SNE Is Not NLE Is Not GME

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It was with great interest that we read the article by Park et al[1] reporting their comparative immunohistochemical studies on canine necrotizing meningoencephalitis (NME), necrotizing leukoencephalitis (NLE), and granulomatous meningoencephalitis (GME). As an adjunct to many previous studies, the authors investigated the usual candidates of inflammatory cells (B cells, T cells, macrophages) via immunohistology/immunofluorescence to shed light on the pathobiology of these 3 types of encephalitis in the dog.

Thereby, the study confirmed already published data on breed prevalences, T-cell predominance in GME, and the macrophageal contribution to GME. The overall immune cell composition was similar throughout the different types of encephalitis and led the authors to conclude that all types appear to share a common pathogenesis and that NME and NLE even may resemble the same disease entity. The plethora of disease mechanisms that converge to a phenotypically similar immune cell composition renders this statement premature without elucidation of the triggers, epitopes, cytokine panels, and T-cell subsets. Future studies also should omit immunosuppressed animals, and sampling needs to be more concise regarding the primary inflammatory foci and zones, affected by seizure (excitotoxicity)–related pathologies, including secondary inflammation.

With their double labeling studies, the authors also approached the rather controversial topic of astrocytes as possible immunological targets. Strikingly, they observed the invasion of astrocyte processes by CD3-positive T cells in GME and NLE. Unfortunately, this statement cannot be supported by planar immunofluorescence images and rather requires z-dimensional scans to confirm. Astrocytes of NLE and, even more so, NME-affected brains, on the other hand, presented with a significant IgG positivity that appears to support previous studies on glial fibrillary acidic protein (GFAP)–specific antibodies. As one of the hot spots, the authors name astrocytes within the leptomeninges. This is just one example of erroneous statements, wrong citations, and misuse of neuropathological terminology that pepper the article.

It definitely is beyond our ambition to provide a second review of this article. Being the victims of incorrect citation, however, we would like to identify 2 wrong statements. First, neither Okamoto et al[2] nor Weissenböck et al[3] ever considered Borna disease virus a possible etiology of GME because these authors did not report GME-like histopathological changes at all.

Second, our communication on Leigh-like subacute necrotizing encephalopathy (SNE) in Yorkshire Terriers[4] by no means refers to mutations associated with NLE. SNE is a neurodegenerative disorder and has in common with NLE nothing but the predisposition of Yorkshire Terriers and the word *necrotizing* within the disease term. To prevent any further confusion, we would appreciate a respective corrigendum and removal of these incorrect citations from the online version of the article.

With the advancement of veterinary science, it remains the authors' responsibility to guarantee the accuracy and authenticity of extrapolated data in wording and content. Otherwise, this rapidly translates into misinformation of the scientific community and has an adverse impact on the professional development of our people in training.

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