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Effect of reactive species photogenerated by the UV irradiation of TiO_2 on the peroxidation of linoleic acid

Shortened version: **Reactive species photogenerated by TiO_2**

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Abstract

This paper shows that hydroxyl radicals adsorbed on the surface of TiO_2 (Ti-$^{\bullet}$OH groups) account for about 70-100% of the photocatalytic peroxidation of linoleic acid under UVB irradiation, which results into malondialdehyde production. The experimental data are silent concerning the involvement of $^1O_2$ in the process, although an upper limit of 30% can be hypothesized, while an involvement of surface-bound holes into the lipoperoxidation process can be excluded.

Keywords TiO_2 nanoparticles, Aeroxide P 25, UVB irradiation, linoleic acid peroxidation, MDA

INTRODUCTION

Ultraviolet radiation is the component of the sunlight spectrum reaching the ground that is most dangerous for living organisms. In humans it can induce skin damage such as acute sunburn, photoallergy, photoaging, immunosuppression or even skin cancer. For this reason it is particularly important the protection against sunlight with products containing specific UV filters. There are both organic and inorganic UV blockers. The former are also called UV absorbers because they mainly absorb UV rays, while inorganic filters are often semi-conductor oxides such as TiO_2, ZnO, SiO_2 and ZrO_2 that reflect, scatter or absorb UV radiation. The inorganic particles are considered broad spectrum substances due to their ability to counteract the harmful effects induced by both UVA and UVB radiation. They are preferred for children and sunscreen-allergic individuals, because they do not cause skin irritation and sensitization. The only inorganic filters approved by the Food and Drug Administration are zinc oxide and titanium dioxide. They are rather photostable, and a thick layer is required to achieve adequate reflection of sunlight. Titania is the most widely used mineral pigment in suntan lotions because of its superior UVB protection and higher refractive index compared to ZnO [1].

Due to cosmetic acceptability issues, TiO_2 is employed in micronized form in topical formulations. In fact, because of its ability to reduce visible light scattering, the micronized form is used to avoid the traditional opaque whiteness that is aesthetically unappealing in sunscreen products [2]. However, microfine TiO_2 particles are reactive and tend to form aggregates in cosmetic formulations. To minimize these drawbacks, they are coated with organic compounds or inorganic materials and they are dispersed with oils, solubilizers and emollients. Such operations reduce free radical formation, increase photostability and enhance dispersion in sunscreen lotions [2, 3].

TiO_2 has important applications which exploit its photocatalytic properties. It is a favorite agent in the industrial technology for wastewater detoxification, environmental purification and

antibacterial applications. It is also a key component of the dye-sensitized solar cells. Moreover, this pigment is widely used in the industry as an additive for products ranging from paint, food coloring, drugs and cosmetics (for instance in make-up products). Because of its well-known high absorption of UV radiation, TiO_2 is also used for food packaging to prevent damage from UV radiation and increase the food shelf life [4].

Titania has been considered for a long time as a safe material, biologically and chemically inert, but since the advent of nanotechnology it has been necessary to better investigate its health effects, and contrasting results have been obtained.

After inhalation, TiO_2 nanoparticles produce severe pulmonary damage such as lung inflammation and cytotoxicity. Moreover, such nanoparticles might be involved in lung cancer development [5-10]. Many researches were carried out on TiO_2 mediated-skin damage. Although early *in vitro* experiments have shown that microfine titanium dioxide particles are not able to penetrate the skin *stratum corneum* [10] and cannot be detected in deeper *stratum corneum*, epidermis and dermis [11], some new studies have yielded different results. Jianhong *et al*. have demonstrated that after *in vitro* or *in vivo* chronic dermal exposure, TiO_2 nanoparticles can penetrate the *stratum corneum* and enter the deeper layer of epidermis. The pigment can thus reach various organs and induce different pathological damages, most notably in skin and liver [12].

As a semiconductor oxide, TiO_2 shows photocatalytic activity upon radiation absorption. The absorption of UV radiation by TiO_2 induces the production of valence-band holes (h^+) as well as of reactive oxygen species (ROS) such as hydroxyl ($HO^\bullet$) and superoxide ($O_2^{\bullet-}$) radicals, hydrogen peroxide (H_2O_2) and singlet oxygen ($^1O_2$), all being able to initiate oxidative processes. Various and adverse reactions could be induced on the skin as a result of the production of ROS: for instance, photoactive TiO_2 causes decomposition of DNA and RNA [13], DNA damage in human skin cells cultures [13-15] and genotoxicity [16].

Titanium dioxide can also induce photooxidation of unsaturated lipids, such as the cell-membrane phospholipids. Unsaturated lipids are also widely adopted in emulsion systems because of their ability to provide structure and stability to formulations [17]. To tackle this problem, a considerable effort has been dedicated to achieve reduction of the photocatalytic activity of TiO_2 [18-20].

