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RESEARCH ARTICLE

Lipid peroxidation and antioxidant defense system in spawn of brown trout (*Salmo trutta* m. *trutta* L.) affected by ulcerative dermal necrosis

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Abstract. The objective of the present study was to investigate the importance of the antioxidant enzymes: superoxide dismutase (SOD), catalase (CAT), glutathione reductase (GR), and glutathione peroxidase (GPX) in the defense against lipid and protein oxidation in spawn from female brown trout, *Salmo trutta* m. *trutta* L. affected by ulcerative dermal necrosis (UDN). UDN induced increased lipid and protein oxidation levels. The current results show that UDN infection led to oxidative stress with the inhibition of antioxidant defense mechanisms. The inhibition of glutathione defense system activity might be responsible for this failure in cellular antioxidant defenses. UDN induces irreversible changes in pro- and antioxidative function which effected decreased spawn survival as well as reductions in spawning efficiency.

Keywords: brown trout, ulcerative dermal necrosis, spawn, oxidative stress, antioxidant defense system

Introduction

Ulcerative dermal necrosis (UDN) is a serious fish disease which has been spreading across Poland in recent years. The etiology of UDN outbreaks remains unknown, and there is no conclusive evidence of the involvement of any particular organism as the primary pathogen (Ribelin and Migaki 1975, Roberts 1993). It was suggested that these fungal infections are triggered by metabolites of necrotic epidermal cells (Khoo 2000). Epidermal cells are shed, and the fungus determines the further course of the disease which terminates in large ulcers covered with fungal hyphae (Roberts and Hill 1976, Roberts 1993, Khoo 2000).

The afflicted fish develop severe skin lesions over large parts of their bodies that penetrate into the muscle (Bullock and Roberts 1979, Johansson et al. 1982, Bruno et al. 2007). The onset of symptoms only occurs after migration into fresh water. Lesions quickly become infected with *Saprolegnia* fungus, and this gives infected fish the appearance of being covered in slimy, white pustules (Ribelin and Migaki 1975, Khoo 2000). The initial symptoms of the disease are circles of pathologically-altered epidermis. Subsequently, inter-cellular spaces dilate and communicate with the exterior. The necrosis of the epidermal cells occurs simultaneously with fungal infections and the marked responses of melanophores (Roberts and Hill 1976, Roberts 1993). Histological symptoms include necrotic, granulomatous dermatitis, and myoositis associated with invasive, non-septate fungal hyphae. The fungus can penetrate visceral organs, such as the kidney and liver, after it has penetrated through the musculature. Frequently, the most severely affected fish die before spawning.

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Factors intrinsic to the fish itself, such as age, phylogenetic position, and feeding behavior, as well as environmental factors such as the type of diet supplied, daily or seasonal changes in temperature, dissolved oxygen, toxins present in the water, pathologies, or parasites, can either fortify or weaken antioxidant defense systems and cause oxidative stress, which is aggravated by UDN (Kane et al. 2000, Law 2001). Brown trout is one of the most common, numerous, and valuable species inhabiting some Pomeranian rivers (Radtke et al. 2006). However, only a few studies have been conducted on the pathology of ulcerative dermal necrosis in brown trout populations (Roberts and Hill 1976, Bullock and Roberts 1979, Johansson et al. 1982, Roberts 1993).

The increased generation of reactive oxygen species (ROS) and lipid peroxidation have been noted in the pathogenesis of many diseases of known and unknown etiology (Yagi 1993). Previous studies by the current authors identified the effects of UDN-induced oxidative stress on the parameters of lipid peroxidation and activities of antioxidative defense system on the livers, muscle tissues, and hearts of brown trout. Selected pro- and antioxidative profile parameters of healthy brown trout were studied and compared with those of UDN-positive fish (Kurhalyuk et al. 2009). The effect of UDN-induced oxidative stress on lipid peroxides and enzymatic antioxidants in the spawn of brown trout, *Salmo trutta* m. *trutta* L., from the River Słupia (northern Poland, central Pomeranian) was conducted to evaluate the effects, if any, of UDN on general antioxidative enzyme activities, lipid peroxidation, and the rate of protein oxidative destruction.

