 2015 AGM can be found on the Society’s website. Current Committee members are listed in this Newsletter and can also be found on the Society’s website.

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### Life Membership in the Genetics Society

Have you reached the age of retirement (65), but wish to continue with your involvement in the Society? If so, and you are an ordinary member who has discharged any arrears the might be due to the Society, then you might consider applying to become a Life Member of the Society. Life members will continue to receive notices and remain eligible to vote in the Society AGM, but will not be required to pay further subscriptions. Recipients of the Genetics Society Medal will also be offered Life Membership. Should you require additional information about becoming a Life Member, please contact The Genetics Society Office (theteam@genetics.org.uk).

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### Upcoming committee vacancies

Five Committee posts will be falling vacant as of 1st May 2017:

1. **Newsletter Editor**
2. **Ordinary Committee Member: Area ‘A’**
   (Gene structure, function and regulation)
3. **Ordinary Committee Member: Area ‘B’**
   (Genomics)
4. **Ordinary Committee Member: Area ‘C’**
   (Cell and Developmental Genetics)
5. **Ordinary Committee Member: Area ‘D’**
   (Applied and Quantitative Genetics)

The nomination deadline for these posts is Friday 25th November 2016. All members in good standing are welcome to nominate individuals for these upcoming vacancies from members of the Society. Nominations should be sent to the Honorary Secretary, Tanya Whitfield (t.whitfield@sheffield.ac.uk), and must be made with the nominee’s consent.
Genetics Society Medal

The Genetics Society Medal is an award that recognises outstanding research contributions to genetics. The Medal recipient, who should still be active in research at the time the Medal is awarded, will be elected annually by the Committee on the basis of nominations made by any individual member of the Society. Those making nominations must be members of the Genetics Society, but there is no requirement for the nominee to be a member, nor any restriction on nationality or residence. Neither current members of the Committee nor those who have retired from office in the past four years may be nominated for the award. The recipient will be invited to deliver a lecture at a Genetics Society meeting, where the medal will be awarded.

The 2016 Genetics Society Medal is awarded to Professor Ottoline Leyser (Sainsbury Institute, Cambridge). See the July 2015 Newsletter for a profile of Ottoline’s career. Details of Professor Leyser’s talk will be announced as soon as they are available.

The winner of the 2017 Genetics Society Medal will be announced at the AGM.

Call for Nominations

Nominations are now being invited for the 2018 Genetics Society Medal. To make a nomination, please confirm that your candidate is willing to be nominated, and then forward a two-page CV of the candidate, together with a list of his or her ten most important publications, plus a one-page letter of recommendation outlining why you feel their contributions to the field have been outstanding. Please submit these supporting documents via email to the Honorary Secretary of the Genetics Society, Tanya Whitfield (t.whitfield@sheffield.ac.uk), by Friday, November 25th, 2016.

The Mary Lyon Medal

This award, named after the distinguished geneticist Mary Lyon FRS, has been established to reward outstanding research in genetics to scientists who are in the middle of their research career. The Mary Lyon medal will be awarded annually, and the winner will be invited to present a lecture at one of the Genetics Society scientific meetings.

The 2016 Mary Lyon Medal is awarded to Dr Duncan Odom (CRUK Cambridge Institute). See the July 2015 Newsletter for a profile of Duncan’s career. Dr Odom will present his lecture at the Genetics Society Autumn Meeting, November 2016, at the Royal Society in London.

The winner of the 2017 Mary Lyon Medal will be announced at the AGM.

Call for Nominations

Nominations are now being invited for the 2018 Mary Lyon Medal. To make a nomination, please confirm that your candidate is willing to be nominated, and then forward a two-page CV of the candidate, together with a list of his or her five most important publications, plus a one-page letter of recommendation outlining why you feel their contributions to the field have been outstanding. Please submit these supporting documents via email to the Honorary Secretary of the Genetics Society, Tanya Whitfield (t.whitfield@sheffield.ac.uk), by Friday, November 25th, 2016.
The Balfour Lecture

The Balfour Lecture, named after the Genetics Society’s first President, is an award to mark the contributions to genetics of an outstanding young investigator. The Balfour Lecturer is elected by the Society’s Committee on the basis of nominations made by any individual member of the Society. The only conditions are that the recipient of the award must normally have less than 10 years’ postdoctoral research experience at the time of nomination. Any nomination must be made with the consent of the nominee. Those making nominations must be members of the Genetics Society, but there is no requirement for the nominee to be a member, nor is there any restriction on nationality or residence.

The 2016 Balfour Lecturer is Dr Felicity Jones, from the Friedrich Miescher Laboratory of the Max Planck Society, Tübingen, Germany. Felicity will present her lecture at the Genetics Society Autumn Meeting in November 2016, and a profile of her career will appear in the July 2016 Newsletter.

The winner of the 2017 Balfour Lecture will be announced at the AGM.

Call for Nominations

Nominations are now being invited for the 2018 Balfour Lecture. Note that there is no restriction on the subject matter of the Balfour Lecture. To make a nomination, please confirm that your candidate is willing to be nominated, and then forward a two-page CV of the candidate, together with a list of his or her ten most important publications, plus a one-page letter of recommendation outlining why you feel their contributions to the field have been outstanding. Please submit these supporting documents via email to the Honorary Secretary, Tanya Whitfield (t.whitfield@sheffield.ac.uk), by Friday, November 25th, 2016.

The JBS Haldane Lecture

The JBS Haldane Lecture recognises an individual for outstanding ability to communicate topical subjects in genetics research, widely interpreted, to an interested lay audience. This speaker will have a flair for conveying the relevance and excitement of recent advances in genetics in an informative and engaging way. The annual open lecture will be delivered on a topic, and in a place, agreed with the Genetics Society. In addition to delivering the Lecture, the recipient will receive an honorarium of £1000 and a three-year membership of the Society.

Date for the diary! – JBS Haldane Lecture 2016 – Aoife McLysaght

The winner of the 2016 JBS Haldane Lecture is Professor Aoife McLysaght (Trinity College Dublin, Ireland). Aoife will be delivering her JBS Haldane Lecture at the Royal Institution on Tuesday 8th November 2016. A profile of Professor McLysaght can be found in the in the July 2015 Newsletter.

The winner of the 2017 JBS Haldane Lecture will be announced at the AGM.

Call for Nominations

Nominations are now being invited for the 2018 JBS Haldane Lecture. The recipient will be selected by a committee chaired by the Genetics Society’s Vice President for the Public Understanding of Genetics from nominations made by Society members. Nominees need not be members of the Society, but should be active researchers working in the UK. To make a nomination, please confirm that your candidate is willing to be nominated, and then submit both a two-page CV and a short explanation of how the candidate meets the criteria above. Please submit nominations to the Honorary Secretary, Tanya Whitfield, by email (t.whitfield@sheffield.ac.uk), by Friday 25th November 2016.
Local Representatives

The Local Representative acts as a key liaison between the membership and the Society’s Office and Committee by helping to recruit new members, publicising the Society’s scientific meetings and other activities, and in providing feedback from the membership on matters of professional concern. The Society normally appoints only one local representative per company, institution or department, but exceptions can be made when there are semi-autonomous sub-divisions containing a substantial number of members or potential members.

We seek to fill vacancies and to update our database of Local Representatives on a yearly basis. Should you wish to volunteer as a local representative or if existing representatives wish to update their contact details, please contact the Honorary Secretary, Tanya Whitfield, by e-mail at t.whitfield@sheffield.ac.uk.

SEE FULL LIST ON PAGE 11
## Genetics Society Local Representatives

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<th>Local representative</th>
<th>Location</th>
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<tr>
<td>Professor Anne Donaldson</td>
<td>Aberdeen</td>
<td>University of Aberdeen</td>
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<td>Dr Glyn Jenkins</td>
<td>Aberystwyth</td>
<td>Aberystwyth University</td>
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<td>VACANT</td>
<td>Ascot</td>
<td>Imperial College London (Ascot and Silwood)</td>
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<td>Dr Araxi Urrutia</td>
<td>Bath</td>
<td>University of Bath</td>
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<td>Dr Declan McKenna</td>
<td>Belfast</td>
<td>University of Ulster, Belfast</td>
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<td>Dr Charlotte Rutledge</td>
<td>Birmingham</td>
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<td>Professor P C H Franklin</td>
<td>Birmingham</td>
<td>University of Sussex</td>
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<td>Dr Felicity Z Watts</td>
<td>Brighton</td>
<td>University of Bristol (Biol. Sci)</td>
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<td>Dr Colin M Lazarus</td>
<td>Bristol</td>
<td>University of Bristol (SOsM)</td>
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<td>Professor Patricia Kuwabara</td>
<td>Brussels</td>
<td>Sainsbury Laboratory</td>
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<td>Dr Philip Wigge</td>
<td>Cambridge</td>
<td>University of Cambridge (Dept of Genetics)</td>
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<td>Dr Ben Longdon</td>
<td>Cambridge</td>
<td>University of Cambridge (Dept of Plant Sciences)</td>
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<td>Dr Ian Henderson</td>
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<td>University of Cambridge (Dept of Zoology)</td>
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<td>Dr Howard Baylis</td>
<td>Cambridge</td>
<td>University of Cambridge (Dept Phys, Dev, Neuro)</td>
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<td>Dr Bénédicte Sanson</td>
<td>Canterbury</td>
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<td>Dr Simon Harvey</td>
<td>Cardiff</td>
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<td>Dr Timothy Rosen</td>
<td>Oxford</td>
<td>University of Wales College of Medicine</td>
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<td>Dr Jose Gutierrez-Marcos</td>
<td>Coventry</td>
<td>University of Warwick</td>
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<td>Professor Michael JR Stark</td>
<td>Dundee</td>
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<td>Professor Ian Jackson</td>
<td>Edinburgh</td>
<td>MR C Human Genomics Unit, Edinburgh</td>
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<td>Dr Doug Vernimmen</td>
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<td>Dr Iain Johnstone</td>
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<td>Dr Fiona Green</td>
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<td>Dr Heather M Sealy-Lewis</td>
<td>Hull</td>
<td>University of Hull</td>
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<td>Professor Michael F Tuite</td>
<td>Kent</td>
<td>University of Kent</td>
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<td>Dr Andrew Peel</td>
<td>Leeds</td>
<td>University of Leeds, School of Biology</td>
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<td>Dr Ed Hollox</td>
<td>Leicester</td>
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<td>Dr Craig Wilding</td>
<td>Liverpool</td>
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<td>Crick Institute</td>
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<td>Alex Blakemore</td>
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<td>Imperial College London (South Kensington)</td>
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<td>Professor Simon Hughes</td>
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<td>Professor Richard A Nichols</td>
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<td>King's College London</td>
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<td>London</td>
<td>Queen Mary and Westfield College</td>
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<td>Dr Claire Russell</td>
<td>London</td>
<td>Royal Botanic Gardens, Kew</td>
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<td>Prof. Harald Schneider</td>
<td>London</td>
<td>Royal Veterinary College</td>
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<td>Professor E M C Fisher</td>
<td>London</td>
<td>The Natural History Museum</td>
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<td>Francesca Mackenzie</td>
<td>London</td>
<td>UCL Institute of Neurology</td>
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<td>Dr Emanuela Volpi</td>
<td>London</td>
<td>UCL Institute of Ophthalmology</td>
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<td>Dr Yalda Jamshidi</td>
<td>London</td>
<td>University of Westminster</td>
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<td>Dr Catherine Walton</td>
<td>Manchester</td>
<td>St George's Hospital Medical School</td>
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<td>Dr Kirsten Wolff</td>
<td>Newcastle</td>
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<td>Professor Enrico Coen</td>
<td>Norwich</td>
<td>University of Newcastle</td>
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<td>Dr Tracey Chapman</td>
<td>Norwich</td>
<td>John Innes Institute</td>
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<td>Dr Richard Emes</td>
<td>Nottingham</td>
<td>University of East Anglia</td>
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<td>Dr Robert Chapman</td>
<td>Nottingham</td>
<td>University of Nottingham (Sutton Bonnington Campus)</td>
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<td>Professor John Brookfield</td>
<td>Nottingham</td>
<td>University of Nottingham (University Park Campus)</td>
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<td>Dr Paul Ashton</td>
<td>Ormskirk</td>
<td>Edge Hill University</td>
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<td>Professor Jonathan Hodgkin</td>
<td>Oxford</td>
<td>University of Oxford (Biochemistry)</td>
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<td>Professor Andrew O M Wilkie</td>
<td>Oxford</td>
<td>University of Oxford (John Radcliffe Hosp)</td>
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<td>Professor Liam Dolan</td>
<td>Oxford</td>
<td>University of Oxford (Plant Sciences)</td>
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<td>Plymouth</td>
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<td>Dr Gonzalo Blanco</td>
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<td>University of York</td>
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The Scottish Drosophila Meeting
8th May 2015, St Andrews

The fourth Scottish Drosophila Meeting (ScotFly) was held this year on the 8th May at the University of St Andrews. The event was a small meeting with representatives from various institutions with new and returning faces. Although the meeting is local, delegates came from as far as Ohio, U.S.A. and Rennes, France. With about 70 registered attendees there were 14 speakers and 22 presented posters. Invited speakers were; Darren Obbard, University of Edinburgh; Megan Neville, University of Oxford; Barry Denholme, University of Edinburgh and Stefan Pulver, University of St Andrews. As expected from a meeting with a focus on such a versatile system, the topics and research programmes presented ranged from the genetic control of organ development and the use of Drosophila as a model for human disease research to host-parasite co-evolution and the genetics of complex courtship behaviours.

The meeting was, once again, very successful and demonstrates a strong and vibrant Drosophila research community in Scotland.

