Adaptative transcriptional response of Dietzia cinnamea P4 strain to sunlight simulator

Procopio, Luciano; Padula, Marcelo; van Elsas, Jan Dirk; Seldin, Lucy

Published in:
Archives of Microbiology

DOI:
10.1007/s00203-020-01879-y

Publication date:
2020

Archives of Microbiology (2020) 202:1701–1708
https://doi.org/10.1007/s00203-020-01879-y

ORIGINAL PAPER

Adaptative transcriptional response of *Dietzia cinnamea* P4 strain to sunlight simulator

Luciano Procópio[1] · Marcelo Pádula[2] · Jan Dirk van Elsas[3] · Lucy Seldin[4]

Received: 26 November 2019 / Revised: 19 March 2020 / Accepted: 1 April 2020 / Published online: 15 April 2020

Abstract

Responses to sunlight exposure of the oil-degrading *Dietzia cinnamea* P4 strain were evaluated by transcriptional levels of SOS genes, photoreactivation and genes involved in tolerance to high levels of reactive oxygen species. The P4 strain was exposed for 1 and 2 h and the magnitude of level changes in the mRNA was evaluated by qPCR. The results described the activation of the SOS system, with the decline of the repressor *lexA* gene levels and the concomitant increase of *recA* and *uvrAD* genes levels. The genes that participate in the photoreactivation process were also responsive to sunlight. The *phrB* gene encoding deoxyribodipyrimidine photo-lyase had its expression increased after 1-h exposure, while the *phytAB* genes showed a progressive increase over the studied period. The protective genes against reactive oxygen species, catalases, superoxides, peroxidases, and thioredoxins, had their expression rates detected under the conditions validated in this study. These results show a fast and coordinated response of genes from different DNA repair and tolerance mechanisms employed by strain P4, suggesting a complex concerted protective action against environmental stressors.

Keywords *Dietzia cinnamea* P4 · UV radiation · Catalase genes · Superoxide dismutase genes · SOS system

Introduction

The ultraviolet (UV) region comprising a range of the solar spectrum, < 400 nm in wavelength, while constituting the smallest part of ultraviolet radiation, exerts a disproportionate effect on living organisms. UV photons belonging to this wavelength range can be absorbed by a wide variety of biomolecules, such as small size molecules, proteins and metabolites. Among the many effects on these molecules, the induction of DNA damage by ultraviolet radiation can cause an important and worrisome event by dramatically influencing the cellular processes of all organisms (Dizdaroglu 1992; Rastogi et al. 2010). In addition, endogenous factors such as free radicals and exogenous factors such as ionizing radiation or ultraviolet radiation in sunlight are widely described as causing damage to genetic material (Taylor 2015). Known damage can be cited: (1) oxidative damage indirectly induced by ionizing radiation; (2) hydrolytic damage, responsible for base deamination, depurination and depyrimidination; (3) incorporation of erroneous bases during the process of DNA replication, and (4) action of alkylating agents, which can modify DNA bases (Lindahl 1993; Taylor 2015; Valko e al. 2006). Although the most harmful solar radiation length is UV-C (100–295 nm), it is completely absorbed during the ozone layer formation process. However, a considerable amount of UV-B radiation (295–320 nm), about 85–95%, depending on latitude, reaches the Earth's surface and can be absorbed by the DNA of living organisms, resulting in damage. Most

Communicated by Erko Stackebrandt.

Electronic supplementary material The online version of this article (https://doi.org/10.1007/s00203-020-01879-y) contains supplementary material, which is available to authorized users.

✉ Luciano Procópio
lucianoprocopio@caxias.ufrj.br

[1] Industrial Microbiology and Bioremediation Department, Universidade Federal Do Rio de Janeiro, Caxias, Rio de Janeiro, Brazil

[2] Laboratório de Diagnóstico Molecular E Hematologia, Faculdade de Farmácia, Universidade Federal Do Rio de Janeiro, Rio de Janeiro, RJ, Brazil

[3] Microbial Ecology Laboratory, Goningen University, Groningen, The Netherlands

[4] Laboratório de Genética Microbiana, Instituto de Microbiologia Paulo de Góes, Universidade Federal Do Rio de Janeiro, Rio de Janeiro, RJ, Brazil

often induced by UV-B are the formation of cyclobutane pyrimidine dimers (CPD) and pyrimidine pyrimidone photoproducts in DNA double strands. Other photochemical reaction types involve the saturation of the 5,6-double bond of pyrimidines producing damaged bases such as cytosine hydrate and thymine glycol (Goosen and Moolenaar 2008).

The possibility of the evolution of life under sun exposure was made possible by several DNA repair mechanisms that allow removing lesions induced by UV radiation to DNA. Bacteria have several genetic and physiological mechanisms that allow them to adapt to unfavorable UV radiation conditions (Simmons et al. 2008). These mechanisms involve a complex system of structured gene expression that responds in a coordinated manner to stress conditions. The most well-known system is the SOS response, which has been described in *Escherichia coli*, and describes a controlled increase in expression levels of different genes in response to UV-induced damage to DNA structure (Sutton et al. 2000; Maslowska et al. 2019). The SOS response involves the participation of *lexA* and *recA* gene expression products (Li et al. 2011; Zhang et al. 2010). In normal physiological conditions, the LexA protein acts as a transcriptional repressor of the genes that participate in the SOS system. However, in conditions of genome damage and consequent single-stranded DNA (ssDNA) production, the RecA enzyme is activated and stimulates auto-cleavage of the LexA repressor protein, decreasing its affinity for the DNA binding site and begins transcribing the SOS genes (Little 1991; Patel et al. 2010; Neher et al. 2003). As a result of LexA derepression, the expression of more than 50 genes that perform different functions in response to DNA damage begins, including excision repair, homologous recombination, translesion DNA replication, and disruption of cell division (Maslowska et al. 2019). Among the participating genes, the Uvr system (e.g., *uvrABCD*) involved in the excision of damaged bases is the first to be transcribed, then *recAN* and *ruvAB* genes, which participate in the homologous recombination, are expressed, and the polymerases encoded by *polB* and *dinB* genes (Kisker et al. 2013).

