Abstract

Food (cassava) linamarin is metabolized into neurotoxicants cyanide and cyanate, metabolites of which we sought to elucidate the differential toxicity effects on memory. Young 6-8 weeks old male rats were treated intraperitoneally with either 2.5 mg/kg body weight (bw) cyanide (NaCN), or 50 mg/kg bw cyanate (NaOCN), or 1 µl/g bw saline, daily for 6 weeks. Short-term and long-term memories were assessed using a radial arm maze (RAM) testing paradigm. Toxic exposures had an influence on short-term working memory with fewer correct arm entries ($F_{2, 19} = 4.57$ $p < 0.05$), higher working memory errors (WME) ($F_{2, 19} = 5.09$, $p < 0.05$) and longer RAM navigation time ($F_{2, 19} = 3.91$, $p < 0.05$) for NaOCN relative to NaCN and saline treatments. The long-term working memory was significantly impaired by cyanide with fewer correct arm entries ($F_{2, 19} = 7.45$, $p < 0.01$) and increased working memory errors ($F_{2, 19} = 9.35$ $p < 0.05$) in NaCN relative to NaOCN or vehicle treated animals. Reference memory was not affected by either cyanide or cyanate. Our study findings provide an experimental evidence for the biological plausibility that cassava cyanogens may induce cognition deficits. Differential patterns of memory deficits may reflect the differences in toxicity mechanisms of NaOCN relative to NaCN. Cognition deficits associated with cassava cyanogenesis may reflect a dual toxicity effect of cyanide and cyanate.