Sponsorship, generously given by the Genetics Society allowed the meeting to run without registration costs along with a lunch for attendees and a wine reception to stimulate discussion after the talks. The next Scottish Drosophila Meeting is due to be held at the University of Edinburgh. ScotFly 2015 was organised by Professor Michael G. Ritchie and Dr. Marcus Bischoff. The full programme and further details are available at: http://synergy.st-andrews.ac.uk/drosophila/

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Annual Conference and AGM 2015
14th July 2015, London

The Genetic Alliance UK annual conference and AGM for 2015 was held on Tuesday 14th July at Amnesty International’s Human Rights Action Centre in central London. This was a celebration of Genetic Alliance UK’s 25th anniversary of achieving charitable status and the progress it has achieved since. The conference was supported in part by a generous Genetics Society award, for which the charity is very thankful. Ninety-four people attended the event, 47 of them representing our patient group members, with other attendees being researchers, clinicians, industry and staff.

Although the annual conference is a key event for Genetic Alliance UK and our membership, providing an excellent opportunity to bring our members together, this was of particular relevance in our silver jubilee year. We looked back at our work since 1990 and progress in healthcare for those living with rare and genetic conditions, but the conference also provided a platform to reflect on some of the successes that our member organisations have achieved.

Our Director, Alastair Kent, opened the conference with his personal highlights of the organisation’s work as he has been with Genetic Alliance UK almost from its inception. Professor Karen Temple of the University of Southampton’s Faculty of Medicine then gave her thoughts on how the clinical and technological environment has changed, ending with a consideration of what genome sequencing could hold for science and patients.

Mark Bale of the Department of Health then spoke in a personal capacity to provide his insights into how policy has evolved to deal with the many significant and often controversial issues that have arisen in the field of genetics.

Genetic Alliance UK has established the tradition of holding an interactive session with attendees to help explore current and future priorities for the organisation based on feedback from our membership. We therefore invited the Academy of Medical Sciences, which is currently conducting a major consultation exercise to establish public priorities for public health in the next 25 years, to lead a session looking at how our members would prioritise key issues. Participants were shown some archive public health information films to reflect on past approaches and were then asked to complete an online survey on their smart phone or tablet, with paper versions also available on the day. This led to a lively debate, moderated by Professor Sarah Harper of the University of Oxford.

The conference culminated with an hour-long session to highlight the work of a cross-section of Genetic Alliance UK’s membership. The AKU Society gave an inspiring presentation on their work to drive patient involvement in a clinical trial examining a potential treatment for alkaptonuria. Alström Syndrome UK then spoke about their efforts to establish a patient support service for their patients alongside the NHS. Niemann-Pick UK closed the presentations with a reflection on how their organisation has grown, encouraging the audience to collaborate with each other to achieve progress for patients with rare and genetic conditions.

Alastair Kent then closed the event by cutting an anniversary cake to celebrate the 25th anniversary.
Second international conference on Mendelian randomization: From population health to pharmaceutical development
22nd-24th June 2015, Bristol

Over 200 scientists from across the world filled the Victoria Rooms at the University of Bristol, for the second international conference on Mendelian randomization methods. The first meeting of about 40 invited scientists was held in 2006, in Brno, Czech Republic.

Mendelian randomization as a method to study disease aetiology and avoid the issues of reverse causation, bias and confounding which have traditionally hindered epidemiological studies, has received serious attention in the intervening years. Work presented at the conference covered methods, major study designs employing Mendelian randomisation and applied examples of the technique. Critically, this meeting engaged with industry and hosted members of the clinical and commercial sectors to illustrate how important this approach is for the development, understanding and repositioning of drugs.

This was well balanced across examples of research activity across the general epidemiology and social science arenas.

Delegates at this conference were unanimous in their enthusiasm to learn more about methodological developments and application in the pharmaceutical pipeline, social sciences, and causality in cancer, cardiovascular disease and other diseases.

Dr Nic Timpson, a programme lead in the MRC IEU said “It seems like the approach (largely experimental and unaccepted at the first meeting in Brno) has come a long way in a relatively short time.

Whilst MR will turn out to be part of the evidence collecting process for many scientific problems, we should not underestimate the ability of this approach to bridge the population and basic science arenas“.

One delegate reported “The conference was great, very informative and interesting, and a fantastic programme”, and a further added “The science was exceptional, with a large number of inspiring and fascinating talks from both influential senior scientists and more junior contributors.” “I really enjoy the methodological sessions”. Some aspects of the conference that pertain to drug discovery have been summarized on the blog [www.plengegen.com/blog/mendelian-randomization-drug-discovery-development-highlights-bristol-meeting-june-2015/] of Dr Robert Plenge, one of the invited speakers in the “Target Development” session.

The conference also hosted the launch of the International Journal of Epidemiology (IJE) [http://ije.oxfordjournals.org/] special issue on Mendelian randomization, of which a copy was distributed to conference delegates.

The conference was kindly sponsored by Illumina [www.illumina.com], DNA Genotek [www.dnagenotek.com] and the Genetics Society [www.genetics.org.uk], and the organisers are grateful for their support.

For further information about the MRC IEU, please visit the IEU website [www.bristol.ac.uk/integrative-epidemiology/].

Work presented at the conference covered methods, major study designs employing Mendelian randomisation and applied examples of the technique.
The science that will transform our future

David Walker  University of Edinburgh

It is almost impossible to mention all of the ‘future-transforming’ inventions, and so I won’t be summarising the future, per se. However, I will say that we find ourselves at a crossroads, which begs the question: “what really makes us human?”

Dearest Present,

I’m sure you’ll agree that what the future holds is an exciting prospect. Good or bad, we are undoubtedly having the most profound impact on the planet. ‘Science’, in its broadest sense, has been at the frontline of change. We have shaped our own environments, solved things deemed to be problems, but have also created our own profound adversities.

It is almost impossible to mention all of the ‘future-transforming’ inventions, and so I won’t be summarising the future, per se. However, I will say that we find ourselves at a crossroads, which begs the question: “what really makes us human?”

Let me explain...

With the exception of identical siblings, each and every one of the 7 billion(ish) Homo sapiens has a unique set of DNA, a genome. This blueprint for life was previously encrypted, a code that needed to be cracked - and so it was. By 1953, James Watson and Francis Crick had published their paper on the structure of DNA.

Skipping forward to 1996, Dolly the Sheep was born at The Roslin Institute. Since her announcement to the public, the first cloned animal from a somatic cell has left behind a legacy of ethical questioning. What if we cloned humans, and what if we made them a little bit better? I know I could do with that extra pair of hands! Many envisioned a transformed future, but these visions are actually not so farfetched.

Let’s consider the dystopian Britain in Kazuo Ishiguro’s 2005 novel, “Never Let Me Go”. Cloned humans are essentially organ farms, harvested until ‘completion’ and considered expendable.

But if you are shocked by this prospect, what if a cloned organ could save your dying loved one? Would you clone parts of yourself, or even a whole new you, as an insurance policy?

Further, consider the superior, designer humans in Andrew Niccol’s 1997 movie “GATTACA”. Those not born as a result of eugenics are still subject to a screen of their genome from birth, told their faults, and shunned for inferior genetics.

However, today, the advent of high throughput genomic sequencing makes cracking somebody’s code a fairly straightforward process.

It is this kind of current science which could transform our future. It could change the course of human evolution. Eugenics may seem unethical to you, but what if a genome screen was a pre-requisite for getting health insurance?

Deviations from the ‘norm’ would be frowned upon, or at least be financially punished. If you had the choice, would you risk having your child being shorter than the rest? Probably. What if they had a gene leading to psychotic tendencies? There’s no harm in changing that… right?

I could tell you that my twin brother wrote this entire article up to this point. I have harvested his ideas. We are genetic clones after all, and as the first-born I have the right to take them for myself. Obviously this may sound rather ridiculous and if true would be unacceptable! But consider the fictitious scenario of organ ‘plagiarism’ in a dystopic future – perhaps word-theft isn’t so bad?
The majority of fictitious peoples’ thoughts aligned to acceptance of organ harvesting and of ‘designer people’. Acceptance is certainly a route that could be taken.

However, we forget, although cloned humans are genetically identical, you cannot create a collection of thoughts and aspirations - these are individual. In essence, this makes identical twins unique. Despite our underlying conformity, my twin and I have been subject to countless arrays of uncontrolled stimuli, resulting in different people. We both rode bikes, and hate peppers, but I am a vet and my brother’s an archaeologist. Should clones therefore be afforded the same rights as anybody else?

What if individual thought is taken away and homogenised? It then has the potential to change the very essence of what makes us all unique – an individuality controlled. What if the obsession with physical perfection is the start of our transformation, by homogenising our thoughts? Perfection and self-preservation by any means?

It is fair to say that science will transform our future, and it will be exciting. Regardless of robot butlers and transportation devices, one certainty is that the future could be transformed by technology that has already been invented.

Kindest regards,
D. Walker
(Natural-Born Human: Class B-UNINSURABLE*)

Interview - Genetic revolutions
Alison Woollard
JBS Haldane prize lecture 2015
Dr Kat Arney . Science Information Manager at Cancer Research UK

Every year the Genetics Society recognises a person with an outstanding ability to communicate genetics through the JBS Haldane lecture and award. This year’s winner is Professor Alison Woollard from Oxford University, whose work focuses on the genetics of ageing. She gave her lecture at the beginning of November at the Royal Institution, focusing on key revolutions in genetic thinking. This article is adapted from Alison’s interview with Naked Genetics podcaster Kat Arney.

Alison: JBS Haldane was a very interesting scientist who was working in the UK in the 1930s and ‘40s, and ‘50s, and beyond. He was fascinated by population genetics – in how to relate Mendel’s ideas of heredity into whole populations – and he applied maths to work that out. He was one of the real proponents of the importance of quantitative analysis in biology.

Kat: He was also quite cool! He was very into debating about ideas and talking about them, wasn’t he?

Alison: Yes – he was amazing! People say that Haldane was the best read of all scientists of his age, but then they said that in order to become that, he only had to read his own work because he was so prolific.

Kat: I love reading about him - he’s my favourite geneticist, I think. He was a bit of a hippie, too.

Alison: He’s like everyone’s favourite granddad I think. He was very left wing – a Marxist and a socialist. He believed in equality and was a great believer in the welfare state. He was passionate about education at all levels, and that education is a great
Haldane was very well-known for his skills in public communication – the Haldane lecture is a public talk where we try to bring genetic ideas to a very wide audience. My take on this was to really think about genetics as revolutionary, because Haldane was a revolutionary, and I wanted to have this idea of revolution in my lecture.

liberator. He also had weird and wacky ideas about all manner of things – he spent a lot of time in India on the hippie trail and wrote some fascinating books about his experiences there, and many other things besides.

Kat: What were you trying to get across in your JBS Haldane lecture?

Alison: Haldane was very well-known for his skills in public communication – the Haldane lecture is a public talk where we try to bring genetic ideas to a very wide audience. My take on this was to really think about genetics as revolutionary, because Haldane was a revolutionary, and I wanted to have this idea of revolution in my lecture. And so I decided I would pick on what I considered to be the most important revolutions in genetics. I was probably a little bit ambitious because I started in 400 BC and ended up in the future, picking out the most important revolution in terms of genetic ideas, starting with Mendel and then moving on from that.

Kat: You had seven revolutionary ideas. Tell me about some of them.

Alison: Of course there were Mendel’s Principles of Heredity. Mendel proposed a mechanism for heredity that was missing from Darwin’s Theory of Evolution by Natural Selection and that was really important. And then we have the idea of relating these hereditary principles to actual tangible things in cells – namely the behaviour of chromosomes. That was the third big revolution, that Thomas Hunt Morgan was involved in.

Kat: That’s all the fruit fly guys?

Alison: Lots of fly stuff! And then after that came the molecular biology revolution. Not just Watson and Crick, but all the people before them that showed that DNA is the hereditary material. And then those that came after, showing how gene expression works and how genes can be switched on and off. There was such a lot of molecular biology that went on it – it was an absolute ferment in the 1940s, ’50s and into the ’60s.

After that, people understood the mechanism of heredity and how it works at the level of molecules, but they didn’t understand the rest of biology. So people started to use genetic techniques to understand other things like cell division and development: how cells end up in the right place doing the right thing, what differentiates one cell from another and so on. That was a very important thing.

Kat: And then we get to the genome and the era of genomics.

Alison: That’s almost a 21st century idea – that you can sequence whole genomes and understand the entire genetic makeup of an organism.

Then you can really drill down into what it is that distinguishes one organism from another and what distinguishes one disease from another. And now we’re in a new era of understanding genomes and also are beginning to manipulate them. That’s my last revolution – genome editing. This is the idea that we can now interfere with our genes and modify our genetic destiny. But we need to be thinking about whether or not that’s a good thing, a bad thing, or an inevitable thing – whether it’s a good way of eradicating disease or whether it’s dangerous and might lead to designer babies and so on. So I think people need to understand the science behind those kinds of ideas if they’re to contribute to the debate about whether or not it should happen in the future.

Listen to the full interview in the Naked Genetics podcast from November 2015 at nakedscientists.com/genetics
Our very own Naked Genetics podcaster Kat Arney has written a new book unpacking the complexities of modern genetics for the general public, engagingly and wittily told through fascinating stories, interviews and anecdotes. The language of genes has become common parlance. We know they make your eyes blue, your hair curly or your nose straight. The media tells us that our genes control the risk of cancer, heart disease, alcoholism or Alzheimer’s. The cost of DNA sequencing has plummeted from billions of pounds to a few hundred, and gene-based advances in medicine hold huge promise.

So we’ve all heard of genes, but how do they actually work?

There are 2.2 metres of DNA inside every one of your cells, encoding roughly 20,000 genes. These are the ‘recipes’ that tell our cells how to make the building blocks of life, along with myriad control switches ensuring they’re turned on and off at the right time and in the right place. But rather than a static string of genetic code, this is a dynamic, writhing biological library.