Another protective mechanism is activated when the UV radiation damage signals events that activate the photoreactivation process, which is conducted by enzymes known as photolyases. These enzymes belonging to the large family of photolyase/cryptochrome, very conserved among the three life domains, have the function of repairing UV-damaged DNA-breaking pyrimidine dimers (Liu et al. 2013, 2018). In addition, other effects caused by UV radiation are the formation of reactive oxygen species (ROS), which are derived from the reduction of oxygen to superoxide (O_2), hydrogen peroxide (H_2O_2) or the hydroxyl radical ($^{\cdot}OH$) (Gao and Garcia-Pichel 2011; Farr and Kogoma 1991; Imlay 2002). Studies show a system called regulon OxyR is responsible for a transcriptional cascade of different groups of genes responsible for the elimination of oxidants (*katG*, *ahpC* and *ahpF*), in maintaining the balance between thiol groups and disulfide bonds (*gprA*, *grxA* and *trxC*). In addition, the SoxS system acts on a second cascade of O_2 response transcription to directly activate transcription of genes directly involved in the elimination of O_2 (manganese superoxide dismutase (*sodAC*) (Castenholz and Garcia-Pichel 2000; Storz and Zheng 2000).

The actinomycete *D. cinnamea* P4 is a Gram-positive bacterium with high G+C content, found in soil, seabed sediments, soda lakes, reed rhizomes, fish skin and intestinal tract (Yassin et al. 2006). The P4 strain was isolated in a study on the diversity of hydrocarbon degraders in tropical forest soil in Brazil (Evans et al. 2004). Subsequently, the genome of P4 strain allowed the identification of several genes involved in the protection and repair of DNA when exposed to stress conditions, such as thermal shock, oxidative shock, and exposure to UV radiation, with the presence of genes belonging to the SOS system (Procopio et al. 2013). The genetic and physiological characteristics of this strain indicate that it can survive in complex environments with various environmental stressors, combined with its ability to degrade hydrocarbons, make this actinomycete a promising candidate for biotechnological remediation processes. In this study, to assess the responsiveness to sunlight exposure, the *D. cinnamea* P4 strain was directly exposed to different periods of exposure to a solar simulator and the transcriptional levels of genes involved in protective responses were evaluated.

Material and methods

Growth, stress conditions and survival experiments

A culture (pre-inoculum) of 10 ml was prepared from an isolated colony of *D. cinnamea* P4, previously growth in Luria–Bertani agar (LB agar: tryptone 1%, NaCl 0.5%, yeast extract 0.5%, agar 1.5%, obtained from Deutsche Sammlung von Mikroorganismen und Zellkulturen, Germany) or in LB broth at 28 °C under agitation. After the culture reached an OD660 of 1.0 (begin of exponential phase), it was centrifuged at 8000 × *g* and the pellet resuspended in 10 ml of phosphate-buffered saline (PBS: NaCl 0.08%, KCl 0.02%, Na2HPO4 0.14%, KH2PO4 0.024%, pH 7.4). The culture was split into three aliquots, of which one was left untreated and the two were employed to sunlight simulation (SSL) treatment. The SSL irradiations were performed using a solar simulator (SS) for 1 and 2 h (Oriel Model 91192-1000, Newport Corp., USA). Atmospheric attenuators AMO 87066 were used, resulting in a final UVB/UVA ratio emission of 1/15, which is in accordance with our mean measurements for summer in Rio de Janeiro, Brazil, at 12:00 noon (− 22.9

latitude and longitude −43.17), and with previous reports of the same order of magnitude (Miller et al. 1998; Mitra et al. 2012; Pitts 1990). The Solar Simulator was 20 cm above the receptor base, which was sufficient to avoid heating to samples.

Real-time quantitative PCR

The transcriptional levels of genes involved in the response to sunlight simulator exposure were assessed by quantitative time PCR. The genes chosen with their respective primers are shown in Table 1. Cell culture of strain P4 was employed to obtain total RNA after exposure to the simulator following the conditions described above. Total RNA was extracted with the RNeasy Mini Kit (Qiagen, Valencia, CA, USA) according to the manufacturer's protocol. Then, total RNAs from each untreated condition, exposure to sunlight simulator for 1 and 2 h, were treated with DNase I (Promega, SP, Brazil). RNA integrity was verified by agarose gel electrophoresis and yield was estimated using a Nanodrop UV spectrometer (Thermo Scientific, Wilmington, DE, USA). About 100 ng of RNA from each sample was used for cDNA synthesis using random hexamers by the ImProm-I Reverse Transcription System (Promega) according to the manufacturer's protocol. The yield of cDNAs was estimated by the Nanodrop UV spectrometer and its concentrations adjusted to about 1 ηg.