Previous studies by some of us have assessed the influence of the inorganic surface coating of TiO_2 nanoparticles [21] and the effects of some additives [22] on the UV-induced peroxidation of linoleic acid, chosen as a model of unsaturated lipids.

It has also been shown that propylene glycol and ethanol, two organic additives commonly used in cosmetic formulations, can significantly reduce but not completely quench the lipoperoxidation of linoleic acid [23].

The aim of this paper is to identify the reactive species produced by TiO_2 under irradiation that are involved into the peroxidation of linoleic acid. We studied the effect on the peroxidation reaction of the addition of ethanol, salicylic acid and sodium azide, with the purpose to selectively or quasi-selectively scavenge hydroxyl radicals, valence-band holes and $^1O_2$, respectively, which should allow quantification of the importance of each process [24-26].

EXPERIMENTAL SECTION

Materials

The uncoated TiO_2 Aeroxide P 25 was a gift from Degussa (Vicenza, Italy). Sulfuric acid was purchased from Merck (Darmstadt, Germany). 1-Butanol, sodium azide (NaN_3), sodium dodecyl sulfate (SDS), sodium hydroxide and salicylic acid (HSal) were from Fluka (Milan, Italy). Absolute ethanol and 85% phosphoric acid were from Carlo Erba (Rodano, Italy). Linoleic acid (LA) was purchased from Aldrich (Milan, Italy), 2-thiobarbituric acid (4,6 dihydroxy-2-mercaptopyrimidine, TBA) and 1,1,3,3-tetraethoxypropane (malondialdehyde-bis (diethyl acetal)

from Sigma (Milan, Italy). All reagents were of analytical grade and were used as received, without further purification.

Methods

UVB-induced peroxidation of linoleic acid

A micellar dispersion of LA (1.00% w/w) in 4.00% w/w SDS was magnetically stirred for 24 hours in the dark. TiO_2 at 1.00% w/w was added to the LA dispersion upon sonication (660/H Transsonic Sonifier, Elma, Singen, Germany). The pH was then adjusted to 5.0 (pH meter Basic 20, Crison, Alella, Spain) employing 1.0 N NaOH or 0.5 N H_2SO_4.

An aliquot (10.0 ml) of each sample was introduced in Pyrex® glass cells and irradiated for 0, 30, 60, 90 and 120 min under magnetic stirring (RO 5, IKA, Staufen, Germany) at a distance of 10 cm from a UVB lamp (G40T10E, Sankyo Denki, Kanagawa, Japan). The lamp had 2.4 W m^{-2} power emission of radiation, measured with a power meter (CO.FO.ME.GRA., Milan, Italy). The irradiation time was up to 2 hours.

UVB-induced peroxidation of LA in the presence of ethanol, salicylic acid and sodium azide

The micellar LA dispersions, prepared as reported above, were added with absolute ethanol at 0.82% w/w, 3.28% w/w, and 7.90% w/w (respectively 5, 20 and 48 folds the LA molar concentration). HSal was employed at 1.17% w/w and 2.00% w/w (respectively 20- and 34-fold the LA molar concentration). Sodium azide was added to the suspensions at 1.00 and 4.00% w/w (respectively 4- and 17-fold the LA molar concentration).

After addition of all the reactants the pH was adjusted to 5.0. Irradiation was carried out under the same conditions that were previously described. After irradiation, all the samples were centrifuged for 10 minutes at 20,000 rpm (Allegra™ 64R centrifuge, Beckman Coulter, Palo

Alto, CA) to separate TiO_2. A sample aliquot (0.2 ml) was taken for the quantification of malondialdehyde (MDA) as end-product of LA degradation.

MDA determination

The concentration of MDA was determined with the TBA assay, which is currently used as an index of lipoperoxidation [27]. The assay is based on the reactivity of MDA with TBA to produce a pink adduct (TBA-MDA-TBA) that absorbs at 535 nm. MDA was detected following a spectrophotometric method, according to Bay *et al*. [28].

The sample (0.2 ml) was introduced into a glass tube closed with a screw cap and added with 0.1 ml of water, 0.2 ml of 8.1% w/w SDS, 1.5 ml of 1.0% w/w phosphoric acid, and 1.0 ml of 0.6% w/w TBA. The mixture was stirred and heated in water bath at 95-100°C for 45 minutes, to allow the formation of the complex. After cooling in ice bath, 4.0 ml of 1-butanol were added to each tube and the TBA-MDA-TBA complex was extracted upon stirring and centrifugation. The organic supernatant was evaluated by spectrophotometry (DU® 730 Life Science UV-VIS spectrophotometer, Beckman Coulter, Fullerton, CA). The final concentration of MDA, derived from the reaction, was expressed as nmol of MDA per mg of lipid substrate.

The calibration curve of the TBA-MDA-TBA complex was obtained employing a solution of 1,1,3,3-tetraethoxypropane in 8.1% w/w SDS, within a concentration range of 9.4-208.0 μM. MDA originates from the acid hydrolysis of 1,1,3,3-tetraethoxypropane in an equimolecular reaction [27].

