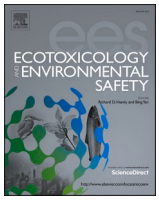
Ecotoxicology and Environmental Safety 281 (2024) 116586

Contents lists available at ScienceDirect

Ecotoxicology and Environmental Safety

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



Microencapsulation of sunscreen reduces toxicity of its components to *A. salina*: Biochemical, behavioral and morphological studies

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ARTICLE INFO

Edited by Dr Hyo-Bang Moon.

Keywords:
Artemia salina
Marine environment
Microencapsulation
Pollution
Sunscreen

ABSTRACT

Sunscreens contain several substances that cause damage to species where they are disposed. New formulations have been created to prevent such marine environmental damages. One promising formulation is the microencapsulated sunscreen. The objective of this study was to evaluate the possible safety to marine environment of one microencapsulated sunscreen formulation. The animal model *Artemia salina* (cists and nauplii) was tested with two sunscreen formulations (microencapsulated and non-microencapsulated) and toxicological, behavioral, morphological parameters as well as biochemical assays (lipoperoxidation and carbonylation tests) were analyzed. Results showed that microencapsulated sunscreen impeded some toxic effects caused by the release of the substances within the microcapsule in the highest concentration, reestablishing the mortality and hatching rates to control levels, while removing the sunscreen microcapsule by adding 1 % DMSO reduced the cyst hatching rate, increasing the nauplii mortality rate and decreased locomotor activity in higher concentrations. Finally, nauplii with 24 hours of life and exposed to sunscreen without the microcapsule showed an increase in mitochondrial activity (assessed at 48 hours after exposure) and presented malformations when exposed to the highest concentration non-microencapsulated concentration (assessed by SEM at 72 hours after exposure), when compared to the control group. These results together allow us to conclude that the microencapsulation process of a sunscreen helps protecting *A. salina* from the harmful effects of higher concentrations of said sunscreens. However, long-term studies must be carried out as it is not known how long a microencapsulated sunscreen can remain in the environment without causing harmful effects to the marine ecosystem and becoming an ecologically relevant pollutant.

1. Introduction

The first references about skin pathologies related to UV radiation date from 1894 (Corrêa, 2015). Thenceforth, it is an unanimity the beneficial preventive effect of sunscreens cosmetics. Brazil represents 82 % of the consumption of sunscreens in Latin America and is the second largest consumer in the world (ABIHPEC, 2014).

Sunscreens are commonly divided into two groups: inorganic and organic. Both inorganic (e.g., zinc oxide and titanium dioxide) and organic filters are considered effective against UVA and UVB radiation (Geoffrey et al., 2019). The most common formulations have chemical agents such as oxybenzone, avobenzone, octocrylene (OCR) and octylmethoxycinnamate (Bratkovics et al., 2015; Zhong et al., 2020). Like other aromatic compounds, organic sunscreens are often fat-soluble and less stable than inorganic sunscreens (Bouillon, 2000). Avobenzone is commonly associated with other compounds commonly found in the sunscreens, such as OCR and bemotrizinol (Chatelain and Gabard, 2001).

Both inorganic and organic sunscreens can pose health risks. *In vitro* and *in vivo* studies showed that sunscreen cosmetics in excessive or chronic use may penetrate the *stratum corneum* of skin and generate reactive oxygen species (ROS) in the cytoplasm of the skin's keratinocytes (Hanson et al., 2006), induce DNA damages and oxidative stress (Makumire et al., 2014; Shrivastava et al., 2014) melanomas (Carrera

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https://doi.org/10.1016/j.ecoenv.2024.116586
Received 1 March 2024; Received in revised form 29 May 2024; Accepted 9 June 2024
Available online 15 June 2024

et al., 2008), and disrupt the hypothalamic-pituitary-thyroid axis and the reproductive axis (Krause et al., 2012; Yang et al., 2018). However, the use of sunscreen cosmetics is highly recommended because of its unquestionable and preventive health role.

Marine environmental pollution caused by sunscreen cosmetics needs to be urgently discussed, given that sunscreen have been detected in many beaches around the globe, with beaches that are frequently visited by tourists presenting with the higher concentrations of sunscreen (Chatzigianni et al., 2022). Homosalate, octisalate, oxybenzone, octocrylene (OCR) were among the sunscreens found, with all of them being toxic in some level (Mitchelmore et al., 2019a). For example, oxybenzone causes DNA damages in coral reefs leading to bleaching, which can be reversible if the exposure to oxybenzone is brief; on all other cases, death of the coral is imminent after bleaching (Hanson et al., 2006; Baker et al., 2008). OCR is responsible for causing mitochondrial dysfunctions in coral polyps (Stien et al., 2018). In organic ultraviolet filters, reproductive, developmental, genetic, and neurological toxicities were the most identified effects in vertebrates and invertebrates aquatic biota (Carve et al., 2021a), including dolphins (Alonso et al., 2015), fish (Balmer et al., 2005; Blüthgen, 2014; Blüthgen et al., 2012, 2014a), mollusks (Nakata et al., 2009) and corals (Mitchelmore et al., 2019b). These toxic effects were associated with molecular interactions with steroid receptors and DNA, or with the production of ROS (Carve et al., 2021b).

