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REVIEW

Prion protein glycosylation

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Abstract
The transmissible spongiform encephalopathies (TSE), or prion diseases are a group of transmissible neurodegenerative disorders of humans and animals. Although the infectious agent (the 'prion') has not yet been formally defined at the molecular level, much evidence exists to suggest that the major or sole component is an abnormal isoform of the host encoded prion protein (PrP). Different strains or isolates of the infectious agent exist, which exhibit characteristic disease phenotypes when transmitted to susceptible animals. In the absence of a nucleic acid genome it has been hard to accommodate the existence of TSE strains within this protein-only model of prion replication. Recent work examining the conformation and glycosylation patterns of disease-associated PrP has shown that these post-translational modifications show strain-specific properties and constitute in the molecular basis of TSE strain variation. This article will review the role of glycosylation in the susceptibility of cellular PrP to conversion to the disease-associated conformation and the role of glycosylation as a marker of TSE strain type.
Keywords: Creutzfeldt–Jakob disease, glycosylation, prion, transmissible spongiform encephalopathy.
J. Neurochem. (2005) **93**, 793–801.

Transmissible spongiform encephalopathies (TSE) are invariably fatal neurodegenerative disorders affecting humans and animals. In humans they arise sporadically (~85% of cases), are inherited through mutations in the prion gene (~15%) or acquired through dietary or medical exposure to infected tissues (< 1%). The transmissible nature of these diseases sets them apart from other neurodegenerative disorders such as Alzheimer's disease and Parkinson's disease, which arise either sporadically or are inherited through mutations in a host gene. According to the protein-only hypothesis, the transmissible agent of these diseases, the 'prion', is comprised solely or principally of an abnormal isoform (termed PrP^{Sc}) of the host-encoded prion protein, PrP^{C} (Table 1). PrP^{Sc} has an increased β-sheet content, and can be distinguished from PrP^{C} by its relative insolubility in detergents and partial resistance to protease degradation. The relative resistance of PrP^{Sc} to degradation by proteinase K has been utilized as an end-stage diagnostic marker of disease. Under defined conditions, proteinase K completely digests PrP^{C}, whereas approximately 70 N-terminal residues of PrP^{Sc} are digested by this treatment, leaving a 27–30 kDa protease-resistant core. Support for the role of PrP^{Sc} as the transmissible agent in TSE disease comes from the observation that it copurifies with infectivity (Bolton et al. 1982) and from a recent study that demonstrated the generation of infectivity from an prion-functionally truncated form of recombinant PrP comprising amino acid residues 89–230: preparations folded into β-rich conformers (Lange et al. 2004). The mechanism by which PrP^{C} undergoes this conformational change to form PrP^{Sc} and acquires its infectious properties is poorly understood. An appreciation of the molecular events that facilitate this process is paramount to the development of effective prophylactic and therapeutic strategies.

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Abbreviations used: BSE, bovine spongiform encephalopathy; CJD, Creutzfeldt–Jakob disease; DY, drowsy strain of hamster-adapted transmissible mink encephalopathy; ER, endoplasmic reticulum; GPI, glycosylphosphatidylinositol; HY, hyper strain of hamster-adapted transmissible mink encephalopathy; PrP, prion protein; N_2N_2, asparagine-linked mouse neuroblastoma N_2a cells; TSE, transmissible spongiform encephalopathy; vCJD, variant Creutzfeldt–Jakob disease.

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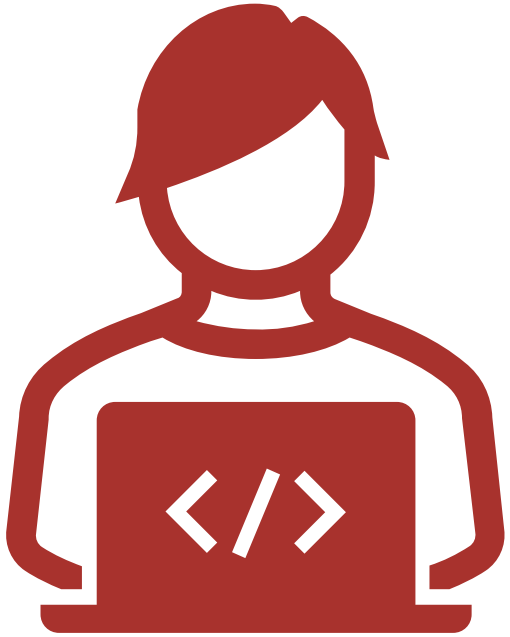

