with other recently published data.<sup>3-5</sup> In addition, our kinetic data suggest seeded reactions are an ordered, on-pathway amyloid formation mechanism, as previously described.<sup>6</sup> The consistencies in our reaction behaviour bestow the confidence required to satisfy rigorous diagnostic demands. Future research is aimed toward qualitative analysis of our elk CWD-seeded RT-QuIC reaction products.

## References

- Bian J, Napier D, Khaychuck V, Angers R, Graham C, Telling G. Cell-based quantification of chronic wasting disease prions. J Virol 2010; 84:8322-6; PMID:20519392; http://dx.doi.org/10.1128/JVI.00633-10
- Gray JG, Dudas S, Czub S. A study on the analytical sensitivity of 6 BSE tests used by the Canadian BSE reference laboratory. PLoS One 2011; 6:e17633; PMID:21412419; http://dx.doi.org/10.1371/journal.pone.0017633
- Elder AM, Henderson DM, Nalls AV, Wilham JM, Caughey BW, Hoover EA, Kincaid AE, Bartz JC, Mathiason CK. In vitro detection of prionemia in TSEinfected cervids and hamsters. PLoS One 2013; 8:e80203; PMID:24224043; http:// dx.doi.org/10.1371/journal.pone.0080203
- Henderson DM, Manca M, Haley NJ, Denkers ND, Nalls AV, Mathiason CK, Caughey B, Hoover EA. Rapid antemortem detection of CWD prions in deer saliva. PLoS One 2013; 8:e74377; PMID:24040235; http://dx.doi.org/10.1371/journal. pone.0074377
- Wilham JM, Orrú CD, Bessen RA, Atarashi R, Sano K, Race B, Meade-White KD, Taubner LM, Timmes A, Caughey B. Rapid end-point quantitation of prion seeding activity with sensitivity comparable to bioassays. PLoS Pathog 2010; 6:e1001217; PMID:21152012; http://dx.doi.org/10.1371/journal.ppat.1001217
- Bocharova OV, Breydo L, Parfenov AS, Salnikov VV, Baskakov IV. In vitro conversion of full-length mammalian prion protein produces amyloid form with physical properties of PrP(Sc). J Mol Biol 2005; 346:645-59; PMID:15670611; http://dx.doi.org/10.1016/j.jmb.2004.11.068

## P.172: BSE exposure risk from bovine intestine and mesentery

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Bovine intestines and mesenteries in the European Union (EU) are considered among the tissues potentially containing the highest level of BSE infectivity and have to be removed from the food and feed chain. A quantitative assessment of the BSE infectious load potentially entering the food and feed chain yearly in the European Union (EU) was developed. The evolution of the BSE infectious titre and of the weight of the structures accumulating infectivity was considered. The number of BSE infected cattle entering undetected in the food and feed chain yearly was

estimated. A model (TSEi) was developed to estimates the evolution of the BSE infectious load in animals and the total yearly infectious load that could enter the food and feed chain. In a BSE infected bovine, the distribution of infectivity in intestines and mesentery varies with the age. Up to 36 months of age the infectivity is mainly associated (on average more than 90%) with the last 4 metres of small intestine and the caecum, over 36 and under 60 months of age, there is an inter-individual variability, from 60 months of age the infectivity is mainly associated (on average more than 90%) with the mesenteric nerves and the celiac and mesenteric ganglion complex. The total amount of infectivity peaks, about 15 BoID<sub>50</sub>, in animals younger than 18 months, it declines to 8-9 BoID<sub>50</sub> (24-48 months of age) and it drops to 0.7 BoID<sub>50</sub> in animals older than 60 months. The ileocaecal plate is the most infectious part of the intestine and it can be used to estimate the potential maximum level of exposure for an individual consumer. In the EU, between 2007 and 2012, the yearly amount of BSE infectivity associated with intestine and mesentery from animals entering the food and feed chain was reduced by a factor of 10 (from about 23,000 to about 2,000 BoID<sub>50</sub>). However, the maximum level of exposure to the BSE agent from intestine remained stable (on average about 1.5-1.6 BoID<sub>50</sub> per meter). In case of re-emergence of BSE in the EU there would be an increase of the potential maximum level of exposure to BSE from intestine. According to the TSEi model the removal of the last four metres of the small intestine and of the caecum from the food and feed chain would result in a major reduction of the BSE exposure risk associated with intestine and mesentery in cattle.

## P.173: Evaluation of immunogenicity of prion vaccine administered together with vaccine enhancing agent

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Transmissible spongiform encephalopathy (TSE) is a neuro-degenerative disorder characterized by pathologic accumulation of a misfolded form of a normal cellular protein in neurons. Emergence of TSEs in wildlife populations and the ability of some TSEs to cross species barriers have prompted concern regarding the lack of treatment options or prevention strategies. Efforts at vaccine development have been hampered by the difficulty of overcoming self-tolerance. Studies in our lab have demonstrated that vaccine induced immunity is often diminished due to the recruitment of anti-inflammatory myeloid cells. We hypothesized that utilizing an effective antigen while simultaneously inhibiting monocyte migration could elicit a more effective anti-prion response.

The vaccine was formulated using a peptide fragment of the human prion protein (PrP106-126). This peptide spontaneously forms fibrillar aggregates and is thought to mediate the