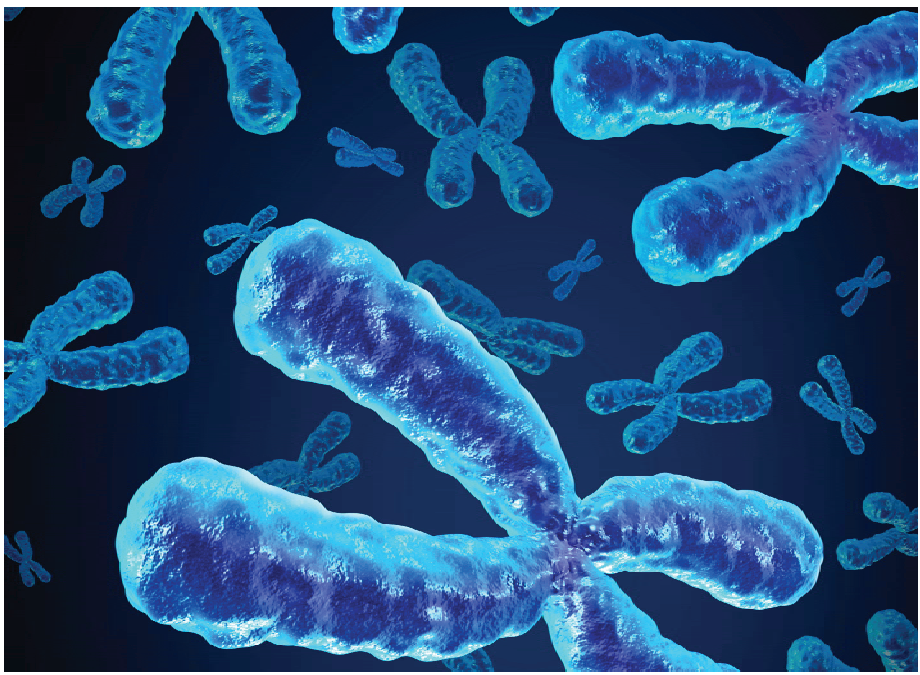
ISSN 2397-9917

Issue 3
Winter 2016-2017

Galton Institute

Exploring Human Heredity

The Galton Review



www.galtoninstitute.org.uk

CONTENTS

Front Cover Image: Depositphotos.com

Published by:

The Galton Institute, 19 Northfields Prospect, London, SW18 1PE

Tel: 020 8874 7257 www.galtoninstitute.org.uk

General Secretary: Mrs Betty Nixon
executiveoffice@galtoninstitute.org.uk

Review Editor: Mr Robert Johnston

EDITORIAL

The highlight of the Institute's year is always the Annual Conference and this year's event proved to be extremely popular with over 250 delegates in attendance. A full report of the proceedings can be found on page 4.

The Galton Lecture is, in turn, the highlight of the Conference and the 2016 edition was especially noteworthy as it was the 100th in the series. It was first given in 1914, and there has since been an almost unbroken run, with only three omissions in all that time. The list of speakers reads like a veritable 'who's who' of eminent social and biological scientists who have been associated with the Galton Institute. Many were experts of world renown, including three Nobel laureates, and all considered themes which were of great significance at the time.

In 1943 Sir William Beveridge delivered his Galton Lecture on the very day the 'Beveridge Report' was being debated in the House of Commons. There was William Bateson, the man who coined the word 'genetics', who spoke in 1921. Julian Huxley had two Galton lectures to his name; 1936 and 1962. John Maynard Keynes, the most influential economist of the 20th century gave perhaps the most famous Galton Lecture of all in 1937, the year after his magnum opus, *The General Theory of Employment, Interest and Money,* was published. His lecture was entitled 'Some economic consequences of a declining population'.

To this esteemed list can be added this year's lecture on 'Nutrition and Lifespan' by the premier expert on biogerontology, Dame Linda Partridge. A video of her inspirational lecture can be viewed on our website.