Figuring out how it all works – how your genes make you, you – is a major challenge for researchers around the world. And what they’re discovering is that far from genes being a fixed, deterministic blueprint, things are much more random and wobbly than anyone expected.

Drawing on stories ranging from six-toed cats and stickleback hips to Mickey Mouse mice and zombie genes – told by researchers working at the cutting edge of genetics – Kat Arney explores the mysteries in our genomes with clarity, flair and wit, creating a companion reader to the book of life itself.

Published by Bloomsbury Sigma in January 2016, Herding Hemingway’s Cats features interviews with researchers working at the cutting edge of genetics – from Nobel prize-winners and Genetics Society president Wendy Bickmore to the next generation of young scientists.

Read an extract online now at bit.ly/HHCextract and use the special member discount code GENES to save money when you buy direct from the publisher’s website: bit.ly/HHCBloomsbury

Kat Arney explores the mysteries in our genomes with clarity, flair and wit, creating a companion reader to the book of life itself.

A sprightly, energetic tour through the minds of those trying to understand genes. Each snappy chapter is a remarkable feat of information and fascination. (Robin Ince, comedian, writer and co-presenter of The Infinite Monkey Cage and The Quest for Wonder)

If you want to find out for whom the cell mutates, then Herding Hemingway’s Cats is for you. Kat Arney decodes the greatest works of nature, written in the language of the genes. (Roger Highfield, author, science journalist and museum executive)

Kat is one of the world’s finest science communicators and enthusiasts. Herding Hemingway’s Cats is a joy to read and a masterclass in making the complex story of life accessible, entertaining and relevant. (Mark Stevenson, author of An Optimist’s Tour of the Future)
I attended the actin and microtubule meeting organised by Thomas Surrey (UK) and Marie-France Carlier (France), which was held from 26-30 May 2015. This was a large, international meeting on the Cytoskeleton at which I was selected to present my work as a poster entitled “Multiple modes of establishing polarised growth in fission yeast S. pombe”. The meeting was organized at the beautiful seaside town of Roscoff (Brittany) in the marine biological station.

The meeting started with an amazing keynote lecture by Tim Mitchison whose lab has done outstanding work in improving the understanding of microtubule nucleation and dynamics. His talk focused on how a very large cell divides using quantitative microscopy and modelling to understand this process.

The conference was divided into different sessions focusing on roles of actin and microtubule cytoskeleton in cell movement, cell division, migrations etc. The first session of the conference focused on “control of self-assembly: chemical and structural aspects”. In this session Eva Nogales (Berkeley, USA) gave an excellent talk on the structural basis of microtubule dynamic instability and its regulation. This session also had a wonderful talk by Jan Lowe (Cambridge, UK) about the bacterial cytoskeleton titled “pushing and pulling of DNA through bacteria. Both these talks focused on using advanced cryo-EM techniques to address fascinating questions in cytoskeleton biology.

Second session of Day 1 focused on nucleation and polymerization. In this session, David Kovar (Chicago, USA) gave a very interesting talk on how profilin regulates f-actin homeostasis by favouring formin over Arp2/3 complex. In the afternoon, we had the poster session where I presented my work for a 2 hour-long session.

The poster session was very interactive and I was able to get lot of constructive feedback and suggestions. The long day of talk ended with an amazing dinner in a sea-facing dining area, which was a great opportunity to meet lot of people and have very cool scientific discussions in a more informal manner.

A second key note lecture was given by Victor Small (Vienna, Austria) who gave a fascinating talk about lamellipodium. The conference took place over four days and had a wide range of interesting talks on different aspects of actin and microtubule cytoskeleton. One of the things that struck me at this meeting was the truly fascinating imaging and image analysis techniques used by different groups to address various biological problems. The meeting ended with a banquet dinner where Tim Mitchison thanked the organizers for such an amazing conference where both the actin and microtubule communities could come together and discuss the progress and challenges faced in each fields.

I would like to thank the Genetics Society for providing me with this wonderful opportunity to attend this conference.

The meeting started with an amazing keynote lecture by Tim Mitchison whose lab has done outstanding work in improving the understanding of microtubule nucleation and dynamics. His talk focused on how a very large cell divides using quantitative microscopy and modelling to understand this process.
In June, three international cancer societies joined forces to host an interdisciplinary conference entitled “Anticancer Drug Action and Drug Resistance: From Cancer Biology to the Clinic.” Held in the beautiful Renaissance city of Florence (Italy), and attracting nearly a thousand delegates from fifty-one countries, the event showcased cutting-edge research from a diverse range of topics including tumour evolution and heterogeneity, liquid biopsies, next-generation sequencing, epigenetics and immunotherapy.

The conference comprised a series of keynote lectures, topical symposia, proffered papers and poster sessions, in addition to a range of evening networking events that were sponsored by exhibitors. I found Professor Charles Swanton’s lecture on intratumoural heterogeneity particularly stimulating; he has been a key opinion leader in this area for several years and has greatly influenced the way in which we view the clonal architecture of malignant tumours, particularly in the context of targeted cancer therapies. Relating to this concept, Professor Alberto Bardelli provided recent evidence that therapeutic resistance can be monitored in the blood using droplet digital PCR and next-generation sequencing, and, in many cases, this preceded the detection of relapsing disease via conventional imaging modalities. Professor Caroline Dive shared her work on the isolation and analysis of circulating tumour cells (CTCs), which she uses to generated CTC-derived xenografts as models of small-cell lung cancer. She uses these models to screen existing and novel compounds, with the aim of identifying effective drug strategies for this aggressive and poorly understood lung cancer subtype. Professor Dive is also investigating the utility of CTCs as prognostic biomarkers and tools to understand the genetic heterogeneity of primary and metastatic tumours.

I had the opportunity to present my PhD research as a poster, which gave me experience in communicating my work to a range of people outside my immediate subject area. I am investigating the utility of circulating cell-free DNA (cfDNA) as a non-invasive biomarker for the early detection of lung cancer, using blood samples from both mouse models and patients with lung cancer. In addition to disseminating my work to fellow participants, this was a fantastic opportunity to network with fellow scientists and leaders in the fields, and I also received career advice from several scientists working in the biotechnology sector.

I am indebted to The Genetics Society for their generosity in funding my attendance at this stimulating and influential conference, and I recommend multidisciplinary conferences of this kind to other PhD students and early career researchers.
Gordon Research Conference on Red Cells
28th June-3th July, 2015. Holderness, USA

Matthew Shannon . King’s College London

This year the Gordon Research Conference on Red Cells was held at Holderness School, New Hampshire, USA. The conference focusses on the biology of erythrocytes, looking at development, maturation, disorders, epigenetics and transcriptional control, with a particular emphasis on presenting unpublished data. As with many Gordon Research Conferences, this was preceded by a Gordon Research Seminar; a short meeting targeted at PhD students and early career researchers, providing an opportunity to present current work in a less formal environment.

I was fortunate enough to be able to attend both the conference and the seminar this year, with the generous support of a Genetics Society travel grant. I am a second year PhD student in the department of Medical and Molecular Genetics at King’s College London. My PhD focusses on genetic and epigenetic factors that influence the phenotypic severity of Sickle Cell Anaemia, so the Red Cells conference was of great relevance to my project. The conference was a great experience, and was invaluable to my PhD work, particularly due to the feedback during the poster sessions, from both PhD students and PIs, there were some really useful comments from researchers that are using similar techniques and have encountered similar obstacles.

This was a great opportunity to listen to talks by some of the leading researchers in the field, and to see unpublished work that these lab groups are currently carrying out, providing great insight into the future directions of red blood cell research.

As well as the conference itself, the Holderness School site was a great experience, the school is quite isolated in the picturesque countryside of New Hampshire, and as a result the conference has a very sociable atmosphere, with roughly 150 attendees spending the entire week on the conference site.

I am extremely grateful to the Genetics Society for awarding me a travel grant, and I would strongly recommend anyone working on erythrocytes to consider the next Red Cells conference, and especially the Gordon Research Seminar, which is a great chance to meet other researchers in an informal environment.

Dicty 2015 - Annual International Dictyostelium Conference
9th-13th August, 2015, Egham, UK

Fu-Sheng Chang . University of Oxford

The Annual International Dictyostelium Conference 2015 was held in a beautiful Victorian-style campus of Royal Holloway, University of London. The International Dictyostelium Conference is the most prestigious conference in the realm of Dictyostelium research and it has been a privilege to be able to be part of it. This annual meeting featured five days of the most cutting-edge and exciting science covering a broad spectrum of Dictyostelium research ranging from Biochemical and Host Pathogen Interactions, Chemotaxis and Development to Cytokinesis and Nuclear Organization. Researchers came from all over the world sharing their ideas and discoveries.

During the meeting, I learnt about new developments covering this range, in particular new techniques for Dictyostelium imaging, signalling pathways, cell development and cell movement. One of the most memorable talks I attended, by Dr Zhi-Hui Chen, of the University of Dundee, described a novel mode of gene regulation by c-di-GMP in Dictyostelium, which could be a potential direction for my future research. My supervisor, Dr Catherine Pears, of the University of Oxford,
also gave a talk on ADP-ribosylation of histone H2B in double strand DNA repair from which we have gained valuable feedback from other researchers.

My research poster was entitled “Analysis of Intracellular Calcium Channels in Dictyostelium Development” in which I presented work characterizing of the role of Calcium release from intracellular stores, which was presented on the second day. By presenting my research in the meeting, I was fortunate enough to be able to discuss my discoveries with eminent researchers in Calcium signalling. In particular I had a valuable discussion with Dr David Traynor (University of Cambridge) and Dr Tsuyoshi Araki (University of Dundee), who kindly shared their important findings and suggestions for my work. Dr Zhi-Hui Chen also gave me valuable suggestions based on his work on the role of c-di-GMP in development of Dictyostelium. These suggestions are extremely important for me to set further research goals for my research and are well timed within my PhD.

In the conference, we were offered a number of great opportunities to get to know and interact with other scientists in many social events. I was able to visit the Fuller’s Griffin Brewery in London, join a conference formal dinner and experience an English Village dance. This was really an unforgettable experience.

Overall, I enjoyed greatly the conference and I would like to take this chance to thank the Genetics Society and University College Oxford for giving me generous support enabling me to attend the conference and make a fruitful trip.

The 15th European Society for Evolutionary Biology (ESEB) is a bi-annual meeting which brings together some of the leading international scientists from a very wide range of fields. The conference was attended by around 1400 researchers, featuring 35 different symposiums in topics ranging from social interactions to the evolution of genomes.

The meeting started each morning with a plenary session covering varied topics, including the evolution of sexual dimorphism, behavioural genetics and the evolution of proteins. The plenary talks comprising these sessions were delivered by some of the leading researchers in the respective fields and introduced some of the most cutting edge ideas floating around today in the field of Evolutionary Biology as a whole.

Following the plenary sessions, there were 4 parallel symposia delivering an enormous range of talks, often leading to some tough decisions on which talk to attend. Some of the most interesting discussions, and from which I profited massively in terms of personal development, covered key ideas in the understanding of the molecular basis for adaptation and the finding of loci under selection from genomic data. These two ideas were a recurrent theme throughout the conference, with many talks demonstrating how the field is making huge leaps in pinning down the precise genes involved in both morphological and behavioural evolution. Without a doubt, one of the most exciting talks given in this respect was by Hopi Hoekstra from Harvard University, who is one of the few people beginning to provide evidence for the genomic basis of “the extended phenotype” hypothesis.

Following the talks, there were two days were researchers presented in excess of 950 posters, providing me with the opportunity to present my...
work to the conference attendees. These sessions gave me a chance to acquire valuable feedback on my project from some of the leading scientists in the field - a truly invaluable experience that will no doubt help shape my future questions and experimental approaches. During the last day of the conference I had the opportunity to listen to talks given to the John Maynard Smith prize winners of the previous and current years. Both were excellent talks on the topics of recombination in great apes and the mathematical basis for evolution in sex and disease. Laurent Keller, a leading researcher in the field of social evolution, gave one of the most inspiring and energetic talks of the conference, praising the importance of Serendipity in science and, at one point, even standing up on the desk of the auditorium to deliver his message, in an act reminiscent of Robin Williams in his Dead Poet's Society. Overall, the conference was an immensely enjoyable experience. I would like to express my gratitude to The Genetics Society for their generous support in allowing me to attend the conference.

Cold Spring Harbor Laboratory -
The Eukaryotic DNA Replication & Genome Maintenance

1st-5th September, 2015. Cold Spring Harbor, USA

Carolin Muller . University of Oxford

Cold Spring Harbor laboratory is known for its research excellence. It is also the venue for many scientific conferences, including one of the most anticipated meetings in the DNA replication and genome stability field. At the beginning of September 2015, more than 300 delegates from around the world congregated to present and discuss recent scientific findings, to set up new or advance existing collaborations and generally to enjoy great science.

The first session featured talks on a rather controversial topic in the replication field – the influence of G-quadruplex structures on replication origin function in metazoa. The data presented by M. Prioleau and M. Méchali indicates G4 structures as major determinants of replication origin specification in metazoa. In contrast, work from S. Gerbi's lab suggests that a subset of these G4 structures at origins are false positives. The partially contradictory views led to a lively discussion, which was reignited due to G4 structures being a reoccurring topic throughout the meeting. For example, G4's are known to briefly stall replication forks. As a result, G4-stabilizing compounds can severely impede replication, particularly in cells deficient in homologous recombination. An interesting talk by J. Zimmer addressed the potential of such stabilizing compounds as cancer drugs.

