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Letters to the Editor

Editor:

Absence of evidence is not always evidence of absence. In the article ''Failure to detect prion protein (PrPres) by immunohistochemistry in striated muscle tissues of animals experimentally inoculated with agents of transmissible spongiform encephalopathy,'' recently published in *Veterinary Pathology* (**41:**78–81, 2004), PrPres was not detected in striated muscle of experimentally infected elk, cattle, sheep, and raccoons by immunohistochemistry (IHC). Negative IHC, however, does not exclude the presence of PrPSc. For example, PrPres was detected in skeletal muscle in 8 of 32 humans with the prion disease, sporadic Creutzfeldt-Jakob disease (CJD), using sodium phosphotungstic acid (NaPTA) precipitation and western blot.[1] The NaPTA precipitation, described by Wadsworth et al.,[3] concentrates the abnormal isoform of the prion, PrPres, from a large tissue homogenate volume before western blotting. This technique has increased the sensitivity of the western blot up to three orders of magnitude and could be included in assays to detect PrPres. Extremely conspicuous deposits of PrPres in muscle were detected by IHC in a recent case report of an individual with inclusion body myositis and CJD.[2] Here, PrPres was detected in the muscle by immunoblotting, IHC, and paraffin-embedded tissue blot. We would therefore caution that, in addition to IHC, highly sensitive biochemical assays and bioassays of muscle are needed to assess the presence or absence of prions from muscle in experimental and natural TSE cases.

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