RESULTS

The extent of lipid peroxidation was determined in different samples constituted by a dispersion of LA in 4.00% w/w aqueous SDS, to which various scavengers of $^{\bullet}OH$ radicals, holes and

singlet oxygen were added in different concentrations. The results were compared with those obtained in the absence of the additives.

The occurrence of MDA (up to 20 nmol/mg LA) was observed in some samples before irradiation. To facilitate the comparison between the different curves, the time evolution of MDA was corrected for its initial concentration and the different plots report the axes intersection as the starting point before irradiation.

Figures 1-3 show the MDA time evolution upon photodegradation of LA in the absence and in the presence of TiO_2. It is reported the effect on the MDA trend caused by the addition of ethanol at 0.82, 3.28 and 7.90% w/w, respectively. It can be observed that some MDA was formed even in the absence of TiO_2, but the addition of the photocatalyst significantly enhanced the production of MDA. Ethanol decreased the MDA formation, although it was definitely not able to prevent it even at the highest adopted concentration value (48-fold molar excess compared to LA). If the MDA production without TiO_2 is taken into account and is subtracted to the TiO_2 data as a baseline value, one finds that ethanol at the highest adopted concentration inhibits LA peroxidation by about 50%.

Figures 4 and 5 report the effect on the MDA trend of 1.17 and 2.00% w/w HSal, respectively. In both cases it can be concluded that HSal has practically no effect on the formation of MDA, because the limited differences observed in the reported MDA trends with and without HSal can be attributed to the experimental incertitude.

The decomposition reaction of LA was also carried out in the presence of sodium azide. The additive was used at 1.00 and 4.00% w/w (Figures 6, 7). The Figures show that the azide enhances the formation of MDA and that the level of enhancement increases with increasing azide concentration. This effect is particularly evident in the run with 4.00% w/w NaN_3, while in the presence of 1.00% w/w NaN_3 the (limited) enhancement of MDA formation is about the same in the absence and in the presence of TiO_2.

DISCUSSION

The three additives were intended to scavenge the reactive species photogenerated by the UV irradiation of TiO_2. In particular, ethanol is able to scavenge homogeneous $^{\bullet}OH$ with a reaction rate constant of 1.9×10^{9} M^{-1} s^{-1} [24] and it is also able to react with the somewhat similar Ti–$^{\bullet}OH$ groups on the surface of titanium dioxide [29]. Interestingly, ethanol reacts very slowly with singlet oxygen (second-order rate constant < 10^{3} M^{-1} s^{-1}; [30]) and it is not reported to react with the surface holes of TiO_2 to a significant extent [29].

HSal forms surface complexes with TiO_2 [31] and can react with the photogenerated holes on the semiconductor surface [25,32]. The photocatalytic degradation of HSal is not inhibited by alcohols to a significant extent [32]. Therefore, it is suggested that HSal transformation in the presence of titanium dioxide mainly takes place upon reaction with the holes, and only to a minor extent via the surface-bound hydroxyl radicals.

Sodium azide (NaN_3) is a well-known scavenger of singlet oxygen [26,33], with which it reacts with a rate constant of 7.8×10^{8} M^{-1} s^{-1} [30]. The azide can also react with the $^{\bullet}OH$ radicals (rate constant 1.4×10^{10} M^{-1} s^{-1}; [24]. Moreover, because the standard reduction potential of the $N_3^{\bullet}/N_3^{-}$ couple (1.33 V; [34]) is significantly lower than that of the TiO_2 valence band (2.4 V; [35]), the one-electron oxidation of N_3^{-} to $N_3^{\bullet}$ by the surface holes of TiO_2 is thermodynamically allowed.

The inhibition by ethanol of LA lipoperoxidation with TiO_2 suggests that the Ti–$^{\bullet}OH$ surface groups can be involved into the process. Interestingly, a large excess of ethanol with respect to LA was able to decrease the rate of MDA formation by no more than 50% (see Figure 3). The fact that ethanol was not able to completely quench the formation of MDA can be explained as follows: *(i)* LA lipoperoxidation could also be caused by other reactive species that are not scavenged by ethanol, or *(ii)* the radicals derived from the oxidation of ethanol by Ti–$^{\bullet}OH$ (*e.g.*

CH_3-$CH_2O^{\bullet}$) could take part to the lipoperoxidation process, which would partially compensate for the scavenging of Ti–$^{\bullet}$OH.

HSal has practically no effect on LA lipoperoxidation. Considering that such a substrate would mainly scavenge the valence-band holes entrapped on the surface of TiO_2, this finding would exclude that the holes are involved into the process. The reactivity of the TiO_2 surface holes bears some resemblance with that of the sulfate radical anions [36], and $SO_4^{-\bullet}$ reacts with carboxylic acids via oxidation of the –COOH group to CO_2 [37]. Accordingly, the valence-band holes of TiO_2 could oxidize the LA carboxylic group rather than induce lipoperoxidation.

