The 6th edition of the *Science* magazine *Dance Your Ph.D.* contest

The American Association for the Advancement of Science (AAAS), the publisher of the *Science* magazine, has announced the 6th edition of *Dance Your Ph.D. Thesis* contest. For the past 5 years, *Science* has sponsored *Dance Your Ph.D.* competition to see how international scientists can express their graduate work through an interpretive dance. The latest edition of the contest was announced on May 16, 2013 and the submission deadline passes on October 1, 2013.

Participants are to create a dance inspired by their Ph.D. research. It does not matter if they are currently Ph.D. students, or if they obtained the degree 50 years ago. The performance can be a solo, a duet, or as the whole lab group, but the author of the Ph.D. thesis must be a part of it. The dance should be recorded and the link with the video should be posted on www.vimeo.com. A panel of judges, who are scientists and artists, will score each Ph.D. dance based on three parameters: scientific merit, artistic merit, and creative combination of science and art. Entrants will be classified into one of four categories based on the scientific field of their Ph.D. thesis: physics, chemistry, biology, and social sciences. The winners of each category will receive an award of $500. The category winner receiving the highest total score awarded by the panel of judges will win the grand prize of an additional $500.

The winner of the contest in 2012 was Peter Liddicoat, a materials scientist at the University of Sydney (Sydney, Australia), who was encouraged to enter the competition by his boss. Liddicoat's Ph.D. topic was *Evolution of nanostructural architecture in 7000 series aluminium alloys during strengthening by age-hardening and severe plastic deformation*. Explaining a scientific Ph.D. thesis to nonscientists is never easy, even with words, but after 6 months of preparations and the help of dozens of friends, he turned his Ph.D. into a burlesque artwork. The performance employs juggling, clowning, and a big dance number, representing the crystal lattices that he studied with atomic microscopy.

Many useful tips for video preparation, FAQs and specific rules of the contest are available at http://gonzolabs.org/dance.

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Common genetic pathway of Parkinson's disease and melanoma

The association of Parkinson's disease (PD) with melanoma has raised much interest in recent years (reviewed by Pan et al. (2011) in the *International Journal of Cancer*). Although PD and melanoma are two distinct diseases that result from abnormal signaling by apparently opposite mechanisms that cause either cell degeneration or over-proliferation, respectively, the link between these diseases has been demonstrated in a number of epidemiological studies. PD is caused by the loss of melanin-positive dopaminergic neurons, and PD patients have been found to have two times higher melanoma risk. Moreover, individuals with cutaneous melanoma have two times higher risk of PD, and PD in these patient has a more severe clinical course. The genetic background of this correlation has been recently elucidated by the group of Prof. Nadem Soufir from Département de Génétique, Hôpital Bichat-Claude Bernard, and Université Paris Diderot, Sorbonne Paris Cité, Paris, France. Their work was presented by Hui-Han Hu (INSERM U976 Centre de Recherche de la Peau and Université Paris 7, Paris, France) during the annual conference of the European Society of Human Genetics in Paris, France, on April 8-11, 2013.

PARKIN (*PARK2*) gene encoding Parkinson protein 2, E3 ubiquitin protein ligase (parkin) is a component of a multiprotein complex that mediates the targeting of substrate proteins for proteasomal degradation. A mutation in this gene leads to a genetic predisposition to PD, but *PARK2* has been described as acting as a tumor suppressor gene as well. Therefore, Hu and colleagues investigated the role of *PARK2* in melanoma susceptibility and oncogenesis in a large cohort 500 melanoma patients and 24 melanoma cell lines. Hu et al. (2013) searched for the point mutations and copy number variations

VIEWS and NEWS section is prepared by Joanna Przybył

(CNVs) in *PARK2,* and examined PARKIN expression and its impact on cell proliferation in melanoma cell lines. The authors identified 15 inactivated *PARK2* alleles leading to a truncated protein in 16 melanoma patients. Additionally, they demonstrated *PARK2* CNVs in 60% of cell lines, and loss of heterozygosity (LOH) in half of the cell lines. PARKIN was absent in 90% of the cell lines but it was present in normal melanocytes. An introduction of a wild type *PARK2* expression clearly inhibited cell proliferation in three melanoma cell lines.

