



Prion2007 Special Issue

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The weeks have gone by, the dust has settled and we can finally see clearly enough to look back at *Prion2007* and be happy with this year's Edinburgh based event. *Prion2007* proved once again that TSEs are a subject that remains one of the most high profile of research areas. The conference was attended by 870 delegates from 40 different countries and featured a record number (430) of poster presentations. I feel very honoured to have been involved with the organisation of this event and to have worked with all my colleagues on the Local Organising Committee and the Scientific Committee; to them I offer my sincerest thanks for spending so much of their time to make this meeting the success it was.



Prion 2007 Gala dinner

The Gala Dinner was held in true Celtic style at the Royal Museum of Scotland. This spectacular venue is one of the finest examples of Victorian architecture in the city.

Prior to the main event, we were also pleased to be able to hold satellite meetings in some of Edinburgh's finest buildings. The South Hall of the University of Edinburgh was the venue for the Chronic Wasting Disease workshop which was as successful as it had been in Torino. Much reference to its talks followed during the main conference and there is clearly a good deal of interest in this emerging disease. A quick dash across Edinburgh took everyone to The Public Lectures held in the Assembly Hall with all its gothic splendour and outstanding views of the city centre. *The Scottish TSE Network*, which hosted *Prion2007* along with *Neuroprion*, was proud to invite two of its members Prof James Ironside and Prof Marc Turner to present the latest views on vCJD in light of the identification of transmission of the disease via blood transfusion.

The main conference was held in the stunning Edinburgh International Conference Centre and we are very grateful to Prof Stanley

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Prusiner who kindly opened *Prion2007* with news of his latest research. Prof Prusiner, and Dr Jean Philippe Deslys helped me set the scene and the pace for what became a tour de force of excellent presentations covering all aspects of the prion diseases in all the affected species. We really couldn't have asked for better presentations. One major subject of the meeting was the amplification and detection of the protease resistant form of PrP. The PMCA method is clearly a technical part of TSE research that will be thoroughly tried and tested in the coming months and we look forward to hearing at *Madrid2008* how development continues. Early detection of the abnormal form of PrP is becoming a major area of focus and we must maintain the strong level of discussion around the nature of infectivity that took place at *Prion2007*. On the final day we all had our minds refocused on why we work on these mysterious diseases. Florence Krantz and Suzanne Solvyns from the CJD International Support Alliance gave two very moving presentations reminding us that no matter how challenging our research might be, it is nothing compared to the challenge of living with CJD.

To wrap up, I would like to use this opportunity to repeat my thanks to those that contributed to the success of the *Prion2007* conference. We were very grateful for the generous support of the UK government funders and I would like to thank the Medical Research Council, Defra, Food Standards Agency, Biotechnology and Biological Sciences Research Council and Department of Health for supporting *Prion2007* along with funding from Neuroprion EU Network of Excellence (Framework 6). We also had considerable industry support and I thank them all for attending *Prion2007*. Of-course the meeting was made by the many speakers and delegates who gave *Prion2007* its life and energy (especially on the Ceilidh dance floor at the Royal Museum!). I thank everyone for their contribution and now invite you all to revisit any of the presentations that are now available on the NeuroPrion members' pages. See you in Madrid in 2008.

Prof Jean Manson.

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PMCA on the move

By Vincent Béringue, INRA, *Virologie Immunologie Moléculaires, France.*

Protein misfolding cyclic amplification (PMCA) facilitates the combining of PrPC substrate with undetectable amounts of PrPSc to achieve levels of PrPSc visible by conventional methods.

This is achieved by subjecting samples to repetitive cycles of incubation to enlarge the PrPSc seed and sonication to fragment it and generate new catalytic units.

Originally developed by Claudio Soto and colleagues, this technology has been the hot topic at the meeting. First, its use has been exported worldwide and is no longer restricted to the inventing laboratory. Consequently, a broad spectrum of PrPSc species has now been successfully amplified, including CWD (T. Kurt et al., Fort Collins; K. Green et al., Lexington), mouse-adapted scrapie or BSE prions (R. Morales et al., Galveston) and noteworthy, human sporadic and variant CJD, whose amplification efficiency is tightly controlled by PrPC substrate genotype at codon 129 (M. Jones et al., Edinburgh).