New techniques have been created to prevent such marine environmental damages, one of the most promising being of microencapsulation of sunscreen components. Microencapsulation consists of packaging solid, liquid, or gaseous materials in micro-scale capsules (Alvarez-Román, 2001). This technique offers, in addition to protection to the encapsulated material, the possibility of releasing it in a controlled manner under specific conditions (Rebello, 2009). There is still needed to verify the safety of the microencapsulation process through adequate testing and compare the effects of the microencapsulated product with its free components, given the possibility of environmental changes (pH, temperature changes, mechanical shock) rupturing the microcapsule, releasing the chemicals within.

A. salina is a microcrustacean of hypersaline environments used as a classic model for ecotoxicity testing due to its easiness of maintenance in laboratory conditions, as living larvae can be obtained by cheap commercially available dry cysts that can last for years in the right conditions. Adult animals can also be maintained in laboratory and its non-selective diet coupled to its high fecundity and high yield of living nauplii (larvae) or cysts make the entire genus a viable and attractive option for ecotoxicity testing (Nunes et al., 2006).

The objective of this study was to evaluate the safety to an aquatic animal model to a particular microencapsulated sunscreen formulation. The animal model *A. salina* was used as testing organism for comparison of the sunscreen microencapsulated formulae Stabyl ® against the free compounds present in these formulae through removal of the lipidic microcapsule through addition of 1 % DMSO.

2. Materials and methods

2.1. Animals

A. salina was used as animal model. Cysts were obtained from a commercial certified breeder (Maramar® Pet, Arraial do Cabo, Rio de Janeiro, Brazil). They were maintained under standard conditions in the Behavioral Neurobiology Laboratory of the Federal University of ABC (Santo André, Brazil). Nauplii were kept in 1 liter glass containers (named housing tanks) with artificial sea water labelled ASW (30 g/L sea salt, Hilker Ocean®), in a constant aeration system, water temperature (28ºC), pH (8.0), and light-dark light cycle (16–8 hours) control. Animals were fed accordingly with standard specifications.

2.2. Sunscreen, groups, and experimental design

The chemical formulation of the commercial sunscreen studied included avobenzone, octylmethoxycinnamate and OCR (Stabyl®, Attivos Magisttrais Ind. Com. Ltda, Anápolis, Goiás, Brazil) and the formulation for human use was microencapsulated in fat-soluble capsules.

The following concentrations of microencapsulated sunscreen were used in tests that evaluated toxicological, morphological and biochemical aspects in *A. salina*: 1:10 (10 %), 1:30 (3,33 %), 1:50 (2 %), 1:75 (1,33 %), 1:100 (1 %), 1:150 (0667 %), and 1:200 (0,5 %) (v:v) and this group was called the **E1** group. Another experimental group, called **E2,** was also exposed to the same concentrations of the sunscreen diluted in 1 % of dimethyl sulfoxide (DMSO) for removal of the sunscreen's lipidic microcapsule, as per recommended by the manufacturer. Changes in the solution liquid phases and density were observed as DMSO was resuspended with the microencapsulated sunscreen with a micropipette. The control group remained in the ASW (and also labelled negative control group) and the positive control remained in the 3 % iodized saline, considering the lethality caused by iodine ions to this model (Lo Nostro et al., 2015). 1 % DMSO toxicity to *A. salina* was negligible according to previous data obtained in our laboratory (data not shown) which corroborates with literature findings (Huang et al., 2018).

Prior to the assays, cysts were kept safe from humidity. Then, a total of 1 g of cysts were added to a 1 L beaker containing ASW at pH 8.0 where they remained for 24 hours under 100-watt light and constant aeration. After hatching, nauplii were transferred to a housing tank containing ASW and under constant aeration. These 24 h-old nauplii were used in experiments for toxicological and behavioral assessments. Nauplii with 72 h-old were used for evaluations of locomotor activity and with 96 h-old they were subjected to SEM for the morphological analysis.

2.3. Hatching and mortality rates analysis

Hatching rates were evaluated based on Meyer et al. (1982) protocols, using *A. salina* cysts. Ten cysts were placed in wells of flat-bottomed ELISA plates (Corning®), containing ASW (control group) and different concentrations of microencapsulated sunscreen (E1 group) or sunscreen + 1 % DMSO (E2 group) and all in triplicate. The cysts were maintained for up to 48 hours, being monitored at 24 and 48 hours to determine the percentage of hatching rate.