Materials and methods

Fish

Adult brown trout aged 3-5 years were collected from sites on the Słupia River, near Słupsk in northern Poland. The fish were caught in co-operation with the Dolina Słupia Landscape Park and the Słupsk chapter of the Polish Angling Association. Sea trout were sampled from November to December in both 2007 and 2008.

Sampling

The samples used in the analysis were collected immediately following catch, and comprised 47 healthy brown trout females (control group) and 38 brown trout females affected by UDN (study group). The average body weight of the fish was 1.98±0.06 kg (healthy females) and 2.41±0.28 kg (UDN-affected females). The trout were caught in the River Słupia with electrofishing. They were anesthetized quickly and then sacrificed. Microbiological tests suggested that the *Aeromonas hydrophila* complex caused UDN (Szewczyk 2005). Individuals from both groups were transported to the Department of Animal Physiology, Institute of Biology and Environmental Protection, Pomeranian University (Słupsk, Poland) in tanks filled with water from their natural environment and analyzed within one day of capture.

Treatment of samples

The specimens from each group were dissected, and one fish was used for each preparation. The spawn from the trout were homogenized in 0.1 M tris-HCl buffer (pH 7.4). The protein content of the samples was determined according to Bradford (1976) using bovine serum albumin as the standard.

Analytical methods

Thiobarbituric acid (TBA), oxidized and reduced glutathione (GSSG and GSH), NADPH, and 5,5-dithiobis-2-nitrobenzoic acid were purchased from Sigma (St. Louis, MO, USA). Ethylenediaminetetraacetic acid (EDTA), thrichloroacetic acid (TCA), quercetin, hydrogen peroxide, ammonium molibdate, sodium aside, t-butylhydroperoxide, Tween-80, urea acid, 2,4-dinitrophenyl hydrazine (DNFH) were obtained

from Fluka (Buchs, Switzerland). All other chemicals were of analytical grade.

All enzymatic assays were carried out at 25±0.5°C using a Specol 10 spectrophotometer (Carl Zeiss Jena, Germany). The enzymatic reactions were started by adding the homogenate suspension. The specific assay conditions are presented subsequently. The lipid peroxidation level was determined by quantifying the concentration of 2-thiobarbituric acid reactive substrates (TBARS) with the Kamyshnikov (2004) method for determining the malondialdehyde (MDA) concentration. This method is based on the the reaction of the degradation product of lipid peroxidation, MDA, with TBA under conditions of high temperature and acidity to generate a colored adduct that is measured spectrophotometrically. A 2.1 mL sub-sample of the spawn homogenate was mixed with 1 mL of TCA and 1 mL of TBA reagent. The mixture was heated in a boiling water bath for 10 minutes. After cooling, the mixture was centrifuged at 3000 g for 10 minutes. The MDA level was expressed in μmol MDA mg^{-1} protein by using 1.56 10^5 mM^{-1} cm^{-1} as the extinction coefficient.

The rate of protein oxidative destruction was estimated from the reaction of the resultant carbonyl derivatives of amino acid reaction with DNFH as described by Levine et al. (1990) and as modified by Dubinina et al. (1995). DNPH was used for determining the carbonyl content in soluble and insoluble proteins. A quantity of 1 mL 0.1 M DNPH (dissolved in 2 M HCl) was added to 0.1 mL of the tissue sample after the proteins were denatured. After adding the DNPH solution (or 2 M HCl to the blanks), the tubes were incubated for 1 h at 37°C. The tubes were centrifuged for 20 min at 3000 g. After centrifugation, the supernatant was decanted, and 1 mL of an ethanol-ethylacetate solution was added to each tube. Following the mechanical disruption of the pellet, the tubes were allowed to stand for 10 min and then centrifuged again (20 min at 3000 g). The supernatant was decanted and the pellet was rinsed with ethanol-ethylacetate two more times. After the final rinse, the protein was solubilized in 2.5 mL of 8 M urea solution. To speed up the solubilization process, the samples were incubated in a 90°C water bath for 5-10 min. The final solution was centrifuged to remove any insoluble material. The carbonyl content was calculated from absorbance measurements at 370 nm and 430 nm and an absorption coefficient of 22,000 M^{-1} cm^{-1}. Carbonyl groups were determined spectrophotometrically from the difference in absorbance at 370 (OMP_{370}) and 430 (OMP_{430}) nm and expressed as E mg^{-1} protein.