Second, PMCA faithfully maintains *in vitro* the specific biochemical and neuropathological strain properties of the starting material, as shown by reinoculation of the amplified material into recipient animals (K. Green, R. Morales), adding to the view that distinct PrP conformations are biologically active and encipher strain-specified characteristics. The specific infectivity of the amplified mouse prions material also approached that of the PrPSc seed (R. Morales). Third, PMCA appears to overcome the species barrier encountered during cross-species transmission more rapidly than *in vivo*. By “forcing” the technique with lower dilutions of the PrPSc seed and more amplification rounds, mouse RML PrPSc can now convert hamster PrPC (R. Morales) or cervid PrPC (K. Green), a conversion that might be observable *in vivo* but associated with extremely long incubation periods.

Conversion of mouse PrPC by hamster 263K PrPSc is now also possible and produces a strain that seems to differ from all other conventional ones by its higher resistance to protease-mediated digestion (R. Morales). In perhaps the most extraordinary presentation at the meeting, J. Castilla (Jupiter) described how prions can be amplified using PMCA from healthy brains unchallenged with prions. PrPSc was generated from 11 different species, including bank vole, mouse, cattle, human, sheep and rabbits - a species notoriously resistant to prions, generating a variety of electrophoretic profiles, whose infectivity is currently under scrutiny. Thus far, 2 of them generated “spontaneously” from voles have been shown to be produce distinct strains upon reinoculation to recipient voles. While these data suggest that pre-existing PrP seeds might be present in normal brain, perhaps providing an explanation for the sporadic generation of prion disease in humans and animals, it is certainly possible that PMCA generates spontaneous prions *in vitro*. Finally and perhaps surprisingly, few advances using PMCA in diagnosis were reported at the meeting and there was discussion about its reliability. For example in one study, known positive samples failed to amplify (H. Tattum et al.). Furthermore assay time is also an issue with 20 rounds of PMCA taking 2 months (K. Green; R. Morales; J. Castilla). However on the positive side, PMCA was able to detect PrPSc in as little as 1 μ L of blood taken of an asymptomatic prion-infected mouse (H. Tattum). Given the increasing evidence of human to human transmission by blood products, all are waiting for PMCA to be incorporated into a reliable test with the ability to identify blood donors that are asymptomatic carriers.

Taken together, the ability to amplify prions *in vitro* is proving to

be the most exciting development in the prion field for a number of years. Further advances in amplification technology are to be expected and the replacement of PrPC by recombinant PrP as a substrate as well as the use of intermittent shaking rather than sonication might circumvent some of the difficulties in the near future (S. Prusiner et al., San Francisco; B. Caughey et al., Hamilton).

Reporting on the CWD workshop

By Michael Stack, *NeuroPrion Cervid Group Coordinator, Veterinary Laboratories Agency, Weybridge, UK*



Prion 2007 started a day early for participants of the NeuroPrion cervid group workshop which was held at South Hall near Arthur's Seat in Edinburgh. Before the workshop started, the NeuroPrion cervid group members had a progress meeting where the agenda covered the minutes of the last meeting (3rd October 2006, Torino), a brief review of the cervids group activities in 2007 so far, the input into the NeuroPrion Task 2 Annual Report, and the result of the NeuroPrion mid-term review of projects (which was held in Baden in May 2007). This was followed by a group discussion about new members, the planning of future activities, and a funding update.

The workshop began with Mick Stack, the CWD group coordinator, welcoming the 150 participants and giving a brief overview of the NeuroPrion group activities since its instigation in 2005.

Session I, focused on the current knowledge of CWD in North America and Canada.

Dr Michael Samuel from the Wildlife Research Unit at the University of Wisconsin-Madison gave an overview of the epidemiology, surveillance and control measures in white-tailed deer populations. Wisconsin has attempted to eradicate CWD using an aggressive culling strategy, but modelling and other research studies indicate that CWD will be a long-term disease problem in free-ranging cervids, management to contain or eradicate the disease will be challenging, and that higher prevalence may affect recreational harvest and sustainable populations.

