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# B6 vitamers as generators and scavengers of reactive oxygen species

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ABSTRACT

B6 vitamers are important natural compounds for human life, principally for amino acid metabolism, and have been reported as potent scavenger molecules of both endogenous and exogenous reactive oxygen species (ROS). However, under UV-light excitation from UVC (200–280 nm) to UVA (315–400 nm), B6 vitamers act as endogenous photosensitizers that promote both cellular photodamage and enzyme inactivation. This occurs through classical photochemical mechanisms mediated by ROS, toxic photoproducts, and the formation of adducts with active protein sites. This minireview aimed to present and discuss the dual roles of B6 vitamers as generators and scavengers of singlet oxygen (an important reactive species in photodynamic processes) and various other ROS. We also examine the basic photophysical, photochemical, and protolytic equilibria principles of B6 vitamers and their role as photodamaging and photoprotecting compounds in UV-light mediated photobiological processes.

## Introduction

In 1926, Goldberger and coworkers discovered a "P-P" nutritional factor from yeast extract that contained water-soluble B vitamins, suggesting that it was involved in the prevention and cure of pellagra.[1] In 1934, Paul György demonstrated that the observed effect of the cure was from vitamin B6.[2] Four years later, Samuel Lepkovsky isolated the factor; it was identified as 3-hydroxy-4,5-dihydroxy-methyl-2-methyl-pyridine.[3] In 1939, György and Eckardt named it Pyridoxine (PN, Scheme 1 **[I]**).[4] In 1942, Snell and coworkers reported two other forms of vitamin B6; these are now known as Pyridoxal (PL, Scheme 1 **[III]**) and Pyridoxamine (PM, Scheme 1 **[V]**). Although PN is frequently used as a synonym for vitamin B6, the term defines a family of six biologically-interconvertible compounds with a common basic structure of 2-methyl-3-hydroypyridine (Scheme 1). They are PN, PL, PM, and their bioactive 5'-phosphate forms essential for both eukaryotic/prokaryotic organisms and serve as versatile coenzymes in more than one hundred types of enzymatic reactions.

Under UV-radiation from UVC (200–280 nm) to UVA (315–400 nm), B6 vitamers act as endogenous photosensitizers that generate reactive oxygen species (ROS), including singlet oxygen ($^1O_2$), hydrated electrons (a powerful starter of many other reactive species), and reactive photoproducts.[6, 7] Conversely, B6 vitamers are also potent ROS antioxidants; in some cases, they are even more efficient than ascorbic acid.[8–10]

This minireview examines the dual roles of B6 vitamers as generators and scavengers of various ROS, including $^1O_2$. We also examine the basic photophysical, photochemical, and protolytic equilibria principles of B6 vitamers and the role of B6 vitamers as photodamaging and photoprotecting compounds in UV-light mediated photobiological processes.

## Physical-chemistry, photophysical, and photochemical properties

### *Physical-chemistry properties*

With common 2-methyl-3-hydroxypyridine structures, different vitamers occur with $R_1$ substitution and with conventional or phosphate forms in $R_2$ (Scheme 1). Due to phenolic oxygen and pyridine ring nitrogen, cationic species of B6 predominate in acid pH; basic environments are favorable for their anionic forms. At physiological pH (~7.4), $R_1$ drives major changes to protolytic equilibria. While most PM presents cationic forms with complex tautomerism,[11] PN is mostly a zero net charge species (98% at pH 6.86) under a simple zwitterion-neutral equilibrium.[12] Zwitterion occurs in aqueous medium, but neutral forms prevail in nonpolar and aprotic polar solvents.[13,14] PL also exhibits a zero net charge, but it assumes different forms (e.g., free aldehyde, hydrate, hemiacetal). Cyclic hemiacetal seems to be the favored form in water.[12] Schemes 2–4 illustrates the protolytic equilibria of pyridoxine (Scheme 2), pyridoxal (Scheme 3), and pyridoxamine (Scheme 4), respectively. The pKa values are dependent on the electron donor-acceptor ability of the $R_1$ group and the electronic state of vitamers.[15] While pyridine ring nitrogen is slightly more basic in the first excited state than in the ground state, phenolic oxygen is approximately 8-fold more acidic with electronic excitation.[15]