Finally, I should also like to draw your attention to the latest *Genetics in Medicine* booklet on precision medicine produced by our 'Manchester Team' of Helen Middleton-Price, Andrew Read and Dian Donnai. It can be seen on our website or is available as a hard copy from Betty Nixon.

Robert Johnston

The Galton Institute Annual Conference
Environmental Factors in Gene Regulation
16 November 2016 at the Royal Society

This year's conference attracted over 250 delegates to listen to experts in the field of 'gene regulation'. The full programme is available on our website. It was organised by the President, **Professor Veronica van Heyningen, FRS** and **Dr Branwen Hennig.** The President started proceedings with a brief account of the aims and activities of the Galton Institute. She described the various activities we undertake including publications, conferences, prizes and grants. (For details please see www.galtoninstitute.org).

Dr Hennig chaired the first session in which **Professor Sir Peter Ratcliffe** from the University of Oxford and the Francis Crick Institute in London discussed **'Oxygen sensing and hypoxia signalling'**. He is particularly interested in the role of oxygen in tumour growth. The transcription factor Hypoxia-inducible factor (HIF) plays an important role in cellular response to systemic oxygen levels in mammals and many processes are associated with regulation of HIF.


Professor Sir Peter Ratcliffe, FRS

HIF is overexpressed in many human cancers and this overexpression is heavily implicated in promoting tumour growth and metastasis through its role in initiating angiogenesis and regulating cellular metabolism to overcome hypoxia. Professor Ratcliffe questioned whether HIF activation causes cancer and he described various experiments to test this. HIF rarely mutates but it seems that genetic predisposition may shift the HIF pathway to become 'more oncogenic'. The HIF pathway is under strong selection once activated by the VHL switch. He finished by suggesting that cancer cells should be easier to kill as they're 'living on the edge' but there may be many other pathways affected. These discoveries pave the way for translational research and development of targeted therapies.

The second session was chaired by **Professor David Burt** and the first speaker was **Professor Akhilesh Reddy** from the University of Cambridge and the Francis Crick Institute in London**.** He spoke on **'Regulation of circadian clocks by redox homeostasis'**. Circadian rhythms are not driven by the environment but are endogenous, being re-set by the environment when appropriate. They allow organisms to anticipate regular changes in light, temperature etc. He noted that the mechanisms involved vary dramatically between plants and animals suggesting that they evolved independently but he wondered whether this is true.

Professor Akhilesh Reddy

It is now known that these 'internal clocks' are in every cell and are not simply located in places such as the suprachiasmatic nucleus in the hypothalamus of the brain in humans. Indeed isolated cells of many types in the laboratory can be shown to 'keep time'. These clocks have a profound effect on many aspects of metabolism in all animals. The classic explanation is the negative feedback oscillator system but Professor Reddy questioned whether this actually drives the rhythm. He showed that in fact redox circadian oscillations are pervasive across life, from bacteria to humans, and described how peroxiredoxin proteins are the drivers of such rhythms. This may have important implications for human health as it has been shown that people subjected to frequent jet-lag or disrupted shift patterns are at a greater risk of developing some cancers.

The second talk of this session was on **'Circadian regulation of innate immunity and inflammation'** by **Dr Julie Gibbs** from the University of Manchester. She described how all immune cells have intrinsic clocks including 'resident' immune cells. Healthy clocks are needed for healthy immunity. She described various experiments using LPS as a test substance and found that microphage response is gated by internal cellular clocks. She found that club cells in lungs show rhythm of cytokine production at different times of the day and that they are affected by glucocorticoid signalling but only when the clock is functioning. She went on to consider whether such clocks affect long-term inflammation in

conditions such as rheumatoid arthritis. Studying similar conditions in mice, it has been found that symptoms are worse during daylight hours even when the mice are subjected to constant light, ie a circadian rhythm is acting. She also observed an increase in the number of circulating T-cells, microphages, neutrophils and fibroblast-like synoviocytes during daylight hours, which may explain how symptoms may fluctuate during the day.

Dr Julie Gibbs

Adaptive immunity is a growing field and it has been found that it is subject to circadian rhythms with levels of B and T cells fluctuating. This work should eventually lead to a search for more effective therapeutic targets and a better understanding of how time of day may influence the effectiveness of vaccine and drug delivery.