Dr Aru Balachandran, from the Canadian Food Inspection Agency, Ottawa, talked about their current CWD situation and, in particular, the susceptibility of different cervid species and ruminants to CWD. The most notable result, from the European perspective, was the oral transmission of CWD to Canadian red deer. Dr Thomas Gidlewski

from the US Department of Agriculture, Animal Plant Health Inspection Service, Fort Collins, Colorado kindly took the place of Dr Terry Spraker, who had to drop out at the last minute, and presented the pathological aspects of CWD, including data showing that cattle orally challenged with CWD have so far not succumbed to disease, but intracerebral (ic) challenge can lead to some "non-classical" histopathological and immunohistochemical observations. In addition, 2 out of 4 sheep with Q at position 171 of the PrP gene orally challenged with CWD had histopathological changes which were identical to scrapie. Dr Delwyn Keane from the University of Wisconsin then gave a detailed account of a white-tailed deer farm which was heavily infected with CWD and showed positive results from recto-anal mucosal tissue and the retina in some of these deer.

Dr Christina Sigurdson from the University Hospital, Zurich presented her work on the prion protein loop region (amino acids 165-175) which appears to be critical for templating and propagating incoming strains, and may be relevant for predicting species barriers in CWD. Professor Glenn Telling of the University of Kentucky described his transgenic approaches to address the origins of CWD and the prevalence of CWD strains. He, and the next speaker, Dr Timothy Kurt from Colorado State University, were investigating protein misfolding cyclic amplification (PMCA) to possibly detect PrP^{CWD} in body fluids and excreta from infected animals; the previous study conducted by these investigators and published by Candace Mathiason demonstrated that blood and saliva from experimentally infected deer was infectious. In fact, Dr Candace Mathiason was next to present and described some additional bioassay studies where healthy deer were fed saliva, urine and faeces and transfused with blood from infected deer; these studies are in progress. Dr Katherine O'Rourke, from US Department of Agriculture in Pullman Washington, presented results on long term experiments studying the susceptibility of Rocky Mountain elk to CWD. Katherine has previously shown that there is a predisposition to CWD in homozygous 132MM elk following oral challenge and that there was the approximate doubling of incubation in 132LM elk. This talk described a prolonged incubation time, approximately triple that for 132MM, in 132LL elk. In addition the PrP^{CWD} in 132LL elk exhibited a reduction in molecular weight and the loss of the mAb P4 epitope compared to the 132LM and 132MM elk samples.

Session II focused on the European CWD studies.

Wider surveillance of cervid populations in Europe has been introduced by the EU this year and, to date, no TSE infections of European red deer have been reported. Should BSE infection have been transmitted into the UK cervid population, the CWD precedent in North America would suggest there would be a great danger of both spread and maintenance of the disease in both free living and captive UK deer populations. Stuart Martin from VLA Lasswade, UK, presented very interesting new data comparing the immunohistochemical (IHC) and Western blot profiles for experimental BSE inoculated ic into UK red deer and the Canadian red deer orally challenged with CWD by Dr Balachandran. Essentially, the BSE infected red deer exhibited a BSE profile and CWD infected deer the CWD profile, which can be readily discriminated from BSE. These experiments are important to further support assessment of the potential risk of BSE and CWD in red deer to human health.

The NeuroPrion cervid group has been active, in the few years leading up to the wider EU surveillance, in carrying out successful proficiency testing of negative and positive deer samples, kindly supplied by Dr Balachandran from Canadian sources. This year there was a European Commission requirement for each National Reference Laboratory responsible for confirmation of the statutory cervid surveillance, to participate in annual IHC proficiency testing. Dr Paul

Webb from VLA Weybridge, UK, presented these results at the workshop and, although there were a variety of antibodies and different IHC protocols used, all laboratories detected the blinded samples correctly. This year, the TSE Community Reference Laboratory (VLA) were overseeing the proficiency testing of front-line rapid tests being used to screen the cervid samples in the EU surveillance. Blinded samples were sent to 19 NRLs, of which 7 were NeuroPrion cervid group members. The results were presented at the workshop by Melanie Chaplin from VLA Weybridge, UK, and all 19 laboratories had at least one test which could detect CWD at all dilutions with no false positive reactions.