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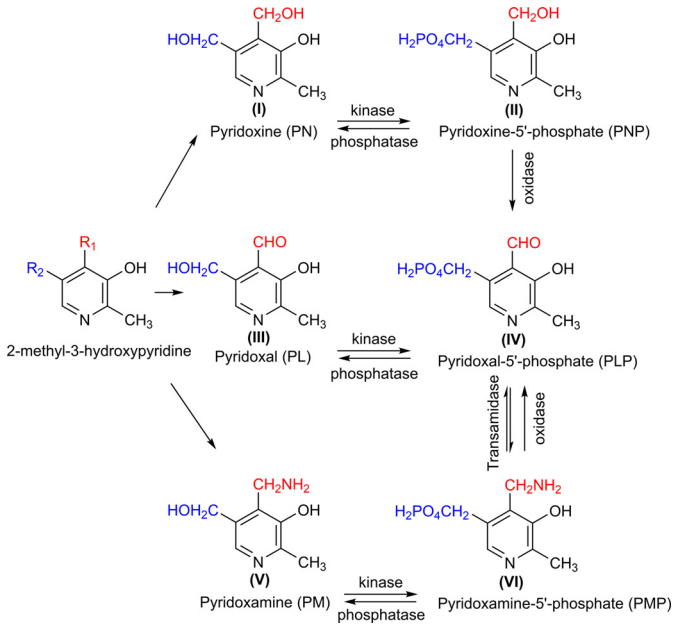



**Scheme 1.** Structures of 2-methyl-3-hydroypyridine, B6 vitamers, and their biological conversions into enzymatically-active phosphate forms. $R_1$ = —$CH_2OH$, —CHO, and —$CH_2NH_2$ for PN, PL, and PM, respectively. For conventional and phosphate forms $R_2$ = —$CH_2OH$ and —$CH_2PO_4H_2$, respectively. Adapted from [5] with permission.

*Photophysical properties*

Except for 4-aminomethyl group (Scheme 1 **[V]** in red) of PM and phosphoryl dissociations, ionizations of the acid/basic groups directly affect the absorption spectra of B6 vitamers. In general, the absorption spectra show two bands within the UV-vis region from a range of 230 to 350 nm; these bands differ in both molar absorptivity coefficient ($\varepsilon$) and maximum absorption wavelength (Fig. 1, Table 1). Neutral forms (one observable band between 285–290 nm [15]) and PL 5-'phosphate (a single detectable band at 388 nm) are exceptions.