Also interesting is the enhancement of MDA formation by sodium azide. At the adopted concentration values the azide would cause complete scavenging of singlet oxygen. If the reaction between singlet oxygen and the azide anion is just a physical quenching ($^1O_2$ loses energy to give ground-state O_2 and N_3^- is not modified; [38]), one expects the reactivity of the system to be completely suppressed. Considering that $^1O_2$ would take part to the lipoperoxidation reactions [39], the expected effect of N_3^- would be an inhibition of MDA formation. However, there is some literature evidence that the reaction between $^1O_2$ and N_3^- yields the azide radical $N_3^{\bullet}$ [40], in which case the oxidizing species $N_3^{\bullet}$ (and/or $O_2^{-\bullet}$ that would also be formed in the process) could be involved into LA lipoperoxidation. Furthermore, production of $N_3^{\bullet}$ from N_3^- could also take place upon oxidation of the azide by Ti–$^{\bullet}$OH or the surface holes, and there is evidence that $^1O_2$ on the TiO_2 surface would have a shorter lifetime compared to the other reactive species [41]. Accordingly, Ti–$^{\bullet}$OH and the holes could account for the oxidation of dissolved compounds (including possibly N_3^-) to a larger extent than $^1O_2$.

The experimental data concerning the azide-enhanced peroxidation of LA can be explained if $N_3^{\bullet}$ is involved into MDA production. If this is the case, azide addition would completely shadow the possible involvement of $^1O_2$ into lipoperoxidation and the present data would be silent in this respect. Unfortunately it is not clear if and to what extent $N_3^{\bullet}$ is produced by the

reaction $^1O_2 + N_3^-$. If the process $^1O_2 + N_3^- \rightarrow N_3^{\bullet} + O_2^{-\bullet}$ is important in the studied system, one has to assume that $N_3^{\bullet}/O_2^{-\bullet}$ is able to induce lipoperoxidation more efficiently than $^1O_2$. Alternatively, $N_3^{\bullet}$ could be produced upon oxidation of N_3^- by Ti–$^{\bullet}$OH or the surface-entrapped holes. The latter possibility is particularly interesting because the HSal data suggest that the holes would not be directly involved into LA lipoperoxidation. The hole-mediated oxidation of N_3^- could produce a lipoperoxidizing agent ($N_3^{\bullet}$) and, therefore, account for the observed increase of MDA generation in the presence of the azide. Interestingly, also the addition to the system of furfuryl alcohol, another scavenger of $^1O_2$ [42], enhanced the production of MDA from linoleic acid and TiO_2 (data not shown).

From the experimental data it can be deduced that the holes trapped on the TiO_2 surface are not able to induce lipoperoxidation reactions, at least in the absence of additives such as N_3^-. Moreover, the data do not allow to conclusively demonstrate or exclude the involvement of singlet oxygen into LA peroxidation. In contrast, the Ti–$^{\bullet}$OH groups could play a very important role in the process. Under the reasonable hypothesis that the reactivity of Ti–$^{\bullet}$OH is similar to that of homogeneous $^{\bullet}$OH [36], and based on the reaction rate constants of the latter [24], in the presence of 1% w/w linoleic acid, 4% w/w SDS and 7.9% w/w ethanol, ethanol would scavenge about 70% of the available Ti–$^{\bullet}$OH. Under such conditions, the inhibition by ethanol of the MDA photogeneration was around 50%. A possible explanation of this finding is that about 70% of the total lipoperoxidation is caused by Ti–$^{\bullet}$OH. The remaining 30% could be accounted for by $^1O_2$, but this is just an upper limit because there is no evidence that $^1O_2$ is actually involved into LA peroxidation. Moreover, it is not possible to exclude that the radical species produced upon oxidation of ethanol by Ti–$^{\bullet}$OH are also involved into the formation of MDA. Under this hypothesis, the experimental data could even be compatible with LA peroxidation being entirely accounted for by Ti–$^{\bullet}$OH.

CONCLUSIONS

This paper studies the contribution of different reactive species generated under photocatalytic conditions to the lipoperoxidation of LA, which causes the production of MDA. From the experimental data it is possible to exclude that the holes trapped on the surface of TiO_2 are involved into lipoperoxidation to a significant extent. Probably this happens because the main reaction between carboxylic acids and holes is the oxidation of the –COOH group rather than the peroxidation of the double bonds. Among the other candidate reactive species, the Ti–$^{\bullet}$OH groups (the reactivity of which is similar to that of $^{\bullet}$OH) are very likely to play a significant role. The data concerning the effect of ethanol on the photocatalytic production of MDA suggest that Ti–$^{\bullet}$OH could account for 70-100% of LA peroxidation. Inconclusive data are available for the involvement of $^{1}O_2$ in the peroxidation process, but an upper limit of 30% can be proposed based on the ethanol/Ti–$^{\bullet}$OH data.