Mick closed the meeting with a short overview of the workshop highlights:- We now know, from this workshop, that many cervid species are likely to be genetically susceptible to prions, with some of the least susceptible giving long incubation periods. We also now know that red deer succumb to oral challenge with CWD and develop disease after intracerebral inoculation with BSE. So far, red deer orally challenged with BSE have not succumbed. CWD, orally fed to cattle, has not so far produced disease and ic inoculation gives a non-classical histological and IHC pathology. Sheep with Q at position 171 of the PrP gene were fed CWD and appear to produce a scrapie-like biochemical and pathological profile. Recto-anal lymphoid tissue biopsy can now be used routinely and offers some ante-mortem diagnosis without being too invasive to the deer. Proficiency testing has been set up to provide confidence in the front-line tests and confirmatory tests being used in Europe. If field evidence of a TSE disease in European cervids were discovered, we have the techniques to further evaluate such cases. In particular we have discriminatory tests to apply to determine that the TSE had not been derived from a BSE infection, and the subsequent unknown question of whether it would be communicable to humans.

By way of this workshop, the NeuroPrion cervid group has instigated the sharing of knowledge between the experts in North America and Canada, and the Institutes in Europe that are involved in the surveillance of cervids or carrying out funded research which involves CWD. From the verbal feedback obtained the workshop content and format was well received by all participants and many are keen to continue this growing collaboration which, apart from highlighting CWD studies, has several "spin-offs" for studying scrapie and BSE. We hope to organise a similar pre-conference workshop for Prion 2008 in Madrid.

Mick Stack – NeuroPrion cervid group coordinator.

CWD workshop at the Prion 2007 Conference

by Dirk Motzkus, PhD, German Primate Center, Göttingen, Germany (dmotzkus@dpz.eu)



Article for NeuroPrion Newsletter by Dirk Motzkus, PhD, German Primate Center, Göttingen, Germany. While the incidence of BSE further declines, the spread of Chronic Wasting Disease (CWD) is gaining ground. Due to the long-term surveillance that already started a decade ago the epidemiology of the disease is well-documented and methods to analyze CWD prions have been successfully adapted from the BSE-field. Not only immunohistochemistry and rapid tests are being

performed, but also cervid transgenic mice and PMCA are already used.

But there are some crucial differences between BSE and CWD, that have been addressed at the workshop. CWD is the only known TSE that affects free ranging wildlife species. Deer and elk can pass the US-Canadian border in both directions. Therefore it is not surprising that local eradication programs were unable to reduce the overall incidence of CWD in North America. New strategies to hold up the spread of CWD are urgently needed, but how can we fight a disease when the route of transmission remains unclear? Once an animal in a captive herd is diagnosed positive for CWD follow up studies revealed a prevalence of as much as 20% - 80% in the remaining animals, which may be a result from persistence of CWD-infectivity in the environment. It is now that these features have raised concerns about impacts on human and commercial health, after all only in Wisconsin about 600,000 hunters prey on 1.7 million deer corresponding to an economic factor of about 1 billion Dollar each year. And CWD will persist. The experts pointed out that CWD was first observed about 30 years ago and that it will most probably hold out for decades.

To date, there is no evidence that CWD can be transmitted to humans. However, the recent appearance of CWD in moose, almost 25 years after the first reports in deer and elk, may be the result of crossing the species barrier under free-ranging conditions. Therefore comprehensive risk assessment studies for CWD are urgently needed.

PhD Day - NeuroPrion reaches out to the students

By Karine Delmouly



For the first time, Neuroprion 2007 hosted a full-day dedicated to PhD student work. Once the building was found and the right room to fit the 25 students, we were welcome by Dr Jean Philippe Deslys, coordinator of Neuroprion and responsible of the CEA prion team, and Dr Jens Schell, coordinator of the student day and scientific manager at the CEA. After a short presentation of the student day schedule, Dr Christian Dumpitak, from the University of Dusseldorf, made an introduction of the scientific presentation techniques and how to give and to receive proper feedback.