Superoxide dismutase (SOD, E.C. 1.15.1.1) activity was measured with the method by Kostiuk et al. (1990) using quercetin as the substrate after suitable dilution. A total volume of 1 mL of the assay mixture comprised 0.08 mM EDTA and 0.1 M sodium phosphate buffer (pH 7.8). After dilution, the tissue homogenate was added to the assay mixture with EDTA and the sodium phosphate buffer. One unit of SOD activity was defined as the quantity of enzyme that inhibited quercetin oxidation by 50% under given experimental conditions. This activity was expressed in units of SOD mg^{-1} protein.

Catalase (CAT, E.C. 1.11.1.6) activity was determined with the Koroliuk et al. (1988) method by measuring the decrease in H_2O_2 in the reaction mixture using a spectrophotometer at a wave length of 410 nm. The reaction was started by adding 0.1 mL of tissue homogenate to 2 mL of the 0.03% H_2O_2 solution and 1 mL of 4% ammonium molibdate. One unit of catalase activity was defined as the amount of enzyme required to clear μmol of H_20_2 min^{-1} mg^{-1} protein.

Glutathione reductase (GR, E.C. 1.6.4.2) activity in the spawn homogenate was measured according to the method described by Glatzle et al. (1974). The enzyme assay mixture contained 2.4 mL of 67 mM sodium phosphate buffer (pH 6.6), 0.2 mL of 7.5 mM oxidized glutathione, and 0.1 mL of tissue homogenate. The rate of NADPH oxidation was followed spectrophotometrically at 340 nm. A control without NADPH was used and the GR activity was expressed as nmol NADPH min^{-1} mg^{-1} protein.

Glutathione peroxidase (GPX, E.C. 1.11.1.9) activity in the spawn homogenate was measured spectrophotometrically as described by Moin (1986). The assay mixture contained 0.8 mL of 0.1 M Tris-HCl with 6 mM EDTA and 12 mM sodium aside (pH 8.9),

0.1 mL of 4.8 mM GSH, 0.2 mL of tissue homogenate, 1 mL of 20 mM t-butylhydroperoxide, and 0.1 mL of 0.01 M 5,5-dithiobis-2-nitrobenzoic acid. The rate of GSH reduction was followed spectrophotometrically at 412 nm. GPX activity is expressed as μmol GSH min^{-1} mg^{-1} protein.

The level of the total antioxidant activity (TAA) in the spawn homogenate was estimated spectrophotometrically at 532 nm following the method with Tween-80 oxidation as described by Halaktionova et al. (1998). The mixture containing the tissue homogenate and Tween-80 was heated in a boiling water bath for 48 hours at 37°C. After cooling, TCA was added and the mixture was centrifuged for 15 min at 3000 g for 10 min. After centrifugation, 2 mL of TBA reagent and 2 mL of supernatant was mixed. The mixture was heated in a boiling water bath for 15 minutes at 90°C. The total antioxidative activity in the spawn was expressed in %.

Statistical analysis

The results were expressed as means ± standard error. Student *t*-tests with 95% confidence intervals (α = 0.05) were applied to determine the significance of differences between groups. Correlations between the level of oxidative stress and antioxidant enzyme activities at set significance levels were determined with the regression method. Interactions were identified by the Pearson test for linear correlation (Zar 1999). All statistical calculations were performed on separate data from each individual with Statistica 8.0 (StatSoft Inc., Tulsa, OK, USA).

Results

The values of lipid peroxidation in the spawn for the females from the control group (healthy specimens) and UDN-affected trout are summarized in Figure 1. The enhanced TBARS levels indicated a significant increase in lipid peroxidation levels in the spawn of UDN-affected trout. The TBARS levels in the spawn homogenates of UDN-positive females were

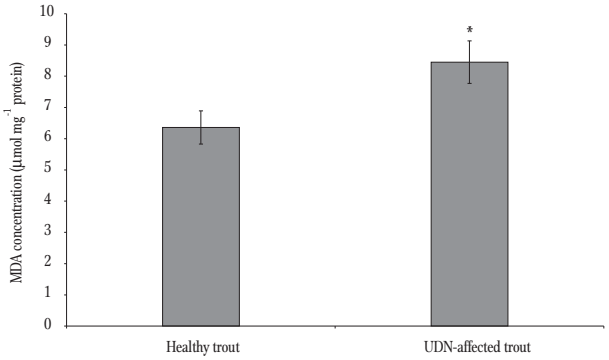

Figure 1. Malondialdehyde concentration in the spawn from control (healthy specimens) and UDN-affected trout. * significance of differences between healthy trout and UDN-affected trout values (P < 0.05, test t).

