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Energetic costs and biochemical biomarkers associated with esfenvalerate exposure in *Sericostoma vittatum*

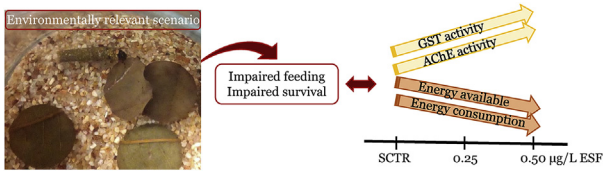
Andreia C.M. Rodrigues [a, b], Carlos Gravato [a], Carla Quintaneiro [a], Maria D. Bordalo [a], Carlos Barata [b], Amadeu M.V.M. Soares [a], João L.T. Pestana [a, *]

[a] Departamento de Biologia & CESAM, Universidade de Aveiro, Campus Universitário de Santiago, 3810-193, Aveiro, Portugal
[b] Department of Environmental Chemistry (IDAEA-CSIC), Jordi Girona, 18-26, 08034, Barcelona, Spain

HIGHLIGHTS

- Environmentally relevant levels of ESF impaired caddisflies survival and feeding behaviour.
- ESF exposure increased AChE and GST activities of *S. vittatum* larvae.
- ESF increased energetic costs related with detoxification and physiological mechanisms.

GRAPHICAL ABSTRACT



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ABSTRACT

Pyrethroid insecticides have been used for decades and their worldwide market continues to increase, despite their high toxicity to non-target insects. Recent studies reveal that it is essential to investigate the secondary mechanisms of action of type II pyrethroids to understand their cellular effects on invertebrates. The aim of this study was to evaluate the lethality, behaviour and physiological alterations and energetic costs in caddisfly larvae exposed to environmentally relevant concentrations of esfenvalerate (ESF). ESF caused both mortality and feeding inhibition of exposed caddisfly larvae: nominal ESF 96 h LC_{50} was 2.29 µg/L; feeding activity was impaired at concentrations equal or above 0.25 µg/L. At the cellular level, glutathione-S-transferase (GST) activity was increased on caddisfly larvae exposed to 0.25 and 0.5 µg/L ESF, which might contribute to prevent oxidative damage since levels of lipid peroxidation (LPO) were not altered. The energy budget of exposed caddisfly larvae was impaired by exposure to 0.25 µg/L ESF since sugar and protein contents decreased, while a decline of energy consumption was observed. The analysis of feeding, energy reserves and consumption data through structural equation modelling (SEM) allowed to quantify the direct and indirect effects of ESF exposure on bioenergetics of caddisfly larvae. SEM analysis showed a strong negative direct influence of ESF onto feeding activity, sugars content and energy consumption, highlighting a significant positive relationship between sugars and protein contents. These results show that energy expenditure is related to oxidative defense mechanisms induced by ESF stress that may lead to deleterious effects on growth and development.

1. Introduction

The global and widespread use of pyrethroids, from agricultural to urban activities, make this class of insecticides one of the most frequently found in freshwater environments at concentrations that pose risk to freshwater life (Allinson et al., 2015; EFSA, 2014; Rasmussen et al., 2013; Siegler et al., 2015). Despite their high hydrophobicity (log Kow > 4.5), and contrary to what was expected and believed in the past, the high sorption of pyrethroids to

* Corresponding author.
E-mail address: jpestana@ua.pt (J.L.T. Pestana).

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particulate organic matter and inorganic particle surfaces does not seem to cause a major decrease of its bioavailability to biota (Rasmussen et al., 2016).

Pyrethroids bind and modulate the activity of the voltage-gated sodium channels, which cause hyperactivity of the nervous system with a subsequent lack of its normal function (Casida and Durkin, 2013; Narahashi, 2000). Pyrethroids are divided into two groups, type I compounds cause a prolonged opening of sodium channels, leading to repetitive firing, while type II pyrethroids act by membrane depolarization leading to conductance block of the nervous system, resulting in prolonged sodium tail current (Narahashi, 2000). Recently, the neurotoxic effects related to most type II pyrethroids (the α-cyano pyrethroids) have been also described as a consequence of secondary sites of action, namely voltage-gated calcium channels and chloride channels, defined as potent enhancers of both calcium uptake and neurotransmitter release (Soderlund, 2012). Therefore, studying the toxicity of type II pyrethroids, such as esfenvalerate (ESF), through biochemical and physiological biomarkers is of central importance to understand their high toxicity to target and non-target invertebrates and also evolution of insect resistance.

Nowadays, the growing knowledge on cellular biomarkers allows a better understanding of their role in detoxification process in several insect species. As pyrethroid molecules are esters, cleavage by esterases has been described as one major route of their biodegradation (Sogorb and Vilanova, 2002). Moreover, resistance to pyrethroids, organophosphates and carbamates in several arthropod pests has been correlated with an enhanced production of esterases through gene amplification or upregulation (Bass and Field, 2011; Li et al., 2007). In the present study, we focus on acetylcholinesterase (AChE), the main cholinesterase described on the trichoptera species selected (Pestana et al., 2014). AChE has been used as biomarker of neurotoxicity and has been correlated with behavioural alterations under organophosphates and carbamates induced stress (Amiard-Triquet, 2009; Xuereb et al., 2009). Recently, inhibition of AChE activity due to type II pyrethroid exposure was observed in rats brain (Mani and Sadiq, 2014).