I was so fortunate to conclude the second session with my talk on the “Physiological Requirements for Temporal Regulation of Replication Timing Control”. Not only was it a huge privilege to give a presentation to so many outstanding scientists; importantly I received constructive feedback, which will be instrumental for the future direction of the project. Many presentations throughout the meeting highlighted the informative power of in vitro systems reconstituting origin licensing, helicase activation and replication elongation. In particular, presentations from J. Diffley’s as well as S.P. Bell’s lab provided valuable insights into replication of chromatin. Both labs presented compelling evidence that nucleosome chaperones, for example FACT, and remodelers, such as Ino80 or ISW1, are required for efficient chromatin replication.

Overall, I found this CSH conference immensely beneficial. I am very grateful to the Genetics Society for subsidizing my attendance at this exceptional meeting!
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*2012 Journal Citation Reports®* (Thomson Reuters, 2013.)
EMBL Symposium - The Mobile Genome: Genetic and Physiological Impacts of Transposable Elements

16th-19th September, 2015.
EMBL Heidelberg, Germany

Andrew Mason. University of Edinburgh, UK

In September 2015 I was able to attend the first EMBL symposium on “The Mobile Genome” with the support of a Genetics Society Junior Scientist Travel Grant. My PhD project includes the identification and characterisation of chicken LTR retrotransposons and endogenous retroviruses, so it was a fantastic opportunity to attend a conference completely focused on the repetitive elements of the genome. Normally it is rare to even have a full conference session on these incredibly diverse features! I really hope EMBL decide to make this a regular slot in their busy and varied symposia programme.

Over 250 attendees were treated to four days of varied and interesting talks in the advanced training centre at EMBL Heidelberg.

This featured keynote presentations from Evan Eichler (RNA mediated gene duplication in primate genome evolution), Philip Zamore (piRNA-mediated transposon silencing), Haig Kazazian (LINE characterisation and genomic effects) and Dixie Mager (Human Endogenous Retroviruses), as well as 127 posters split over the double helix that makes up the inside of the building. I was selected to present a poster – “A New Look at the LTR retrotransposon content of the chicken” – and it was really useful to have so many leading researchers there to discuss my results and suggest possible new research questions.

Having attended much larger conferences before, this smaller, more focused setting made networking both easier and relevant to your own work in some way. This was helped by generous coffee and lunch breaks as well as a “meet the speakers” table after each talk session. I was able to make some good connections with others in the UK working on endogenous retroviruses, and also a group in Sweden working more widely on avian repeat content. It felt particularly good that these connections are of mutual benefit, rather than unidirectional.

I would like to take this opportunity to thank the Genetics Society again for their support. This conference was really fantastic, both in terms of the scientific content and networking success, and I feel being able to attend has made a big difference to my research.

After a number of interesting talks on the first evening of the meeting, Professor Feng Zhang, a CRISPR/Cas pioneer from MIT, presented his groups ground-breaking research on a novel nuclease that mirrors Cas9, Cpf1.

Genome Engineering: The CRISPR/Cas Revolution 2015

Cold Spring Harbor, USA

David Courtney. Ulster University

In September 2015 I had the opportunity to attend the first ‘Gene Engineering: CRISPR/Cas revolution’ conference, held at Cold Spring Harbor, New York. Attending a scientific conference at this world-renowned venue was exciting in itself, never mind having the opportunity to spend 4 days with the world’s most influential researchers in the fast evolving field of CRISPR/Cas gene editing. The meeting itself was not slow to start. After a number of interesting talks on the first evening of the meeting, Professor Feng Zhang, a CRISPR/Cas pioneer from MIT, presented his groups ground-breaking research on a novel nuclease that mirrors Cas9, Cpf1. This discovery was the talking point amongst researchers for the rest of the evening, and continued through the following day as the scientific paper describing this researcher was published online in Cell that morning.

Throughout the following 3 days conference sessions comprised of presentations on the various applications of CRISPR/Cas9, including the development of transgenic animals, therapeutic applications and gene screens for various cancer targets. One panel discussion comprising of esteemed Professors including Jennifer Doudna, Jonathan Weissman and Hank Greely, focused on the ethics surrounding gene editing. Across the world this is an on going hotly debated topic, as the ability to manipulate the DNA of species at the germline level becomes less expensive and easier to perform...
in any molecular biology laboratory. The therapeutic advantages and disadvantages of this were thoroughly discussed over the course of the session. At the end of the session a vote of all in attendance was taken, with most agreeing that restrictions must be put in place to, for the meantime, halt germline editing of viable human embryos.

On Saturday evening I presented some of my PhD research as a poster. During my PhD I performed research into the development of allele specific therapeutic systems for dominantly inherited corneal dystrophies. I developed a CRISPR/Cas9 system highly specific to the mutant allele, with no effect on wild type expression. Just before leaving for the conference this research was published in Nature Gene Therapy. I enjoyed the opportunity to present this work to well established researchers of CRISPR/Cas9 based gene editing. During the poster session I received positive comments on my research and interesting ideas for moving this work forward in the future.

Overall the conference was an excellent assortment of presentations detailing the possibilities for CRISPR/Cas9 systems in both the lab and the clinic. Talks from leaders in the field, and researchers tipped for Nobel Prizes in the future, were fascinating, and I was sent back to Northern Ireland with a wealth of knowledge and ideas to incorporate into my PhD thesis and future postdoctoral studies.

The conference was well organised and executed and I express my thanks to the conference committee for this. I would also personally like to thank the Genetics Society for giving me the opportunity to attend this excellent conference and have no doubt it will aid in my future scientific endeavours.

Conference Cold Spring Harbor - Neurobiology of Drosophila
29th September-3rd October, 2015. Cold Spring Harbor, USA

Niki McAllister . University of Birmingham

The neurobiology of Drosophila meeting is a prestigious biannual international conference set in the beautiful surroundings of the Cold Spring Harbor Laboratory, USA. It is renowned for attracting some of the top researchers in this field from far and wide to gather, present and discuss current research, and it certainly did not disappoint!

The schedule was filled with an exciting amount of different presentation sessions, poster sessions and plenary lectures. New to this year this meeting also contained numerous workshops including, but not limited to, development of new techniques and well as professional development courses that enabled participants to broaden their knowledge base and thus facilitate the spread of new techniques and ideas to the others to increase the scientific scope of Drosophila research.

The first evening started with sessions on brain, behaviour and evolution with an incredible mix of presentations detailing important pathways, chemical components as well as individual specific cells all of which are key in the regulation of behaviours such as sleep, voluntary movement, emotions, courtship and mate choice. Following this the neural development session had important findings in axon guidance, neuronal remodelling as well as growth and maintenance.

The variety of presentations continued throughout the conference with sessions including but not limited to sensory systems, glial biology, mechanisms of neurological diseases as well as synaptic transmission and plasticity. Each presenter provided new and exciting data as well as transferring knowledge of some of the advances in techniques that will no doubt be beneficial to all attendees.

It was inspiring to listen to some of the high quality research that is produced by many of the well known figures in Drosophila neurobiology but I was also particularly impressed with the calibre of the up and coming new researchers.

The penultimate evening was fun filled with a piano concert followed by a lobster banquet and finally a screening of The Fly Room which follows the birth of modern genetics as witnessed by the daughter of Calvin Bridges during a period known as the original fly room laboratory at Columbia university under the supervision of Thomas Hunt Morgan. What an amazingly appropriate setting to be able to view this film!

I feel honoured to have attended such large and prestigious conference where I have been able to strengthen and develop my communication skills, receive feedback and build essential collaborations from key people in the Drosophila Neurobiology field. I cannot thank the genetics society for giving me the opportunity to attend and I look forward to returning for the next conference!
The Naked Genetics Podcasts

Download, or subscribe for FREE, at www.thenakedscientists.com/genetics.
Thanks to the grant from the Genetics Society I could attend a really great workshop on analysing data produced by an innovative and promising sequencing approach called restriction site-associated DNA sequencing (RAD-Seq). This technique produces a reduced representation of the whole genome which involves not only much lower costs than whole genome sequencing but is also sufficient to address many relevant research questions in population genetics or genomics. A variety of tools for dealing with RAD-seq data and its special features have been developed. Understanding the concepts behind these programs, getting familiar with RAD-seq data analyses and the general workflow is of central importance to my PhD project aiming to identify regions under selection in a South African bee species. The workshop was held by some of the top experts in the field of RAD-seq and thus, did not only give me a very good overview over the analyses workflow but also allowed me to address specific questions concerning my own project. There was a good balance between the lectures part introducing us to the theory behind and the practical bioinformatics part. Lectures were well structured and easy to follow. Exchanging with the other workshop participants also provided me with new ideas and feedback for my own project. The general atmosphere was very friendly which facilitated networking and made working much fun. The workshop is highly recommendable for everyone interested in starting to work with RAD-seq data and I am grateful to the Genetics Society which gave me the great opportunity to participate.

CHRO 2015 conference
1st-5th November, 2015. Rotorua, New Zeland

I presented some of my research during that conference and received positive echoes afterwards. The work I presented was a side project I worked on, based on the study of Accessory and Core genomes of strains of Helicobacter from different geographic and ethnic origins.
TRAVEL GRANTS FOR JUNIOR SCIENTISTS

65th Annual Meeting of the American Society for Human Genetics

6th -10th October, 2015. Baltimore, Maryland, USA

Gaia Andreoletti . University of Southampton

Thanks to the Genetics Society Conference Grant I was able to able to participate at the 65th Annual Meeting of the American Society for Human Genetics (ASHG). The meeting was held at the Baltimore Convention Centre in Baltimore, Maryland, from Tuesday 6th October until Saturday 10th October 2015. The ASHG Annual Meeting is the largest human genetics meeting and exhibition in the world; it represents an excellent opportunity for geneticists to network and share the latest cutting-edge research breakthroughs. This year’s meeting attracted over 7,000 delegates and more than 200 exhibitor booths from across the world, which typifies the evolving field of genetics and its integration into mainstream healthcare. The programme was packed with presentations across the 5 days delivered by top researchers in the field, including 390 talks and 2,800 posters on genetics, genomics, statistics and epidemiology.

As a PhD student within the Genetic Epidemiology and Bioinformatics group at the University of Southampton, I had been invited to present a poster on my research in the application of next generation sequencing (NGS) technologies in paediatric inflammatory bowel disease (IBD). My research includes the analysis and interpretation of huge amounts of rare and common genetic data collected from hundreds of children diagnosed with IBD. Specifically, we apply whole-exome sequencing methods, which targets DNA analysis to the coding parts of the genome. The poster was well perceived and discussed with colleagues, and I was happy to receive positive feedback and constructive criticism. Overall, I found the experience intellectually stimulating and this has precipitated new directions for my future research. In addition, I was able to engage in interesting discussions with international colleagues with the view to form new research collaborations. This experience has proven to be beneficial, inspiring and fruitful with regards to my on-going research and future career. Without the support of the Genetics Society this opportunity would not have been possible.

of course, but also some others that were focused on other microorganisms but were using techniques that could be of interest with what I am doing.

One presentation particularly raised my curiosity and interest. A researcher from Canada presented her work with a native community in far North Canada. We had a passionate discussion over her project which might lead to collaboration between our groups, as we could bring the bioinformatics knowledge and infrastructure than her group lacks of to handle that amazing dataset they managed to gather. I also discussed the possibility of adapting a Phase Variation study runned on Campylobacter to my Helicobacter strains. This could lead to a project as well.

I presented some of my research during that conference and received positive echoes afterwards. The work I presented was a side project I worked on, based on the study of Accessory and Core genomes of strains of Helicobacter from different geographic and ethnic origins. I developed a method which allows a visualisation of the sharing genes patterns between groups of strains. This method was well received and raised interest. A few interesting questions were raised which will allow me to make a few more analysis and strengthen my results.

None of this would have been possible without the financial help from the Genetics Society, so I would like to thank the Society for offering me the opportunity to attend and present my work in that conference.
Join the online debate

Keep in touch with your colleagues via the Genetics Society Group on LinkedIn

We have added another way to keep in touch with society and your colleagues by creating a Genetics Society group on LinkedIn. In order to ensure that all content on that group is meaningful to you, we have set this up as a moderated group. This means that when you join the group this needs to be formally approved, but as long as we can see you are active in a genetics related area this is not a problem.

This prevents a lot of indiscriminate postings from online recruiters that have affected some of the Genetics related groups. As a member of the LinkedIn group you will be updated on our activities but you can also comment and add your own events. If you are not already on LinkedIn please consider joining. Especially young scientists hunting for a job outside academia do well to build up their profile on LinkedIn.
Multi-host pathogens of honeybees and wild bumblebees: Is the *Varroa* mite a game changer for disease dynamics?

Robyn Manley. University of Exeter

Honeybees and wild bumblebees share a range of viral pathogens. Researchers have identified emerging pathogens as a primary cause of decline in both honeybees and bumblebees, alongside habitat loss and pesticide use. In honeybees, the recently emerged ectoparasitic mite *Varroa destructor* - which is specific to honeybees - is a major factor. It can cause colony mortality by vectoring and amplifying otherwise asymptomatic viruses. Could the presence of *Varroa* in honeybee populations indirectly affect wild pollinators such as bumblebees? We aim to test how *Varroa* changes the disease dynamics of three multi-host viruses in honeybees and wild bumblebees by comparing viral prevalence, disease intensity and sequence diversity between *Varroa*-infested and *Varroa*-free populations. With the support of the Genetics Society and the CB Dennis trust, I have sampled the relevant field populations this summer. *Varroa* arrived and spread swiftly across Europe around fifty years ago, leaving few places untouched. However, a few islands around the British Isles and France still remain *Varroa*-free. Funding from the heredity field grant allowed me to take advantage of these unique field sites – providing a natural experimental set-up to test our research questions. This summer I sampled 120 foraging bees (30 honeybees, 60 *Bombus terrestris/lucorum* complex, and 30 *B. pascuorum*) from 14 field sites: including four *Varroa*-free islands, four *Varroa*-infested islands and 6 *Varroa*-infested mainland sites. I am currently extracting RNA from these samples to determine molecularly the presence and prevalence of three target viruses with contrasting epidemiology: Deformed wing virus (DWV) – the virus most clearly affected by *Varroa* and an emerging bumblebee virus in the UK; Acute bee paralysis virus (ABPV) – a true multi-host pathogen with similar distribution across honeybees and bumblebees in the UK; and Black queen cell virus (BQCV) – a multi-host pathogen, which appears not to be actively transmitted by *Varroa*. qPCR will provide data on infection strength, measured as within-individual virus titres and all virus-positive samples will be Sanger sequenced to measure viral diversity.