The session after lunch was chaired by **Professor Dian Donnai**. **Dr Amanda Drake** from the University of Edinburgh spoke first on **'Early environmental regulation of glucocorticoid receptor gene expression'**. Her talk focussed on how pre- and post-natal environments influence disease risk in later life. It has been known for some time that a high birth weight leads to an increased risk of hypertension, type 2 diabetes, hypothalamic–pituitary–adrenal axis activation and neurodevelopmental disorders. Genes, maternal size, maternal nutrition and glucocorticoids given antenatally all influence this. More recent work with rats has shown that a rise in cortisone results in reduced birth weight probably by causing epigenetic dysregulation by increased methylation of DNA or histone modification.

Dr Amanda Drake

Dr Drake went on to consider how such knowledge can help us to address the major problem of 'preterm babies' which also leads to health issues in later life including hypertension, glucose intolerance and various neurodevelopmental conditions. She

concluded with the concern that there may be intergenerational effects with such epigenetic conditions passed on to children and grandchildren.

The second talk of the session was by **Professor Michèle Ramsay** from the University of Witwatersrand in Johannesburg and concerned how **'Foetal exposure leads to altered gene expression'**. She began with the prediction that over the next 30 years, there is expected to be a huge rise in the number of women of childbearing age across Africa. The major medical concerns for many women in South Africa are exposure to alcohol and maternal obesity. Foetal alcohol syndrome has become worryingly common in South Africa and can lead to changes in foetal facial morphology, reduced birth weight and neurological disorders. It is a complex trait which is affected by a number of environmental factors but is also influenced by genetic predisposition. Work in mice has revealed that epigenetic mechanisms are involved but also that effects may be due to damage of placental DNA rather than DNA in the embryo itself.


Professor Michèle Ramsay

Maternal obesity is becoming increasingly widespread in South Africa and has in turn caused a steep increase in gestational diabetes. The increase in maternal blood glucose causes epigenetic changes, largely de-methylation, in a significant number of genes in both mother and infant and these may turn out to be critical to the future of the child. There are however very positive developments in the field with regard to the expansion of genetic and genomic analyses of African populations. Given greater genetic variability in such populations, combined with many more large-scale consortium-led research undertakings, real headway is being made towards a better understanding of complex traits.

The final session was chaired by the President, Professor Veronica van Heyningen, FRS, and began with **the 100th Galton Lecture** given by **Professor Dame Linda Partridge, FRS** from University College London and Max Planck Institute for the Biology of Ageing in Cologne. The topic of her lecture was **'Nutrition and Lifespan'**. Dame Linda began with a few pertinent questions: Is there a limit to lifespan? If living conditions and health care continue to improve, why shouldn't

longevity increase? Is ageing a disease? Is ageing the major risk factor for many fatal diseases? She admitted it is difficult to study ageing in typical laboratory model organisms and many of the longest-lived animals are rare in the wild eg the ocean quahog (an edible clam) examples of which have been aged at 507 years. Human longevity has been shown to be associated with the insulin/insulin-like signalling and target of rapamycin (IIS/TOR) network. This regulates lifespan and reproduction, as well as metabolic diseases, cancer, and ageing.

Professor Dame Linda Partridge, FRS

As long ago as the 1930s, it was known that 'somewhat' undernourished rats have extended lives and are healthier in many tissues. But dietary restrictions also have negative effects. In *Drosophila*, they extend lifespan but reduce egg laying. The key ingredient to fecundity appears to be the amino acid methionine but its addition does not reduce lifespan. Dame Linda also described differences in the ageing patterns of male and female fruit flies.

Finally, investigations into drugs to prolong lifespan were considered. *Rapamycin*, an immunosuppressant used following organ transplantation, has been found to extend lifespan in mice and fruit flies by inhibiting mTOR activity. *Trametinib* also increases lifespan in fruit flies by suppressing a pathway regulated by Ras. *Lithium*, again in fruit flies, seems to extend life in low dosages by blocking GSK-3. Further work is being undertaken to investigate whether combinations of these three drugs would be more effective and may also cancel out each other's side effects.