Toxicity tests were performed based on (Veiga and Vital, 2002) studies, using the endpoint the lethality of nauplii after 48 hours. To obtain toxicity results, nauplii were monitored daily in a Zeiss Stemi DV4 stereomicroscope (Carl Zeiss®) 24 and 48 hours after the beginning of the exposures. The nauplii mortality rate was evaluated.

2.4. Locomotor activity

After the toxicity tests, the nauplii had their locomotor activity recorded (pen-microscope, Novadigital®) in the same wells in which they were kept during all analyzes. The data were analyzed using ImageJ® (Schindelin et al., 2012) software and MtrackJ (available at https://imagescience.org/meijering/software/mtrackj/) plugin. Locomotor activity was evaluated using the parameter total traveled distance (cm).

2.5. Lipoperoxidation and carbonylation assays

Nauplii with 72 h-old and for all groups were filtered separately in a vacuum pump and washed with 25 mM phosphate buffer to remove the samples. The extension of lipid peroxidation was evaluated by the thiobarbituric acid reactive substances (TBARS) assay. Progressive lipid oxidation in crude homogenates was blocked by adding 20 μL of 4 % butylated hydroxytoluene solution in ethanol (BHT). Pinkish TBARS

adducts were measured at 535 nm after reaction with 0.25 % thiobarbituric acid in 0.25 M HCl and 1 % Triton X-100, at 100°C, for 15 min (blanks lack thiobarbituric acid). Malondialdehyde prepared by acid hydrolysis of 1,1′,2,2′-tetraethoxypropane (TEP) was used as a standard (Fraga et al., 1988). Protein oxidation was here evaluated as protein carbonyls by the classic method using 2,4-dinitrophenylhydrazine (DNPH) (Levine et al., 1990a). Briefly, the nauplii of different groups were filtered and washed as aforementioned for the lipoperoxidation assay. Nauplii homogenates were prepared in RIPA buffer (pH 7.2), containing the protease inhibitor PMSF. The homogenates were then treated with 2 mM DNPH, and the protein fractions were isolated by precipitation with 20 % trichloroacetic acid followed by centrifugation at 14,000 rpm for 3 minutes, room temperature. The pellet was then washed 3 times with ethanol:ethyl acetate (1:1), and totally resolubilized in 6 M guanidine-HCl, at 37 ºC under slight agitation, for further reading at 370 nm in a microplate reader. Total protein content was also quantified by absorbance at 280 nm (Levine et al., 1990b; Matias et al., 2012).

2.6. Reactive oxygen species (ROS) assay

For detection of ROS in nauplii with 72 h-old of different groups (5 nauplii per triplicate per group), the dye 6-carboxy-2′, 7'dichlorodihydrofluorescein diacetate (H2DCF-DA) was used, following a modified protocol of Leite et al. (2017). Nauplii were incubated with the dye in a concentration of 5 mM for 1 hour, at 38.7 °C in the dark. The samples were then washed twice in PBS and the slides were made. The images were captured after 24 hours in a Leica® DMI 6000B epifluorescence microscope, using a green fluorescence filter. For all samples, all nauplii were used to verify the intensity of fluorescence of pixels in each image with ImageJ software, which was considered the dependent variable of the analysis. Each nauplius was considered an experimental unit.

2.7. Mitochondrial activity assay

Mitochondrial activity in nauplii with 72 h-old and of all groups (5 nauplii per triplicate per group) was evaluated using the fluorescent probe MitoTracker1 Red CMXRos (M 7512; Life Technologies ™) for a period of 1 hour, in the dark, being subsequently washed in PBS. The fluorescent dye is detectable at a wavelength of 599 nm. The images were obtained using a Leica® DMI 6000B epifluorescence microscope. For all samples, the total area of the nauplii was used for fluorescence analysis. After this procedure, the number of pixels of each image was verified using ImageJ software, which was considered the dependent variable of the analysis. Each nauplius was considered an experimental unit. This is a modified protocol based on de Lima et al. (2020).