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FIGURE CAPTIONS

FIG. 1. MDA (nmol/mg LA) time evolution upon UVB photodegradation of 1.00 % w/w LA in the absence and in the presence of 1.00% w/w TiO_2 (P 25), in 4.00% w/w SDS micellar dispersion. The MDA data are compared with those obtained in the presence of ethanol (EtOH) at 0.82% w/w and otherwise identical conditions.

FIG. 2. MDA (nmol/mg LA) time evolution upon UVB photodegradation of 1.00 % w/w LA in the absence and in the presence of 1.00% w/w TiO_2 (P 25), in 4.00% w/w SDS micellar dispersion. The MDA data are compared with those obtained in the presence of ethanol (EtOH) at 3.28% w/w and otherwise identical conditions.

FIG. 3. MDA (nmol/mg LA) time evolution upon UVB photodegradation of 1.00 % w/w LA in the absence and in the presence of 1.00% w/w TiO_2 (P 25), in 4.00% w/w SDS micellar dispersion. The MDA data are compared with those obtained in the presence of ethanol (EtOH) at 7.90% w/w and otherwise identical conditions.

FIG. 4. MDA (nmol/mg LA) time evolution upon UVB photodegradation of 1.00 % w/w LA in the absence and in the presence of 1.00% w/w TiO_2 (P 25), in 4.00% w/w SDS micellar dispersion. The MDA data are compared with those obtained in the presence of salicylic acid (HSal) at 1.17% w/w and otherwise identical conditions.

FIG. 5. MDA (nmol/mg LA) time evolution upon UVB photodegradation of 1.00 % w/w LA in the absence and in the presence of 1.00% w/w TiO_2 (P 25), in 4.00% w/w SDS micellar dispersion. The MDA data are compared with those obtained in the presence of salicylic acid (HSal) at 2.00% w/w and otherwise identical conditions.

FIG. 6. MDA (nmol/mg LA) time evolution upon UVB photodegradation of 1.00 % w/w LA in the absence and in the presence of 1.00% w/w TiO_2 (P 25), in 4.00% w/w SDS micellar dispersion. The MDA data are compared with those obtained in the presence of sodium azide (NaN_3) at 1.00% w/w and otherwise identical conditions.

FIG. 7. MDA (nmol/mg LA) time evolution upon UVB photodegradation of 1.00 % w/w LA in the absence and in the presence of 1.00% w/w TiO_2 (P 25), in 4.00% w/w SDS micellar dispersion. The MDA data are compared with those obtained in the presence of sodium azide (NaN_3) at 4.00% w/w and otherwise identical conditions.

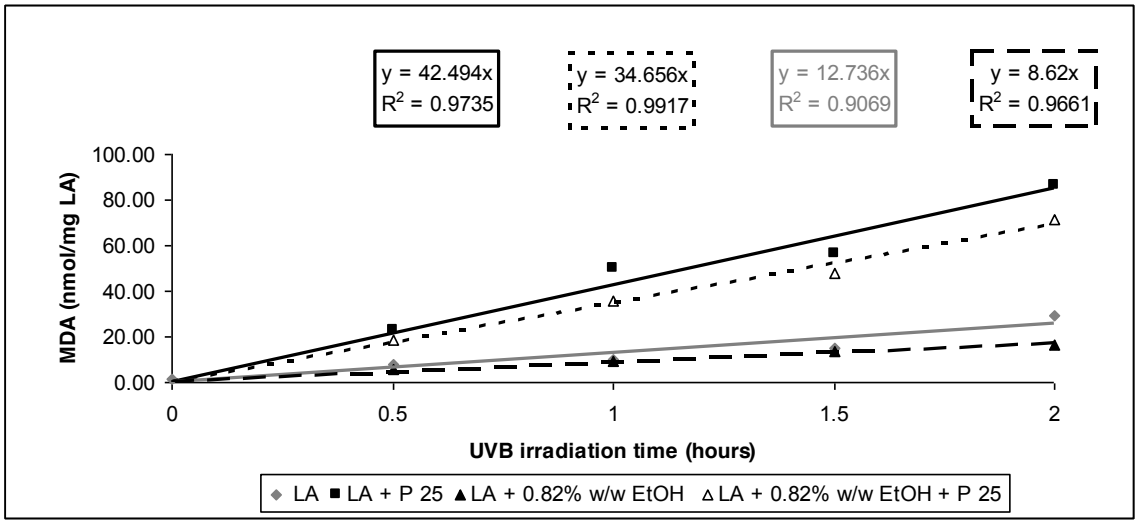
y = 42.494x
R2 = 0.9735
y = 34.656x
R2 = 0.9917
y = 12.736x
R2 = 0.9069
y = 8.62x
R2 = 0.9661
MDA (nmol/mg LA)
100.00
80.00
60.00
40.00
20.00
0.00
0
0.5
1
1.5
2
UVB irradiation time (hours)
LA
LA + P 25
LA + 0.82% w/w EtOH
LA + 0.82% w/w EtOH + P 25