At the conclusion of the lecture, the President presented Dame Linda with the Galton Plate as commemoration.

The final talk was given by **Professor Anne Ferguson-Smith** from the University of Cambridge on **'Parental nutrition can modify gene expression in the offspring'**. She began with an overview of epigenetic mechanisms including chromatin folding, packaging of DNA around nucleosomes, covalent modification of

histone tails (acetylation, methylation, phosphorylation) and DNA methylation, and the influence of regulatory small or long non-coding RNAs on gene transcription. Some specific questions raised included how much these epigenetic markers vary and whether their effects can be observed across generations. An intergenerational model of mice fed either a full or restricted calorie diet has been used to see if the variable phenotype of differences in glucose handling were transmitted to the F2 generation.

Professor Anne Ferguson-Smith

The viable yellow Agouti mouse provides a classic model of metastable epialleles, identical alleles that are variably expressed due to epigenetic modifications of the Av locus during embryogenesis. Therefore a group of such mice may be genetically identical but can differ in fur colour from light yellow to dark brown due to epigenetic modifications that are established very early in development. The extent of DNA methylation can be dependent on maternal nutrition, as demonstrated with the Agouti mouse model, and other environmental factors such as drugs or toxins given to the mother. The effects may result from the insertion of a murine retrotransposon upstream of the transcription start site of the Agouti gene. With regard to transgenerational inheritance there are two re-programming events of epigenetic markers before embryogenesis starts but it appears that several loci may not be re-programmed in such a way and may provide the sites for transgenerational memory. Alternatively, there may be endogenous polymorphic retrotransposons that could contribute to such a memory. This suggests that epigenetic gene regulation serves as a link between nature and nurture.

We thank all the speakers of 2016 Galton Institute Conference for an inspiring programme and Betty Nixon for her tireless management of all organisational matters.

Robert Johnston
David J Galton

THE ARTEMIS TRUST

The Birth Control Trust (BCT) of the Galton Institute was established in 1977 with capital derived in part from the estate of Marie Stopes and in part provided by the Institute itself. Its objectives were very narrowly defined in line with the title of the Trust itself. As a result of the many changes in the last four decades in social attitudes, socio-economic circumstances, reproductive technologies, and medical knowledge and practice, the Institute Council considered that the time had come to widen the objectives of the Trust to enable it to play a more effective and wider role in the very different conditions of the twenty first century, whilst maintaining much of the spirit underlying the BCT.

Following a submission to the Charity Commission, new objectives for the Trust have been approved as follows:

To preserve and protect the physical and mental health of people, particularly but not only those from poorer communities, in particular by:

a) assisting in the provision of fertility control and other measures to improve reproductive and sexual health; and

b) advancing education in all aspects of reproductive and sexual health

The name, Birth Control Trust, being clearly no longer appropriate, the Council has resolved that the new name should be **The Artemis Trust** drawing on the knowledge that, in the Greek pantheon of deities, Artemis was deemed to be a goddess with special responsibilities for women and childbirth .

In line with the new framework, the Trust will be making grants to applicants who are considered qualified to organise and execute high quality projects which demonstrate progress towards one or both of the Trust's objectives. The dates of grant availability and application forms will be placed on the Galton Institute website.

John Beardmore

The Alzheimer's Association International Conference 2016 24 – 28 July 2016 Toronto, Canada

Thanks to the generous bursary from The Genetics Society and **The Galton Institute**, I was lucky enough to travel to the vibrant city of Toronto in order to attend The Alzheimer's Association International Conference 2016. This 4-day event is the world's largest dementia research conference and provides a platform where the worldwide dementia research community can meet every year to discuss the very latest findings in the field.

The first thing that struck me about this conference was its size; with a vast array of talk options on offer, four booklets worth, not to mention the 200,000 square feet of exhibition space which would host 1,700 posters over the course of the conference. I knew this event would be big, but this was a scale beyond my imagination.

