Environmental Toxicology and Pharmacology 64 (2018) 70–77

Contents lists available at ScienceDirect

Environmental Toxicology and Pharmacology

journal homepage: www.elsevier.com/locate/etap

Dietary co-exposure to methylmercury and monosodium glutamate disrupts cellular and behavioral responses in the lobster cockroach, *Nauphoeta cinerea* model

Blessing A. Afolabi[a,b,c], Isaac A. Adedara[d], Diogo O. Souza[b], João B.T. Rocha[a,*]

[a] Departamento de Bioquímica e Biologia Molecular, Centro de Ciências Naturais e Exatas (CCNE), Universidade Federal de Santa Maria, 97105-900 Santa Maria, RS, Brazil
[b] Departamento de Bioquímica, Instituto de Ciências Básicas da Saúde, Universidade Federal do Rio Grande do Sul, Rua Ramiro Barcelos 2600-Anexo, 90035-003 Porto Alegre, RS, Brazil
[c] Department of Biochemistry, Bowen University Iwo, Osun State, Nigeria
[d] Drug Metabolism and Toxicology Research Laboratories, Department of Biochemistry, College of Medicine, University of Ibadan, Ibadan, Nigeria

ARTICLE INFO

Keywords:
Nauphoeta cinerea
Methylmercury
Monosodium glutamate
Locomotor behavior
Oxidative stress

ABSTRACT

The present study aims to investigate the effect of monosodium glutamate (MSG) both separately and combined with a low dose of methylmercury (MeHg) on behavioral and biochemical parameters in *Nauphoeta cinerea* (lobster cockroach). Cockroaches were fed with the basal diet alone, basal diet + 2% NaCl, basal diet + 2% MSG; basal diet + 0.125 mg/g MeHg, basal diet + 0.125 mg/g MeHg + 2% NaCl; and basal diet + 0.125 mg/g MeHg + 2% MSG for 21 days. Behavioral parameters such as distance traveled, immobility and turn angle were automatically measured using ANY-maze video tracking software (Stoelting, CO, USA). Biochemical end-points such as acetylcholinesterase (AChE), glutathione-*S*-transferase (GST), total thiol and TBARS were also evaluated. Results show that MeHg + NaCl, increased distance traveled while MeHg + MSG increased time immobile. AChE activity was significantly reduced in cockroaches across all the groups when compared to the control. There was no significant alteration in GST activity and total thiol levels. It could be that both NaCl and MSG potentiates the neurotoxic effect of MeHg in cockroaches.

1. Introduction

Methylmercury (MeHg) has been established as a precarious environmental contaminant that causes neurological deficits in both experimental animals and humans (Syversen and Kaur, 2012). The electrophilic nature of MeHg makes it react with nucleophiles such as sulfhydryl-, and selenol-containing proteins and low molecular mass thiol-containing molecules, which will be responsible for most of its toxicological effects. The potent involvement of MeHg with the activation of Nrf2, the transcription factor involved in the regulation of antioxidant system also suggests there are several mechanisms involved MeHg-induced oxidative stress and cell toxicity (Antunes dos Santos et al., 2018; Unoki et al., 2018). Experimental points of evidence have shown that methylation of inorganic mercury by methanogenic bacteria in an aquatic environment allows MeHg bioaccumulation in the aquatic food chain, eventually reaching the human diet (Honda et al., 2006; Moreira and Farina, 2014). Accordingly, seafood represents a major source of MeHg, and about 90–95% is absorbed in the gastrointestinal tract (Bradley et al., 2017). Depending on the exposure, MeHg can cause long-term neurological disturbances and can also be teratogenic (Abbott et al., 2017). An increased severity of methylmercury poisoning in the population of a MeHg polluted area in Japan who fed on high intake of fish has been observed, demonstrating for the first time dose-response effects of methylmercury in Japan (Takaoka et al., 2018). A wide range of sensory disturbance and other complications such as malignant diseases, renal diseases, respiratory diseases, amongst others were also observed in these subjects suggesting other symptoms of MeHg poisoning (Takaoka et al., 2018).

A broad toxic effect of MeHg toward different systems should be expected considering the pervasive distribution of sulfhydryl-and selenohydryl containing proteins in the nervous system, as well as the significance of the proper redox state in such proteins, which allows their correct functioning (Farina and Aschner, 2017). Apparently, metabolites of selenium have been shown to mitigate the toxicity of methylmercury (Oliveira et al.,2017). In view of this, MeHg has been described to disrupt the homeostasis of different neurotransmission

* Corresponding author.
E-mail address: jbtrocha@yahoo.com.br (J.B.T. Rocha).

https://doi.org/10.1016/j.etap.2018.09.003
Received 4 April 2018; Received in revised form 3 September 2018; Accepted 12 September 2018
Available online 16 September 2018