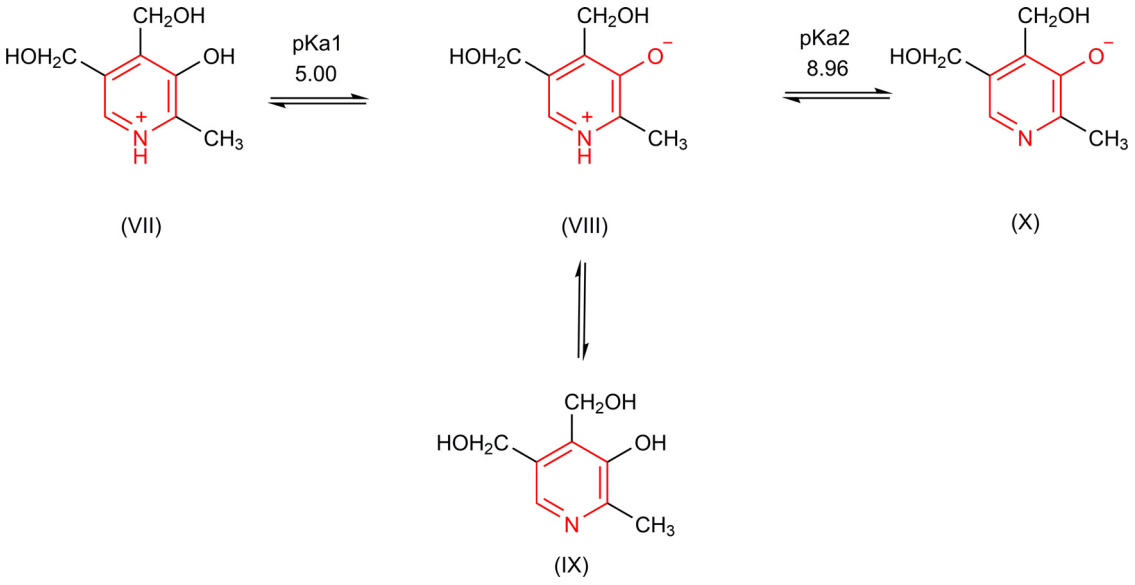
For fluorescence emission, B6 vitamers show distinct fluorescence characteristics for each protolitic species (except with strong hydrogen-donating trifluoroethanol) and only one band with a maximum emission wavelength between 380–395 nm.[15] In aqueous solution, phenol deprotonation causes a redshift in emission spectra. Contrarily, a blueshift is generally seen in organic solvents due to the shift from protonated phenolic to neutral. Nitrogen deprotonation of the pyridine ring leads to a blueshift in the emission spectra and an approximately two-fold decrease in the fluorescence quantum yield.[15] Conversely, the deprotonation of phenolic oxygen increases the fluorescence quantum yield.[15] Apart from PM, fluorescence quantum yields and fluorescence lifetimes of B6 vitamers are considerably low (< 0.1 and < 1.0 ns., respectively) in physiological pH (~7.4). The cationic (Scheme 2 VII, XI, and XV) and anionic (Scheme 2 X, XIV, XVIII, and XIX) species tend to have short lifetimes.[15] The higher fluorescence yields and longer lifetimes of PM (0.15 and 1.60 ns, respectively) compared to other vitamers have made it useful as a luminescent label and tool for distribution analysis in fungus by fluorescence microscopy.[7, 10]

*Photochemical properties*

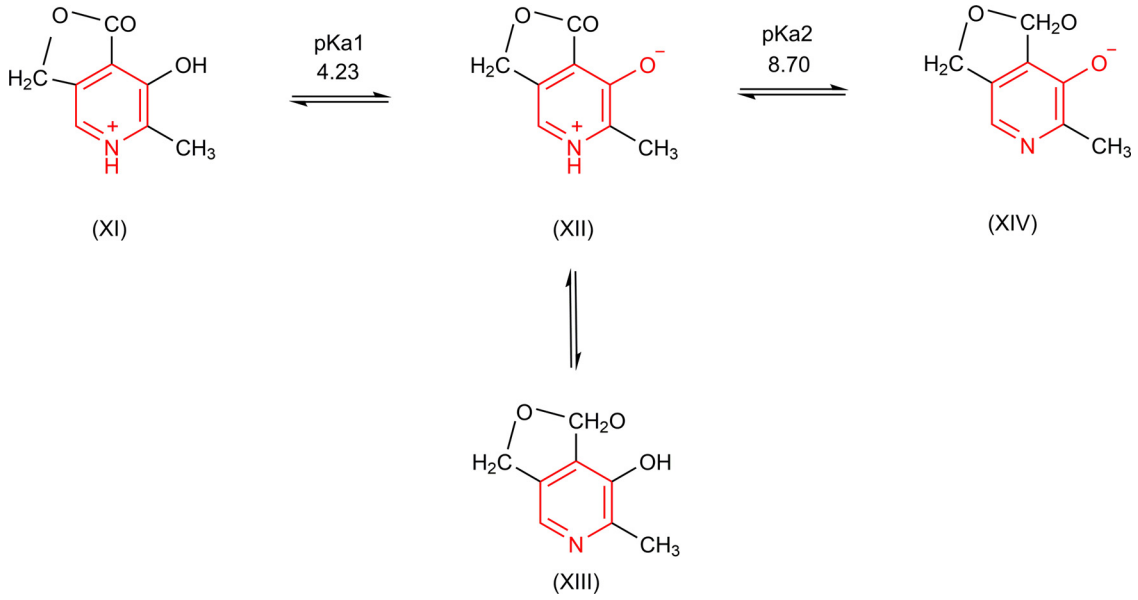
Neutral and basic forms of PN are unstable under UV-light irradiation. Their two peaks of absorption (at approximately 250 and 310 nm) gradually disappear during irradiation. In 1968, Ikeda and coworkers reported that PN's irradiation at 254 nm promoted the destruction of the chromophoric PN ring under aerobic conditions.[18] Further observations have shown that this occurs even in an anaerobic atmosphere, suggesting that the process is guided by photolytic oxidation,[19] although computational calculations indicate that the barriers are too high along the reaction coordinates in the excited states for the occurrence of photolytic ring-opening and dehydroxymethylation, demethylation, and dehydroxylation from the PN aromatic ring.[20] In an *in silico* study, Wu and coworkers suggested that PN UV-trigged photolysis occurs by dehydroxylation of the C4-bound hydroxymethyl group and dehydrogenation of the ring-bound hydroxyl substituent.[20] Definitive descriptions of the photolysis mechanisms for B6 vitamers are still needed.