During the first session of the conference, I had the opportunity to present a poster of my research. My PhD project aims to investigate how the protein encoded by *BIN1*, a susceptibility locus for Alzheimer's disease, functions in brain endothelial cells that comprise the blood-brain barrier. With the sheer number of posters on display, coupled with the early start, potential jet-lag and it being a pre-coffee session, I wasn't expecting a huge audience! However, to my pleasant surprise, I had a constant stream of researchers coming to discuss my work. It was a fantastic experience for me to engage with scientists from all over the world, many of whom were authors of some of the key papers I had studied. While I was daunted by this prospect at first, everyone who I spoke to was both supportive and encouraging of my work, posing intriguing questions and also providing suggestions for future studies which I found extremely valuable.

Many of those I spoke to during the poster session were presenting talks themselves which I could then attend to learn about their own research. I had an interesting conversation with **Dylan Kwart** (Rockefeller University) who presented an immense amount of work on creating induced pluripotent stem cell (iPSc)-derived neurons which harboured familial Alzheimer's disease mutations

through gene editing technology. Having been involved with iPSc differentiation at the start of my PhD, I understood first-hand the difficulties such studies presented and it was very impressive to listen to what his lab had achieved.

The oral presentations were divided into several themes encompassing a broad variety of interests, from molecular and cell biology to neuropsychology and clinical therapeutics. This meant there was always something interesting to attend and I often found myself struggling to decide between sessions as a 15 minute walk between halls didn't really allow for session jumping! However, this often encouraged me to stay for presentations that weren't necessarily within my specific research area, allowing me to discover new avenues of interest. As a cell biologist, it was refreshing to learn about clinical approaches to research in addition to insights into other forms of dementia and how there is much overlap in terms of the basic mechanisms involved in different pathologies.

The programme of plenary sessions provided a diverse array of speakers from different backgrounds. Of particular interest for me was Professor John Hardy's lecture entitled "*Genomic Analysis of Neurodegeneration Gives Clues to Pathways to Selective Cell Loss: The Hypothesis of the Catastrophic Cliff of Neuron Failure".* **Professor Hardy** (University College London) firstly described the various genetic analyses that have been carried out in neurodegenerative research, including several studies conducted by our group in Cardiff. He then discussed the idea of selective vulnerability shown in different brain regions to different neurodegenerative diseases and proposed a 'catastrophic cliff' theory whereby each neuronal cell type is closer to different types of catastrophic cliffs, depending on their functions. I thought this was a very interesting proposition, drawing together what we know from genetics, cell biology and clinical phenotypes, which may lead to new approaches in dementia research.

While the extensive scientific program was both fascinating and inspirational, it was the overall ethos of the conference that made it such a motivating experience for me. The organisers truly made this meeting an absolute spectacle; from the stunning welcome reception featuring themed rooms depicting the seasons of Ontario complete with circus performers, musicians and dancers to the CN tower completely illuminated in purple to mark the occasion. Many of the local people we met while exploring the city, whether it be at the Toronto Blue Jays baseball game

or the local country music bar, were fascinated when we described our reason for visiting. Most, if not all, went on to mention how they had personally been affected by Alzheimer's disease.

This repeatedly reminded me how important each and every piece of research presented at the conference was to provide hope for patients and their families. I feel honoured to be able to participate and witness such cutting-edge research and this conference has provided much personal motivation as I progress into the final year of my PhD.

I would like to take this opportunity to again thank both the Genetics Society and the **Galton Institute** for making this experience possible.

Anna Burt
Cardiff University

Gordon Research Conference, 23-24 July 2016, Hong Kong

Celebrating its second birthday, the GRC conference took place on the mesmerizingly scenic campus of the Hong Kong University of Science and Technology situated at the coastline of the New Territories. The conference was focused on latest developments in the field of genomic instability.

Run for the first time, the seminar was designed to relieve the burden of the main conference to accommodate all the speakers and allow students and postdocs to present their work in a more relaxed and informal environment. Run mainly by the students themselves, it was a roaring success, igniting stimulating discussions.