For real-time quantitative PCR, 5 µl cDNA was mixed with LightCycler FastStart DNA MasterPLUS SYBR Green I (Roche, IN, USA), for 0.5 µg forward and reverse primers of each gene to be analyzed in a final volume of 20 µl in three replicates for each gene analyzed. Also, controllers were included in three replicates for each gene to be analyzed. 16S rRNA gene expression was used as a reference gene to normalize the expression of the tested genes. Real-time quantitative PCR was performed with Light Cycler 1.5 (Roche), following the protocol: one cycle at 95 °C for 10 min, 40 cycles at 95 °C for 10 s, followed by 60 °C for 10 s, 72 for 25 s. Amplification specificity was verified at the end of PCR by LightCyler 4.1 software and the normalized relative fold change in mRNA levels were calculated for the gene of interest in each sample using the 2-ΔΔCT method (Livak and Schmittgen 2001). The primers used in this study are available in Table S1 in the supplementary file.

Statistical analyses

Changes in gene expression patterns in this study were presented as mean ± standard deviation (± SD). Statistics were performed using the Past3 software. Differences among groups were analyzed by one-way analysis of variance (ANOVA), followed by post hoc Tukey's test for intergroup and intragroup comparison. p value < 0.05 was considered statistically significant.

Results

SOS system activation

The levels of the genes that participate in the initial response to ultraviolet radiation were evaluated after 1 and 2 h. The genes measured were the *lexA* repressor, the *recA* gene involved in DNA damage response, the *uvrA* and *uvrD* genes from the excision of damaged bases system, the *nheJ* gene encoding DNA ligase, and the *metA* and *metB* genes encoding methyltrasferases. The assessment of the transitional measures of the *lexA* repressor under both conditions indicated clear repression of prolonged exposure to sunlight.

Table 1 Levels of SOS gene expressions related to strain *D. cinnamea* P4 response to sunlight simulator exposure after 1 and 2 h

Access number	Gene	Description	Sunlight simulation		
			1 h	2 h	*p* value
ZP_08022314	*lexA*	LexA repressor	17.39 (±3.7)	0.4 (±0.02)	0.001*
ZP_08022556	*recA*	Recombinase A	17.14 (±6.72)	217.77 (±3.39)	
ZP_08023420	*metA*	Methylated-DNA-protein-cysteine methyltransferase A	3.60 (±0.12)	56.36 (±2.40)	
ZP_08025258	*metB*	Methylated-DNA-protein-cysteine methyltransferase B	N.D	N.D	
ZP_08025300	*uvrA*	Excision repair protein	1.04 (±0.30)	76.99 (±1.85)	
ZP_08022138	*uvrD*	Excision repair protein	4.35 (±1.29)	39.40 (±2.87)	
ZP_08025107	*nheJ*	DNA ligase	8.98 (±2.98)	N.D	
ZP_08024163	*mug*	G/U mismatch-specific uracil-DNA glycosylase	66.89 (±13.21)	122.22 (±3.25)	

Standard errors of mean are indicated in parentheses

Data are expressed as mean ± SD of three independent experiments

ND no detectable signal

*$p \leq 0.05$ vs control conditions (LB-medium), based on Tukey's test

After 1 h of exposure, there was an increase of about 17 times when compared to the control system, with a sudden drop in the levels after 2 h. Ultraviolet-induced repression of *lexA* levels was followed contrary to the measured levels of the *uvrAD* genes. The *uvrA* gene increased from 1.04 in 1 h of incubation, to 76.99 times after 2 h, while the *uvrD* gene increased from 4.35 to 39.4 times, when both were compared to the control system. When evaluating the expression of the *metA* and *metB* genes, there was a different behavior for both gene products. The *metA* gene showed an appreciable lift after 2 h and no signal could be detected for *metB* gene under the evaluated conditions. In addition, the *nheJ* gene also showed near-bottom transcriptional levels after 2 h of the experiment (Table 1 and Fig. 1).

Photoreactivation responses

The main genes with participation in the photoreactivation process were evaluated. The genes measured were *phytAB* genes, which encode phytoene synthase/dehydrogenase, the *phrB* gene encoding the deoxyribodipyrimidine photo-lyase, and *crtY* gene encoding lycopene cyclase enzyme. The transcriptional response of the *phrB* gene was shown to be precocious when compared to other photoreactivation genes. While after 1 h of exposure, the *phrB* levels were 718.99 compared to the control, after 2 h the exposure value decreased to 5.8. *phytAB* gene levels showed different performance, with a slight elevation between 1 (3.83) and 2 h (14.99) of exposure. The transcriptional level of *crtY* showed a variation similar to the *phrB* gene, with a slight increase after 1 h of exposure, while after 2 h no signal could be detected (Table 2 and Fig. 2).