**Photodamage**

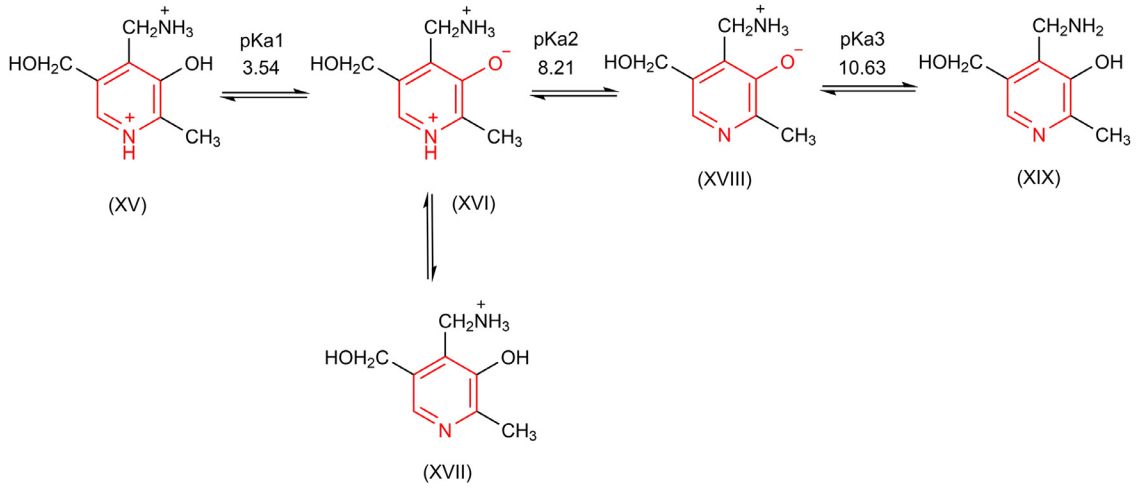
B6 vitamers mediate photooxidative stress in human skin cells. Skin phototoxicity is typically due to high therapeutic doses and abnormal metabolism of B6.[21,22] Skin photoirritation can result from excitation of endogenous chromophores under UV-light (from UVC to UVA) and the intersystem conversion to an excited triplet state that can react