We will test whether *Varroa* leads to an increase in viral prevalence and infection strength across host species, beyond *Apis mellifera*, using generalised linear models. A population genetic approach will allow us to test whether *Varroa* exerts selection on multi-host pathogens by comparing viral diversities, measuring population differentiation and testing for positive selection in the viral sequence data. We will contrast patterns for DWV and ABPV, which are actively vectored by *Varroa*, with BQCV. If *Varroa* drives viral disease emergence in pollinators we would expect to see high prevalence, reduced diversity and high titres in *Varroa*-infested populations for DWV and ABPV, but not in BQCV strains because *Varroa* is not involved in its transmission. Independent of the question of *Varroa*, the data will provide much needed information on the biogeography of viral strains present across pollinator species.

Our work will elucidate the role of *Varroa* in multi-host pathogen dynamics of pollinators. It will provide an indirect test of whether beekeeping efforts to manage and reduce *Varroa* infestations reduce the prevalence and transmission of multi-host pathogens. In turn, this data will be able to inform policy on the maintenance of apiaries in the UK, the transportation of honeybees and commercial bumblebees.

I would like to thank the Genetics Society for providing funding towards the costs of field work, without which my sample size would be much diminished. I’d also like to thank NERC and The CB Dennis Trust for providing further financial support. I’m also grateful to Lena Wilfort for giving me the opportunity and support to carry out this work, Martin I. Jones, Emma Davey and Daisy Gates were invaluable in the field, and Ken Haynes and his lab group for welcoming me into his lab at Streatham campus.
Geometric Morphometrics and Phylogeny course – 6th edition

Mairead Bermingham . University of Edinburgh

Geometric morphometrics is the analysis of shape using Cartesian geometric coordinates rather than areal, volumetric or linear variables. My future research plans include the incorporation of geometric morphometrics and human genetics. I had learned what I knew of the field from reading books and the literature. The ‘Geometric Morphometrics and Phylogeny’ course therefore provided an opportunity to increase my knowledge base and gain some practical hands on experience in this emerging field.

The course has been held annually over the last six years in different locations around Barcelona, Spain. This year the course was held at the premises of the Centre de Restauració i Interpretació Paleontològic, Els Hostalets de Pierola, Barcelona (a village of 2000 people within the Catalonia province of Barcelona). The area is known for its dryland farming; particularly cereal and wine. The beautiful landscape around Els Hostalets de Pierola provided a wonderful setting to explore during my free time.

Fifteen people from across the globe participated in this course, coming from as far afield as the US and Canada. The course ran for five days and Prof. Chris Klingenberg, a world authority in the field of geometric morphometrics was the course instructor. The daily schedule was packed full. Every day we began at 9am with a three hour lecture. Following an hour long lunch break, we convened for a 3-4 hour practical computer session. The lectures and practicals were very well planned and the material covered was current and clearly presented. This course overall provided a general overview of the interface between geometric morphometrics and phylogenetics including the different approaches and methodologies that link the two fields. In my opinion, the best aspect of this course is that we applied what we learned to our own datasets during the practical sessions. I very much appreciated Prof. Klingenberg teaching style, he always took the time to answer our questions, during and after lectures and practical sessions. I especially thank Dr. Soledad De Esteban-Trivigno, the course coordinator, she really looked after us from collecting us in Barcelona, organising accommodation and meals and organising transport back to Barcelona, we had nothing to do but learn which was very much appreciated by all.

The workload was quite heavy; however it was structured in such a way as to not be overwhelming. Nevertheless, despite the busy work schedule, there was still plenty of time to converse with Prof. Chris Klingenberg, Dr. De Esteban-Trivigno and the other participants in a more informal setting during meal times and in the evenings. These conversations are important part of learning in courses like this, as they facilitate a free and open exchange of ideas, provide an opportunity to observe the application of
what we had learned outside our area of expertise, and also foster the development of strong collaborative links for the future. I thoroughly enjoyed my time in the Els Hostalets de Pierola and believe that the training provided by this course has provided me with a solid footing from which to expand my knowledge and practice of geometric morphometrics.

I would very much recommend that others who are new to the field of geometric morphometrics should also consider attending this course. The information about next edition of this course can be found at: http://www.transmittingscience.org/courses/gm/gm-and-phylogeny/

I have recently attended the “Optical Microscopy & Imaging in the Biomedical Sciences” workshop, from the 9th until the 19th of September, hosted at the Marine Biological Laboratory (MBL) in the United States. The purpose of the course is to provide an in-depth introduction to the fundamentals of microscopy and digital imaging to research scientists, post-doctoral trainees and advanced graduate students.

The topics covered by the workshop were divided into five broad themes: a) microscope design, image formation, resolution and contrast, b) microscopy techniques that use the properties of light, such as brightfield, darkfield, phase contrast, differential interference contrast (DIC), which were followed by the principles of fluorescence microscopy, c) latest technology cameras- detection and record information in the form of digital imaging, an understanding of signal to noise ratio, d) the latest advancements in microscopy such as FLIM, FRET, two-photon, SIM, light sheet and e) digital imaging restoration/deconvolution, wide-field microscopy, confocal scanning microscopy etc.

Following our everyday lectures, we greatly benefited from hands-on microscope practice, problem solving and open discussions, which ingrained to our minds the importance of proper setting up and the practical uses of each of microscope technique, depending on the biological question that is to be answered.

The course would not have been possible without the endless dedication, friendship and enthusiasm of the academic staff during the lecture and laboratory sessions. Furthermore, the course would not have been possible without the expertise of the commercial staff from Zeiss, Olympus, Leica, Life Technologies, GE, Hamamatsu and others. They kindly provided and demonstrated the proper setting up and use of the latest technology microscopes and cameras. A big thank you to both the academic and commercial staff, who were always present and helpful during those very late night hours of our problem solving exercises!

I have truly benefited from this workshop. I realised that there are so many parameters when imaging our samples that can be taken for granted or that are not even considered- to the very simplest: which is the most suitable microscope to image my samples? Therefore, the background knowledge provided by the workshop will vastly improve my decisions on the proper microscopy technique to be applied, depending on the experiment, maximise the image quality and therefore save precious
time and effort. It is important to stress that revision of the material covered and post-course practice on microscopes are vital components of the learning process.

To sum up, I would like to thank the Genetics Society for kindly providing a grant to cover part of the workshop’s cost.

It was an excellent opportunity to gain further understanding of the principles of microscopy that will benefit my future career, network with scientists from different disciplines of the life sciences, exchange ideas and experimental observations and set up new collaborations.

I really enjoyed this workshop and recommend it to everyone whose project relies heavily on microscopy.

CTR Placental Biology Course

Dr Claire Dent . Queen Mary University London

The research I have undertaken during my scientific career has focused on imprinted genes. These are genes subject to epigenetic changes resulting in monoallelic expression, and are associated with a number of different neurological and developmental disorders. Having completed a PhD in Neuroscience, my research focused on the role of imprinted genes in the brain and their effect on symptoms of psychiatric disease. Following the completion of my PhD I was lucky enough to continue research in the field of imprinted genes, but this time focusing on their role in the placenta. The placenta is exceptionally important in the field of genetic and epigenetic research, as it is entirely responsible for the healthy development of the fetus. Furthermore the prolific expression of imprinted genes in the placenta has led to the belief that imprinted genes may be responsible for not only the growth and development of the embryo, but programming of later-life diseases.

With little knowledge of the placenta, embarking on my first post-doc position was challenging, to say the least. However my supervisor encouraged me to attend the highly acclaimed ‘Placental Biology Course’, run by the Centre for Trophoblast Research (CTR) at the University of Cambridge, UK. The training program is a week-long, residential course that teaches the biology of the placenta; including the development, morphology and function of the placenta, as well as the genetic and epigenetic mechanisms. The course was attended by 17 participants selected by applications from around the world. The participants therefore represented an extremely diverse group of countries and fields of work; this made the course even more interesting and made for very stimulating dinner conversation!

The course was set in the beautiful surroundings of the University of Cambridge, and delegates were lucky enough to reside at the impressive St John’s College for the duration of the course. The course comprised an intense series of taught lectures including human and mouse placental development, epigenetics, mouse genetic models of placental function and immune genes and placentation; as well as a number of practical workshops including mouse and human placental dissection, bioinformatics, and stereology. Furthermore the course also included a number of workshops that developed transferable skills which are highly important in scientific research, including journal club presentations and grant writing workshops. The impressive list of course instructors and lecturers included some of the most influential scientists in the field of placental biology and epigenetic research, this meant the quality of lectures was incredible, and not just highly informative but inspirational. As a result of attending the course I have improved my knowledge of the placenta immeasurably, I have gained new skills, ideas and inspiration for my research, and importantly now have the confidence to work in the field of placental biology.

I would like to thank the Genetics Society for awarding me a training grant which significantly contributed to the cost of my attendance, I would also like to thank my supervisor Dr Marika Charalambous for actively encouraging me to attend the course, and finally I would like to thank the Centre for Trophoblast Research for allowing me to attend such a fantastic course.
The wrt-2 gene is a member of the warthog gene family (the name was used to show that these genes contain a domain homologous to a domain found in hedgehog genes, which control the formation of paired limbs and organs in animals). The function of wrt-2 in *C. elegans* had not previously been explored, although a different gene of the same family, wrt-5, was studied in detail. Wrt-2 was believed to be a signalling molecule, and that it may undergo protein cleavage and then degradation of the C terminal fragment, like other warthog proteins.

First, I used methods involving bioinformatics to compare wrt-2 promoter sequences and check for sequence homology in *C. elegans*, *C. japonica*, *C. briggsae* and *C. remanei* (the other three are the closest relatives of *C. elegans*). Highly conserved sequences were then tested to check for presence of transcription factor binding sites (using the JASPAR and ModenCode search engines). The actual sequences of the wrt-2 gene were also compared, and introns were aligned as well to check for any possible control elements (none were found). After researching the physiological relevance and expression patterns of these transcription factors in *C. elegans*, I selected 6 transcription factors to inactivate in worms using RNAi (by ingestion of E. coli with RNAi constructs).

These were egl-13, blmp-1, ets-4, brc-1, che-1 and cfi-1. I would use the intensity and presence/location of fluorescence to determine whether a particular transcription factor had a significant effect on driving wrt-2 gene expression.

Initially, we were planning to use a worm strain with a modified gene sequence for wrt-2, with an mCherry (red fluorescent protein) and GFP construct on both ends of the protein. The strain was also mutant for the unc-119 gene (uncoordinated movement of worms). Because of this, and also poor transmission of this modified gene to new offspring, the strategy was changed. An alternative construct was used, where two different GFP proteins were added to the wrt-2 promoter, while wrt-2 itself was removed. One of these GFP proteins associates with the H2B histone, while the other one associates with the cell membrane. Therefore, if one of the transcription factors driving wrt-2 expression (in normal wildtype N2 worms) had a significant effect on wrt-2 expression while inactivated by RNAi, I would see this as decreased GFP expression while looking at the worms under a fluorescence microscope.

RNAi experiments were performed for each transcription factor separately, but three transcription factors (together with controls) were tested in each RNAi run due to the time-consuming nature of taking images of worms, and the fact that worms continue to grow on the plates for the duration of the experiment. Three controls were used each time, the GFP control (knocking out the GFP constructs themselves), the empty vector control without an RNAi construct, and the pop-1 control (used to check whether the RNAi procedure itself was successful; pop-1
is an embryonic lethal mutation, and if the procedure did work, worms could not develop past the egg stage.

The images below show the difference between a successful GFP knockout (adjacent cells show no fluorescence at all, some cells show reduced fluorescence) and a worm where the RNAi did not work (full fluorescence, this was the che-1 TF RNAi experiment).

The RNAi procedure was repeated several times for each transcription factor, and two plates with *E. coli* containing the RNAi constructs were used per transcription factor per run (the same for the controls). The final stage included taking snapshots of worms using imaging software linked to the fluorescence microscope, and images of worms that had ingested transcription factor RNAi constructs were compared to those of worms that had ingested controls to see whether a significant difference could be observed.

It was found that there was no significant difference between the transcription factor RNAi worms and the empty vector (L4440) control worms (this means that GFP was not underexpressed to a significant or uniform extent for any single transcription factor inactivated by RNAi). Based on this, we can assume that no single transcription factor has a significant role in controlling the expression of wrt-2. The next step in continuing the project would need to be testing pairs or triplets of transcription factors together to see whether a number of them, when knocked out together, significantly reduce wrt-2 expression in *C. elegans*.

The aim of my project was to develop a new progenitor line for transgenic experiments where the entire *FLC* locus was deleted from the genome.

Using CRISPR-Cas9 to generate an *FLC* deletion in the Arabidopsis genome

**Student** Olivia Tasker  
**Supervisor** Professor Caroline Dean, John Innes Centre, Norwich

Plants align their flowering to spring/summer conditions to maximise pollination and seed set. A focused effort on the molecular control of flowering time in Arabidopsis has identified multiple pathways involving both promoters and repressors of the floral transition. These pathways monitor different environmental cues and converge to regulate a common set of gene targets that induce the transition of the meristem to a floral fate. A locus central to repression of the floral transition is *FLOWERING LOCUS C* (*FLC*).