2.8. Scanning Electron Microscopy (SEM)

Nauplii of *A. salina* with 96 h-old used in previous experiments were evaluated by SEM. For this, each sample was constituted by the triplicate pool of each group and concentration. After sunscreen exposure, these nauplii with 96 hours of life were fixed in 3 % glutaraldehyde / 4 % paraformaldehyde solution in 0.1 M phosphate saline buffer (PBS, pH 7.2) for 2 hours and washed 3 times with 0.1 M PBS. This was followed by post fixation in 1 % osmium tetroxide (OsO4) for 1 hour, followed by 3 washes with 0.1 M PBS (pH 7.2) and a progressive dehydration protocol with ethyl alcohols (30 %, 50 %, 70 % and 90 %). One hour before the beginning of the procedures, 90 % alcohol was replaced by 100 % alcohol. After this replacement, specimens were critical point dried, and gold sputtered with a 10 nm layer. Samples were observed using a Quanta 250 scanning electron microscope (FEI®). This method was adapted from Rodrigues et al. protocol (Rodrigues et al., 2012).

2.9. Statistical analysis

Homoscedasticity was verified using an F-test or Bartlett's test. Normality was verified by a Kolmogorov-Smirnov test. For hatching and mortality assays, two-way ANOVA followed by Dunnett's test was used. For the data from fluorescence assays, initially an outlier detection test was performed (Grubb's test with $\alpha = 0{,}05$) and Pearson's and D'Agostino's normality tests. Two-way ANOVA followed by the Tukey's multiple comparisons test was used. In all cases, the results were considered significant if $p < 0.05$. The results were expressed as the means ± SD or as percentage.

3. Results

The results showed that the removal of microcapsule of sunscreen (E2 group) significantly reduced hatching rates ($F_{(2.24)}$=594.50, $p < 0.0001$) and increased mortality rates in a dose-dependent manner in all evaluated concentrations ($F_{(2.224)}$=31.40, $p<0.0001$) when compared to control (Fig. 1A). The mortality rate increased in a dose-dependent manner in the same group when compared with control group (Fig. 1B).

In relation to locomotor activity (total traveled distance, in cm), all concentrations reduced this parameter in the E2 group when compared to control group ($F_{(1.224)}$= 150.10, $p < 0.0001$) as showed in Fig. 1C. No statistical difference was observed between control groups (ASW or ASW + 1 % DMSO), though the effect of DMSO on this genus has been reported by Huang et al. (2018). This difference in the results could be due to the difference in the techniques used and should be further investigated.

The evaluation of oxidative stress by lipoperoxidation analysis (Fig. 1D) revealed that there were no changes in these values between the groups ($F_{(1.252)}$= 0.30, $p = 0.61$). However, the study of carbonylation aspects (Fig. 1E) revealed that the positive control (nauplii in 3 % iodized salt) presented higher carbonyl levels, as well as E2 group at 1:75 and 1:100 sunscreen concentrations ($F_{(1.252)}$= 222.70, $p < 0.0001$) when compared to control negative group.

Nauplii of both groups of exposure did not show differences in ROS compared to the control group (Fig. 2A). Fig. 2B illustrates photomicrographs of these ROS evaluations in and it is possible to

observe that E1 and E2 groups and exposed at 1:10 concentration revealed an increase in the green fluorescence, which is characteristic of the marker for ROS, although without statistical differences when compared to the positive and negative controls. On the other hand, nauplii of E2 group revealed an increase in mitochondrial activity in 1:10 and 1:30 concentrations as showed in Fig. 3A ($F(1.252)$= 68.95, $p < 0.0001$). Fig. 3B illustrates photomicrographs of these mitochondrial evaluations in nauplii. It is noted that the group exposed to non-encapsulated sunscreen show a brighter red, meaning higher mitochondrial activity.

SEM analysis revealed the occurrence of malformations in *A. salina* nauplii of E2 group only at 1:10 concentration and when analyzed after 72 hours of exposure to sunscreens in different concentrations (96-hour-old nauplii). The most evident morphological aspects were the malformation of the first pair of antennae, decreased size of the second pair of antennae, and absence of both mandible and labra. Other concentrations showed no signs of morphological alterations (Fig. 4). In other groups the SEM analyses did not show any alterations in morphological aspects (data not showed).

4. Discussion

Microencapsulation of the compounds present in the sunscreen formulae have shown to protect *A. salina* from the toxic effects of the substances within the microcapsules at its highest concentrations. The microencapsulated sunscreen didn't impede hatching or caused mortality even at the highest concentration tested while the compounds freed by 1 % DMSO addition in the testing solution impeded hatching