Transcriptional responses to ROS

To measure the responses of genes with functions on detoxification processes of reactive oxygen species, the transcriptional levels of four different catalases, *katE1*, *katE2*, *katE3* and *katMn*, two *sodA* and *sodC* genes coding for superoxide dismutase, and the *gpx1* and *gpx2* genes, which encode two different glutathione peroxidase, the *ahpC* and *ahpF* genes for hydroperoxidase, the *trxA* and *trxB* genes for tioredoxins, and the *tpx* gene for thiol presoxidase (Table 3 and Fig. 3) were measured. Among the four catalase genes, two had

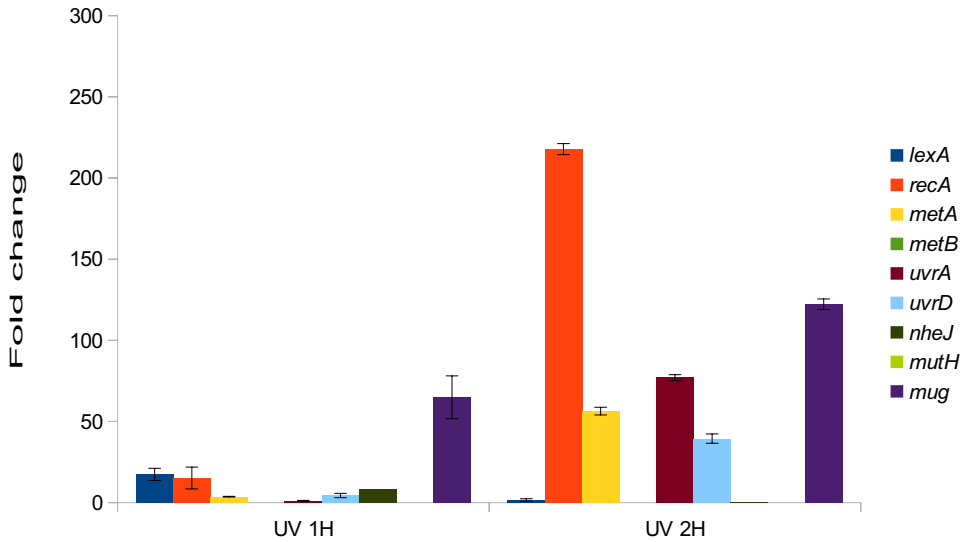

Fig. 1 Expression levels of putative SOS genes of *D. cinnamea* P4 strain under UV radiation exposition. Relative expression levels were determined with real-time quantitative RT-PCR. The error bars shown indicate the standard deviation of the analyzed genes. Standard errors of mean are indicated in parentheses. Data are expressed as mean ± SD of three independent experiments; *$p \leq 0.05$ vs control conditions (LB-medium), based on Tukey's test

Table 2 Levels of photoreactivation gene expressions related to strain *D. cinnamea* P4 response to sunlight simulator exposure after 1 and 2 h

Access number	Gene	Description	Sunlight simulation		
			1 h	2 h	*p* value
ZP_08023567	*phrB*	Deoxyribodipyrimidine photo-lyase	718.99 (±218.83)	5.80 (±2.79)	0.001*
ZP_08024379	*phytAB*	Bifunctional phytoene synthase/phytoene dehydrogenase	3.83 (±0.1488)	.99 (±1.06)	
ZP_08022078	*crtY*	Lycopene cyclase	2.56 (±1.35)	N.D	

Standard errors of mean are indicated in parentheses

Data are expressed as mean ± SD of three independent experiments

ND no detectable signal

*$p \leq 0.05$ vs control conditions (LB-medium), based on Tukey's test

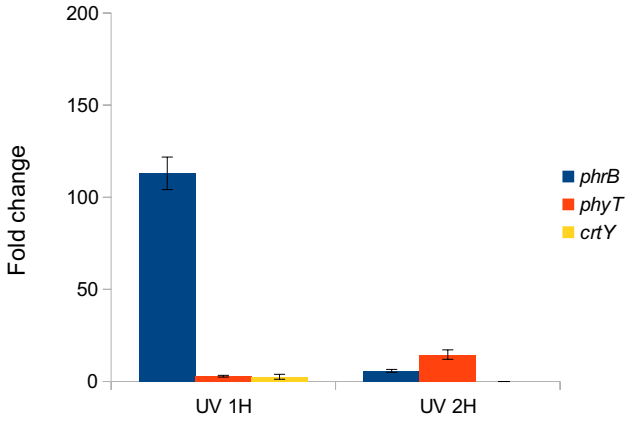

Fig. 2 Expression levels of putative photoreactivation genes of *D. cinnamea* P4 strain under UV radiation exposition. Relative expression levels were determined with real-time quantitative RT-PCR. The error bars shown indicate the standard deviation of the analyzed genes. Standard errors of mean are indicated in parentheses. Data are expressed as mean ± SD of three independent experiments; $*p \leq 0.05$ vs control conditions (LB-medium), based on Tukey's test

signal detected in all conditions. The *katE1* gene showed a high transcriptional level after 1 h, more than 393.4 times when compared to the control, however, with 2 h of incubation in the sunlight had a dramatic drop in expression level. The *katE2* gene showed an increased ratio after 1 h of exposure, above 197.7 times, and continued to rise to the level of 615.9 times above the control experiment (Table 3 and Fig. 3). The *sodAC* genes showed high levels under both conditions. However, while *sodA* had a higher index after 2 h of exposure (406.7), *sodC* had a higher level at 1 h (282.6), followed by an expressive decline after 2 h (61.1) (Table 3 and Fig. 3). The *gpx1* and *gpx2* genes also showed similar results. The *gpx1* gene was detected only at 1 h of sunlight exposure (13.9), while the *gpx2* signal rose dramatically after 2 h (322.5). Both *trxAB* genes were expressed positively over the period. *trxA* showed strong expression soon after 1 h (196.7), and after 2 h the levels remained high (776.2), and *trxB* showed similar results, with 1.4 above control after 1 h and one peaked after 2 h (645.1). The *tpx* gene showed a slight increase throughout the experiment from 31.5 to 33.5 times, after 1 and 2 h, respectively (Table 3 and Fig. 3).

