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Effects of sophoroflavonoside and narcissin flavonoids on the amount of malondialdehyde, a product of lipid peroxidation, and the activity of the enzyme cytochrome c oxidase in rat liver mitochondria poisoned with indoxacarb pesticide

M.J. Parpieva[1], P. Mirkhamidova[2], M.K. Pozilov[3], R.A. Alimova[4]*

1. Andijan State University, Andijan City
2. Tashkent State Pedagogical University Named after Nizomi, Tashkent City
3. M. Ulugbek National University of Uzbekistan, Tashkent City
4. Tashkent State Agrarian University, Tashkent City, Republic of Uzbekistan

** Corresponding author's E-mail: alimovdt@gmail.com*

ABSTRACT

In this study, the effects of soforaflavonolonoside and narcissin flavonoids on the amount of malondialdehyde, a product of peroxidation of lipids in the liver mitochondria of rats poisoned with indoxacarb pesticide, and on the activity of the enzyme cytochrome-s-oxidase were studied depending on the dynamics of 10, 20, 30 and 40 days. The experimental group was injected with indoxacarb pesticide at a dose of 1/10 LD_{50} through a special probe. After administration of indoxacarb pesticide, animals were administered orally with soforaflavonolonoside and narcissin flavonoid at a dose of 10 mg kg^{-1} once a day for 10 days.

Key words: Pesticide, Indoxacarb, saphoroflavonoside, Flavonoid, Saphoroflavonoside (SFL), Narcissin, Lipid peroxidation (LPO).
Article type: Short ommunication.

INTRODUCTION

Currently, many types of pesticides are produced all over the world and are widely used in agriculture against crop pests, the production of pesticides is 1 million tons per year (Dzhurabekova & Saidov 2015). The concentration of pesticides in cultivated fields around the world is 300 g ha^{-1} of cultivated land or 30 mg m^{-2}. In European countries, this figure is 295 mg m^{-2} per year (Dremova 2011; Dzhurabekova & Saidov 2015). This has a negative impact on the environment, enters all living organisms in different ways, and accumulates in tissues as a residual pesticide. The amount of residual pesticides can affect the biochemical and physiological processes of liver mitochondria and cause a number of changes (Alimbabaeva *et al.* 2005; Mirkhamidova *et al.* 2022; Assi *et al.* 2022). Acute and chronic poisoning with pesticides causes damage to all tissues, structural changes in bone marrow elements, peripheral blood, endocrine glands, liver, kidney, heart muscle and brain cells (Akinshina & Gudnikova 2003). Pesticide poisoning affects all tissues and organs, however, the most sensitive organ to their effects is the liver (Alimbabaeva *et al.* 2005). Detoxification takes place mainly in the liver. Liver damage affects metabolism. The emergence and development of a number of pathological conditions in humans and animals is caused by LPO activation of cell membranes (Mirkhamidova *et al.* 2020). LPO activation by pesticides weakens the antioxidant defense system in mitochondria, and antioxidant enzymes can degrade and reduce the amount of free radicals ($\bullet O_2^-$, •ON, and N_2O_2) in the cytosol [8]. However, the molecular basis of the effect of the newly synthesized indoxacarb pesticides on tissues and cells has not been fully investigated, and currently, the effects of these pesticides on the disruptions in the liver mitochondrial membranes, including LPO and cytochrome C

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oxidase activity, and their correction with herbal preparations have not been studied. For this purpose, in this experiment, the effects of SFL and narcissin on the amount of LPO product MDA and cytochrome c oxidase activity of rat liver mitochondria poisoned with indoxacarb were studied based on the dynamics of 10, 20, 30 and 40 days. The purpose of the study was to examine the dynamics of the correcting effect of sophoraflavonolonoside and narcissin flavonoids on the liver mitochondrial membrane dysfunction of indoxarab-poisoned rats for 10, 20, 30 and 40 days.