*FLC* is the target of several chromatin mechanisms mediating its temperature and developmental silencing. Detailed mechanistic dissection of these *FLC* regulatory pathways has been an important objective of the Dean lab for many years. However, to date the transgenic experiments have introduced transgenes into an Arabidopsis line carrying a loss of function *flc* mutant that carries a large deletion/inversion (*flc-2*). This rearrangement left a 3’ fragment of *FLC* elsewhere in the genome, which complicates analysis of the transgenes. The aim of my project was to develop a new progenitor line for transgenic experiments where the entire *FLC* locus was deleted from the genome.

It was found that there was no significant difference between the transcription factor RNAi worms and the empty vector (L4440) control worms (this means that GFP was not underexpressed to a significant or uniform extent for any single transcription factor inactivated by RNAi).
The new developments of CRISPR/Cas9 provided an opportunity to generate a clean FLC deletion. My work began using four previously made CRISPR constructs. They each contained a Human Codon Optimized Cas9 protein and an AtU6-26 promoter and terminator for the guide RNAs. There were also two different pairs of pairwise combinations of the guide RNAs and Cas9 promoters and terminators. These constructs had already been introduced to Arabidopsis using the floral dip method and T1 progeny had been generated. I continued to screen these lines as well as beginning new transformation experiments using a tissue culture root transformation protocol. We wanted to see if different transformation procedures influenced the frequency of CRISPR deletions being inherited.

Since research in the Jones lab (TSL) and Coupland lab (MP Koeln) had found other promoters and terminators to increase the efficiency of CRISPR/Cas9 targeting I also wanted to incorporate a new Arabidopsis codon optimized Cas9 and a pINCURVATOR2 promoter with agsT terminator in my experiments. I began to create these using golden gate cloning, a technique for easy assembly of gene fragments. I successfully placed the guide RNAs and promoters into a vector and transformed them into E.coli.

By the end of my stay there were positive transformants from both types of transformation experiments and the lab has continued with the work. Completion of the project will hopefully lead to a clean deletion of FLC from the genome and the potential for more experimentation to enhance our knowledge of flowering time.

I would like to thank the Genetics Society for the studentship as well as the John Innes Centre International Summer School Programme. Many thanks to Professor Caroline Dean for the fantastic project and to Dr Rebecca Bloomer, Dr Jo Hepworth and Dr Penny Hundleby for endless amounts of help.

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Novel roles for the exoribonuclease Dis3L2 in cell proliferation

**Student** Oliver Rogoyski  **Supervisor** Dr. Sarah Newbury, Brighton and Sussex Medical School

The gene *dis3L2* is known to encode a highly conserved exoribonuclease (Dis3L2) involved in the cytoplasmic degradation of RNA which is necessary for proliferation of cells. It has been shown that the knockdown of Dis3L2 expression in the wing imaginal discs of fruit fly *Drosophila melanogaster*, leads to development of wings 20% larger than those of wild type flies due to increased cell proliferation. Reflecting this in humans, Dis3L2 has been associated with Perlman Syndrome, a rare condition of overgrowth, and predisposition to Wilm’s tumours in neo-natal babies.

The aim of this studentship was to shed light on the underlying genetic basis of the control of proliferation by Dis3L2, using molecular and genetic techniques, by testing at which stage in larval development Dis3L2 is required to control the final size of the wings.

It was hoped that by using an *engrailed-GAL4 (en-GAL4)* system to drive the knockdown of *dis3L2* by *UAS-dis3L2RNAi* only in the posterior compartment of the wing, an internal control could be generated, allowing quantification of overgrowth without having to normalise wing size to *Drosophila* weight. By combining this with temporal control of knockdown, provided by using a GAL80ts system, knockdown could be induced at timepoints throughout development, and the effect it had on *Drosophila* development could be observed.

The project started by setting up genetic crosses which would produce *dis3L2* knockdown progeny to test the viability of the *en-GAL4* driver we planned to use. Using qRT-PCR on whole larvae, it was shown that the knockdown cross had levels of *Dis3L2* approximately 70% lower than that of the parental stocks. Contrary to
expectations however, measuring the wings of the knockdown flies showed no posterior overgrowth to be occurring in the knockdown Drosophila. By subsequently using the same en-GAL4 system to drive GFP expression, we found that the system was not in fact driving expression in the predicted region i.e. the posterior of the wing disc.

Following this, a second cross was set up, using an alternative en-GAL4 driver, which had previously been used successfully to drive dis3L2 knockdown in combination with GAL80ts providing temporal knockdown control. This system was known however to cause a significant portion of wings to blister to a point where they are impossible to measure. Pre-empting this occurrence, additional crosses were set up, in the hope that flies with the blistered wing phenotype could be discarded, while leaving a sufficient number of non-blistered flies.

Knockdown was temperature induced by transferring the developing flies from 19°C (a temperature that inhibits the dis3L2 knockdown) to at 29°C (allowing the system to drive dis3L2 knockdown), at a series of timepoints from larval stage 2 to pupal stage, with a negative control kept at 19°C, and a positive control kept at 29°C. The positive control group showed a 70% knockdown of Dis3L2 expression compared to the negative control group, indicating that the system was viable for inducing the desired time-controlled knockdown. Unfortunately, severe blistering was induced in 100% of wings of knockdown flies, making them impossible to measure.

Using a third en-GAL4 driver with the GAL80ts system, along with later experiments after the end of my studentship, it was confirmed that the combination of en-GAL4 with GAL80ts causes the occurrence of the observed blistered wing phenotype, for reasons yet unknown.

Therefore, although we confirmed that the GAL80ts could be combined with an en-GAL4 driver to add a layer of temporal regulation to the knockdown, the unforeseen interaction seen in the presence of both together prevents the system from being useful to investigations into wing development. This means that other wing disc drivers, such as nubbin-GAL4 will have to be used despite the fact that they do not provide an internal control.

Due to the time I was able to spend working on this project, my understanding of both the theory, and the practice of molecular biology, genetics, and developmental biology have all grown significantly. The experience has confirmed for me my interest in hopefully pursuing a career in research, and hopefully allowed me to contribute to the work of the Newbury lab group, who I look forward to working with again over the course of my third year research project.

I would like to express my gratitude to the Genetics Society for providing the funding and opportunities that allowed me to develop my research skills and take part in this fantastic project. I would also like to thank Dr Sarah Newbury and Ben Towler, as well as all the other staff and students in the Newbury lab, for their help, support and time spent teaching me, allowing me to make the most of this wonderful opportunity.
Abnormal heart rhythms (cardiac arrhythmias) affect a large percentage of the population (for example, over 2 million people in the UK are affected every year), however they can often go unnoticed. Some can be life-threatening leading to sudden cardiac arrest, and are associated with underlying coronary heart disease or rare inherited arrhythmias. Hence, research into the genetic basis of these abnormalities is clinically important.

The ADAMTS (A Disintegrin and Metalloproteinase with Thrombospondin motifs) family in humans comprises nineteen members, of which six are orphan enzymes. ADAMTS6, one of these orphan enzymes, was the focus of this project. Rare coding variants in this gene were recently found to be associated with a measure of cardiac repolarisation (the time it takes for ions to return to their state during a resting potential, as the cardiac muscle relaxes). It was identified in an exome chip based association study in a large population of healthy individuals. Very little is known about the role and function of this gene in cardiac conduction and therefore my project aim was to investigate this in an in vivo model.

Zebrafish (Danio rerio), originally from the River Ganges in India, are
good model organisms due to their transparency, fast development, cheap maintenance and minimal space requirements. They are especially suitable for heart studies as they share a similar heart physiology to humans but are simpler with only two chambers rather than four, and one valve instead of two. Zebrafish can survive up to 5 days without a functioning heart, thereby allowing investigation of cardiac abnormalities which lead to a non-functioning heart.

The gene of interest, ADAMTS6, is predominantly conserved between zebrafish and humans, with 75% conservation for nucleotides, and 83% for amino acids.

In order to investigate the function of ADAMTS6 I knocked down the gene expression using morpholinos. There are two types of morpholino, both 25 bases in length: ATG and Splice. The ATG morpholino binds over the translation start codon, thus blocking translation of the entire gene. Contrastingly, the splice morpholino binds to the exonic and intronic DNA of a splice site thus preventing correct splicing of the pre-mRNA. The most likely outcome is nonsense-mediated decay at the point of translation although the splice morpholino is generally associated with less severe gene knockdown as it has the potential to produce a truncated protein.

Zebrafish eggs were collected, made easier by the external nature, and the yolk microinjected with a morpholino. The morpholino concentration was optimised to balance a good survival rate with a common phenotype. Survival counts were taken at 6, 24, 48 and sometimes 72 hours post fertilisation (hpf). Dechorionation (removal of the embryo from the chorion) was performed soon after 24hpf to eliminate this affecting the tail curvature. Embryos were anaesthetised prior to imaging at 48hpf in methylcellulose to give a clear, still image. Gross morphological changes were investigated by light microscopy.

However no conclusive defect was observed. Some anomalous phenotypes were observed in single fish but these did not occur at a level sufficient to be linked to the morpholino injection. Heart rate was also measured between the wild-type and morphant fish but again no statistically significant difference was observed. A cardiac phenotype in fish cannot be excluded until cardiac conduction investigations have been performed using the electrocardiogram (ECG).

Further research could also focus on using another type of genetic modification technique such as CRISPR to insert human ADAMTS6 mutations into zebrafish. It is also possible that within such a large gene family, different isoforms may play different functional roles between species.

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This placement was a wonderful eye-opener to in vivo research and the practices employed to reduce the ethical issues involved (in particular the 3R’s: replace, refine and reduce). I learnt an incredible amount and was fascinated by the potential of genetic investigations using zebrafish embryos. The workshop was an amazing bonus as it provided both the opportunity to present our work and to meet other like-minded people, of a similar age, from other universities.

I would like to say a very heartfelt thank you to the Genetic Society for funding this placement, and to Dr Yalda Jamshidi for giving me the opportunity to work as part of her group. I really enjoyed the challenges it presented and the introduction to lots of new techniques. I would also like to say thank you to Evmorfia Petropoulou, Jaipreet Bharj and Dan Osborn for their help during the project.
Genetic analysis of AGMO-1: a mystery enzyme in *C. elegans* lipid ether metabolism

**Student** Francesca Donnellan  
**Supervisor** Jonathan Hodgkin, University of Oxford

**Alkylglycerolmonooxygenase-1** (AGMO-1) is an enzyme involved in the degradation of ether lipids; however, its exact physiological role remains uncertain. Previous work in *C. elegans* has shown *agmo-1* mutant nematodes have fragile cuticles with different phospholipid composition to wild type. This presumably accounts for the fact that *agmo-1* worms are also hypersensitive to bleach and detergent, and, most interestingly, are resistant to the *C. elegans* bacterial pathogen *Leucobacter Verde1*. Previous work from the Hodgkin lab has shown that this resistance can be attributed to an inability of the Leucobacter Verde1 bacteria to adhere to the *agmo-1* cuticle. The primary aim of this studentship was to investigate the role of this enzyme further, principally by conducting a forward genetic screen to identify potential suppressor mutations of the detergent sensitivity phenotype of *agmo-1* worms (suds mutations).

Three allelic variants of *agmo-1* worms (e3016, e3019, e3047) were chemically mutagenized with EMS and both F1 and F2 generation screens were conducted by placing populations of worms on plates containing detergent (0.007% SDS). After approximately one week only *agmo-1* worms that were detergent resistant, i.e. worms with potential suds mutations were still alive. These worms were selected and crossed with a mapping strain. This facilitated the possibility of identifying dominant suds mutations by testing the detergent sensitivity of the heterozygous progeny of these crosses. None of the suds candidate worms tested had dominant suppressor mutations. After re-testing the suds candidates by placing them on detergent plates for a second time, the *agmo-1* genes of two of the most detergent resistant suds candidate worms were sequenced. Neither showed any change in the *agmo-1* sequence suggesting that the suppressor mutations were not intragenic or *agmo-1* revertant mutations. After sequencing these two candidates, further stronger suds candidates were identified from subsequent screens. Further mapping and sequencing of these and the other candidates will be required to identify the loci and nature of the suds mutations.

In addition to the genetic screen a drug sensitivity assay was conducted to assess whether the *agmo-1* mutants had a more permeable cuticle than wild type worms. Worms in M9 buffer were treated with nicotine, ivermectin or phenoxypropan-2-ol and time to paralysis was measured. *Agmo-1* (e3016 and e3019), *bus-5* (br19) and *bus-17* (e2800) worms were paralysed significantly more quickly than N2 (wild-type) worms. *Bus-5* and *bus-17* worms have previously been shown to be sensitive to all three drugs using the same assay. The three drugs have different structures: sensitivity to all three suggests that *agmo-1* worms have a generally more permeable cuticle than wild type worms. This finding is consistent with the observation of the bleach and detergent hypersensitivity phenotypes of *agmo-1* worms. Further corroboration could come for example from Hoechst staining.

In summary, after screening over twenty thousand mutagenized genomes using SDS selection, seven SDS resistant strains carrying candidate suds mutations were isolated. In addition, it was confirmed that *agmo-1* worms are drug sensitive suggesting that they have more permeable cuticles than wild-type worms. With further investigation, these suds mutants should reveal more both about the function of AGMO-1 and interactions between the worm and its environment via the cuticle surface.