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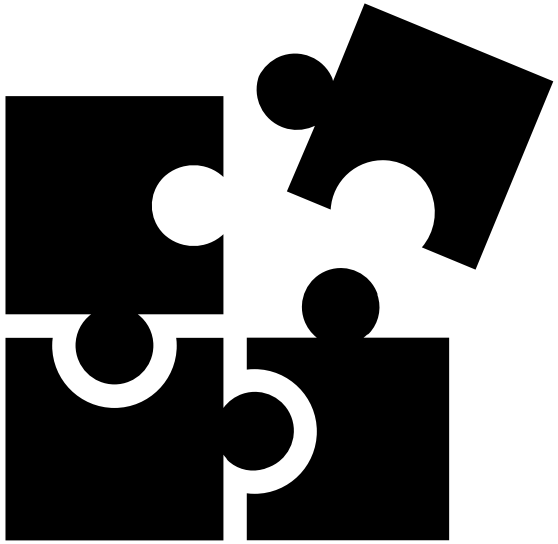

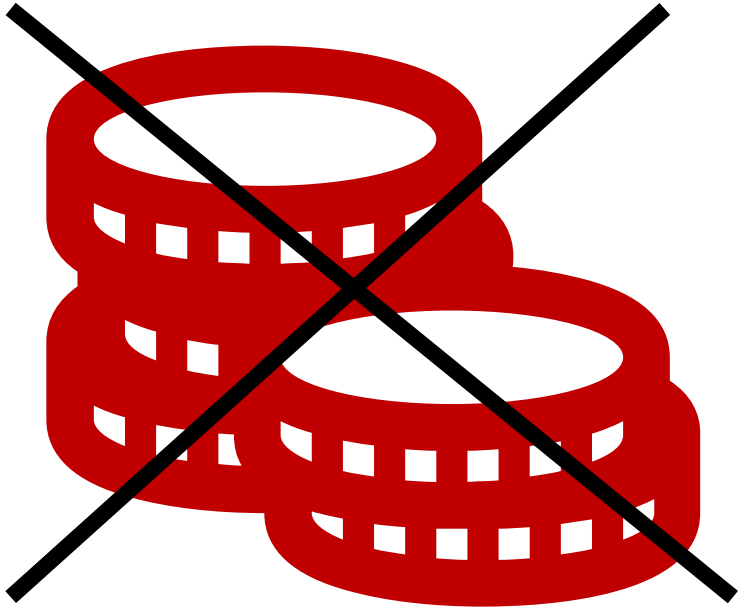

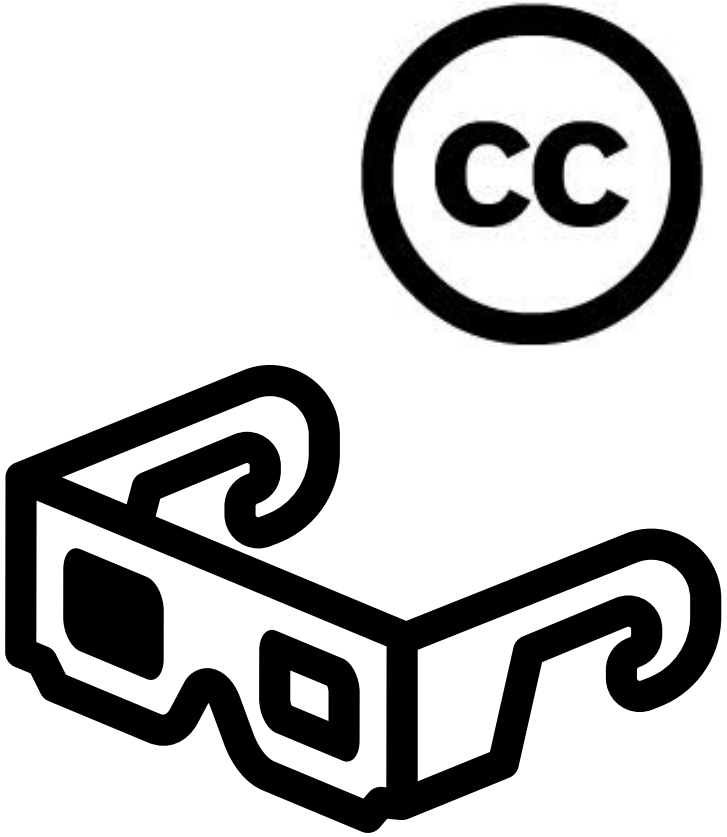

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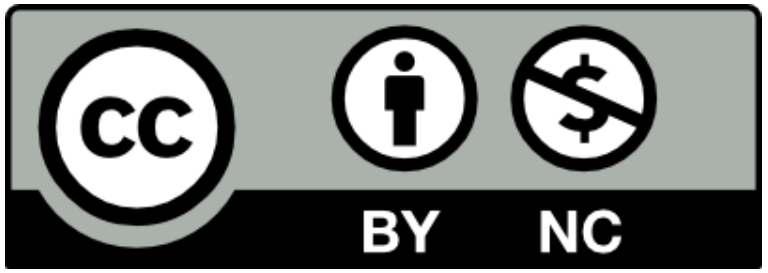