FIG. 1

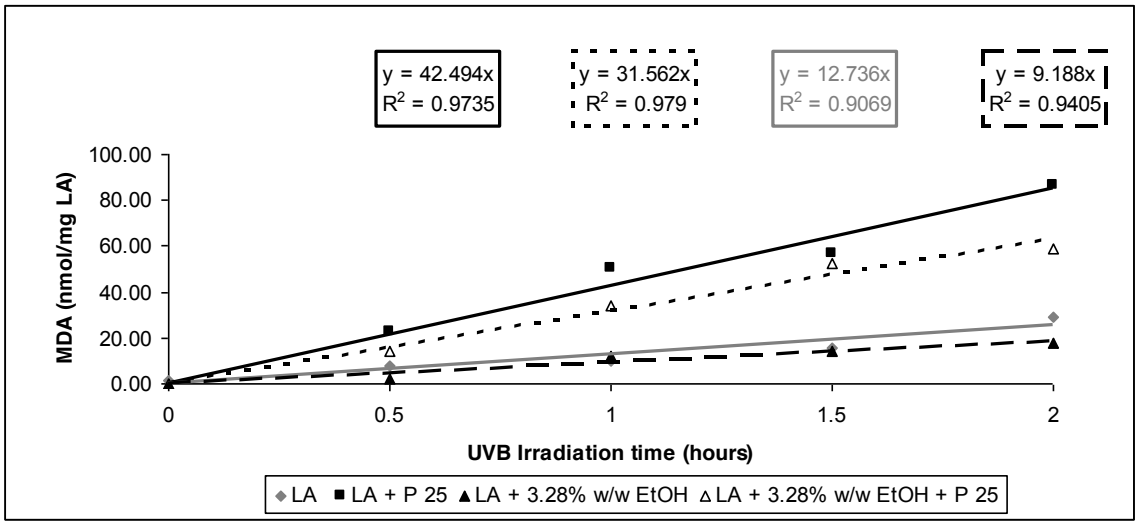
y = 42.494x
R2 = 0.9735
y = 31.562x
R2 = 0.979
y = 12.736x
R2 = 0.9069
y = 9.188x
R2 = 0.9405
MDA (nmol/mg LA)
100.00
80.00
60.00
40.00
20.00
0.00
0
0.5
1
1.5
2
UVB Irradiation time (hours)
LA
LA + P 25
LA + 3.28% w/w EtOH
LA + 3.28% w/w EtOH + P 25

FIG. 2

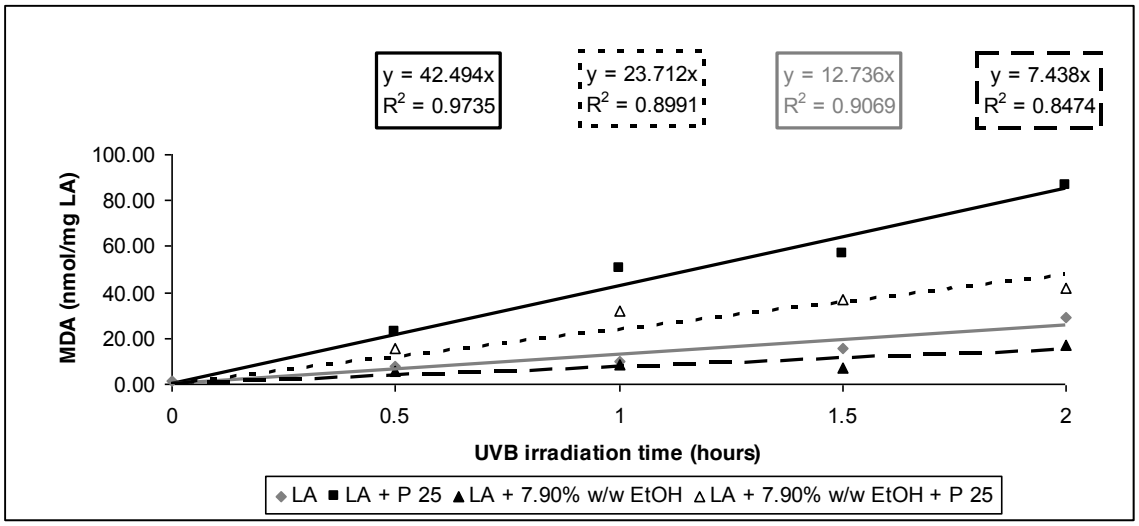
y = 42.494x
R2 = 0.9735
y = 23.712x
R2 = 0.8991
y = 12.736x
R2 = 0.9069
y = 7.438x
R2 = 0.8474
MDA (nmol/mg LA)
100.00
80.00
60.00
40.00
20.00
0.00
0
0.5
1
1.5
2
UVB irradiation time (hours)
LA
LA + P 25
LA + 7.90% w/w EtOH
LA + 7.90% w/w EtOH + P 25