**Scheme 2.** The protolitic equilibria of pyridoxine. The pKa values were obtained from [16].




**Scheme 3.** The protolitic equilibria of pyridoxal. The pKa values were obtained from [16].




**Scheme 4.** The protolitic equilibria of pyridoxamine. The pKa values were obtained from [16].

with substrate molecules or molecular oxygen-yielding ROS. A variety of ROS are generated by electron transfer with a biologic substrate (Type-I reaction), such as superoxide anions ($O_2\bullet^-$), hydrogen peroxide ($H_2O_2$), and hydroxyl radicals (•OH). In parallel, energy transfer to molecular oxygen (type II reaction) forms $^1O_2$. This nonradical form of molecular oxygen has a strong oxidizing potential[9] capable of reacting with biomolecules, especially those containing electron-rich double bonds (e.g., amino acids, DNA, lipids, and other macro components). This reaction leads to cell damage and triggers different cell death pathways. Toxic B6 photoproducts are other important species in the photooxidative action of B6 vitamers. Also, the formation of hydrated electrons by vitamers might inactivate DNA, although these electrons' inactivation efficiency is low (approximately eight percent) [23].

With a type I mechanism, Sato and coworkers showed that PN (100 $\mu$g mL$^{-1}$) yielded about 0.04 $\mu$g mL$^{-1}$ of $H_2O_2$ when it was irradiated with UVA for 16 minutes (17.5 kJ cm$^{-2}$). Maeda and coworkers demonstrated that B6 vitamers produced $H_2O_2$ *in situ*, but in an insufficient amount to cause significant cytotoxicity in the human fibroblastic cell lines where B6 photoproducts most effectively cause cytotoxicity. In this work, PMP was the most cytotoxic vitamer in fibroblasts (P3Ka and PX3305 cell lines); PLP showed non-toxicity.[24] In contrast, Wondrak and coworkers demonstrated that PLP and PL were efficient in inducing fibroblast apoptosis (CF3 cells line) under similar light doses and concentrations.[25] Differences in findings might be due to distinct cell lines' specificity, although further data is needed for more consistent conclusions.

Dzhagarov and coworkers extensively investigated the formation of $^1O_2$ from primary and derivative $B_6$ vitamers.[7,26] The $^1O_2$ quantum yield ($\Phi\Delta^1O_2$) was established in the following order: pyridoxal > pyridoxine > pyridoxamine[26] ( Table 2). This outcome was related to triplet generation influenced by the robust electron-donating characteristics of $R_1$ substituents. Phosphorylation increases $\Phi\Delta^1O_2$ values; PLP linkage to amino acids decreases $\Phi\Delta^1O_2$ values.[7]

PLP binds to various enzymes through reversible Schiff-base linkage with lysine or histidine residues due to the aldehyde group. This linkage is a primary requisite to photoaction in protein active sites. The linkage makes PL much less phototoxic than PLP against phosphoglucose isomerase. As phosphoglucose isomerase has both histidine and lysine as catalytic critical residues, PLP can effectively photomodulate its activities. Hathaway and Noltmann observed a significant loss of activity of this enzyme when under aerobic conditions. This effect is mostly related to $^1O_2$ formation, as evidenced by higher inactivation

