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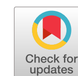
Gill bioenergetics dysfunction and oxidative damage induced by thiamethoxam exposure as relevant toxicological mechanisms in freshwater silver catfish *Rhamdia quelen*



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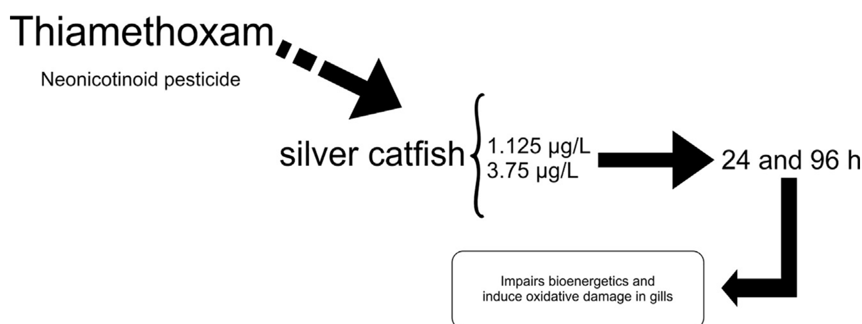
HIGHLIGHTS

- Thiamethoxam (TMX) impairs creatine kinase and sodium potassium pump activities, as well as ATP metabolism
- TMX induced branchial lipid peroxidation and protein carbonylation
- Oxidative stress-mediated inhibition of branchial CK activity

GRAPHICAL ABSTRACT



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ABSTRACT

Thiamethoxam is a neonicotinoid pesticide utilized on a worldwide scale, it has been reported in freshwater ecosystems, and detected in fishery products. Nevertheless, there is a lack of information about thiamethoxam sublethal effects on the gills of freshwater fish, principally linked to energetic metabolism. In this context, creatine kinase (CK) is an enzyme of the phosphoryl transfer network that provides a temporal and spatial energy buffer to maintain cellular energy homeostasis in tissues with high energy requirements, such as gills. Based on this evidence, the aim of this study was to evaluate whether exposure to thiamethoxam impairs the cytosolic and mitochondrial CK activities in gills of *Rhamdia quelen*, and the involvement of oxidative stress in the energetic imbalance. Branchial CK (cytosolic and mitochondrial) activity and sodium potassium pump (Na^+, K^+-ATPase) were inhibited, and adenosine triphosphate (ATP) levels decreased after 96 h exposure to 1.125 and 3.75 μg/L thiamethoxam compared to the control group. Moreover, levels of branchial thiobarbituric acid reactive substances (TBARS) and protein carbonylation increased at 3.75 μg/L thiamethoxam after 96 h of exposure compared to the control group, while the non-protein thiol (NPSH) content did not differ between groups. It is important to emphasize that all evaluated parameters did not recover after 48 h in clean water. To summarize, the data presented here clearly demonstrated that thiamethoxan exposure severely impairs cytosolic and mitochondrial CK activities, a key enzyme for gill energy buffering to maintain cellular energy homeostasis, and this effect appears to be mediated by oxidation of lipid and protein molecules, which consequently thereby induces oxidative stress.

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1. Introduction

Pesticides have been widely used in several sectors of agricultural production in order to enhance food production and improve the quality of the product (Doruchowski et al., 2017). Despite numerous merits, the abusive and indiscriminate use of pesticides represents an important environmental and public health problem worldwide due to toxicity, bioaccumulation, and environmental contamination, including the aquatic ecosystem (Zheng et al., 2016; Rodrigues et al., 2018), as only 0.1% of pesticides reach their target. Aquatic organisms, such as fish, are inevitably exposed to a several pesticides via different routes, principally run-offs or spray drifting from agricultural fields, including the thiamethoxam. This pesticide was one of the most used in rice-prawn concurrent systems of south-west Bangladesh (Sumon et al., 2016). In this sense, recent study conducted by Clasen et al. (2018) demonstrated that EngeoPleno® (Lamda-cyhalothrin + thiamethoxam) caused alterations on oxidant/antioxidant status of common carp (*Cyprinus carpio*) reared in a rice-fish system in a concentration recommended for rice-culture use in Brazil (0.2 L/ha), i.e., in fish exposed to real environmental thiamethoxam levels. However, the direct effect of thiamethoxan remains poorly understood.

Thiamethoxam is the first commercial second-generation neonicotinoid insecticide from the thianicotinyl sub-class and is used for the effective control of several commercially important insect pests on a variety of crops (Tang et al., 2017). Its mechanism of action is exerted through associated antagonistic effects on the insect synaptic and extrasynaptic nicotinic acetylcholine (nACh) receptor (Thany et al., 2007). Despite thiamethoxam representing a low risk for certain non-target organisms (e.g. mammals) (Jeschke et al., 2011), the low soil absorption, high leaching capability, high solubility in water, and resistance to biological treatment make thiamethoxam a potential contaminant of surface and underground waters (Zhang et al., 2012), which represents an important risk for fish health. Several studies have demonstrated the toxic effects of thiamethoxam on fish species, such as hepatotoxicity in zebrafish (*Danio rerio*) and bighead carp (*Aristichthys nobilis*) (Yan et al., 2016; Stoyanova et al., 2016) and hematological alterations in pacu (*Piaractus mesopotamicus*) (Carraschi et al., 2017). However, the effects of thiamethoxam contamination on gills remain poorly understood and are limited only to histopathological alterations (Georgieva et al., 2014; Ugurlu et al., 2015). Thus, additional studies are needed to understand the toxic mechanisms of thiamethoxam-induced branchial lesions, such as the involvement of creatine kinase (CK) activity. This multifaceted enzyme is linked to bioenergetic homeostasis, oxidative stress, and the normal functioning of enzymes dependent on adenosine triphosphate (ATP), such as the sodium potassium pump (Na^+, K^+-ATPase) (Amaral et al., 2012).