FIG. 3

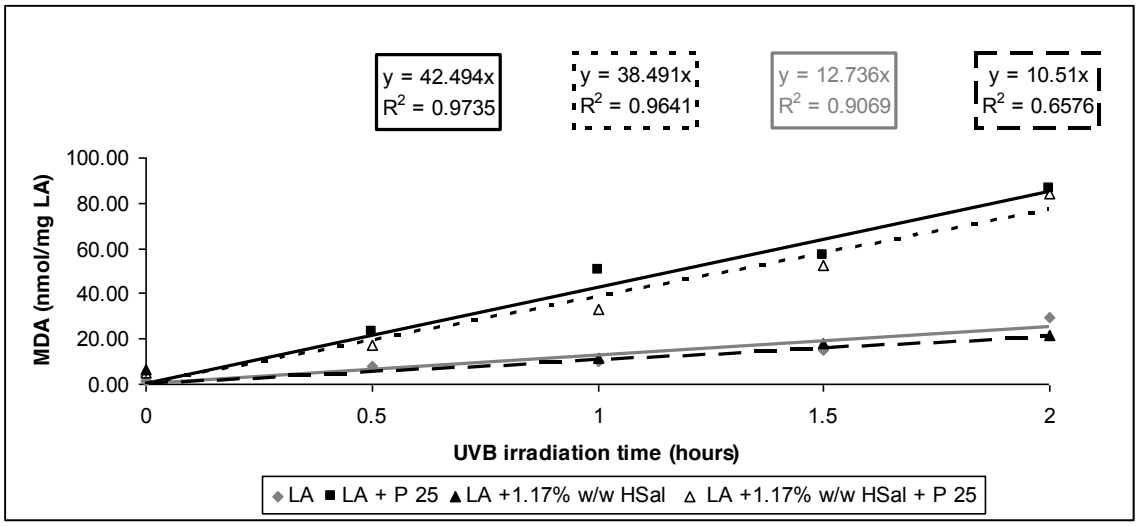
y = 42.494x
R2 = 0.9735
y = 38.491x
R2 = 0.9641
y = 12.736x
R2 = 0.9069
y = 10.51x
R2 = 0.6576
MDA (nmol/mg LA)
100.00
80.00
60.00
40.00
20.00
0.00
0
0.5
1
1.5
2
UVB irradiation time (hours)
LA
LA + P 25
LA +1.17% w/w HSal
LA +1.17% w/w HSal + P 25

FIG. 4

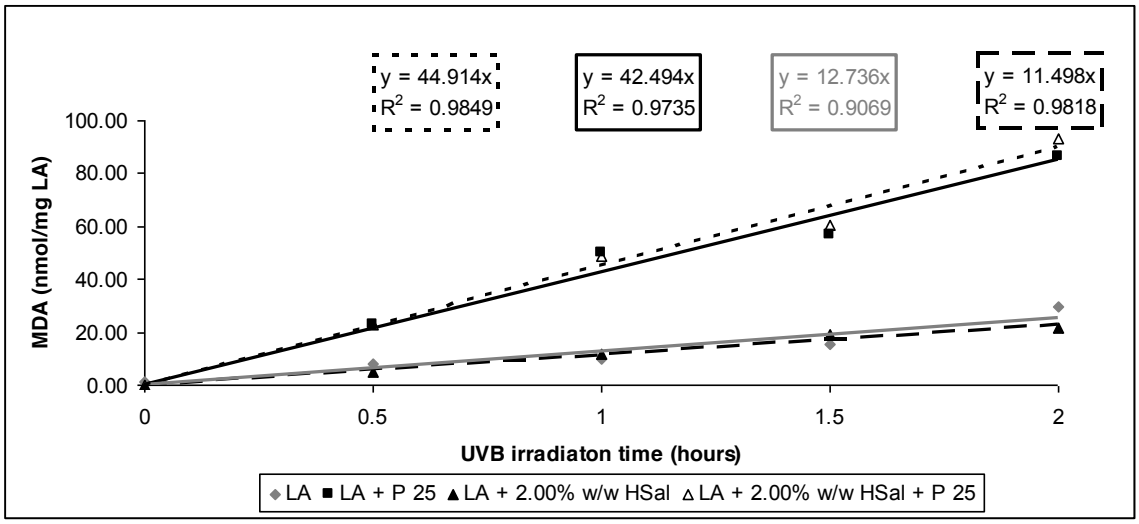
y = 44.914x
R2 = 0.9849
y = 42.494x
R2 = 0.9735
y = 12.736x
R2 = 0.9069
y = 11.498x
R2 = 0.9818
MDA (nmol/mg LA)
100.00
80.00
60.00
40.00
20.00
0.00
0
0.5
1
1.5
2
UVB irradiaton time (hours)
LA
LA + P 25
LA + 2.00% w/w HSal
LA + 2.00% w/w HSal + P 25