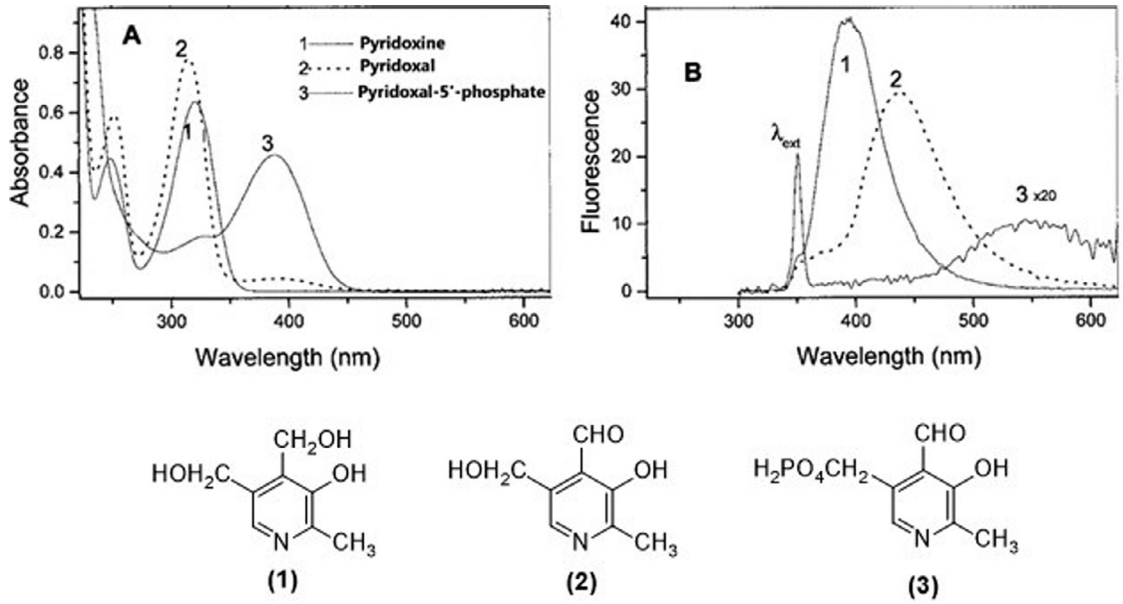



**Fig. 1.** Absorption and fluorescence spectra of B6 vitamers in phosphate buffer solution (pH 7.4). A) absorption spectrum of pyridoxine (1), pyridoxal (2), and pyridoxal-5'-phosphate (3); the spectrum of PM was nearly identical to 1 and is not shown. B) Corrected and normalized fluorescence spectrum of pyridoxine (1), pyridoxal (2), and pyridoxal phosphate (3); the amplitude of spectrum 3 was enlarged 20 times for illustration; the spectrum of PM was practically identical to 1 and is not shown. Excitation wavelength 350 nm is marked by the signal from scattered light. Reprinted with permission from [9].

**Table 1**
Maximum absorption wavelength and molar absorptivity coefficient ($\varepsilon$) of the protolitic forms of B6 vitamers. Adapted with permission of [17].

| Compound | Condition | $\lambda_{máx}$ (nm) | $\varepsilon$ ($10^{-3}$ $M^{-1}$ $cm^{-1}$) | $\lambda_{máx}$ (nm) | $\varepsilon$ ($10^{-3}$ $M^{-1}$ $cm^{-1}$) | Ref. |
|---|---|---|---|---|---|---|
| **Pyridoxine** | | | | | | |
| Cation | 0.1 N HCl | 232 | 2.1 | 291 | 8.6 | [12] |
| Neutral | Alcohol | - | - | 286 | 5.7 | [12] |
| Dipolar ion | pH 6.8 | 254 | 3.9 | 324 | 7.2 | [12] |
| Anion | 0.1 N NaOH | 245 | 6.3 | 310 | 6.8 | [12] |
| **Pyridoxal** | | | | | | |
| Cation | 0.1 N HCl | - | - | 288 | 9.0 | [12] |
| Neutral | 60%dioxane | - | - | 280 | 4.1 | [12] |
| Dipolar ion | pH 6.9 | 252 | 5.8 | 317 | 8.9 | [12] |
| Anion | pH10-11 | 240 | 8.4 | 302 | 5.7 | [12] |
| **Pyridoxamine** | | | | | | |
| | 0.1 N HCl | 226 | 2.0 | 292 | 8.2 | [12] |
| Monohydrochloride | 98%dioxane | - | - | 287 | 3.4 | [12] |
| | pH 6.7 | 252 | 4.5 | 326 | 7.9 | [12] |
| | 0.1 N NaOH | 245 | 6.2 | 310 | 7.2 | [12] |
| **Pyrixodal 5′-** | pH 8.2 | - | - | 388 | 4.93 | [10] |
| **phosphate** | pH 7.2 | - | - | 388 | 5.14 | [10] |
| | pH 6.3 | - | - | 388 | 4.92 | [10] |

rates in $D_2O$ than in $H_2O$.[27] In parallel, a slow inactivation rate was obtained under a nitrogen atmosphere; this effect was attributed to the formation of an adduct with a reversible PLP-enzyme bond mediated by UV-light.