Discussion

The environmental factors present in tropical soils can represent a challenge for the evolution and survival of microbial species. The presence of humic acids, metabolites, temperature and humidity variations, as well as the presence of a competitive microbial community, can limit nutrient availability and influence the success of certain bacterial species. However, many bacteria species have global response systems to adapt and survive many environmental stresses, including UV radiation (Micevski and Dougan 2013; Foster 2007). Previous work on the genetics and physiology of the oil-degrading *D. cinnamea* P4 strain described the

Table 3 Levels of ROS detoxification gene expressions related to strain *D. cinnamea* P4 response to sunlight simulator exposure after 1 and 2 h

Access number	Gene	Description	Sunlight simulation		
			1 h	2 h	*p* value
ZP_08023119	*ahpF*	Alkyl hydroperoxide reductase	1.3 (±0.5)	14.6 (±1.81)	0.001*
ZP_08023120	*ahpC*	Alkyl hydroperoxide reductase	19.6 (±5.15)	758.3 (±5.55)	
ZP_08024224	*tpx*	Thiol peroxidase	31.5 (±19.35)	33.5 (±2.59)	
ZP_08024195	–	Glutathione S-transferase	6.7 (±1.57)	N.D	
ZP_08023504	*trxB*	Thioredoxin reductase	1.4 (±1.10)	645.1 (±14.99)	
ZP_08024053	*trxA*	Thioredoxin	196.7 (±0.15)	776.2 (±11.01)	
ZP_08024106	*gpx1*	Glutathione peroxidase	13.9 (±10.32)	N.D	
ZP_08023200	*gpx2*	Glutathione peroxidase	2.78 (±1.1)	322.5 (±0.85)	
ZP_08021853	*sodC*	Cu/Zn Superoxide dismutase	282.6 (±98.44)	61.1 (±3.50)	
ZP_08023526	*sodA*	Superoxide dismutase	195.6 (±57.51)	406.7 (±10.35)	
ZP_08022106	*katE1*	Catalase	393.4 (±144.62)	11.1 (±1.50)	
ZP_08022939	*katE2*	Catalase	197.7 (±91.44)	615.9 (±17.53)	
ZP_08025226	*katE3*	Catalase	N.D	N.D	
ZP_08022130	*KatMn*	Mn catalase	N.D	N.D	
ZP_08024268	*alkD*	DNA alkylation repair enzyme	N.D	N.D	

Standard errors of mean are indicated in parentheses

Data are expressed as mean ± SD of three independent experiments

ND no detectable signal

$*p \leq 0.05$ vs control conditions (LB-medium), based on Tukey's test

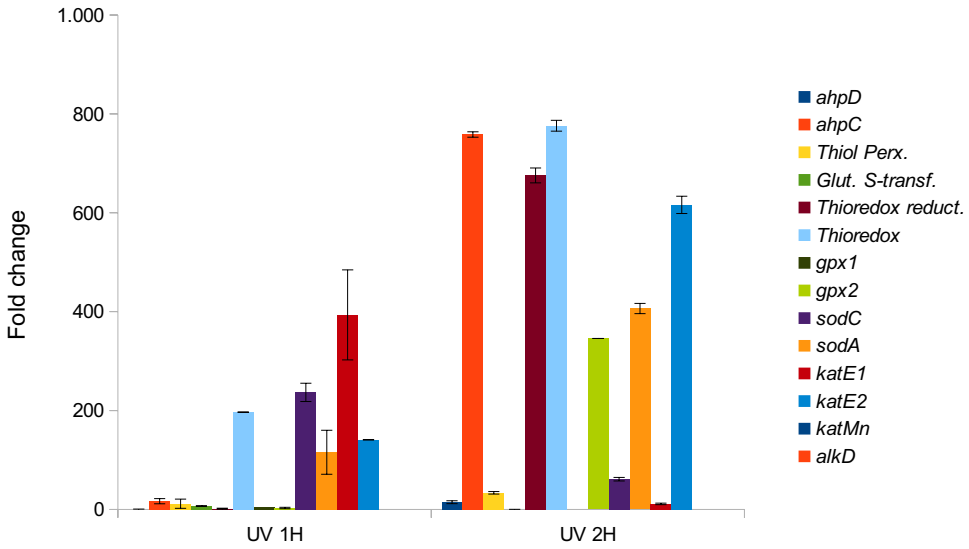

Fig. 3 Expression levels of putative ROS detoxification of *D. cinnamea* P4 strain under UV radiation exposition. Relative expression levels were determined with real-time quantitative RT-PCR. The error bars shown indicate the standard deviation of the analyzed genes. Standard errors of mean are indicated in parentheses. Data are expressed as mean ± SD of three independent experiments; $^{*}p \leq 0.05$ vs control conditions (LB-medium), based on Tukey's test

complex gene system responsible for adaptation under different conditions of environmental stresses (Procópio et al. 2012). In addition, strain P4 was tolerant to chemical agents and induced mutagenesis compared to *E. coli* K12A14, which is also described as resistant to induced mutagenesis and UV radiation, in the three wavelengths UVA, UVB and UVC (Procopio et al. 2013). Further work evaluating the resistance of *D. cinnamea* P4 to treatment with different concentrations of hydrogen peroxide also demonstrated its refractory capacity to this compound (Procopio et al. 2019).