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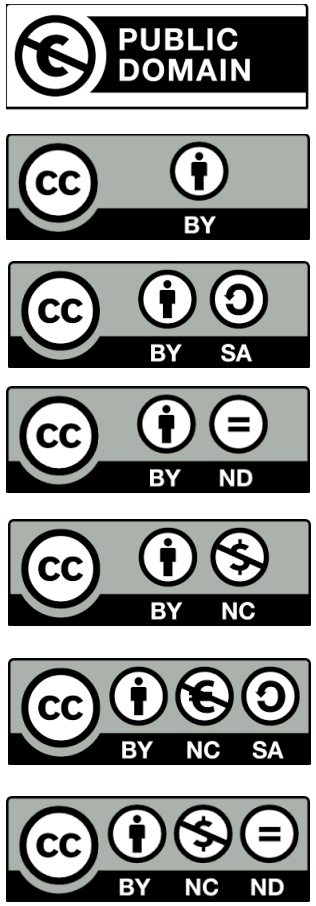

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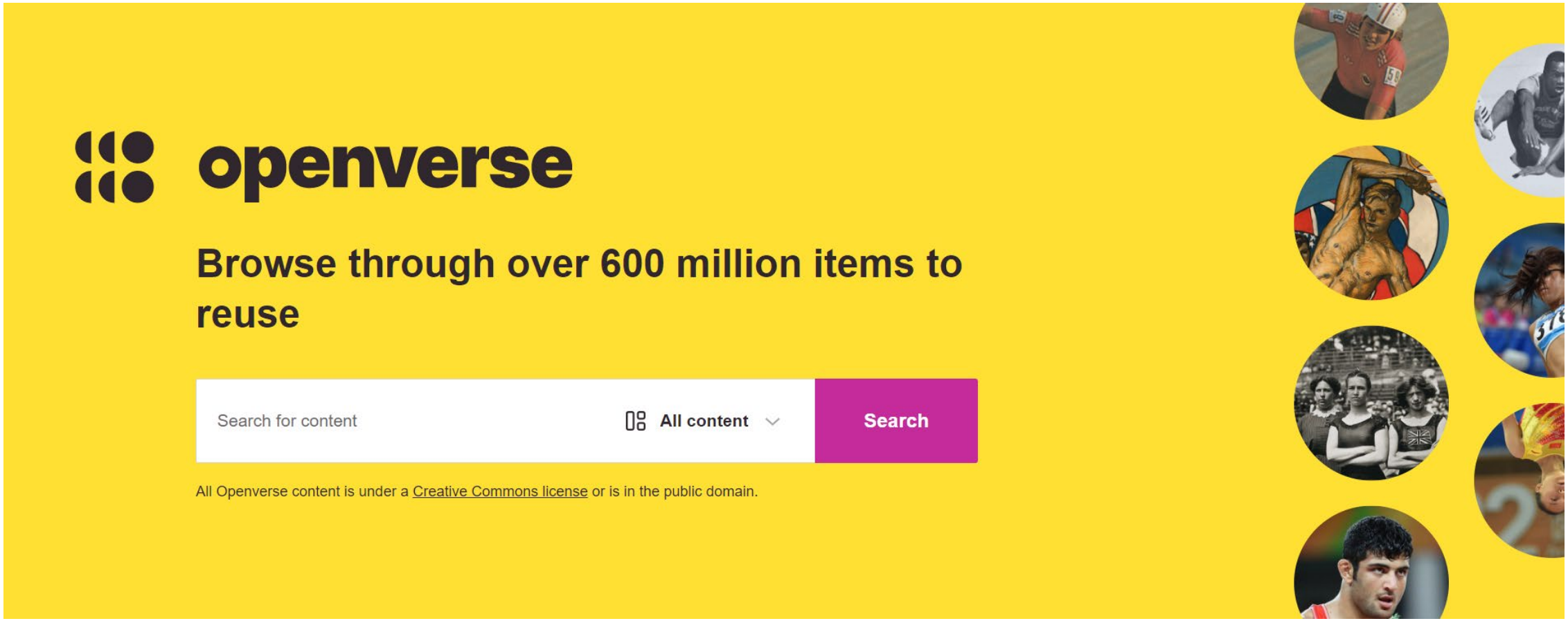

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