Similarly to esterases, glutathione S-transferases (GSTs) in insects have caught the attention because of their role in insecticide resistance and detoxification of pyrethroids, organophosphorus, organophosphate and organochlorine compounds (Bass and Field, 2011; Kostaropoulos et al., 2001; Li et al., 2007). Further, most of the toxic effects are known to be due to failure of phase II reactions and capability to excrete resultant metabolites. GSTs and reduced glutathione (GSH) are known to have a major role in conjugation reactions and excretion of pesticides metabolites, since they turns pyrethroids into more polar compounds easier to excrete (Regoli and Giuliani, 2014; Vontas et al., 2001). Plus, GSTs and GSH have also a vital role in the repair processes by detoxification of peroxides and oxidized DNA bases, being a key protection mechanism (Forman et al., 2009; Vontas et al., 2001). Glutathione is also a ROS reductant, reducing H_2O_2 via glutathione peroxidase (Forman et al., 2009). Another frequently studied biomarker is the anti-oxidant enzyme catalase (CAT), a ROS scavenging, which decomposes H_2O_2 (Felton and Summers, 1995), and therefore, counteract pyrethroid-induced oxidative stress.

Moreover, and since biotransformation and some antioxidant defenses require energy, the energy related parameters have also been used as indicators of chemical induced-stress and, as a reflection of trade-offs that govern organisms' life-history (Monaghan et al., 2009; Sokolova et al., 2012). It is known that the increased energy demand due to stress conditions results in a depletion of glycogen and lipid reserves (Sokolova, 2013). Additionally, under prolonged moderate to high stress condition, proteins can be metabolized to produce energy under aerobic or anaerobic metabolism (Arrese and Soulages, 2010; Sokolova et al., 2012), and several amino acids can be used to prevent oxidative damage (Monaghan et al., 2009). Also, the measurement of the energy consumption, through the electron transport system (ETS) activity, gives an insight into the metabolic activity of organisms, and energy consumption under stress conditions (Smolders et al., 2004). As energy balance is essential to an organism to be able to adapt and tolerate stress (Sokolova et al., 2012), the consequences of chemical stress to the bioenergetic parameters evaluated at the organismal level can be extrapolated, to predict effects on the growth and development of organisms (Parsons, 2007; Sokolova, 2013).

Furthermore, the ultimate effects of neurotoxicants, like ESF, will be translated into behavioural changes, as behaviour is the final outcome of a chain of neurophysiological steps (Amiard-Triquet, 2009; Lagadic et al., 1994). Indeed, behaviour results from cumulative and integrative sequences of physiological alterations, and sometimes, it is even more sensitive to chemical stress than biochemical biomarkers (Amiard-Triquet, 2009). One behavioural endpoint that has been frequently used and proved to be a sensitive and reliable ecotoxicological endpoint is feeding behaviour (Maltby et al., 2002; McWilliam and Baird, 2002). Impairment of feeding behaviour in both target and non-target invertebrate species due to pyrethroids exposure has been documented and associated with effects at individual and population levels, such as reduced growth and offspring production (Barata et al., 2006; Hannig et al., 2009; Lagadic et al., 1994). Further, the complementary use of behavioural parameters with biochemical and physiological biomarkers is recommended, and may give important clues to link sub-lethal biochemical changes to harmful effects in natural populations (Amiard-Triquet, 2009).

That said, we hypothesized that environmentally relevant concentrations of ESF would cause deleterious effects in exposed organisms through behavioural effects and also the metabolic costs of detoxification. The aim of this study was thus to evaluate behavioural and biochemical responses of the trichopteran *Sericostoma vittatum* Rambur, 1842 under exposure to environmentally relevant concentrations of ESF. Neurotransmission function was evaluated as the enzymatic activity of acetylcholinesterase – AChE – EC 3.1.1.7. Parameters related with oxidative stress, such as, the antioxidant enzyme catalase (CAT – EC 1.11.1.6), total glutathione levels (TG), phase II biotransformation enzyme glutathione S-transferase (GST – EC 2.5.1.18), and levels of lipid peroxidation (LPO) were also assessed. Energy homeostasis of exposed larvae was assessed through feeding activity and cellular energy allocation (CEA) methodology. Direct and indirect effects of exposure to ESF on feeding and energy fractions of the organisms were also analyzed and compared through the structural equation modelling (SEM) methodology.

2. Material and methods

2.1. Test organisms and alder leaves

Sericostoma vittatum Rambur (Trichoptera: Sericostomatidae) is a freshwater detritivore, described as a very efficient shredder (Campos and González, 2009; Feio and Graça, 2000). Also, its feeding behaviour has been shown to be a sensitive and reliable endpoint for ecotoxicological studies (Campos et al., 2014; Pestana et al., 2009b). *S. vittatum* larvae were sampled using a hand net, in the fourth-order São João stream, Lousã, Portugal (40°06′N, 8°14′W). At the laboratory, the organisms were acclimated for at least 1 week at constant temperature (20 ± 1 °C), 16 h light:8 h dark, in American Society for Testing Materials (ASTM) hard water (ASTM, 1980) constantly aerated and 1 cm layer of inorganic fine