In this study, we report the transcriptional responses of *D. cinnamea* P4 strain to exposure to sunlight, after 1- and 2-h periods. The evaluation of different gene groups involved in transcriptional expression shown a coordinated and powerful response. The *lexA* repressor gene decreased in expression after a long period of exposure to sunlight, with the concomitant increase in *recA* and *uvrAD* levels, suggesting that activation of the SOS system is essential for the survival of the P4 strain over sunlight radiation. The function of the RecA enzyme is widely described in studies on DNA repair damaged by different agents, including UV radiation treatment (Prada Medina et al. 2016). Study of the direct function of the RecA enzyme in *E.coli* mutants for the *recA* gene showed the role of this gene in protecting against damage caused by exposure to sunlight (Doudney and Rinaldi 1989). The nucleotide excision repair (NER) process protects the cellular genome against damage caused by UV radiation and involves the pairing of UvrA and UvrB enzymes (Barnett and Kad 2019). A study on recombination catalyzing after UV treatment indicated the participation of UvrA enzyme in *E. coli* cell genome reorganization (Estévez Castro et al. 2018).

In addition, to the repair processes conducted by the SOS system, photoreactivation consists of an important mechanism for cell survival under different stress conditions. Photoreactivation is conducted by a group of highly conserved enzymes among the life domains, called "photolyases", which closely resemble cryptochromes (Rastogi et al. 2010; Cashmore et al. 1999). The genome elucidation of the bacterium *D. cinnamea* P4 identified the putative genes for the PhrB enzyme, in addition, enzymes involved in the protection of the Bifunctional phytoene synthase/phytoene dehydrogenase sunlight and the Lycopene cyclase CtrY enzyme were identified. *phrB* expression levels indicated that expression occurred soon after 1 h of exposure, suggesting an immediate response to sunlight. A study on transcriptional *phrB* levels was also performed in *Rhodococcus sphaeroides* (Hendrischk et al. 2007). The results of these studies indicated an upregulated expression of the phr gene, suggesting its protective function of this enzyme in the *R. sphaeroides* genome. Although few studies have been conducted on the function of pigment compounds, such as carotenoids, in the action against DNA damage by environmental factors, their protective action on carotenoid compounds is demonstrated in UV radiation studies (Glaeser and Klug 2005; Tandori et al. 2001). Our analyses show that there was an early expression of *crtY*, followed by the absence of signal after 2 h, while *phytAB* there was a gradual increase over the analyzed period. Both gene products, *crtY*, and *phytAB*, are described as enzymes that respond positively to DNA damage by preventing the formation of oxygen singlet by quenching of triplet bacteriochlorophyll (Ziegelhoffer and Donohue 2009; Glaeser and Klug 2005).

Although the toxic effects of UV radiation are complex, one of the mechanisms is ROS formation, which includes superoxide anion radical ($O^{\cdot}_2$), hydroxyl radical ($^{\cdot}OH$), hydrogen peroxide (H_2O_2), and singlet oxygen ($O_2^{\cdot}$) (He and Hader 2002; Hoerter et al. 1989; Kramer and Ames 1987). The molecular responses to these agents constitute expression and activation of enzymes of the catalase,

peroxidase, and superoxide families (Ziegelhoffer and Donohue 2009; Storz and Zheng 2000). The measured levels of catalase genes in this study showed that only two of the five described in the genome of P4 strain were found to be responsive to UV radiation. Similar results were also identified in the expression of the two genes for superoxide dismutase. Distinct activity between different enzymes present in bacterial specimens has been reported in other studies also. Tolerance experiment on *Acinetobacter* sp. isolates under UVABC radiation exposure have shown that genes coding for different catalases and superoxides enzymes were expressed differently (Di Capua et al. 2011). A similar study was also conducted with nine different bacterial isolates, showing different survival responses of isolates under different UV wavelength conditions (Santos et al. 2013). The *ahpCF* genes in *D. cinnamea* P4 showed a sudden elevation after 2 h of exposure to UV radiation. In addition, *trxAB* levels also had an abrupt increase in the conditions analyzed in this study. Scientific studies show that these enzymes play an essential role against ROS, generated by environmental factors, including exposure to sunlight (Matallana-Surget 2009).

Conclusion

Our results allow us to conclude that strain *D. cinnmaea* P4 has a complex system of response to external environmental factors, as UV radiation. Different genes were expressed in a coordinated manner against simulated sunlight exposure. The genes that make up the SOS system presented transcriptional levels similar to those described in the scientific literature in other bacterial isolates. Also, genes involved in protection by photoreactivation mechanisms and detoxification activities of reactive compounds have also been shown to be responsive to UV radiation exposure.

