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Oxidation, glycooxidation, lipoxidation, nitration, and responses to oxidative stress in the cerebral cortex in Creutzfeldt-Jakob disease.

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Gel electrophoresis and Western blotting of frontal cortex homogenates have been carried out in sporadic Creutzfeldt-Jakob disease (CJD) cases and age-matched controls to gain understanding of the expression of glycation-end products (AGEs). N-Carboxymethyl-lysine (CML) and N-carboxyethyl-lysine (CEL) were used as markers of glycooxidation; 4-hydroxynonenal (4-HNE) and malondialdehyde-lysine (MDAL) as markers of lipoxidation; and nitrotyrosine (N-tyr) and neuronal, endothelial and inducible nitric oxide synthase (nNOS, eNOS and iNOS) as markers of protein nitration and as sources of NO production, respectively. Age receptor (RAGE) and Cu/Zn superoxide dismutase (SOD1) and Mn superoxide dismutase (SOD2) expression levels were also examined. The results showed a significant increase in the expression levels of AGE ($p<0.05$), CEL ($p<0.001$), RAGE ($p<0.05$), HNE-modified proteins ($p<0.01$), nNOS, iNOS and eNOS ($p<0.01$ and $p<0.05$, respectively), N-tyr ($p<0.05$), and SOD1 ($p<0.05$) and SOD2 ($p<0.05$). No relationship was observed between PrP genotype, PrP type, PrP burden, and expression levels of oxidative stress markers. The present findings demonstrate oxidative, glycooxidative, lipoxidative and nitrative protein damage, accompanied by increased oxidative responses, in the cerebral cortex in sporadic CJD. **These results provide support for the concept that oxidative stress may have important implications in the pathogenesis of prion diseases.**

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