Many thanks to the Genetics Society both for funding the project and for running the workshop for all of us receiving summer studentships: it was a fabulous way to round off a summer of hard work and scientific endeavour. I would also like to thank my supervisor Professor Jonathan Hodgkin for all his much appreciated support and teaching throughout my project. I am also very grateful to all the other members of the lab: they made the experience extremely enjoyable and were always happy to help.
Can torsional stress in genomic DNA directly regulate RNA polymerases co-transcribing a DNA Sequence?

Student Claire Brown. Supervisor Dr Christoph Baumann, University of York

DNA supercoiling is a fundamental process involved in DNA replication, transcription and recombination. Genomic DNA is a topologically constrained molecule with dynamic regions of overwound and underwound DNA. Using a novel single-molecule manipulation technique, horizontal magnetic tweezers (MT), it is possible to probe the supercoiling-dependent mechanics of DNA transcription.

In vivo, DNA has an average superhelical density ($\sigma$) of -0.05, indicating an excess of underwinding or negative supercoiling which facilitates transcription initiation. Previous studies have shown that translocation of an actively transcribing RNA polymerase leads to local increases or decreases in DNA torsional stress (twin-supercoiled domain), which cannot be resolved in vivo due to interactions of the template DNA, nascent RNA and RNA polymerase with the crowded cellular environment. Local changes in DNA supercoiling are biologically relevant as they have been shown to regulate transcription initiation at promoters located downstream.

Single-molecule biophysical studies allow for the manipulation and visualisation of a molecular system in vitro, which can be systematically probed for the purpose of in-depth analyses. To achieve this, horizontal MT can be combined with epi-fluorescence imaging to track RNA polymerases co-transcribing DNA under defined torsional stress in real-time. Horizontal MT were chosen for their compatibility with epi-fluorescence illumination.

This technology offers the capacity to directly probe transcription as a function of applied torsion. Horizontal MT enable the relative extension and linking number of the DNA tether to be manipulated, whilst the relative positions of RNA polymerases are viewed indirectly by annealing fluorescently-labelled DNA oligonucleotides to the nascent RNA chains.

This project involved the validation and optimisation of a previously devised protocol for the production of a DNA construct that can be torsionally constrained (Figure 1). $\lambda$ phage DNA is ligated to handles labelled with digoxigenin or biotin in both strands. The handles are labelled during PCR amplification using digoxigenin or biotin labelled dUTP. Each handle is then digested using the same restriction enzymes as the respective end of the $\lambda$ DNA-construct to generate complementary sticky ends. The handles are ligated to each end of the $\lambda$ DNA-construct with T4 DNA supercoiling is a fundamental process involved in DNA replication, transcription and recombination. Genomic DNA is a topologically constrained molecule with dynamic regions of overwound and underwound DNA. Using a novel single-molecule manipulation technique, horizontal magnetic tweezers (MT), it is possible to probe the supercoiling-dependent mechanics of DNA transcription.

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DNA ligase. The digoxigenin handle is bound by a 9μm diameter microsphere through anti-digoxigenin IgG. The biotin labelled handle is bound to a streptavidin-functionalised M280 paramagnetic bead. This yields a single DNA tether in which the twist can be manipulated using horizontal MT.

Additional work was carried out to optimise the sample chamber construction so that the applied force was sufficient to extend the DNA molecule while allowing collapse due to changes in DNA winding to be observed. To do this, a range of mechanical modifications were made to the horizontal MT, including a new motor assembly that actively spins the magnets to add or remove twist to torsionally-constrained DNA, new slide holders and a new air cushioned vibration-free table. Research was also undertaken into sample preparation methods to minimise non-specific interactions that prevent stable DNA tether formation. A combination of acetylated bovine serum albumin and tRNA was found to be most effective in terms of limiting non-specific interactions at the chamber surfaces. These modifications were described in an article published in Methods in Molecular Biology on which I was a co-author.

DNA tethers were frequently observed and manipulated. Tether collapse induced by DNA supercoiling was observed. Through image analysis, it was possible to show tether shortening by 1.03 μm, which is a 4.2% reduction in the relative extension of the DNA. This is a highly significant result and demonstrates the possibility of probing the effects of torsional stress in DNA at the single-molecule level using horizontal MT.

Throughout this project, the conditions have been optimised to enable the production of DNA tethers that are capable of being supercoiled. From here, transcription-induced supercoiling and the effects of supercoiling on the rate of RNA polymerase translocation can be studied, as well as other biological processes that are supercoiling-dependant.

I would like to thank the Genetics Society for funding this research and enabling me to develop highly specialised skills in molecular biophysics. I am indebted to my supervisor, Dr Christoph Baumann, for his help and support during the studentship. I look forward to contributing to the completion of this research.

Analysis of the synthetic Spindle Assembly Checkpoint arrest

Student Eleni Papachristoforou . Supervisor Prof. Kevin Harwick University of Edinburgh

Introduction
The spindle checkpoint is a mechanism which acts as a surveillance system and ensures that during mitosis segregation of replicated chromosomes to the opposite spindle poles are correct. The spindle assembly checkpoint (SAC) senses the interactions between kinetochores and spindle microtubules and if any defects are detected it delays the onset of anaphase by inhibiting the Anaphase Promoting Complex (APC). Unattached kinetochores or ones that lack tension induce the SAC which blocks the cell cycle and allow time to sort incorrect attachments. SAC proteins identified in budding yeast(S. pombe) screens including Mad1, Mad2, Mad3, Bub3 as well as the conserved kinetochore kinase Mps1. SAC proteins are responsible for generating the Mitotic Checkpoint Complex (MCC) which consists of Mad3, Cdc20 and Mad2, which inhibits APC/C-Cdc20 activity.

Activation of the spindle checkpoint
Spc7 and Ncd80 are core kinetochore components that form a scaffold for checkpoint protein recruitment. The molecular mechanism of their recruitment and activation is not yet clear. A ‘tension-sensing’ model proposes that Aurora B kinase works as a ‘kinetochore stretch sensor’. It loads and activates Mps1 kinase at kinetochores and their activity is widely conserved in activating the recruitment of SAC components forming a scaffold.
Lab and Project Background
The interest of the certain lab I was involved was how MCC assembles and then functions away from the KTs. A synthetic array with Tet Operators at the arms of the chromosomes was made in S. pombe. An active form of Spc7 was tethered to the array and shown to be sufficient in recruiting Bub1, Bub3 and Mad3 to the TetO. However, it was not sufficient to activate the checkpoint or to cause a checkpoint dependant arrest. It has recently been found that when one of the checkpoint kinases was co-tethered with Spc7 on the array, then the checkpoint arrested cells.

Aims and Results
Using this array some questions were raised on how the synthetic signalling-checkpoint functions. My first approach was to cross in various checkpoint mutants, such as mad3A. After performing this cross and confirming it with the use of PCR and fluorescent microscopy techniques I came up with the result that yeast cells did not arrest and this indicates that Mad3 was necessary. Mad3 binds to the Bub1/Bub3 complex and then with Mad2 at the KTs can form the MCC which inhibits APC. Generally, when Mad3 is absent the synthetic arrest is not observed. I also built various strains to test the importance of other protein kinases (Bub1, Aurora etc.) in generating/amplifying the synthetic checkpoint arrest. These strains are now being thoroughly tested in the lab.

Conclusion
Working in the lab for about 8 weeks it was mutually beneficial as I had the opportunity to consolidate, develop and expand laboratory methods and techniques that I have acquired through my practical’s curriculum. Part of the methods I have been using in the lab include yeast genetic crosses where I had to cross two known strains and select for the right outcome, molecular genetics technique which involves PCR used when wanted to find out whether the protein of interest was present, western-blotting as to confirm fusion protein expression and lastly live-cell imaging with the use of fluorescent microscopy. These have given me a full understanding on why, when and how to apply each technique which is an important skill especially for someone like myself that really looks forward for a future career in research. Furthermore, while working with others I have particularly strengthen my understanding of team dynamics, as well as developing my team working skills and the ability to follow rules. I soon mastered the ability to prioritise tasks through flexible, structured planning to meet various deadlines. In addition, I was given the chance to attend and experience how group meetings work, listen to research presentations and institute scientific seminars. This undoubtedly helped to realise and identify some of the important skills that a scientist must acquire including determination, enthusiasm, passion and commitment.

Last but not least, being involved for 8 weeks in a research project I have explored the working life of the lab, comprehend the general principles of synthetic biology and improve my laboratory skills. Essentially, these kinds of work have given me an invaluable experience and an insight of my future career. It has provided me with enough information and knowledge so to make right decisions regarding my future academic plans.

Developmental profile of alternative isoforms of Cdkl5, a gene associated with severe brain disorders

Student Kyriaki Savva
Supervisor Dr. Mark Bailey, University of Glasgow

The cyclin-dependent kinase-like 5 (CDKL5) gene is located on the X chromosome at position Xp22. Patients with mutations in CDKL5 are characterised by intellectual disability, early-onset seizures with infantile spasms and severe epileptic encephalopathy. The clinical picture resembles Rett syndrome (RTT) to an extent. RTT is also an X-linked disorder and is associated with mutations in MECP2. The overlap of phenotypes amongst individuals with pathogenic mutations in either of the two genes is supported by recent studies that have placed CDKL5 and MeCP2 in the same molecular pathway. However, the molecular characterisation of CDKL5 and its functions is at an early stage. CDKL5, formerly known as STK9 (Serine Threonine Kinase 9), contains a kinase domain, but its other roles and the function of the remainder of the protein are unclear.

CDKL5 is approximately 240 kb in size, with at least 24 exons. Exons 2-22 contain the coding regions, but there are variable 5’- and 3’-UTRs encoded by a variety of 5’ and 3’ exons, and alternative splicing within the coding region also occurs. There are currently six known human CDKL5 mRNA isoforms and three mouse isoforms. Isoform m1 in mouse and h1/h11a in human are
Lipid Tissue Mini Kit and the RNA was quantified using a Nanodrop spectrophotometer. RNA quality was assessed using the Bioanalyser.

Three samples of good-quality RNA from mouse brain at each sampled developmental stage subjected to cDNA synthesis and RT-PCR using primers designed to amplify each specific isoform being assessed. No-RT and No Template Controls (NTC) were used, and two control gene transcripts (ACTB and HPRT) were also amplified. Results showed that isoform 1 (m1) is strongly present at early embryonic timepoints (E13 and E17), with expression decreasing later in development (E20, P1 and P7) and being almost absent in adult mice (P42). Isoform 2 (m2) is present at all developmental stages while isoform 3 (m3) is absent early in development (E13, E17 and E20) but appears postnatally and reaches its highest expression level in adult mice (P42).

qRT-PCR was subsequently carried out on RNA samples from E13, E20, P1 and P42. ACTB was used as a housekeeping gene control. Serial dilutions were prepared for the standard curve and also no-RT and NTC controls were used. All of the samples were run in triplicate (technical replicates). The results confirmed the previous results from the RT-PCR. We therefore conclude that expression of Cdkl5 isoforms is under developmental regulatory control, which may have implications for our understanding of the genotype-phenotype pathway in CDKL5 disorders.

The aim of this project was to investigate how this mRNA isoform tissue-specificity in the adult mouse is achieved, by characterising the expression of the different Cdkl5 isoforms in the mouse brain at different stages of development. This was achieved by performing a series of semi-quantitative RT-PCR experiments to estimate the relative levels of each mRNA isoform at each developmental stage. This was then followed by quantitative RT-PCR of some samples.

Brain tissue (whole brain) from mice was collected at embryonic stages E13, E17 and E20, and cortical/forebrain samples were collected at postnatal time points P1, P7, P21 and P42 (adult). Tissue samples were placed immediately in RNAlater, which stabilises and protects the RNA. Purification of total RNA from the brain tissue was carried out using the RNeasy Lipid Tissue Mini Kit and the RNA was quantified using a Nanodrop spectrophotometer. RNA quality was assessed using the Bioanalyser.

Validating weighted burden gene based association tests via simulated studies

Student Martin Kelemen. Supervisor Prof Dave Curtis, University College of London

One of the most difficult problems in genome wide association studies originates from the fact, that usually, the number of variants tested far exceeds the number of samples. This then results in a lack of statistical power due multiple testing, which means that the standard \( \alpha \)-threshold of 5% may yield a large number of false positives: assuming just a 1000 variants, 50 ‘significant’ false positives are expected.

One solution is to apply multiple testing correction, such as Bonferroni or FDR, which will then either greatly reduce power and/or will mean that amongst the hits we would still have to accept a certain number of false positives.

To overcome this challenge, one of the more successful strategies are gene based (burden) tests. In short, these combine all variants within one gene, which will increase power as all variants have been pooled into a...
sample t-test. Its main innovation is that its weighting scheme follows a parabola that permits weights to fall off more gradually, which then allows us to be able to fine tune its parameters.

However every time parameters are user defined rather than being driven by the data, the danger exists that if those parameter values are arbitrarily set, this may amplify signal purely as a function of those user-defined parameters and not reflect underlying biological reality.

My work involved assessing this analytical method using simulated datasets and exploring the sensitivity of such a gene based pooling strategy to different parameters to determine if there were any such weaknesses.

My project consisted of simulating Crohn’s disease studies from the 1000 genomes project’s PIII VCFs’ NOD2 gene via the following procedure (performed by a Java app I’ve written):

All haplotypes were pooled, then two were randomly picked to generate an individual.

As the dataset did not include phenotype data for Crohn’s disease, these had to be inferred by checking how many known risk alleles (Lesage et al, 2002) these generated subjects carried by calculating a total LOR and assigning them into either case or control groups based on the resulting probability. This step was repeated until we’ve had enough individuals for both groups.

In addition, we’ve also considered both common and rare variants, where rare variants were digitally ‘inserted’ onto the chromosomes, if they were not originally present in the dataset.