The seminar opened with the keynote speech from **Thanos Halazonetis** who spoke about DNA replication stress in cancer and the relevant repair pathways. He started off with the general overview of the cancer progression and how genomic instability leads to cancer, with the focus on p53 inactivation, in order to refresh the concept in the audience's jet-lagged minds. He spoke about his research progressing from the cell lines to the human tissues, mainly focusing on the double stranded breaks at the common fragile sites caused by the replication stress. The second part of his talk covered base excision repair, break induced replication and the role of Rad52. The seminal sessions included emerging trends

in DNA repair, as well as chromatin dynamic and DNA replication. I had the pleasure of presenting my work on one of the replication-associated mutational processes that might have a role in increasing mutation burden at protein binding sites in germline cells.

Additional panel discussion on personal-professional life balance in academia and industry involved **Anindya Dutta**, **Eric Lightcap** and **Thanos Halazonetis** talking about how they reached their current positions, giving advice on which mistakes to avoid and what should be learned along the way.

The main GRC conference began on the Sunday evening with the arrival of many more jet-lagged, but excited attendees. The session on telomere replication and stability commenced with **Rodger Greenberg** giving a talk on the mechanisms of ALT, a telomere extension pathway alternative to that executed by telomerases, and telomere recombination. He talked about the work done to try and monitor the multi-step process of homology-directed DNA repair at ALT and if the 'break-induced replication' is identical to 'canonical replication'.

Jan Karlseder focused on more novel unpublished research done on the CYREN protein (Cell Cycle Regulator of non-homologous end joining), a cell cycle regulator of the pathway choice. **Rodger Reddel** then spoke about the role of shelterin in signalling telomere-induced growth arrest. Subsequent sessions covered such topics as chromosome cohesion, condensation and segregation, double-stranded repair and recombination, challenges on the replication fork, replication of normal and damaged DNA templates, chromatin dynamics and epigenome maintenance, DNA repair in development and aging, DNA damage signalling and DNA repair as a therapeutic target.

Lars Jansen gave a talk on histone variant inheritance and assembly at the centromeres, presenting his work on CENP-A protein, its turnover and deposition on the daughter strands during replication, and highlighted differences in turnover rates between centromeric and non-centromeric locations. Using time-ChIP, a novel approach that combines pulse labelling and chromatin immunoprecipitation, he was able to show that enhancers and promoters have a higher rate of nucleosome turnover. In one of the following talks, **Huilin Zhou** focused on SUMO as a master regulator of chromosome biology in relation to chromosome segregation, gene

silencing and chromosome translocations. **Jessica Woodward** presented her recent work on regulation of condensing II during mitosis, showing that it correlates with S phase kinetics during lymphocyte development. **Yoshinori Waranabe** spoke about the role that the Shugoshin (SGO1) protein plays in protection of centromeric cohesion in human mitotic cells and its importance in chromosome segregation. **Xuebiao Yao** gave a very nice talk on molecular delineation of CDK -TIP60-AuroraB signalling axis in chromosome segregation, focusing on the interactions of the TIP60 and AuroraB proteins. **Karen Wing Yuen** delved into the discussion of the epigenetic regulation of centromere propagation and establishment in yeast, rather than humans. She focused on the interactions between the CENP-A and RbAp46/48 proteins and also talked about the centromere formation from scratch. **Hong Wang** presented work on the cohesion SA1 and SA2 subunits, their abilities to bind single- and double-stranded DNA and preference to the centromeric or telomeric functions and rates of their diffusion.

Another session of note was titled 'Power hour' and was led by passionately driven **Bik Tye**. Designed to address challenges women face in science, it prompted an active discussion of how the problems could be approached, and how they are dealt with in different parts of the world. While it did not solve issues, it felt like an important step towards a world of more equal opportunities. The session did not solely confine to the discussion of what kind of challenges woman face, but also included the issues that ethnic minorities encounter on their scientific career path.

Conference concluded with an authentic banquet, followed by a grand round of applause, in recognition of all the hard work of organisers **Anindya Dutta** and **Ania Groth**. They promised, among other exciting things, karaoke for the next meeting in two years' time… Not to be missed!