Creatine kinase is considered a central controller of cellular bioenergetics through the reversible interconversion of creatine (Cr) into phosphocreatine (PCr), this creates a large pool of rapidly diffusing phosphocreatine for temporal and spatial buffering of ATP levels (Schlattner et al., 2006). The control of cellular bioenergetics occurs due to the interplay between the cytosol and mitochondria by the compartmentalized subcellular sites of energy production (mitochondria) or energy consumption (cytosol by cellular ATPases) (Sauer and Schlattner, 2004). In this sense, the presence of microcompartments of CK with a variety of cellular ATPases highlights the importance of CK activity in the energetic support (by providing ATP) for the normal function of Na^+, K^+-ATPase activity, a crucial enzyme to the maintenance of branchial internal homeostasis (Bystriansky and Schulte, 2011). Recently, a study conducted by Baldissera et al. (2017) demonstrated that impairment of renal cytosolic CK activity elicited an inhibition of renal Na^+, K^+-ATPase activity, resulting in a severe energetic imbalance. Thus, our hypothesis is that impairment of the activity of CK isoenzymes leads to a downregulation of branchial Na^+, K^+-ATPase activity during thiamethoxam exposure, resulting in energy depletion.

Some evidence has suggested inhibition of CK activity occurs with oxidative stress under pathological conditions, including during exposure to contaminants, such as methylmercury (Glaser et al., 2010). Both CK isoenzymes are extremely susceptible to damage and inactivation by reactive oxygen species and by the oxidative stress framework (Wang et al., 2001), with the mitochondrial CK being more vulnerable than the cytosolic CK due to its mitochondrial localization, since the mitochondria are a rich source of reactive oxygen species (ROS), such as hydrogen peroxide (H_2O_2) and nitric oxide (NO) (Raha and Robinson, 2000). Based on the evidence that thiamethoxam induces hepatotoxicity via oxidative stress (Yan et al., 2016), our hypothesis is that exposure to thiamethoxam causes branchial oxidative stress, and consequently contributes to impairment of CK activity.

Based on this evidence, the aim of this study was to evaluate whether exposure to thiamethoxam impairs the cytosolic and mitochondrial CK activities in the gills of *Rhamdia quelen*, and the involvement of oxidative stress in the energetic imbalance.

2. Materials and methods

2.1. Chemicals

Thiamethoxam was purchased in the Brazilian market as commercial name of Actara® (Syngenta). The concentrations of 1.125 and 3.75 μg/L were choiced based on it is use in irrigated rice (Teló et al., 2015), and was already used experimentally in silver catfish by Baldissera et al. (2018). The real thiamethoxam concentration was determined in the water in the beginning (0 h) and in the end (96 h) of experiment by high-performance liquid chromatography with tandem mass spectrometry (LC-MS/MS), as published in detail by Baldissera et al. (2018).

2.2. Fish maintenance and experimental study

Juvenile *R. quelen* (106.27 ± 20.21 g; 23.5 ± 1.19 cm) were bought from a local fish farm and transferred to the Fish Physiology Laboratory at the Universidade Federal de Santa Maria. In the laboratory, the animals were acclimated during seven days in aerated 250 L tanks with controlled temperature (20.9 ± 0.03 °C), pH (6.7 ± 0.03) and dissolved oxygen (5.88 ± 0.42 mg/L). The fish were given commercial pellets to satiation once a day and feeding continued during the experimental period.

The animals were then allocated to 100 L tanks with continuous aeration and exposed for 24 or 96 h to thiamethoxam (0.0 (control), 1.125, and 3.75 μg/L), and allowed 48 h of post-recovery in water without insecticide (54 fish; three replicates per concentration; six fish/replicate). Water quality parameters, such as temperature, dissolved oxygen, total alkalinity, total hardness, total ammonia nitrogen, non-ionized ammonia and nitrite were evaluated daily, and no changes were detected throughout the experimental period, as already reported in detail by Baldissera et al. (2018).

2.3. Sample collection

Two fish from each tank (six fish per treatment at each given time, totaling 54 animals) were anesthetized and euthanized after exposures to thiamethoxam (24 and 96 h), and at the end of experiment (48 h post-recovery). Thereafter, the branchial tissue was removed for posterior determination of parameters cited below.

2.4. Branchial cytosolic and mitochondrial CK activities

Branchial tissue was washed in sucrose buffer (0.32 M sucrose; pH 7.4) and homogenized (1:10 w/v) in the same buffer with glass homogenizer. The homogenate was centrifuged at 10,000 ×g for 15 min at 4 °C, and the supernatant was collected for determination of cytosolic CK activity. The pellet, containing the mitochondria, was resuspended

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