Once we got informed of what should have been done, two sessions were made where students were placed according to their scientific projects. At this time, we had the opportunity to show what we had learned earlier. The quality of the talks was remarkable, they were really professional and the coordinators were pleasantly surprised. The presentations were linear, well structured and clear. It was easy for the audience to follow and understand the speakers who were well prepared. All the talks, from the first year student to the nearly graduate one, were full of interesting information. We got the opportunity to address to some current research and remaining dark holes in the prion field (strain characterisation, physiopathology in the immune system, cell therapy and model of infection...). At the end of the day, four persons were selected by students and coordinators based on the value of their projects and presentations. Claudia Manzoni, from

the Instituto Ricerche Farmacologiche Mario Negri (IRFMN) of Milan (Italy) and Dana Tzfati, from the department of Neurology of the University Hospital of Jerusalem (Israel) were picked from the first session.

Aroa Relaño Ginés, from the Instituto Nacional de Investigación y Tecnología Agraria y Alimentaria (I.N.I.A.) of Madrid (Spain) and Assunta Senatore from the Instituto Ricerche Farmacologiche Mario Negri (IRFMN) of Milan (Italy) were selected from the second session. After an additional presentation in front of all the students, it was hard for the coordinators to determine which student will have the opportunity to present her work during the Neuroprion congress. This occasion was given to Aroa Relaño Ginés who, although anxious, made a really good presentation on stem cell therapy. At the end of the day, students returned home with valuable advices to make good presentations. It was nice to have this training, to assess our skills to speak in front of an assembly composed of people from outside our proper lab.

In conclusion, many thanks to the Neuroprion team, who arranged this day, and to the team of the National Creutzfeldt-Jakob Disease Surveillance Unit who assure a nice, enjoyable and perfectly organised day.

Prion 2007: Pathology and Pathogenesis session (Thursday 27th September)

By Karen Brown



The science presented covered a wide range of topics including; cellular targeting of infection, sub-cellular localisation of infection, role of the immune system in pathogenesis and immunotherapeutic approaches to TSE infection. The session was chaired excellently by Professor James Ironside of the CJD surveillance unit, Edinburgh.

The session began with a plenary talk given by M. Jeffrey describing his ultrastructural studies with a range of experimental and natural TSE strains. In the peripheral lymphoid system he described membrane alterations of follicular dendritic cells (FDCs) and macrophages in response to TSE infection and also showed that the accumulation of PrP^{Sc} on FDCs led to an increased retention of immune complexes on these cells. Of further interest was the illustration of how PrP^{Sc} was differentially trafficked in the CNS depending on infecting TSE strain.

The important issue of cellular infectivity in blood was addressed by O. Andreoletti who showed that infectivity in blood from orally infected sheep was predominantly associated with CD14 positive cells (mono-nucleated phagocytes). Interestingly, these studies suggested that the presence of infectivity in blood could be correlated to PrP^{Sc} presence in secondary lymphoid tissues.

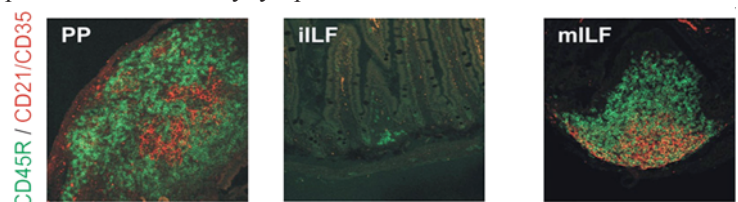


Figure 1. Immunohistochemical characterization of mILFs. iILFs typically comprise a loose aggregation of B lymphocytes at the base of a villus (CD45R-positive cells; green), whereas mILFs contain both B lymphocytes and CR2/CR1-expressing FDCs (CD21/CD35-positive cells; red). PP (peyers patch). *Glaysner et al., (2007) Immunology*

In addressing the effects of host PrP on pathogenesis E. Cancellotti described how host PrP glycosylation can influence incubation period of disease, vacuolar pathology and strain determination.

The importance of the gut associated lymphoid tissue (GALT) in TSE agent neuroinvasion from the intestine was presented by N. Mabbott. In these studies he demonstrated that in the combined absence of GALT and FDCs, TSE transmission was blocked. Interestingly, these studies also found that mature isolated lymphoid follicles (mILFs) were a novel site for TSE agent replication in the gut providing a compensatory mechanism for TSE neuroinvasion in the absence of peyers patches (PP). (Figure 1).

