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Abstract

Atypical myopathy (AM) is a frequently fatal pasture myopathy that emerges in Europe. More than one thousand European cases have been communicated to the AM Alert Group (AMAG) since autumn 2006. This seasonal condition kills 75% of affected horses within 72 hours with signs resulting from acute degeneration in postural and respiratory muscles.

From epidemiological studies performed on European cases [1] and by elucidating the pathophysiological mechanism [2], using several samples collected through the AMAG network, the assumption of a toxin of environmental origin that would alter the energy metabolism has been hypothetised. Indeed, affected horses have acquired deficiencies in multiple acyl-CoA dehydrogenases resulting, among others, from defects in several mitochondrial dehydrogenases [2].

Recently, it was shown that Seasonal Pasture Myopathy (SPM) in the US was caused by the toxic amino acid hypoglycin A present in the seeds of box elder trees (Acer negundo) [3]. Once ingested, hypoglycin A is metabolized into methylenecyclopropyl acetic acid (MCPA) that disrupts energetic metabolism leading to the biochemical derangements seen in both, SPM and AM.

In a preliminary study, the mitochondrial respiration in cultured equine skeletal myoblasts was monitored with high-resolution respirometry with or without addition of serum of AM-affected horses. We observed a dose-dependent inhibition of the mitochondrial respiration (up to the full inhibition) which was not induced by serum of healthy controls but that was similar to the one obtain with MCPA.

Hypoglycin A may be contained in seeds of Acer pseudoplatanus (maple tree; Aceraceae) that was consistently present in pastures of affected horses and currently, sera from European cases are being analyzed to search for MCPA-conjugates in blood. We should know soon if AM is due to the same toxin than SPM in the US.


Keywords: Horse, Myopathy, Rhabdomyolysis, Pasture, Multiple acyl-CoA dehydrogenase deficiency

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