ABSTRACT

We sought to elucidate the protein carbamoylation patterns associated with cyanate neuropathy relative to cyanide poisoning. We hypothesized that under a diet deficient in sulfur amino acids (SAA), the carbamoylation pattern associated with cyanide poisoning is similar to that of cyanate neuropathy. Male rats (6–8 weeks old) were fed a diet with all amino acids (AAA) or 75%-deficiency in SAA and treated with 2.5 mg/kg/body weight (bw) NaCN, or 50 mg/kg/bw NaOCN, or 1 µl/g/bw saline, for up to 6 weeks. Albumin and spinal cord proteins were analyzed using liquid chromatography mass spectrometry (LC-MS/MS). Only NaOCN induced motor deficits with significant levels of carbamoylation. At Day 14, we found a diet-treatment interaction effect on albumin carbamoylation ($p = 0.07$). At Day 28, no effect was attributed to diet ($p = 0.71$). Mean number of NaCN-carbamoylated sites on albumin was 47.4% higher relative to vehicle (95% CI:16.7-86.4%). Only NaOCN carbamoylated spinal cord proteins, prominently, under SAA-restricted diet. Proteins targets included myelin basic and proteolipid proteins, neurofilament light and glial fibrillary acidic proteins, and 2', 3' cyclic-nucleotide 3'-phosphodiesterase. Under SAA deficiency, chronic but not acute cyanide toxicity may share biomarkers and pathogenetic similarities with cyanate neuropathy. Prevention of carbamoylation may protect against the neuropathic effects of cyanate.