(pattern 3), the gitter cells showed neither myelin nor axonal positivity. This might be related to the chronicity of the lesion with the phagocytized components being digested to non-detectable elements.

Conclusion

Although axons hardly showed morphological signs of diseased axons, our results indicate that axonal loss was, to some degree, always present in association with myelin loss and increased interstitial proliferation (fibrillary astrocytosis). This findings, however, do not indicate changes typical of early axonal degeneration. Immunohistochemical investigations using myelin oligodendrocyte specific protein (MOSP) labelled cells with aberrant cytoplasmic processes and did not allow to differentiate between reactive oligodendrocytes or astrocytes. In conclusion, the immunohistochemical data reported here have provided a first insight into the pathogenesis of the disease. Thus, supporting the opinion that cheetah myelopathy constitutes a specific entity of unknown etiology. The pathological process, which triggers myelin or axonal loss in the present investigation and the relationship between tissue environment, such as astrocytic and macrophage response is not yet known. Further investigations, such as in situ hybridization and EM are required to clarify the features of the atypical GFAP-positive/ MOSP-positive oligodendrocytes and the astrocytic involvement in early stage disease. Whether these abnormalities result from an intrinsic genetic defect of oligodendrocytes, abnormal interactions with axons or glial cells, or environmental factors remains to be elucidated.