This study has demonstrated a common genetic pathway that could explain the epidemiological association between PD and cutaneous melanoma, because *PARK2* inactivation apparently plays an important role in the melanoma predisposition and oncogenesis. These results provide a new insight into a cutaneous melanoma oncogenesis that could be considered in the development of a targeted therapy for a subset of melanoma patients, and which may have dermatological clinical implications in PD.

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Exploiting bacteriophages for bioscience, biotechnology and medicine **conference**

EuroSciCon Ltd. (Barnet, United Kingdom) is organizing the *Exploiting bacteriophages for bioscience, biotechnology and medicine* conference on January 23, 2014. The event will take place in the Cineworld: The O2 in London, United Kingdom. This will be the 5th conference focused on this topic, which is held in a biennial series.

Bacteriophages play crucial roles in driving the adaptive evolution of their bacterial hosts, which is achieved through the predator-prey relationship of the phage-bacteria interaction and through the adaptive impacts of lysogeny and lysogenic conversion. Phages are the source of many biochemical reagents and technologies applied in modern molecular biology. Furthermore, they are being exploited in other areas of biotechnology, including diagnostics, prophylaxis and food microbiology. In the recent years, phages have also been considered for use for therapeutic purposes as a natural alternative to antibiotics. The rise in the incidence of antibiotic resistance in bacterial pathogens, coupled with the very low rate of emergence of new, clinically useful antibiotics, provides strong rationale for the potential utility of phages in treating human and animal diseases. Prospective applications of bacteriophages in basic research, and medical and industrial biotechnology will be discussed at the meeting.

The conference will be chaired by Professor George Salmond, who is a research group leader in the Department of Biochemistry at the University of Cambridge (Cambridge, United Kingdom). His laboratory is focused on selected aspects of molecular microbiology, including the biology and exploitation of bacteriophages. Professor Salmond has an interest in isolating novel phages from the natural environment for the development of genetics and functional genomics of diverse bacteria, including plant, animal and human pathogens. His research interest is also focused on how bacteria evade the potentially lethal impacts of viral infection via phage abortive infection systems.

The deadline for abstract submission for oral presentation is October 10, 2013. Abstracts for poster presentation only can be submitted up to two weeks before the event.

Information about invited speakers and registration is available at the conference website: http://www.regonline.co.uk/builder/site/Default.aspx?EventID=1061419.

Mouse Models of Disease: Using pathology techniques to enhance phenotyping outcomes **conference**

Mouse Models of Disease: Using pathology techniques to enhance phenotyping outcomes conference will be held on February 5-7, 2014 at the Wellcome Trust Genome Campus (Cambridge, United Kingdom).

The meeting aims to attract veterinary and human pathologists, along with scientists working with mouse model systems in academic and industrial research institutions, to discuss the use of pathology and related *in vivo*

techniques for phenotypic analysis of mouse models of disease. The program will be focused around three sessions: *Evaluation of embryos and neonatal mice*, *Characterising neurological and associated phenotypes* and *Evaluating haematopoietic and immune systems*. Sessions will cover the normal background anatomy, and development, sampling and analytical methods relevant to these areas, including imaging techniques. The conference will also address the use of histopathology to define morphological phenotypes and distinguish them from common spontaneous changes in the mouse strains. Confirmed speakers are renowned scientists from the European, American and Canadian institutions.

Abstracts should be submitted no later than December 3, 2013 and registration deadline without abstract submission is January 6, 2014. Event details may be accessed at https://registration.hinxton.wellcome.ac.uk/display_info.asp?id=352.

Publication of HeLa genome and transcriptome causes privacy controversy

We have recently reported HeLa genomic and transcriptomic data [*BioTechnologia* vol. 94(1) 2013]. HeLa cell line was derived from cervical cancer tumor specimen, obtained without consent from Henrietta Lacks who died in 1951. After the article describing HeLa genome was published, relatives of Henrietta Lacks expressed concern that the publication of genetic data from HeLa cells may affect their privacy.