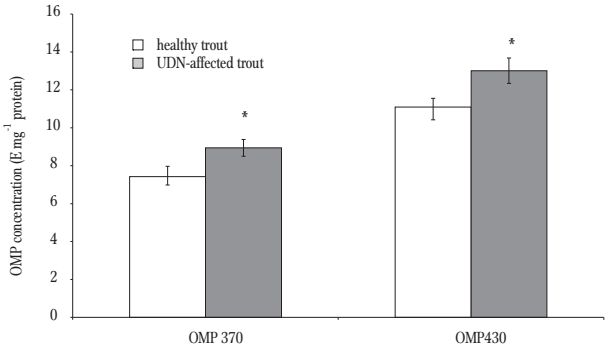

Figure 2. Content of oxidatively modified proteins (OMP), measured by the quantity of carbonyl oxidation in the spawn from control (healthy specimens) and UDN-affected trout. Carbonyl groups were determined spectrophotometrically from the difference in absorbance at 370 (OMP_{370}) and 430 (OMP_{430}) nm. * significance of differences between healthy trout and UDN-affected trout values (P < 0.05, test t).

significantly higher by 32.9% (P < 0.05) in comparison with those of the control group (Fig. 1).

The rate of protein oxidation measured by the quantity of carbonyl oxidation in the spawn homogenates was significantly higher by 20.5% (P < 0.05) and 17.3% (P < 0.05) for OMP_{370} and OMP_{430}, respectively (Fig. 2).

The activities of antioxidant enzymes in the spawn of trout are presented in Table 1. In comparison to the control group, no significant difference was determined in the SOD activity of the UDN-affected group, in which SOD activity was 14% lower

Table 1
Antioxidant enzyme activities in the spawn of UDN-affected females

Antioxidant parameters	Healthy trout	UDN-affected trout	P
SOD (U mg^{-1} protein)	617.75±30.70	532.03±38.55	0.092
CAT (μmol min^{-1} mg^{-1} protein)	6.40±1.49	2.20±0.35	0.010
GR, (nmol min^{-1} mg^{-1} protein)	67.95±7.96	32.99±4.50	0.000
GPX (μmol min^{-1} mg^{-1} protein)	51.49±6.78	29.69±3.04	0.006
TAA (%)	60.28±3.82	62.36±2.01	0.632

Table 2
Correlative analysis of antioxidant parameters in the spawn of trout affected with ulcerative dermal necrosis (UDN)

Variables	r	Regression equation	r^2	P
TBARS-OMP_{370}, control	0.393	y=0,159x+4,28	0.154	0.049
GR-GPX, control	0.510	y=3.818x+89.20	0.260	0.000
GPX-OMP_{370}, control	0.401	y=0.312x+5.88	0.161	0.011
TBARS-GPX, UDN	0.500	y=3.93x+80.88	0.250	0.001
TBARS-OMP_{370}, UDN	0.520	y=0.272x+6.31	0.270	0.020
$OMP_{370\text{-}OMP430}$, UDN	0.795	y=0.085x-2.52	0.632	0.000

(P > 0.05). Statistically significant differences were determined between the CAT activity of the control group and the UDN-affected trout; that of the former decreased by 34.4% (P < 0.05) of that of the control group. Some changes in GPX activity were also observed. The GPX and GR activities in the spawn of UDN-affected fish were lower by 42.3% (P < 0.01) and by 51.4% (P < 0.01), respectively, in comparison to the controls. UDN infection did not significantly increased total antioxidant activity (TAA) in the spawn of UDN-affected females.