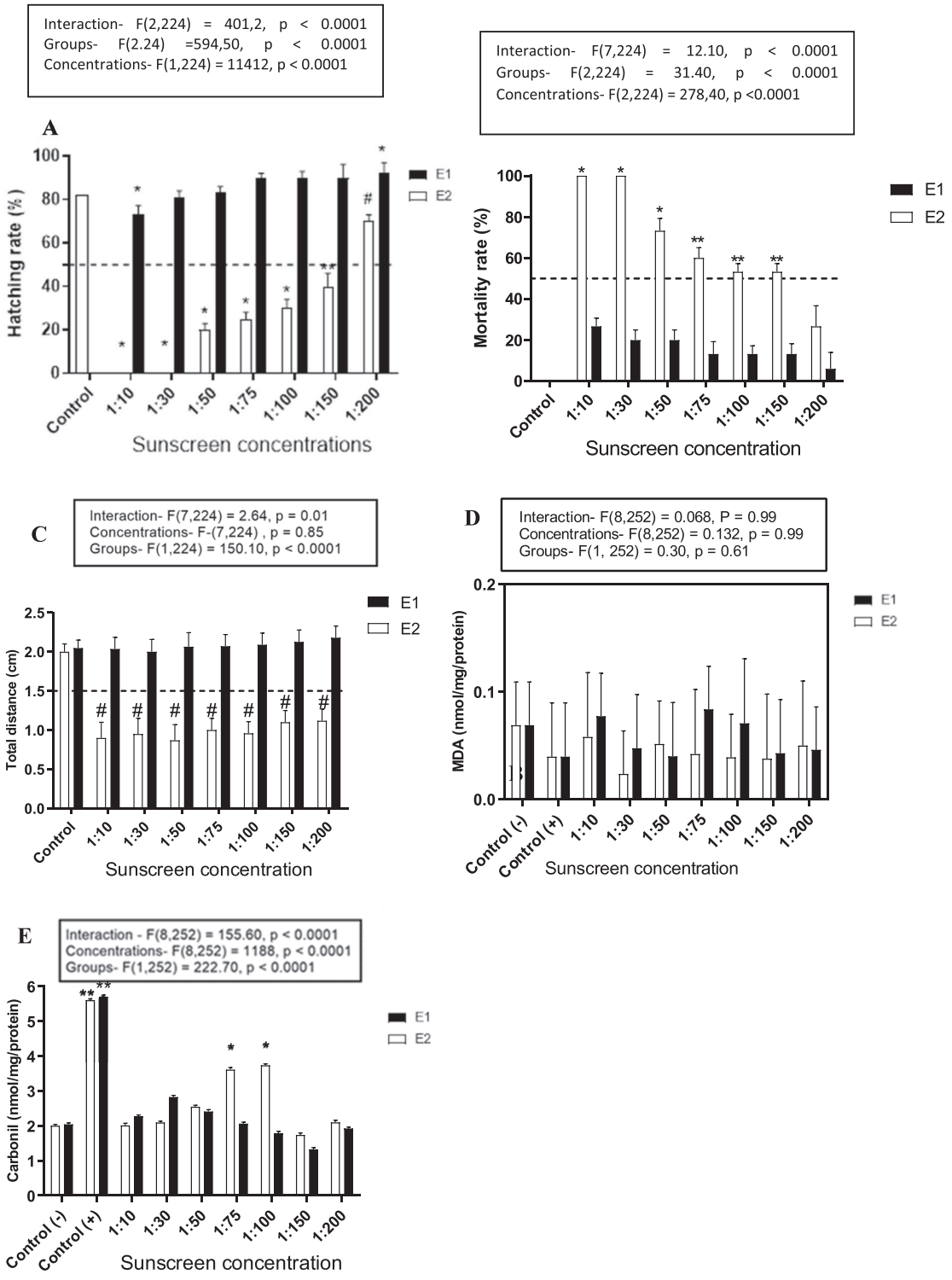

Fig. 1. Hatching and mortality rates, locomotor activity and oxidative damage in A. salina. (A) Hatching rate of cysts and (B) mortality rate of *Artemia salina* nauplii subjected to different concentrations microencapsulated sunscreen (E1 group) or removed microcapsules sunscreen by 1 % DMSO (E2 group) evaluated after 48 hours of exposure. (n = 10/triplicate) ** p <0.0001, * p <0.001, and # p <0.05 in relation to the control group. Bars indicate standard deviation. Two-way ANOVA followed by Dunnet' test. (C). Locomotor activity (total traveled distance) of *Artemia salina* nauplii subjected to different concentrations microencapsulated sunscreen (E1 group) and removed microcapsules sunscreen by 1 % DMSO (E2 group) evaluated after 48 hours of exposure. (n = 10, in triplicates). Bars indicate standard error of the mean of 15 nauplii. Two-way ANOVA followed by Dunnet. Studies of (D) lipoperoxidation and (E) carbonylation (oxidative stress) of Artemia salina nauplii subjected to different concentrations microencapsulated sunscreen (E1 group) and removed microcapsules sunscreen by 1 % DMSO (E2 group) evaluated after 48 hours of exposure. Bars on D are standard deviation and on E standard error of the mean. Two-way ANOVA followed by Dunnet's test.