References

Barnett JT, Kad NM (2019) Understanding the coupling between DNA damage detection and UvrA's ATPase using bulk and single molecule kinetics. FASEB J 33(1):763–769. https://doi.org/10.1096/fj.201800899R

Cashmore AR, Jarillo JA, Wu YJ, Liu D (1999) Cryptochromes: blue light receptors for plants and animals. Science 284(5415):760–765

Castenholz RW, Garcia-Pichel F (2000) Cyanobacteria responses to UV-radiation. In: Whitton BA, Potts M (eds) The ecology of Cyanobacteria. Kluwer Academic Publishers, Dordrecht, pp 591–611

Castro CFE, Serment-Guerrero JH, Fuentes JL (2018) Influence of uvrA, recJ and recN gene mutations on nucleoid reorganization in UV-treated *Escherichia coli* cells. FEMS Microbiol Lett. https://doi.org/10.1093/femsle/fny110

Di Capua C, Bortolotti A, Farías ME, Cortez N (2011) UV-resistant Acinetobacter sp. isolates from Andean wetlands display high catalase activity. FEMS Microbiol Lett 317(2):181–189. https://doi.org/10.1111/j.1574-6968.2011.02231.x

Dizdaroglu M (1992) Oxidative damage to DNA in mammalian chromatin. Mutat Res 275(3–6):331–342

Doudney CO, Rinaldi CN (1989) Evidence that ultraviolet light-induced DNA replication death of recA bacteria is prevented by protein synthesis in repair-proficient bacteria. Mutat Res 217(1):33–38

Evans FF, Rosado AS, Casella R, Sebastian GV, Machado PLOA, Holmström C, Kjelleberg S, van Elsas JD, Seldin L (2004) Impact of oil contamination and biostimulation on the diversity of indigenous bacterial communities in soil microcosms. FEMS Microbiol Ecol 49(2):295–305

Farr SB, Kogoma T (1991) Oxidative stress responses in *Escherichia coli* and *Salmonella typhimurium*. Microbiol Rev 55(4):561–585

Foster PL (2007) Stress-induced mutagenesis in bacteria. Crit Rev Biochem Mol Biol 42:373–397

Gao Q, Garcia-Pichel F (2011) Microbial ultraviolet sunscreens. Nat Rev Microbiol 9(11):791–802. https://doi.org/10.1038/nrmicro2649

Glaeser J, Klug G (2005) Photo-oxidative stress in *Rhodobacter sphaeroides*: protective role of carotenoids and expression of selected genes. Microbiology 151:1927–1938

Goosen N, Moolenaar GF (2008) Repair of UV damage in bacteria. DNA Repair (Amst) 7(3):353–379

He YY, Häder DP (2002) Involvement of reactive oxygen species in the UV-B damage to the cyanobacterium Anabaena sp. J Photochem Photobiol B 66(1):73–80

Hendrischk AK, Braatsch S, Glaeser J, Klug G (2007) The phrA gene of *Rhodobacter sphaeroides* encodes a photolyase and is regulated by singlet oxygen and peroxide in a sigma(E)-dependent manner. Microbiology 153(Pt 6):1842–1851

Hoerter J, Eisenstark A, Touati D (1989) Mutations by near-ultraviolet radiation in *Escherichia coli* strains lacking superoxide dismutase. Mutat Res 215(2):161–165

Imlay JA (2002) What biological purpose is served by superoxide reductase? J Biol Inorg Chem 7(6):659–663

Kisker C, Kuper J, Van Houten B (2013) Prokaryotic nucleotide excision repair. Cold Spring Harb Perspect Biol 5(3):a012591. https://doi.org/10.1101/cshperspect.a012591

Kramer GF, Ames BN (1987) Oxidative mechanisms of toxicity of low-intensity near-UV light in *Salmonella typhimurium*. J Bacteriol 169:2259–2266

Li J, Liu Z, Tan C, Guo X, Wang L, Sancar A, Zhong D (2010) Dynamics and mechanism of repair of ultraviolet-induced (6–4) photoproduct by photolyase. Nature 466(7308):887–890. https://doi.org/10.1038/nature09192

Li X, Wang Q, Yu X, Liu H, Yang H, Zhao C, Liu X, Tan C, Klejnot J, Zhong D, Lin C (2011) Arabidopsiscryptochrome 2 (CRY2) functions by the photoactivation mechanism distinct from the tryptophan (trp)triad-dependent photoreduction. Proc Natl Acad Sci U S A 108(51):20844-9. https://doi.org/10.1073/pnas.1114579108

Lindahl T (1993) Instability and decay of the primary structure of DNA. Nature 362(6422):709–715

Little JW (1991) Mechanism of specific LexA cleavage: autodigestion and the role of RecA coprotease. Biochimie 73(4):411–421

Liu Y, Li X, Ma D, Chen Z, Wang JW, Liu H (2018) CIB1 and CO interact to mediate CRY2-dependent regulation of flowering. EMBO Rep 19(10):e45762. https://doi.org/10.15252/embr.201845762

Liu Z, Tan C, Guo X, Li J, Wang L, Sancar A, Zhong D (2013) Determining complete electron flow in the cofactor photoreduction of oxidized photolyase. Proc Natl Acad Sci USA 110(32):12966–12971. https://doi.org/10.1073/pnas.1311073110