Surprisingly, the family of Henrietta Lacks did not know about the cultivation of the cells until 20 years after her death, when scientists began using her children in research without their knowledge. Later their medical records were released to the press and published also without consent. Now, the publication of HeLa genome without family's consent has generated controversies again. Technically, the permission of Lacks' family was not required for the publication, but the granddaughter of Henrietta Lacks claimed that this is private family information and should not have been published without family's consent. The authors of the publication apologized and took the data off-line, however at least 15 people have already downloaded complete data before the withdrawal. The authors and *G3: Genes|Genomes|Genetics* editors are currently working on the mutual agreement with the Lacks family regarding the availability of the data.

The European Molecular Biology Laboratory (EMBL) which produced the data, has stated that the HeLa cell line sequenced in the study has spent decades in labs, dividing and thus undergoing mutations and changes, and consequently it is very different from the original cells that started growing in 1951. Moreover, they claimed that the DNA of cancer cells is different from that of the patient and the sequenced genome contains a combination of genetic variants originating from the donor's genome, variants that arose during the tumor development, and variants that occurred during the years of evolution of the cell line. The EMBL has also declared that the goal of this study was not to gain insight into Mrs. Lacks' personal biology, but rather to provide a resource for researchers using HeLa cells. However, the EMBL acknowledges that it is possible to make predictions about Henrietta Lacks' or her descendants' genome based on these data.

Rebecca Skloot, the author of *The Immortal Life of Henrietta Lacks*, has also published her opinion regarding this controversial issue on March 23, 2013 in the *New York Times* newspaper. Skloot claimed that although the Lacks family *has been through a lot with HeLa (...)*, they are *proud of HeLa's contributions to society, and they don't want to stop HeLa research. But they do want to learn about the HeLa genome – how it can be used for the good of science while still protecting the family's privacy – so they can decide whether to consent to its publication. And they want researchers to acknowledge that HeLa cells are not anonymous and should be treated accordingly.* Rebecca Skloot pointed out that while the advanced molecular technologies open perspectives for more accurate, personalized medicine, the society is still not ready to apply them, especially regarding the ethical and legal issues.

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The UK proposal to assuage the EU agricultural policies

The United Kingdom (UK) will reportedly prompt the European Union (EU) to ease restrictions on the licensing of genetically modified (GM) crops for human consumption, based on the growing scientific evidence of their safety, and surveys demonstrating that they are supported by the farmers. It has been recently showed that 61% of UK farmers claim that they would like to grow GM crops after a disastrous 12-month cycle of poor weather that is expected to significantly reduce harvest yields.

The Environment Secretary, Owen Paterson, is said to believe that the UK should take the lead in overcoming the current stalemate in debate over GM crops, in order to avoid being left behind in an important technology that has the potential to improve crop yields and help British agricultural industry. This initiative has also been supported by the Science Minister David Willetts, who claimed that *we believe that GM crops can help make agriculture more efficient and also just as importantly more sustainable, by, for example, reducing the use of pesticides and the use of fossil fuels*. Moreover, Martin Haworth, director of policy at the National Farmers Union, emphasized recently that the consequences of climate changes reflected in increasingly extreme weather events, may be overcome using GM technology.

Gemma Masip and colleagues working at the Department of Plant Production and Forestry Science of the University of Lleida-Agrotecnio Center (Lleida, Spain), have recently published a review article in *Trends in Plant Science* journal, which strongly justifies the British concept to revise the EU agricultural policies. The authors pointed out the major paradoxes in the current EU legislation on agriculture, that not only affect agriculture directly, but also the environment, human health and the EU economy. For instance, the EU imports animal feed (mainly soybean and maize) because it cannot meet the in-house demand, but more than 80% of imported feed comes from producers of GM crops. The EU authorizes import of 39 genetically engineered crops but only 2 of them are authorized for cultivation. This means that the member states do not allow their own farmers to grow GM crops even if they are identical to the imported varieties. Likewise, the EU has banned many pesticides, but approves the import of food products treated with the prohibited chemicals. The authors underlined the fact that the suppression of genetically engineered agriculture in the EU is widely recognized to have ideological rather than scientific background, and is driven by short-term political goals instead of long-term strategy to improve agriculture industry. Masip et al. (2013) concluded that the current EU policies are *working against the EU's own goals, driving research, development and innovation abroad, and granting commercial and economic benefits to other countries that then sell the GM products back to EU member states. The EU is thus becoming increasingly uncompetitive and isolated in the international markets, which thrive on innovation and technological development in agriculture*.

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