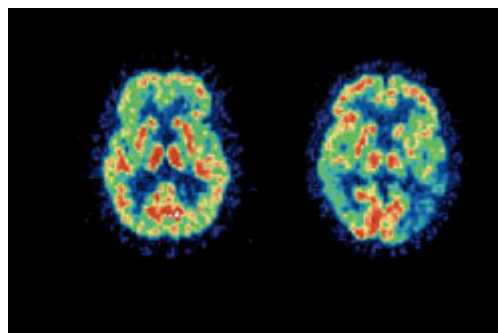




Offers on Biology, Medicine

New data sheds light on prion mystery

Transmissible spongiform encephalopathies (TSEs) are characterised by nerve cell deterioration and share a common causative agent, the prion protein (PrP). PrPs are unlike most pathogens, however, what is known about them is very little.



PrP mechanism of action is still unclear and it becomes more complicated given that neurones produce PrPs in their healthy state. Natural, cellular PrP and pathogenic PrP are structurally different in certain aspects, but even the function of cellular PrP has not been elucidated. The EU-funded project PRP AND NEURODEGENER set out to discern the links between PrP, oxidative stress and ion metabolism in neurones. © PhotoDisc

Existing research data have implied that PrP may have a role to play in ion transport and/or metabolism in or across cells. Project partners, Centre National de Recherche Scientifique studied the effect of PrP in ion metabolism and oxidative stress and how these events are linked. Furthermore, researchers focused on building a hypothesis on how these events can play a role in the onset of neurodegeneration.

The studies that were carried within the framework of the project indicated quite strongly that oxidative stress can cause imbalance in copper ion homeostasis. That imbalance showed signs of correction in the presence of PrP, suggesting a distinct role for PrP on metal homeostasis.

PrP's antioxidant properties have been shown in the past. It is now suggested that the protein also plays a key part in stabilising copper ion levels under conditions of oxidative stress. Shedding light on the function of PrP in healthy cells is a fundamental first step towards establishing the pathology of PrP in infected neurons. That is probably the only way towards an effective treatment for TSEs in all affected mammalian species and therefore this research becomes extremely important.

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