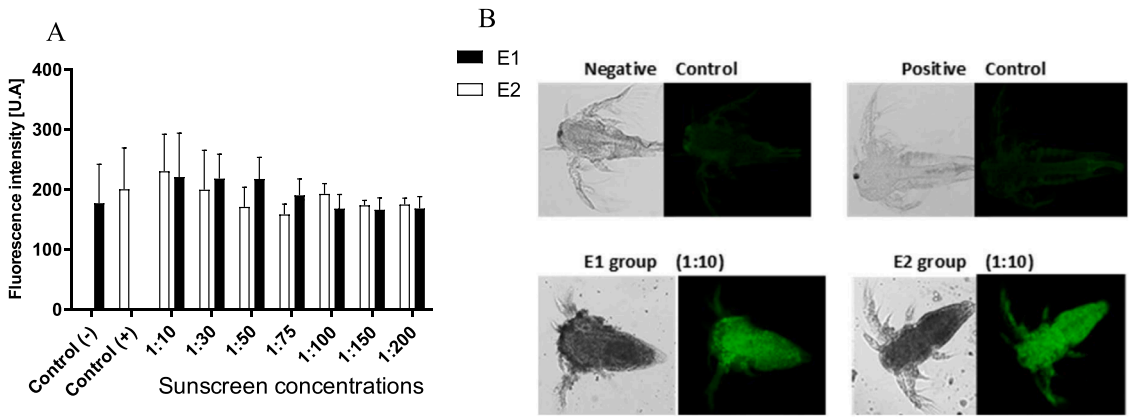

Fig. 2. Reactive oxygen species. (A) Fluorescence intensity and (B) fluorescence images of reactive oxygen species (ROS) of *Artemia salina* nauplii subjected to different concentrations microencapsulated sunscreen (E1 group) and removed microcapsules sunscreen by 1 % DMSO (E2 group) evaluated after 48 hours of exposure. No statistical significance was found between groups.

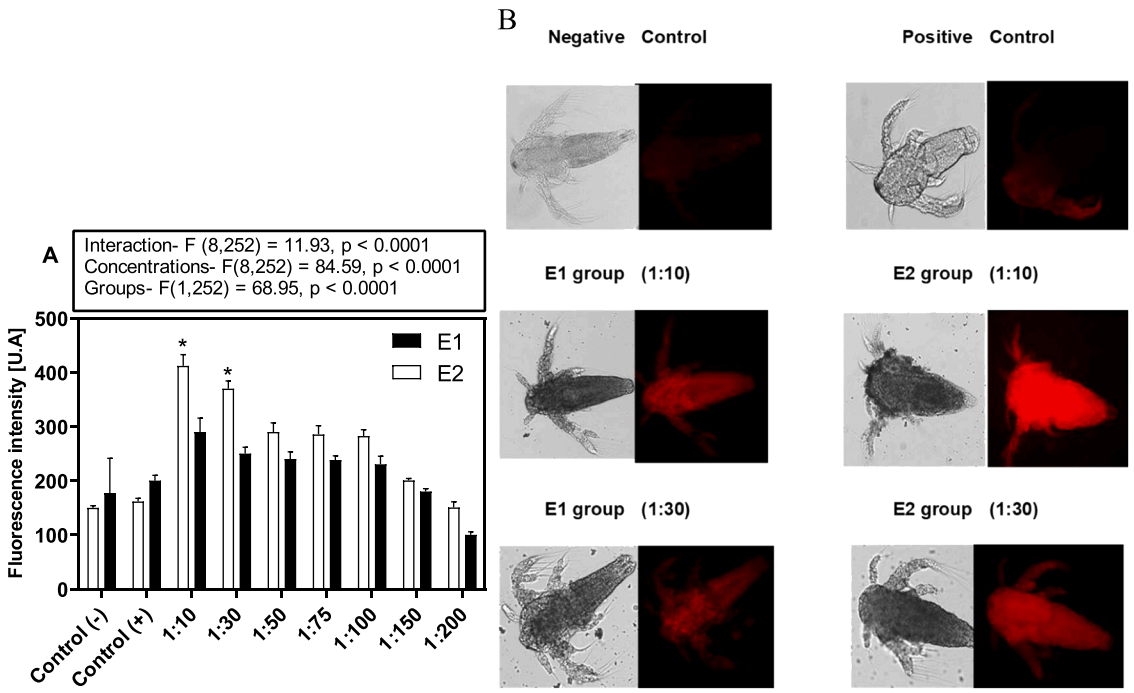

Fig. 3. Mitochondrial activity. (A) Fluorescence intensity and (B) fluorescence images of mitochondrial activity of *Artemia salina* nauplii subjected to different concentrations microencapsulated sunscreen (E1 group) and removed microcapsules sunscreen (E2 group) evaluated after 48 hours of exposure. Bars indicate standard error of the mean. n = 15. * p <0.001. Two-way ANOVA followed by Tukey.

and promoted death of the free-swimming nauplii.