Liu Z, Tan C, Guo X, Kao YT, Li J, Wang L, Sancar A, Zhong D (2011) Dynamics and mechanism of cyclobutane pyrimidine

dimer repair by DNA photolyase. Proc Natl Acad Sci USA 108(36):14831–14836. https://doi.org/10.1073/pnas.1110927108
Livak KJ, Schmittgen TD (2001) Analysis of relative gene expression data using real-time quantitative PCR and the 2(− ΔΔC(T)) method. Methods 25(4):402–408
Maslowska KH, Makiela-Dzbenska K, Fijalkowska IJ (2019) The SOS system: a complex and tightly regulated response to DNA damage. Environ Mol Mutagen 60(4):368–384. https://doi.org/10.1002/em.22267
Matallana-Surget S, Joux F, Raftery MJ, Cavicchioli R (2009) The response of the marine bacterium *Sphingopyxis alaskensis* to solar radiation assessed by quantitative proteomics. Environ Microbiol 11(10):2660–2675. https://doi.org/10.1111/j.1462-2920.2009.01992.x
Micevski D, Dougan D (2013) Proteolytic regulation of stress response pathways in *Escherichia coli*. Subcell Biochem 66:105–128
Miller SA, Hamilton SL, Wester UG, Cyr WH (1998) An analysis of UVA emissions from sunlamps and the potential importance for melanoma. Photochem Photobiol 68:63–70
Mitra D, Luo X, Morgan A et al (2012) A UV-independent pathway to melanoma carcinogenesis in the redhair–fairskin background. Nature 491:449–453
Neher SB, Flynn JM, Sauer RT, Baker TA (2003) Latent ClpX-recognition signals ensure LexA destruction after DNA damage. Genes Dev 17(9):1084–1089
Patel M, Jiang Q, Woodgate R, Cox MM, Goodman MF (2010) A new model for SOS-induced mutagenesis: how RecA protein activates DNA polymerase V. Crit Rev Biochem Mol Biol 45(3):171–184. https://doi.org/10.3109/10409238.2010.480968
Pitts DG (1990) Sunlight as an ultraviolet source. Optometry Vision Sci 67:401–406
Prada Medina CA, Aristizabal Tessmer ET, Quintero Ruiz N, Serment-Guerrero J, Fuentes JL (2016) Survival and SOS response induction in ultraviolet B irradiated *Escherichia coli* cells with defective repair mechanisms. Int J Radiat Biol 92(6):321–328. https://doi.org/10.3109/09553002.2016.1152412
Procópio L, Alvarez VM, Jurelevicius DA, Hansen L, Sørensen SJ, Cardoso JS, Pádula M, Leitão AC, Seldin L, van Elsas JD (2012a) Insight from the draft genome of *Dietzia cinnamea* P4 reveals mechanisms of survival in complex tropical soil habitats and biotechnology potential. Antonie Van Leeuwenhoek 101(2):289–302. https://doi.org/10.1007/s10482-011-9633-7
Procópio L, de Cassia Pereire e Silva M, van Elsas JD, Seldin L (2013) Transcriptional profiling of genes involved in n-hexadecane compounds assimilation in the hydrocarbon degrading *Dietzia cinnamea* P4 strain. Braz J Microbiol 44(2):633–641. https://doi.org/10.1590/S1517-83822013000200044
Procópio L, Pádula M, van Elsas JD, Seldin L (2019) Oxidative damage induced by H(2)O(2) reveals SOS adaptive transcriptional response of *Dietzia cinnamea* strain P4. World J Microbiol Biotechnol 35(4):53. https://doi.org/10.1007/s11274-019-2628-7
Rastogi RP, Kumar A, Tyagi MB, Sinha RP (2010) Molecular mechanisms of ultraviolet radiation-induced DNA damage and repair. J Nucleic Acids 2010:592980. https://doi.org/10.4061/2010/592980
Santos AL, Oliveira V, Baptista I, Henriques I, Gomes NC, Almeida A, Correia A, Cunha Â (2013) Wavelength dependence of biological damage induced by UV radiation on bacteria. Arch Microbiol 195(1):63–74. https://doi.org/10.1007/s00203-012-0847-5
Simmons LA, Foti JJ, Cohen SE, Walker GC (2008) The SOS regulatory network. EcoSal Plus. https://doi.org/10.1128/ecosalplus.5.4.3
Storz G, Zheng M (2000) Bacterial stress responses In: Storz G, Hengge-Aronis R (eds). ASM, Washington, pp 47–59
Sutton MD, Smith BT, Godoy VG, Walker GC (2000) The SOS response: recent insights into umuDC-dependent mutagenesis and DNA damage tolerance. Annu Rev Genet 34:479–497
Tandori J, Hideg E, Nagy L, Maróti P, Vass I (2001) Photoinhibition of carotenoidless reaction centers from *Rhodobacter sphaeroides* by visible light. Effects on protein structure and electron transport. Photosynth Res 70(2):175–184
Taylor JS (2015) Biomolecules. The dark side of sunlight and melanoma. Science 347(6224):824. https://doi.org/10.1126/science.aaa6578
Valko M, Rhodes CJ, Moncol J, Izakovic M, Mazur M (2006) Free radicals, metals and antioxidants in oxidative stress-induced cancer. Chem Biol Interact 160(1):1–40
Yassin AF, Hupfer H, Schaal KP (2006) *Dietzia cinnamea* sp. Nov., a novel species isolated from a perianal swab of a patient with a bone marrow transplant. Int J Syst Evol Microbiol 56(Pt 3):641–645
Zhang AP, Pigli YZ, Rice PA (2010) Structure of the LexA-DNA complex and implications for SOS box measurement. Nature 466(7308):883–886. https://doi.org/10.1038/nature09200
Ziegelhoffer EC, Donohue TJ (2009) Bacterial responses to photo-oxidative stress. Nat Rev Microbiol 7(12):856–863. https://doi.org/10.1038/nrmicro2237