MATERIALS AND METHODS

Indoxacarb is an insecticidal chloroorganic compound, which belongs to a new chemical class - oxadiazines (Khzhaev *et al.* 2009). Indoxacarb ($C_{22}H_{17}ClF_3N_3O_7$,) suspension concentration of 15% is a synthetic drug and an insecticide. Indoxacarb blocks sodium channels of nerve fibers of poisonous insects, stopping feeding. Their coordination is disturbed, and then paralysis and death occur. The LD_{50} dose of indoxacarb in rats is 5000 mg kg^1. It is considered to be less toxic to humans and warm-blooded animals and has a toxicity class 3, but has a toxicity class 1-2 for beneficial insects (www. pesticidy.ru>active_substance>indoxacarb). The antitoxic properties of saphoroflavonoside (SFL) and narcissin flavonoids selected for the experiment were studied against pesticides. Experimental animals were divided into separate model groups for intoxication with indoxacarb pesticides and their correction with flavonoids. Rats poisoned with indoxacarb pesticide were divided into groups. Group I was healthy (control); Group II indoxacarb; Group III indoxacarb + SFL; group IV indoxacarb + narcissin. Animals of the II, III and IV groups of the experiment were poisoned once with a 1/10 LD_{50} dose through a special probe with indoxacarb pesticide. After the indoxacarb pesticide was administered, SFL 10 mg kg^{-1} was administered to experimental group III and narcissin flavonoid 10.0 mg kg^{-1} was administered orally once a day to group IV for 10 days. After 10, 20, 30 and 40 days of administration of SFL and narcissin flavonoids to pesticide-poisoned rats, the levels of mitochondrial membrane LPO product MDA and mitochondrial membrane-dependent enzyme cytochrome-s-oxidase enzyme activity were investigated. Rat liver mitochondria were isolated by differential centrifugation. The detection of LPO is based on the reaction between MDA and thiobarbituric acids (TBK), which results in the formation of a colored trimethine complex at high temperature and acidic pH (Rogozhin 2013). The complex was measured in a spectrophotometer at a wavelength of 532 nm. Mitochondrial membrane-bound enzyme cytochrome-s-oxidase enzyme activity was determined spectrophotometrically by the rate of oxidation of dithionite-reduced cytochrome-C (Parpiyeva *et al.* 2022) and measured in a spectrophotometer at a wavelength of 550 nm.

RESULTS AND DISCUSSION

In our preliminary experiment, the effects of indoxacarb-poisoned rats on the amount of liver mitochondria MDA were studied. According to the obtained results, the amount of LPO product MDA in liver mitochondria was 36.4 ± 2.4%, 36.7 ± 2.8%, 33.3. It was found that it increased by ±1.9% and 25.7±1.3%. In group III rats treated with SFL, the amount of liver mitochondria MDA was 9.1 ± 0.8%, 16.4 ± 1.2%, 18.8 ± 2%, respectively, compared to the values of group II at 10, 20, 30 and 40 days, 1% and 11.5 ± 1.2% decrease (Fig. 1).

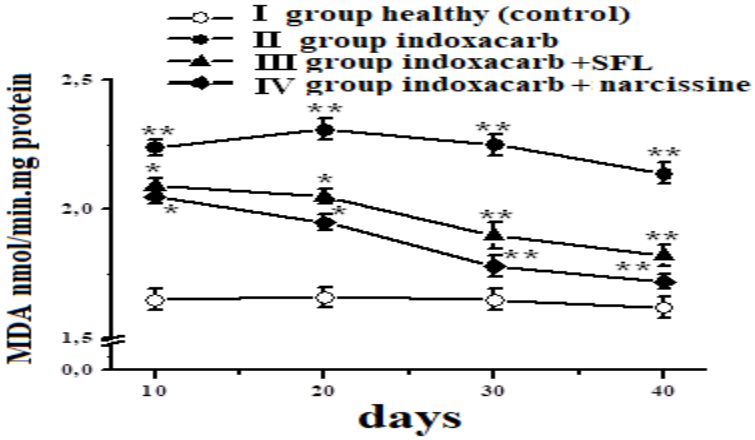

Fig. 1. 40-day dynamics-dependent effects of SFL and narcissine on MDA content in liver mitochondria of indoxacarb pesticide-intoxicated rats (*R < 0.05; *R < 0.01; n = 5-6).