For antimicrobial photodynamic therapy (aPDT), Xu and coworkers evaluated PN's bacterial effect in plasma under UVA-light (6, 12, and 18 J $cm^{-2}$), as depicted in Fig. 2, A-D.[28] The photodynamic response led to a 6.0 log reduction for *E.coli* at an 18 J $cm^{-2}$ light dose. This was consistent with the high $^1O_2$ generation of PN, as demonstrated in Table 2.

## Photoprotection

Although not officially classified as an antioxidant compound, B6 vitamers can scavenge a variety of ROS by attacking an aromatic ring and subtracting a proton from substituent groups or via sequestration of specific metals.[29] Otherwise, PN quenches $^1O_2$ preferably by 1,3-addition at position four, resulting in allylic hydroperoxides; or 1,4-addition at position 6, generating hydroperoxide ketones.[30] Matxain and coworkers have presented further details of these reactions.[9,29,30] While PN efficiently scavenges •OH through the formation of slightly-exergonic adducts,[31] PM and PL seem to be greater quenchers of $^1O_2$.[10] In fact, Natera and coworkers showed that various B6 vitamers quench $^1O_2$ species in constant rates similar to ascorbic acid and effectively decrease $^1O_2$ lifetime.[32]

Hyperglycemia induces ROS production through different pathways in diabetic patients.[33] Chetyrkin and coworkers indicated in several animal models that PM alleviates diabetic nephropathy and retinopathy-protecting lysozyme activity; this occurs partially due to •OH scavenging (Fig. 2E).[34] Such scavenging was also attributed to an indirect mechanism of sequestering of redox-active metal required for •OH production. Natera and coworkers showed that the presence of B6 vitamers quenches $^1O_2$ species at rate constants similar to ascorbic acid, increasing $^1O_2$ lifetime.[32]

**Table 2**
Singlet oxygen quantum yield ($\Phi\Delta^1O_2$) of B6 vitamers and their adducts with amino acids/proteins in $D_2O$ at 25°C. Adapted from [7] and [26] with permission.

| Compounds | $\Phi\Delta^1O_2$ | Ref. |
|---|---|---|
| Pyridoxine (PN) | 0.28 ± 0.04 | [26] |
| Pyridoxamine (PM) | 0.06 ± 0.02 | [26] |
| Pyridoxal (PL) | 0.44 ± 0.06 | [7] |
| Pyridoxal-P (PLP) | 0.56 ± 0.07 | [7] |
| Pyridoxine-P (PNP) | 0.54 ± 0.07 | [7] |
| Pyridoxyl-P-valine | 0.31 ± 0.05 | [7] |
| Pyridoxyl-P-histidine | 0.17 ± 0.04 | [7] |
| Pyridoxyl-P-valine (Schiff base) | 0.13 ± 0.03 | [7] |
| Pyridoxyl-P-lysine (Schiff base) | 0.17 ± 0.04 | [7] |
| Pyridoxyl-P-apohemoglobulin | 0.25 ± 0.05 | [7] |
| Pyridoxyl-P-serum albumin | 0.22 ± 0.05 | [7] |