A significant, positive correlation was observed between antioxidant enzyme activity and peroxidation levels in the spawn of females from the control group and the UDN-affected group (P < 0.05) (Table 2, Fig. 3). TBARS levels in the spawn from UDN-affected females correlated with the ketonic (r = 0.622, r^2 = 0.387) (Fig. 3a) and aldehyde derivate levels of oxidatively modified proteins (r = 0.520, r^2 = 0.270) (Table 2). Catalase activity correlated inversely with aldehyde derivate levels of oxidatively modified proteins from females infected with UDN (r = -0.627, r^2 = 0.393) (Fig. 3b). Thus, GPX activity was connected with lipid peroxidation content in the spawn of UDN-affected females (r = 0.500, r^2 = 0.250). Relationships between ketonic and aldehyde derivate levels of oxidatively modified proteins were also observed (r = 0.795, r^2 = 0.632) (Table 2).

Discussion

The present study demonstrated that there is an antioxidative response in the spawn of UDN-affected brown trout. The specific questions addressed in the current study were whether oxidative stress occurs during UDN and whether the antioxidative defense system is induced. The interplay between defense systems is evidently important for the passive survival of UDN-induced oxidative stress. Recent data suggest that no study has yet focused on the issue of potential oxidative stress in fish affected by UDN.

Cells and tissues are continuously threatened by damage from free radicals and reactive oxygen species (ROS), which are produced during normal

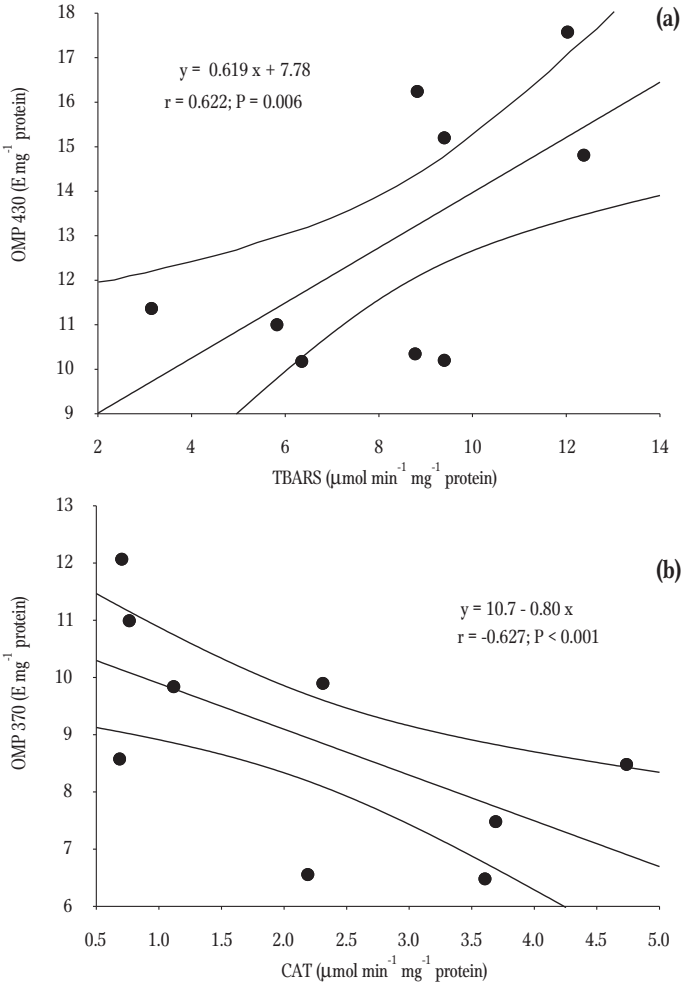

Figure 3. Dependence between levels of oxidatively modified proteins and TBARS concentration (a) and catalase activity (b) in the spawn of trout affected of ulcerative dermal necrosis.

oxygen metabolism or are induced by exogenous damage or diseases (de Groot 1994, Grace 1994). The mechanisms by which free radicals interfere with cellular functions are not fully understood, but one of the most important events seems to be lipid and protein oxidation, which results in cellular membrane damage. This cellular damage causes a shift in the net charge of the cell and changes osmotic pressure, which leads to swelling and eventually cell death (Nijveldt et al. 2001).