FIG. 5

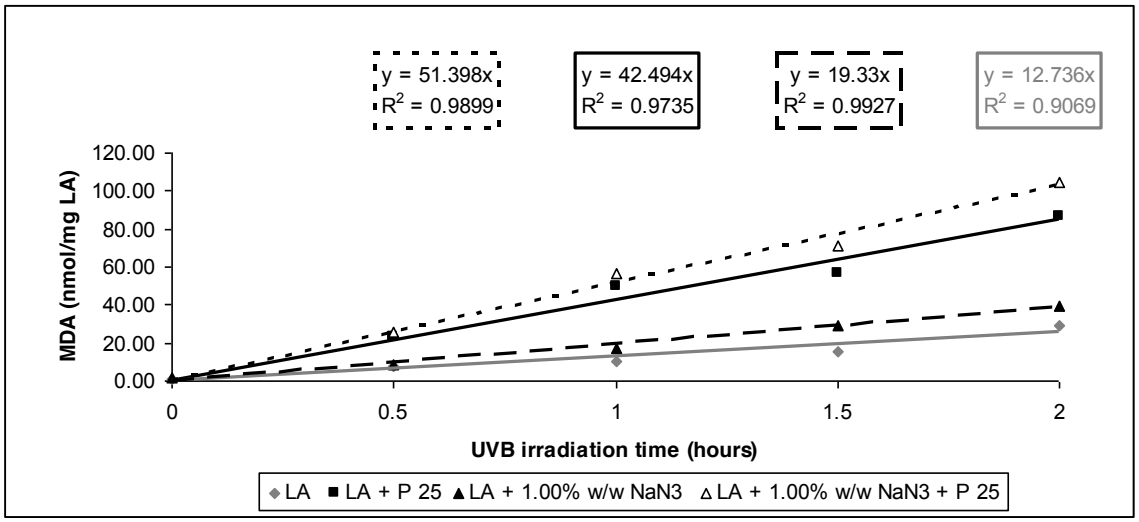
y = 51.398x
R2 = 0.9899
y = 42.494x
R2 = 0.9735
y = 19.33x
R2 = 0.9927
y = 12.736x
R2 = 0.9069
MDA (nmol/mg LA)
120.00
100.00
80.00
60.00
40.00
20.00
0.00
0
0.5
1
1.5
2
UVB irradiation time (hours)
LA
LA + P 25
LA + 1.00% w/w NaN3
LA + 1.00% w/w NaN3 + P 25

FIG. 6

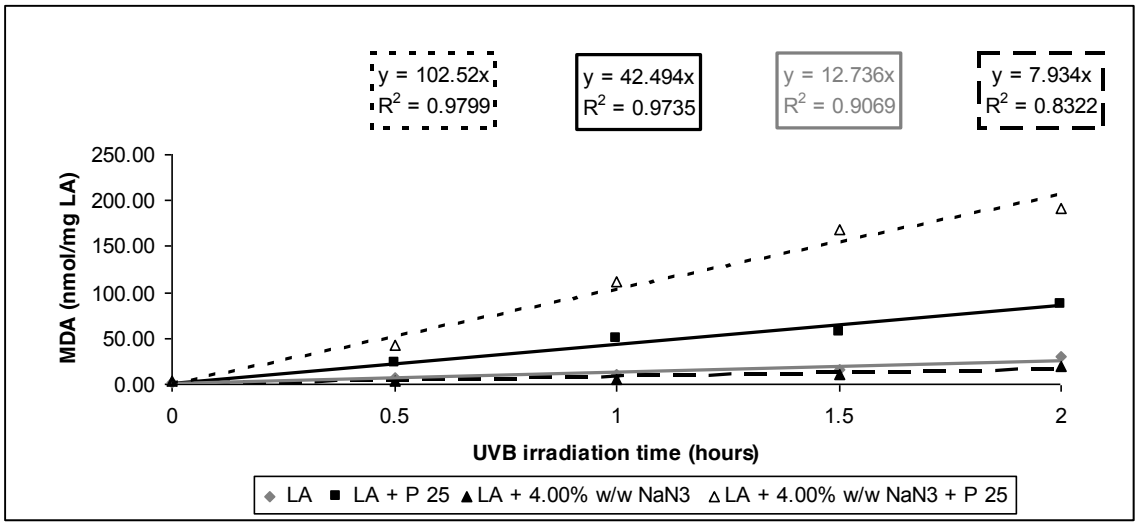
y = 102.52x
R2 = 0.9799
y = 42.494x
R2 = 0.9735
y = 12.736x
R2 = 0.9069
y = 7.934x
R2 = 0.8322
MDA (nmol/mg LA)
250.00
200.00
150.00
100.00
50.00
0.00
0
0.5
1
1.5
2
UVB irradiation time (hours)
LA
LA + P 25
LA + 4.00% w/w NaN3
LA + 4.00% w/w NaN3 + P 25

FIG. 7