In zebrafish, octocrylene causes inhibition of the expression of genes related to the development of the brain (Blüthgen, 2014), whereas octylmethoxycinnamate is genotoxic in in vitro studies (Sharma et al., 2015), cytotoxic to mouse cells (Butt and Christensen, 2000), and causes adverse effects in the offspring of rats, such as locomotor activity impairments (Axelstad et al., 2011). Avobenzone is also reported to induce harmful side effects, such as suppression of the proliferation of human trophoblasts (Yang et al., 2018), and obesogenic effects in keratinocytes of the epidermis and human mesenchymal bone cells (Ahn et al., 2019).

Thorel et al. (2020) assessed the toxicity of several sunscreen compounds on marine organisms from two major trophic levels, including autotrophs (*Tetraselmis sp.*) and heterotrophs (*A. salina*). Octocrylene concentrations used were 15–90 times greater than those reported in occurrence studies for both species. Moreover, Giraldo et al. (2017) showed high toxicity of octocrylene to different marine trophic levels: the microalgae *Isochrysis galbana*, the mussel *Mytilus galloprovincialis*, and the sea urchin *Paracentrotus lividus*. The ubiquitous presence of sunscreen and sunscreen components in the marine environment, though not in concentrations able to induce mortality, can accumulate in tissues of marine fauna (Lozano et al., 2020), transferring through the trophic net, possibly causing adverse effects on consumers. A study published by Blüthgen et al. (2014) evaluated the bioaccumulation and the molecular effects of octocrylene in adult male zebrafish and the transcriptomic analysis revealed that octocrylene mainly affected genes linked to developmental processes and metabolic processes, which coincides with our result of decreased hatching rate and the increased nauplii mortality within E2 group.

Avobenzone can cause indirect DNA damage and higher concentrations can contribute to a higher incidence of melanomas (Carrera et al., 2008), with avobenzone generally formulated in association with octylmethoxycinnamate due to the combination of substances covering

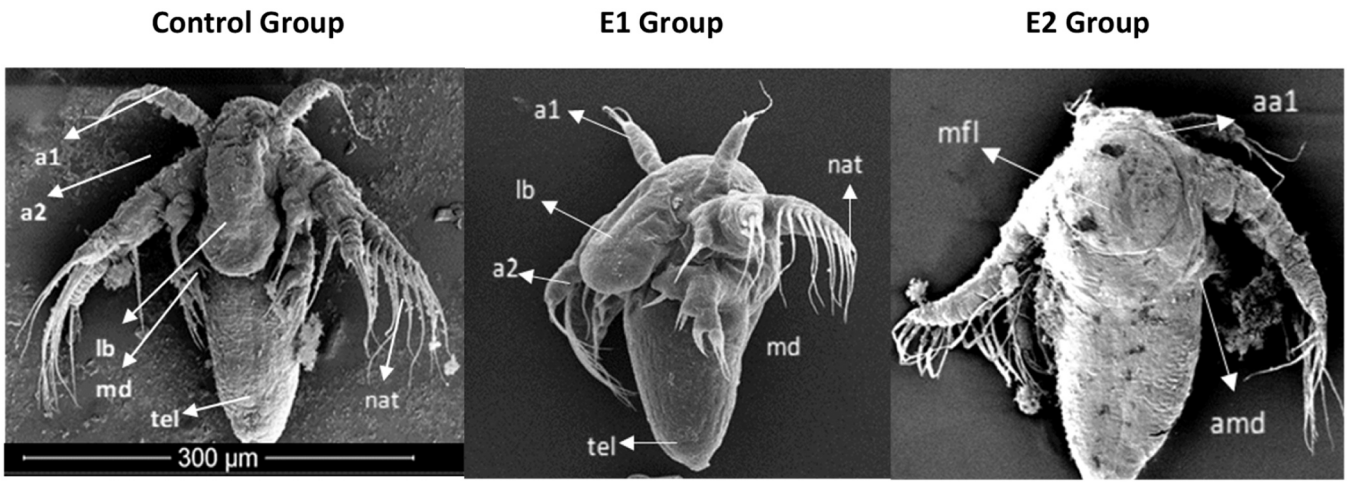

Fig. 4. Scanning Electron Microscopy (SEM). SEM photomicrographs of *Artemia salina* nauplii subjected to 1:10 concentration of microencapsulated sunscreen (E1 group) and removed microcapsules sunscreen in the same concentration (E2 group) evaluated after 72 hours of exposure to these substances and compared with nauplii from the control group. a1: first pair of antennas; a2: second pair of antennas; lb: labrum; md: mandibula; nat: swimming bristles; tel: telson; mfl: labrum malformation; aa1: absence of the first pair of antennas; amd: absence of mandible.

a wide range of UV wavelengths, providing increased protection.