I would like to give a huge thanks to the organisers of both the seminar and conference, and of course the **Galton Institute** and the Genetics Society for the generous grant that has allowed me to take part in this great meeting.

Lana Talmane
Institute of Genetics & Molecular Medicine, University of Edinburgh

16th World Congress on Pain, International Association for the Study of Pain, 26- 30 September, 2016. Yokohama, Japan

These world congresses are held every two years and are the largest meetings in the field of pain study. In this edition, around 7,000 professionals came together from around the globe to share new advances in pain research and development.

As a PhD student in the final write-up stage of my thesis, this was an important experience to meet new professional contacts, extend my knowledge and to share my research on the involvement of epigenetics during pain development.

During my first day at the venue, I attended a refresher course on the topic of *Pain Genetics and Epigenetics*. There, the organizers educated the audience on the major advances in the epigenetic field, in the context of pain research. This helped me gain knowledge and ensure that all relevant data was included in the introduction and discussion of my thesis. Following the course, I had the opportunity to interact with the speakers and thus establish important connections for future projects.

On the third day, my PhD supervisor lead a workshop titled *Epigenetics and the Development of Prolonged Pain*, where he presented data from my thesis to the audience. It was really rewarding to see my experiments being shared and explained in great detail. It also gave me the opportunity to hear the opinions and comments from fellow researchers in the field, which helped me to prepare for my future VIVA exam and ensure the meaningfulness of my data.

Additionally, I presented a poster regarding the involvement of epigenetics following a burn-injury model. Since my poster was presented one day after my PhD supervisor's workshop and due to the interest on the topic, a great number of researchers came to ask further questions and discuss future progresses on the topic. Another poster, on a similar line of investigation, was presented the following day by a former BSc student that I supervised. In that poster, where I was also included as author, she presented complementary data that we gathered during her time in the lab.

Attending the 16th World Congress on Pain also allowed me to listen to plenary lectures and topical workshops on different pain relevant subjects, as well as giving me the opportunity to meet renowned scientists and interact with colleagues. This kept me updated on my line of investigation and helped develop my professional network.

Overall, this was a fascinating meeting and a great opportunity to progress in my career as a researcher. It was only possible thanks to the award of grants from the Genetics Society and the **Galton Institute** to whom I am most grateful.

Jose V. Torres-Perez
Imperial College London

The African Society for Human Genetics Conference 15-17 May 2016

The AfSHG met for the ninth time as a Society on the African continent, this time in the historic city of Dakar in Senegal, the most westerly city in Africa. A total of 210 delegates gathered and represented 26 nationalities including Senegal, Mali, South Africa, Congo, DR Congo, Benin, Cameroon, Burkina Faso, Zimbabwe, Emirates, Morocco, Ethiopia, Nigeria, Uganda, Tanzania, Sudan, Tunisia, Switzerland, Rwanda, Ghana, Egypt, Botswana, USA, UK, Canada and France.

The theme of the conference was ***"Strengthening Human Genetics Research in Africa"*** which is central to the mission of the Society. In the absence of extensive research on the genetic contribution to diseases in diverse African populations, it would not be possible to offer appropriate genetics services or to offer the hope of a precision medicine approach on the continent. This meeting brought together members from country-specific Societies of Human Genetics from Cameroon, Democratic Republic of Congo, Mali and Southern Africa. Many African countries do not have Departments for Human Genetics as a stand-alone discipline and therefore we need to be creative in nesting our discipline and activities in existing structures in our tertiary institutions, hospitals and health care sectors and to promote its contribution to patient care.