The effects of the immune system on the transmission of BSE to mice were investigated by C. Farquhar in studies using immunodeficient and chimaeric mice. These studies also found that functional FDCs had a role in the transmission of BSE to mice.

Although many studies have demonstrated a critical role for FDCs in the peripheral pathogenesis of disease, an interesting talk by M. Heikenwalder presented evidence of disease replication in granulomas in the absence of FDCs. Granulomas are composed of mature mononuclear phagocytes (macrophages and/or epithelioid cells) but not FDCs. In these studies infectivity and accumulation of PrP^{Sc} could be detected within ectopically induced granulomas of wild-type, but not PrP deficient mice that had been challenged with the RML strain. Previously published studies have shown that RML and another experimental TSE strain, ME7, may have different cellular involvement in the lymphoid system.

The session was concluded with a presentation detailing the characterisation and therapeutic potential of a range of anti-PrP monoclonal antibodies. The demonstration of the potential therapeutic effects of these antibodies, via the increase of PrP^c in plasma, was of great interest.

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A PrionStory: Infectious PosSession or Conformational ObSession?

By Vito Vetrugno



Whether or not TSE causative agents are solely altered pathological conformers of the normal cellular prion protein (as strongly referred by Stanley Prusiner during his plenary lecture: "Prions are infectious Prions...") or not yet completely understood, complex, infectious agents is still debated.

Huge amount of experimental data produced by several research groups using PMCA showed that this technique can replicate *in vitro* prion strain characteristics, in terms of clinical signs, brain lesion patterns, biochemical fingerprints, and incubation times. Moreover, dr. Castilla (FC7.4, *De novo generation of prions in a cell free system*) presented new exciting data about *de novo* generation of infectious prions by PMCA.

However, as reported during this congress, some scientists presented evidence in which PrP amyloid is not necessarily a reliable marker

for TSE infectivity (e.g. Langeveld, FC7.2, *Enzymatic degradation of PrP^{Sc} fails to dis-infect bovine BSE brain homogenates*). Some cell biology, transmission, and inactivation studies are contrasting, at least in part, with the prion only hypothesis (e.g. Lasmez, *Science*, 97; Somerville, *JBC*, 02; Silveira, *Nature*, 05; Piccardo, *PNAS*, 07). The common assumption that presence of pathological PrP or PrP amyloid signifies presence of TSE infectious agent is difficult to integrate and reconcile with the infectious protein-only dogma. And to date, only a different etiology for sporadic, genetic and infectious TSE forms might explain these discrepancies.

High quality of oral presentations and massive scientific content of poster sessions characterized Prion 2007. I found interesting the results presented by dr. Lewis, from Australia (i.e. FC3.2, *Investigating the subcellular location of the infectious "Prion" and its relationship to PrPres in a cell culture model of Prion disease*). In this study the authors demonstrated that the most infectious fractions (i.e. after density gradient fractionation) do not correlate with those containing the highest levels of PrPres suggesting that other related PrP species (i.e. PrP*) and/or no-PrP cofactors are associated to the infectious particle and may be likely actors in the infectivity transmission.

Dr. Heiseke, from Germany, showed (i.e. FC4.5 "Induction of cellular autophagy reduces Prions") data about the antiscrapie compound imatinib (i.e. Gleevec, Novartis Pharmaceuticals Corporation; Basel, Switzerland), a tyrosin kinase inhibitor commonly used in the cancer therapy. Imatinib mesylate, increases the cellular clearance of intracellular protein aggregates upregulating lysosomal activity and significantly decreased PrP^{Sc} accumulation in a dose dependent fashion. Moreover, it abolished prion infectivity to almost undetectable level in persistently scrapie-infected neuroblastoma cells (ScN2a). Application of inhibitors of autophagy in parallel with imatinib antagonized its anti-prion effect, viceversa the administration of autophagy inhibitors alone increased PrP^{Sc}. The underlying mechanisms of PrP^{Sc} clearance by imatinib may provide insight into understanding mechanisms of prion replication as well as the development of new therapeutic strategies for prion diseases *in vivo*.