Regarding locomotor activity, the sudden and steady drop in the total distance travelled point to a strong effect of the released sunscreen components from the microcapsule. Though 1 % DMSO has been affirmed to reduce the locomotion of *Artemia franciscana* (Huang et al., 2018), our positive control group (ASW + 1 % DMSO) did not suffer any impairment to its activity.

Results of carbonylation assays demonstrated a significant increase in damaged proteins in two intermediate concentrations of the E2 group (Fig. 4B). The lack of a dose-dependent response impedes any correlation of the increased carbonylation in these groups with the concentrations of the tested sunscreen. Regarding lipoperoxidation, no difference was observed between groups, with high variance between each group. The protocols used for both oxidative damage assays require further refinement and adaptation of these protocols to this model in the future, given the importance of data regarding protein damage and turnover caused by various stressors present in the marine environment, as well as the common increase in ROS seen as a response to pollutants (Niyogi et al., 2001; Livingstone, 2001; Olasehinde and Olaniran, 2022). Furthermore, DMSO has been known to have radical scavenging capabilities (Franco et al., 2007) and interaction with the reaction medium used in these protocols should be further investigated to determine whether these techniques should be applied to this model and with DMSO as a solvent.

Regarding mitochondrial activity, however, the E2 group significantly increased the fluorescence intensity at 1:10 and 1:30 concentrations, while microencapsulated sunscreen was incapable of inducing such response. Increased mitochondrion activity can lead to cellular senescence (Camacho-Encina et al., 2024), and general redox imbalances, possibly leading to ROS increase (Li et al., 2024). Though the increase in green fluorescence was observed, statistical significance might be found increasing the number of nauplii observed. Other techniques to assess ROS damage should also be applied further asses whether or not free sunscreen components can induce formation of ROS.

It was observed the occurrence of malformations in *A. salina* nauplii of E2 group exposed to the sunscreen at the highest concentration. The most evident malformations were the decrease in the size or the absence of the first pair of antennae and labra, and the underdevelopment of the nauplii exposed to this concentration of sunscreen. While nauplii in E1 and less concentrated sunscreen present well-formed and developed mandibulae and labrum, E1 nauplii seem to have malformed labrum and small mandibulae. The importance of these two organs in nauplii is vital. The first pair of antennae is responsible, at this stage of life, for the mobility of the larvae and the labrum is associated with osmoregulation (Russler and Mangos, 1978). Deficiency in osmoregulation can result in irreversible cell damage since the animal is found in nature and thrives in environments of high salinity that have been observed to increase concentration of ROS in other crustacean species. The observed malformation of the labrum and its physiology should be further studied in order to correlate malformations to its function to the organism.

Despite all the harmful results found after removing microcapsules of the sunscreen, such as impairments in hatching and mortality rates, behavior, in metabolic pathways and malformations, the microencapsulated sunscreen formulation prevented all harmful effects.

These results showed that the microencapsulated sunscreen was successful at protecting *A. salina* development and metabolism alterations against the harmful components and effects of the sunscreen versus the free compounds in the same concentrations. The hatching rate and mortality of *A. salina* exposed to the microencapsulated sunscreen were statistically equal to the control group exposed to artificial sea water. We conclude that microencapsulation of sunscreen formulae can greatly diminish the harmful effects caused by the most common sunscreen components even at high concentrations, as long as the microcapsule membrane remains intact.

CRediT authorship contribution statement

Giselle Cerchiaro: Methodology. **Elizabeth Teodorov:** Validation, Supervision, Software, Methodology, Funding acquisition, Conceptualization. **Maria Martha Bernardi:** Writing – original draft, Supervision, Methodology. **Arnaldo Rodrigues dos Santos:** Methodology. **Marcella Pecora Milazzotto:** Methodology, Conceptualization. **Caio Schatzer:** Writing – review & editing, Writing – original draft, Formal analysis, Data curation, Conceptualization.

Declaration of Competing Interest

The authors declare no conflict of interest.

Data Availability

The authors do not have permission to share data.

Acknowledgements

This study was supported by the Coordenação de Aperfeiçoamento

de Pessoal de Nível Superior - Brasil (CAPES) – Finance Code 001 and Fundação de Amparo a Pesquisa do Estado de São Paulo (FAPESP) grant number 2022/05498-6. We are grateful to Attivos Magisttrais Laboratories for providing all the sunscreen used in this study as well to Dr. Thiago Berti Kirsten and Dr. Eduardo Bondan for technical support.

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