The meeting was preceded by the Young Researchers Forum, which is a one-day

meeting for the students to present their work and to have an opportunity to meet with established researchers as mentors and for inspiration in their careers. There were generous prizes for the best oral and poster presentations. The guest speaker was **Professor S. Magueye Gueye** from Senegal who spoke on *Cancer in Africa: challenges and perspectives.*

The first day of the meeting was jointly hosted as a collaborative venture with the 8th Human Heredity and Health in Africa (H3Africa) Consortium meeting (http:www.h3africa.org) and included a panel discussion on *"Funding sustainable genomic research in Africa"* that provided much food for thought. The breaks brought opportunities for rigorous debate about the state of genetic services across the continent and sharing ideas about advancing the discipline and expanding services for the benefit of African patients and their families. The ethics sessions and talks on community engagement highlighted the ingenuity of the researchers in conveying the content of their research to communities that do not have a vocabulary for explaining genetic and genomic concepts.

Since the conference brought together the Human Genetics Communities from across Africa to a Francophone country in order to invigorate the discipline and to provide a platform to present and share research findings, we spared no cost on simultaneous translation for French and English to ensure that everyone would benefit in both languages, this proved to be a great success. The Conference was opened by the Chancellor of University Cheikh Anta Diop of Dakar, **Professor Ibrahima Thioub**. **Dr Charles Rotimi** (founding President of the AfSHG) gave a thoughtful keynote address titled: *"The African Society of Human Genetics: looking back to shape the future"*. It was an inspiration to be reminded of the beginnings of our Society and the considerable challenges we have faced along the way. The scientific sessions included cancer genetics and genomics, medical genetic services, genetics of infectious diseases, human population genetic diversity and health, genomic medicine and the ethics of genomics approaches to patient care and research. Two mini-symposia were held during the meeting one on *Human non-human system studies: TB genomics* and one on *Disorders of sex development*. In addition, there were two training workshops on *Research leadership* (hosted by Nature Genetics) and *Next generation sequence data analysis in com-*

plex traits. The formal deliberations were concluded by the Director General of Research, Ministry of Higher Education and Research Senegal, **Professor Cheikh Becaye Gaye**. Further details of the meeting can be found at: http://www.afshgdakar2016.org/. It was a wonderful opportunity for participants to engage, participate and learn from one another as peers, mentors, colleagues and friends. We were united in our quest to explore the treasure trove of African genetic diversity and to promote an understanding of the genetic contribution to health and disease in African populations with a view to improving health on the continent.

The goals of the African Society of Human Genetics are to expand genetic and genomic research in Africa, to include all regions of the continent; to integrate the work of the African Society of Human Genetics with the activities of other related societies; to increase avenues of inter- and intra-continental collaboration in human genetics research; to increase awareness of human genetics and genomic research and promote the development of effective public policy regarding this research in Africa; and to promote the translation of genetic knowledge into clinical practice throughout Africa. More information on the AfSHG can be found at http://www.afshg.org/

The Society wishes to thank **The Galton Institute** for generously supporting the meeting and making it possible for us to host such an excellent and well attended conference.

Michèle Ramsay, President: African Society of Human Genetics
Rokhaya Ndiaye Diallo, Chair: Local Organising Committee
Ambroise Wonkam, Chair: International Scientific Committee

Post doctoral travel grant

The Galton Institute post doctoral travel grant, is available to outstanding post doctoral researchers, normally within 6 years of receiving a doctoral degree, working in the field of genetics.
The Fellowship, which will be up to £6,000, aims to support visits to carry out research into aspects of human inheritance in laboratories abroad 'to enrich the research experience and help develop the scientific career of the Fellow'. The duration of the Fellowship needs to be well justified and requests for up to 6 months will be considered. Applications will also be considered for attendance at advanced, intensive, high quality laboratory-based courses, e.g.: at Cold Spring Harbor, Woods Hole and similar centres. Full details of the grant can be found on our website.

THE GALTON INSTITUTE

Conferences 2017

Surveying Galton's Legacy

Wednesday 15 November, 2017

To be held at

The Royal Society
6-9 Carlton House Terrace
London, SW1Y 5AG

Advances in Genetics: a second teachers' conference

Wednesday, 28 June, 2017

To be held at the
Nowgen Centre
29 Grafton Street
Manchester M13 9WU

This is a genetics up-date for A-Level biology teachers

Admission to both conferences is free, but strictly by ticket available from:

The General Secretary at: **executiveoffice@galtoninstitute.org.uk**