These datasets were then exported for analysis in SCOREASSOC and finally the results were read back and plotted in R. What we were particularly interested in were the percentage of the results that achieved significance (p<10-6) and of course, if there were any anomalous results. This entire procedure was repeated hundreds of times on a cluster with various weights, variant consequence settings and sample sizes.

The results were reassuring: the method proved robust against various parameters and not easily disturbed by different arbitrary weights. In our particular project, a frequency weight of at around 10 and above, with some extra weight (~10) assigned to missense variants generating the best results. The only important limitation of my work was that we could not consider variants with a protective effect due to time constraints.

In conclusion, my project was a success and gave me invaluable work experience in my chosen area of statistical genetics/bioinformatics, and will surely improve my CV once I start applying for Phd positions later this year.

Thanks go out to my supervisor Prof Curtis, Dr Plagnol and the rest of the Phd students/post-docs in our lab and of course, my project would not have been possible without funding from the Genetics Society, to whom I am especially grateful.
See the relevant web pages and downloadable Funding Application Forms at www.genetics.org.uk

One-off Meeting Sponsorship

Purpose

Sponsorship of genetic research meetings not organised by the Genetics Society.

The Genetics Society receives several requests from members each year to sponsor meetings in the field of genetics. These meetings are usually one-off meetings with an ad hoc organising committee and may be partly sponsored by another Society. The guidelines below indicate a review process for applications and the conditions that must be met for the award of Genetics Society sponsorship.

Review of applications

1) Members may make applications at any time visiting the following website: http://gensoc.fluidreview.com/
2) The application will be circulated to the full committee for review. The review will cover suitability of the meeting for Genetics Society sponsorship and level of support requested.
3) The committee will be asked to respond within two weeks and the Society aims to respond to requests within four weeks.

Conditions of sponsorship

4) Several levels of sponsorship are possible: (a) single lecture: £200 (b) session: £500-1000 (c) major sponsor: £1500-2000.
5) Genetics Society sponsorship must be mentioned in all pre-meeting publicity (e.g. posters, flyers, website) and in the meeting programme. If the Genetics Society is the major sponsor the meeting should be advertised as a “Genetics Society-sponsored meeting”.
6) Details of the programme of the meeting and registration forms should be sent as far in advance as possible to theteam@genetics.org.uk, for inclusion in the Society’s newsletter and on the website.
7) A short report on a meeting that receives sponsorship of £1000 or more, for possible publication in the newsletter and on the website, should be sent to theteam@genetics.org.uk within one month of the conference taking place.
8) Genetics Society sponsorship may be used at the organiser’s discretion, but budget travel and accommodation options should normally be insisted upon. Any unused grant should be returned to the Genetics Society. The Society will not be responsible for any losses incurred by the meeting organisers.
9) An invoice for the grant awarded should be submitted to theteam@genetics.org.uk. The grant may be claimed in advance of the meeting and no longer than one month after the meeting.
10) The meeting organisers agree to make details of how to apply for Genetics Society membership available to non-members attending the sponsored meeting. Meetings that receive maximum sponsorship will be expected to offer a discounted registration fee to Genetics Society members to encourage non-members to join the Society at the same time. New members may then attend at the discounted rate, once confirmation of their application for membership of the Genetics Society has been received from the Society’s Office.
New Sectional Interest Groups

Purpose

Regular sponsorship of genetic research meetings on particular themes. Regular (e.g. annual) funding is available for genetics research communities who wish to run regular series of meetings. Current examples include Arabidopsis, the Population Genetics Group and the Zebrafish Forum.

Members may make applications for new Sectional Interest Groups at any time. Applications should be submitted on the GS Funding Application Form and emailed to theteam@genetics.org.uk using message subject ‘New Sectional Interest Group’ and your surname. The award of Genetics Society support will be subject to review of applications by the committee and subject to the following conditions.

1) The sponsorship of the Genetics Society must be mentioned in all pre-meeting publicity (e.g. posters, flyers, website). It should also be acknowledged in the meeting programme booklet. It is understood that wherever possible, the meeting should be advertised as ‘A Genetics Society Meeting’, however, where the Society’s financial contribution support is only partial, and where this formula of words would conflict with the interests of other sponsors, it is acceptable for the meeting to be advertised as a ‘Genetics Society-Sponsored Meeting’.

2) Details of the programme of the meeting should be made available to all Genetics Society members via the Society’s newsletter, and electronic copy should be sent as far in advance as possible to the newsletter editor, at the latest by the advertised copy date for the newsletter preceding the close of registrations for the meeting. The same details will appear on the Genetics Society website. This information should include the programme of speakers, the topics to be covered, plus details of how to register for the meeting.

3) A report on the meeting, once it has taken place, should be submitted for publication in the newsletter, which is the official record of the Society’s activities. This should be sent as soon as possible after the meeting to theteam@genetics.org.uk, and should include brief factual information about it (where and when it took place, how many people attended and so on), together with a summary of the main scientific issues covered.

4) Genetics Society funds may be used to support speaker travel, accommodation, publicity or any other direct meeting costs, at the organizers’ discretion. It is understood that budget travel and accommodation options will normally be insisted upon. Any unused funds should be returned to the Society. The Society will not be liable for any financial losses incurred by the meeting organizers. Any profits should be retained solely for the support of similar, future meetings, as approved by the Society.

5) A written invoice for the agreed amount of Genetics Society sponsorship should be forwarded to theteam@genetics.org.uk, no later than one month after the meeting date. Funds may be claimed in advance of the meeting, as soon as the amount of support has been notified in writing.

6) Meeting organizers may levy a registration charge for attendance at the meeting as they see fit. However, it is understood that Genetics Society members will be offered a substantial discount, so as to encourage non-members wishing to attend to join the Society at the same time. The meeting organizers agree to make available to non-member registrants full details of how to apply for Genetics Society membership, such as appear on the website and in the newsletter, and may charge such persons the same registration fee as charged to members, upon confirmation from the Society’s Office that their application and remittance or direct debit mandate for membership fees has been received.

7) The meeting organizers are free to apply to other organizations for sponsorship of the meeting, as they see fit. However, organizations whose policies or practices conflict with those of the Genetics Society should not be approached. In cases of doubt, the officers of the Genetics Society should be consulted for advice.
New Sectional Interest Groups (continued)

8) If the meeting is advertised on the Internet a link to the Genetics Society website (www.genetics.org.uk) should be included.

9) For those groupings holding their first such meeting with Genetics Society support, it is understood that the Society’s support for future meetings of the series will be decided on the basis of the success of the first meeting, including adherence to all of the conditions listed above. The first meeting is hence supported on a pilot basis only.

10) The meeting organizers will nominate a responsible person who will liaise with the Genetics Society on all matters relating to the meeting, and whose contact details will be supplied to the Society’s Office. This person will inform the Society if he/she resigns or passes on his/her responsibility for the meeting or series to another person, whose contact details shall also be supplied.

Junior Scientist Grants

Purpose
To support attendance at genetics research meetings by junior scientists. In this section, junior scientists are defined as graduate students and postdoctoral scientists within two years of their PhD viva.

Travel and accommodation to the Genetics Society meetings
Grants up to £150 are available for travel and essential overnight accommodation costs to attend all Genetics Society meetings, including the Genetics Society’s own bi-annual meetings and meetings of our Sectional Interest Groups. The cheapest form of travel should be used if possible and student railcards used if travel is by train. Airfares will only be funded under exceptional circumstances.

How to apply: For the Genetics Society’s own Spring and Autumn meetings, applications should be submitted online (https://gensoc.myreviewroom.com) before the registration deadline of the meeting.

For meetings of our Sectional Interest Groups (e.g. Arabidopsis, Population Genetics Group, Zebrafish Forum), junior scientist travel claims should be submitted on the GS Funding Application Form at any time and emailed to theteam@genetics.org.uk using message subject “Travel to GS meeting” and your surname.

There is no limit to the maximum frequency at which the grants can be awarded for attending the Genetics Society meetings.

Travel, accommodation and registration cost at other meetings
Grants of up to £750 to attend conferences in the area of Genetics that are not Genetics Society meetings (including sectional meetings) are available to junior scientists.

How to apply: Please visit the website https://gensoc.myreviewroom.com in time for one of the quarterly deadlines (1st day of February, May, August and November). The application must be accompanied by a supporting statement from the applicant’s supervisor or head of department, which must be uploaded via the online application form before the deadline.

Other conditions: Recipients of these grants will be asked to write a short report that may be included in the newsletter. A maximum of one grant per individual per two years will be awarded.
GRANT SCHEMES

Training Grants

Purpose
To support attendance at short training courses.

Grants of up to £1,000 are available to enable members to go on short training courses in the area of Genetics research. Eligible expenses include travel, accommodation, subsistence and tuition fees.

How to apply: Applications should be made online via the Genetics Society Grants application site. Deadlines are bi-monthly (1 February, 1 April, 1 June, 1 August, 1 October and 1 December). To apply please visit the website https://gensoc.myreviewroom.com.

Closing date: awards will be announced within two months of the closing date. A maximum of one Training Grant per individual per three years will be awarded.

Heredity Fieldwork Grants

Purpose
To support field-based genetic research and training.

Grants of up to £1,500 are available to cover the travel and accommodation costs associated with pursuing a field-based genetic research project or to visit another laboratory for training. The research field should be one from which results would typically be suitable for publication in the Society’s journal Heredity. The scheme is not intended to cover the costs of salaries for those engaged in fieldwork or training, or to fund attendance at conferences.

How to apply: Applications should be made online via the Genetics Society Grants application site. Deadlines are bi-monthly (1 February, 1 April, 1 June, 1 August, 1 October and 1 December). To apply please visit the website https://gensoc.myreviewroom.com.

A panel of members of the Genetics Society committee will review applications including both information on the student and the proposed project. Feedback on unsuccessful applications will not be provided. Awards will be announced within two months of the closing date.

Other conditions: Only one application from any research group will be admissible in any one year. Recipients of these grants will be asked to write a short report within two months of completion of the project that may be included in the newsletter. A maximum of one grant per individual per three years will be awarded.
Genes and Development Summer Studentships

Purpose

To support vacation research by undergraduate geneticists.

Grants of up to £2,350 are available 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.

Applications must be made by Principal Investigators at Universities or Research Institutes. The application must be for a named student. 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, and the student does not have to attend a UK university.

How to apply: there is one closing date of 31st March each year. The student’s tutor or equivalent must also send a reference. Undergraduate students who wish to do vacation research projects are encouraged to seek a PI to sponsor them and to develop a project application with the sponsor. Both the PI and the student involved must be members of the Genetics Society.

The studentship will consist of an award of £200 per week for up to 8 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.

A panel of members of the Genetics Society committee will review applications including both information on the student and the proposed project. Feedback on unsuccessful applications will not be provided.

Other conditions: Recipients of these grants will be asked to write a short report within two months of completion of the project that may be included in the newsletter.
The Genetics Society

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.

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.

Medals and Lectures
The Mendel Medal, named in honour of the founder of modern genetics, is usually given on alternative years at a Genetics Society Meeting by an internationally distinguished geneticist.

The Society also awards the Genetics Society Medal, the Mary Lyon Medal, Balfour Lecture and JBS Haldane lecture on an annual basis. Winners of the Genetics Society Medal and Balfour lectures present their lecture at a Genetics Society Meeting.

International links
The Society has many overseas members and maintains links with genetics societies in other countries through the International 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.

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.
Please complete this form and return it, along with your cheque, Direct Debit instructions or credit card to The Genetics Society, c/o Portland Customer Services, Charles Darwin House, 12 Roger Street, London, WC1N 2JU. 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: ___________________________

Email: ___________________________

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. 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 membership 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 from the membership charge for full and postgraduate members paying by Direct Debit

4. 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 the web site. In addition, after one year full membership you can apply for a grant 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 degree: ___________________________

Signature of Head of Department/Supervisor ___________________________

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

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

Complete this membership form and a Direct Debit mandate form, which can be downloaded from our website and send them to the address below.

I wish to pay by Direct Debit (tick box if applicable). **Paying by Direct Debit entitles Full members and Postgraduates to a saving of £5.00 from the price of their membership.** Direct Debit Membership Subscriptions are renewed on an annual basis.

**Option 2: Cheque/Bank transfer**

I enclose a cheque for the sum of £____ made payable to Portland Customer Services.


To facilitate identification please confirm:

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<th>Your transfer reference</th>
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**Option 3: Credit/Debit Card**

I wish to pay by Credit Card.

Credit Card Type: Visa □ Mastercard □ Switch □

I authorise Portland Customer Services to use the credit card details below to pay my membership fees.

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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 where 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: theteam@genetics.org.uk.

Signature of nominating FULL MEMBER Print name in block capitals Membership No.

I do not have a signature of a nominating member. 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, Portland Customer Services, Commerce Way, Colchester CO2 8HP, UK marking your envelope MEMBERSHIP APPLICATION.

Please note that the approval of new members is ratified at the Spring Meeting as part of our AGM. However, your membership will begin as soon as your application is processed.
Notification of change of address form

If you wish to notify us of a change of address, you can use our online facility by visiting www.genetics.org.uk or by emailing us at theteam@genetics.org.uk. Alternatively you can complete the form below and return it to:
The Genetics Society, c/o Portland Customer Services, Charles Darwin House, 12 Roger Street, London, WC1N 2JU marking your envelope CHANGE OF ADDRESS NOTIFICATION.

Note that from ___________ my new address will be:

Title:  Prof. ☐ Dr. ☐ Mr. ☐ Miss. ☐ Mrs. ☐ Ms. ☐
(Print or Type)

Last Name: ___________________________ First Name: ___________________________

Institution: ________________________________________________________________

Address: ___________________________________________________________________

Postcode: ___________________________ Country: ________________________________

Telephone: __________________ Fax: __________________________

Email: ________________________________

Previous address: ____________________________________________________________

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