The current paper confirms that the spawn homogenate of trout affected by UDN undergo lipid and protein oxidation due to the oxidizing effects of ROS. UDN induced increased TBARS levels in the spawn of UDN-affected trout. The depletion of the antioxidant defense system and changes in the activities of various antioxidant enzymes indicative of lipid peroxidation were implicated in oxidative tissue damage. Previous studies by the current authors indicated that UDN is quite capable of causing oxidative stress in the livers, muscle tissues, and hearts of brown trout (Kurhalyuk et al. 2009). UDN caused the accumulation of lipid and protein oxidation end products. The peroxidation process was preceded by a decrease in the cell antioxidant defense system followed by the production of lipid and protein oxidation products. Correlative relationships between prooxidative parameters and antioxidant enzyme activities also confer with what is anticipated if the peroxidation process is subsequent to the consumption of the antioxidant defense system. TBARS levels and oxidatively modified protein content were also

potential markers of oxidative stress mediated by UDN.

ROS can attract various mediators that contribute to cell response and tissue damage. To protect themselves from ROS, organisms have developed several effective mechanisms (Halliwell 1995). The antioxidant defense system includes enzymes such as SOD, CAT, GR, and GPX, but also non-enzymatic counterparts such as glutathione, ascorbic acid, and α-tocopherol. The increased production of ROS during diseases results in the consumption and depletion of endogenous scavenging compounds (Yagi 1993). The current results indicate that UDN caused a decrease in the antioxidant enzyme activities in the spawn homogenate from UDN-affected females, including those of CAT, GPX, and GR. This might be due to the inactivation of the above-mentioned enzymes by the end products of lipid and protein oxidation.

SOD is the antioxidant enzyme that catalyzes the dismutation of the highly reactive superoxide anion to O_2 and to the less reactive species H_2O_2 (Buetler et al. 2004). CAT reacts very efficiently with H_2O_2 to form water and molecular oxygen. GPX catalyses the reduction of H_2O_2 and lipid hydroperoxides at the expense of GSH, thereby protecting cells against oxidative damage. GSSG is reduced to GSH by GR (Rikans and Hornbrook 1997). In the current study, the inhibition of GR and GPX activity was observed in the spawn from UDN-affected trout. Decreased GR and GPX activities indicated a reduced capacity to scavenge for H_2O_2 and lipid hydroperoxides produced in the spawn of UDN-affected trout. The drop in CAT activity in the spawn could be explained by the flux of superoxide radicals due to the oxidative stress caused by UDN infection. This corresponds well with a previous study by the current authors in which the inhibition of the antioxidant defense system in the livers, muscles, hearts, and blood of UDN-affected trout was noted (Kurhalyuk et al. 2009).

In conclusion, the current study suggests that UDN causes changes in oxidative stress intensity in the spawn of UDN-affected trout. Moreover, increased lipid and protein oxidation modifies the antioxidant defense system and causes the inhibition of CAT, GPX, and GR activities in the spawn of UDN-affected females. This might be due to the inactivation of antioxidant enzymes by the end products of lipid and protein peroxidation. The current results suggest the importance of the glutathione-mediated antioxidant defense system in the protection against UDN-induced oxidative stress in trout.

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Streszczenie

Intensywność procesów lipoperoksydacji i aktywność enzymów antyoksydacyjnych w ikrze troci wędrownej (*Salmo trutta* m. *trutta* L.) z wrzodziejącą martwicą skóry

Celem badań była analiza poziomu intensywności procesów lipoperoksydacji i oksydacyjnej modyfikacji białek oraz aktywności enzymów antyoksydacyjnych (dysmutaza ponadtlenkowa, katalaza, glutationreduktaza i glutationperoksydaza) w ikrze samic troci wędrownej z wrzodziejącą martwicą skóry (UDN). Wyniki naszych badań sugerują zwiększenie poziomu procesów peroksydacji lipidów oraz ketonowych i aldehydowych pochodnych oksydacyjnej modyfikacji białek w ikrze u osobników z UDN. Zaznaczone schorzenie wywołuje stres oksydacyjny w komórkach ryb z jednoczesną inhibicja mechanizmów antyoksydacyjnej obrony. W największym stopniu te zmiany dotyczą układu glutationowego, który uważa się za podstawowy element bilansu antyoksydacyjnego. Negatywne skutki UDN powodują nieodwracalne zmiany funkcjonowania systemu pro- i antyutleniania komorek, co obniża w następstwie przeżywalność ikry ryb oraz wydajność procesów tarłowych dla tego cennego gatunku.