In group IV rats injected with narcissin, the amount of MDA decreased by 15.2 ± 1.2% and 25.3 ± 1.7%, respectively, compared to group II at 10 and 20 days. However, on days 30 and 40, the amount of MDA in liver mitochondria decreased by 25.5 ± 1.6% and 16.4 ± 1.5%, respectively, compared to the values of group II (Fig. 1). In our next experiment, the effects of SFL and narcissin compounds on cytochrome C oxidase activity in liver mitochondria of rats treated with indoxacarb pesticide were investigated. According to the obtained results, the cytochrome c oxidase activity of liver mitochondria in group II rats poisoned with indoxacarb pesticide on 10, 20, 30 and 40 days were 62.6 ± 4.2%, 55.8 ± 4.4%, 44.8. It was found that it decreased sharply by ± 3.0% and 41.7 ± 2.7%. The highest inhibition of enzyme activity was observed on the 10th day of poisoning. The obtained results showed that pesticides lead to disruption of the function of mitochondria membranes of the liver of poisoned rats. In the literature, under the influence of pesticides (karate, chlorprifos), a deep inhibition of the enzyme cytochrome-s-oxidase was observed [2,12]. Liver mitochondrial cytochrome c oxidase activity in group III rats treated with SFL pharmacotherapy was 22.2 ± 1.6%, 17, 17. It was found that it increased by 0 ± 1.3%, 17.8 ± 1.1% and 25.4 ± 1.7% (Fig. 2). In group IV rats injected with narcissin, cytochrome-c-oxidase activity did not recover compared to group II at 10 and 20 days. However, on days 30 and 40, the activity of cytochrome-s-oxidase of liver mitochondria was found to be restored by 8.0 ± 0.5% and 19.0 ± 1.1%, respectively, compared to the indicators of group II (Fig. 2).

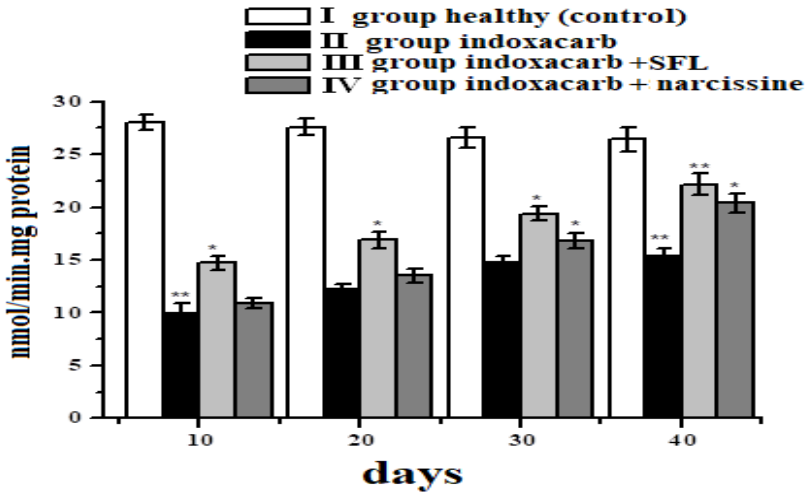

Fig. 2. Dynamic effects of SFL and narcissin on cytochrome c oxidase enzyme activity in liver mitochondria of indoxacarb pesticide-poisoned rats at 10, 20, 30 and 40 days (*R < 0.05; **R < 0.01; n = 5-6).

In these experiments, the corrective effect of SFL flavonoid was proved to be more effective than that of narcissin flavonoid.

CONCLUSION

Thus, under the influence of the indoxacarb pesticide, an increase in the amount of MDA, a product of the liver mitochondrial membrane LPO, and as a result, a sharp decrease in the activity of the membrane-dependent enzyme cytochrome-s-oxidase was observed. An increase in the amount of MDA and a profound inhibition of cytochrome-s-oxidase activity under the influence of pesticides were observed on 10-20 days of poisoning, which in turn leads to a change in the transfer of electrons in the respiratory chain in the mitochondria of the liver of rats. Such a pro-oxidant effect of pesticides can increase the formation of PFSH from the respiratory chain by reducing the activity of cytochrome-s-oxidase, causing an increase in LPO of the membrane. SFL and narcissin flavonoid compounds used in the experiments show antioxidant properties (Parpieva *et al*. 2022), decrease the amount of MDA, a product of liver mitochondrial membrane LPO, and restore the activity of cytochrome-s-oxidase to a certain extent.

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