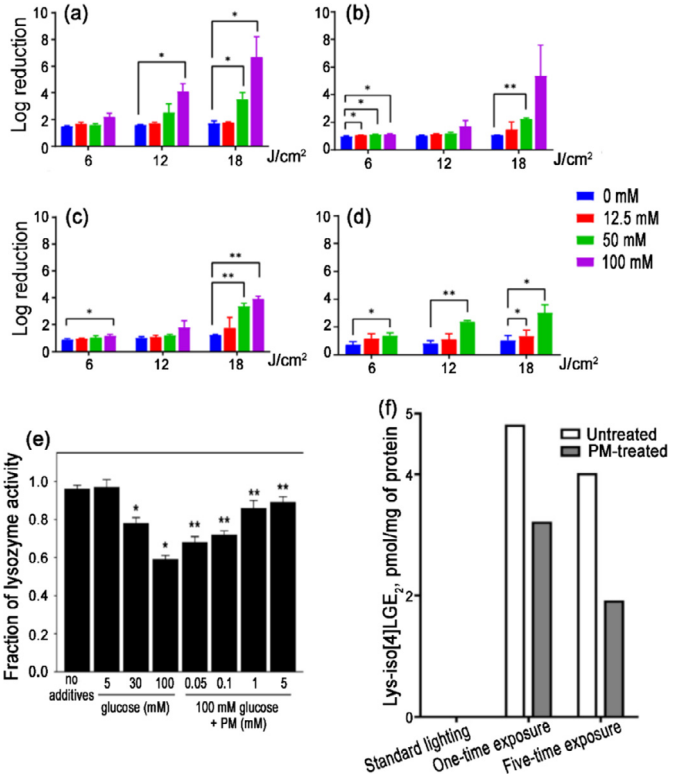



**Fig. 2.** Photodamage and photoprotection in *in vitro* and *in vivo* studies on B6 vitamers. Bacterial effects of UVA-induced B6 vitamin in PAS-diluted plasma on (a) E. coli, (b) K. pneumoniae, (c) B. cereus, and (d) S. aureus. Reprinted with permission from [25]. (e) Inhibition of lysozyme activity by glucose and protection by PM at pH 7.5 after 15 days of incubation at 37°C. *p<0.05, glucose vs control; **p<0.05, glucose/PM vs glucose. Reprinted with permission from 26. (f) Levels of isoLG-protein adducts in retina of mice kept in dim lighting conditions or following one-time or five-time exposures to bright light (10.000 lux / 2 h). Reprinted from [33] with permission.

Wang and coworkers explored PM's effects against UVC-induced death of human keratinocyte cells (HaCaT) using light doses of 20 and 40 J $cm^{-2}$. In different PM concentrations, three effects were observed: abrogation of the phototoxicity under UVC, reversion of apoptosis with both doses, and decreased ROS production. This demonstrated the potential use of PM as an antioxidant agent.[35]

The effects of B6 vitamers *in vivo* have also been studied. Charvet and coworkers designed a mouse retina model to study PM's effects on diabetic retinopathy. IsoLG-protein adducts participated in inflammatory and hypertensive biochemical processes, and PM attenuated isoLG changes of retinal proteins by up to two-fold under bright light irradiation (10000 lux, 2 h). This reduction protected mitochondria from light exposure due to PM's scavenging of lipid peroxide (Fig. 2F). [36] Other *in vivo* studies have corroborated the hypothesis that PM restrains the generation of advanced glycation end-products and lipoxidation end-products for diabetics.[37,38]

## Conclusion

This minireview showed how the B6 vitamin family can act as phototoxic and quenching species against several ROS. Under ultraviolet light, these vitamers generate photodamage in microorganisms and cell lines and cause the photoinactivation of specific sites of various enzymes. The mechanisms involved with high $\Phi\Delta^1O_2$, significant generation of hydrogen peroxide by B6 vitamers (except PM), and toxic photoproducts play an important role in both photodamage and photoinactivation. These phototoxic species contribute to the photoinactivation of specific enzymatic sites (e.g., lysine, histidine, and their surroundings) due to the photo-triggered formation of adducts. The potential antioxidant character of B6 described in the literature involves both the quenching of ROS (e.g., hydroxyl radicals and superoxide anions) and the capturing of redox-metal ions that possibly generate these reactive species. Moreover, it is noticeable that, compared to other vitamers, PM seems to be more effective as a fluorophore than as a photosensitizer. Thus, it could be used as a vitamin B6-based luminescent label for mapping tissues by fluorescence microscopy.

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# Update

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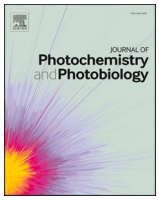

Erratum

# Erratum to: "B6 Vitamers as Generators and Scavengers of Reactive Oxygen Species" [Journal of Photochemistry and Photobiology]

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The authors regret that due to an inadvertent mistake declaration of competing interest was not published in this article, Which is as the following.

The authors would like to apologise for any inconvenience caused.

## Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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