Unfortunately, all compounds, which are able to cure prion infected cell lines and/or increase survival time of TSE injected rodents are efficient only when administered before or in the early post-infection phase. So far, the majority of these anti-TSE compounds are unable to cure or even prolong the disease duration in experimental animals or in patients with CJD.

An effective therapy is extremely needed and an integrated combination of available drugs, which may act at different levels, can synergize effectively in this scope

IPFA session on "progress with blood safety and prions"

IPFA, the international association of not-for-profit organisations involved in plasma fractionation, was delighted to again hold a session within the annual neuropriion tse conference.

Progress with Blood Safety and Prions chaired by Marc Turner of SNBTS and Paul Brown formerly of NIH, had a participative format with each topic being summarised by an expert in the field followed by discussion within the audience of around 200. Noel Gill of the UK Health Protection Agency covered ongoing and planned prevalence studies on the UK population, although to the audience's disappointment interim results from the National Anonymous Tonsil Archive study will not be published until December.

Chris Prowse of the SNBTS described progress with prion removal filters, with one currently being trialled for Red Cell Concentrates and others being developed.

Paul Brown provided an update on blood screening tests, noting progress against key milestones by several organisations but commenting that four leading companies had recently suspended work due to technical problems or commercial uncertainties. Phil Minor of NIBSC described the approaches to validating such assays and the EU regulatory framework for in vitro diagnostic devices (IVDs). He indicated that one company was close to achieving satisfactory performance and that a proposal to incorporate a test for vCJD into the EU Directive on IVDs was due to be considered in November. He was questioned on provision of vCJD samples, release of which is being strictly controlled because of the small amounts available. Stephen Dobra of the UK Department of Health provided updated assessments of risk reduction measures in the UK, importantly also considering the impact on overall blood safety.

Finally Joliette Coste of the French Blood Service reviewed the range of initiatives being taken in France, in epidemiology, risk assessment, prion removal and efficacy evaluations, and input to establishment of a European Blood Sample Collection. Paul Brown also took the opportunity to announce the first recipients of grants from the Foundation Alliance Biosecure with the topic of prion blood safety well represented. Integration of this IPFA session within Prion 2007 was certainly a great success and it is a pleasure to report that Neuroprion has expressed its desire to host a similar session at Prion 2008 in Madrid.

Prion 2008 in Madrid

The international conference «Prion 2008» will be held in Madrid, Spain, on October 8-10, 2008. As in precedent edition, the main purpose of the congress is to discuss the latest research in the field of Transmissible Spongiform Encephalopathies (TSE) and Prion disease. The programme of "Prion 2008" will include state-of-the-art lectures, workshops, oral presentations, poster sessions and other forms of presentation selected from contributed abstracts. The diverse meeting schedule will cover contemporary topics in Prion Disease, always with an emphasis on research data. Scientific sessions will be organised covering all the important subjects in the TSE field. This event will provide a great opportunity for scientists from all over the world to share their findings and progress in an attractive and interesting setting.



Announcements

Prion conferences:

- **Cambridge Healthtech Institute's 12th Annual Transmissible Spongiform Encephalopathies The Definitive American TSE Meeting**
Feb 11 - 12, 2008
Sheraton Inner Harbor Hotel - Baltimore, USA
www.healthtech.com
- **Prion2008**
NeuroPrion Network of Excellence annual TSE conference
Oct 8 - 10, 2008 Madrid, Spain
www.prion2008.com

Job offers:

- **Post-doctoral position**
Decontamination of prions
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CEA, France
Please contact: Emmanuel COMOY
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Tel: (33) 1 46 54 90 05
http://www.neuroprion.com/en/jb_professor.html
- **Postdoctoral position in prion diseases**
(Closing date : December 31, 2007)
Washington University in St.Louis
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Contact information: David A. Harris, M.D., Ph.D.
Dept. of Cell Biology and Physiology
Telephone: +1 314-362-4690
http://www.neuroprion.com/en/jb_professor.html
- **Two postdoctoral positions in Prion Diseases**
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University of Texas
Claudio Soto, PhD
Director Mitchell Center for Neurodegenerative diseases
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