Excited triplet state photophysics of the sulphonated aluminium phthalocyanines bound to human serum albumin

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Abstract

The binding of the sulphonated aluminium phthalocyanines to human serum albumin (HSA) in aqueous phosphate buffer solution at 25 °C has been studied by measuring the properties of the triplet excited states of these dyes. The triplet lifetimes were measured by triplet–triplet absorption flash photolysis. The triplet lifetime of the disulphonated AlS,Pc (2.5 µM) varies from 500 ± 30 ps in the absence of protein to 1100 ps and longer with HSA concentrations above 100 µM. Under identical conditions, the maximum triplet lifetimes of the mono-, tri- and tetrabsulphonated compounds bound to HSA are shorter than those for the disulphonated species. The increase in the triplet state lifetimes is attributed to the ability of the bulk aqueous phase to interact with the sensitizer at the site of binding; the site of binding being dependent on the degree of sulphonation. For AlS,Pc and Al,S,Pc at all HSA concentrations, and regardless of the degree of sulphonation, all the triplet state decay profiles follow simple pseudo-first-order kinetics. The exponential decay of the triplet phthalocyanine at all HSA concentrations is ascribed to the rapid association and dissociation of the phthalocyanine–HSA complex on the time-scales of the triplet state lifetimes. A simplified one-step binding model is utilized to describe the results. The association of Al,S,Pc with HSA results in substantial quenching of the triplet state quantum yield, and a more complex model is required to analyze the results. The tetrabsulphonated compound (Al,S,Pc) binds to the protein at a site where it experiences some protection from the aqueous phase. © 1997 Elsevier Science S.A.

Keywords: Phthalocyanine; Human serum albumin; Dynamic binding equilibrium; Photodynamic therapy

1. Introduction

The water-soluble sulphonated aluminium phthalocyanine dyes AlS,Pc (n = 1, 2, 3, 4) have attracted a considerable amount of attention as second-generation photosensitizers for the treatment of malignant tumours by photodynamic therapy (PDT) [1]. This treatment relies on the site-selective accumulation of the dye in neoplastic tissue, followed by laser irradiation to generate in situ cytotoxic species. The generation of the cytotoxic species—regardless of whether it is superoxide or singlet oxygen—is believed to be mediated by the excited triplet state via a Type I (electron transfer) or Type II (energy transfer) mechanism respectively [2].

In vivo, the localization of the sensitizer at tumour tissue occurs via the blood plasma proteins which serve to transport many endogenous and exogenous molecules. Serum albumin is the major protein blood plasma protein, with a concentration of about 0.6 mM, and delivers bound drug to the vascular stroma [3]. The lipoproteins—especially low density lipoprotein (LDL)—effectively internalize photosensitizers in malignant cells. Tumour cells have been shown to exhibit an elevated concentration of LDL receptors on the cytoplasmic membrane, which selectively recognize the apoprotein B and E moieties of LDL [4]. The association of a photosensitizer with either albumin of LDL depends on the hydrophilic–hydrophobic nature of the dye. Numerous publications have shown that the LDL pathway is especially important for the hydrophobic photosensitizers, whereas the more hydrophilic sensitizers are bound progressively more to the albumin [3,5–7].

Previous studies on the binding equilibrium of the tetrapyrrolic photosensitizers have been undertaken with compounds of mixed or ill-defined compositions, using molar ratios of dye to protein of less than or equal to 1:1, and have identified one or two specific binding sites [8–10]. Because
Table 1
Fluorescence lifetimes and quantum yields for AIS_Pc (0.5 µM), and triplet state quantum yields and lifetimes for AIS_Pc (2.5 µM) in a variety of solvent and model systems [12,13]

<table>
<thead>
<tr>
<th>Solvent</th>
<th>τ (ns) ± τ (µs)</th>
<th>Φ2</th>
<th>Φ1</th>
</tr>
</thead>
<tbody>
<tr>
<td>H2O</td>
<td>5.0 ± 0.1</td>
<td>500 ± 30</td>
<td>0.40 ± 0.04</td>
</tr>
<tr>
<td>D2O</td>
<td>6.8 ± 0.1</td>
<td>1130 ± 30</td>
<td>0.34 ± 0.03</td>
</tr>
<tr>
<td>CH3OH</td>
<td>6.7 ± 0.2</td>
<td>670 ± 15</td>
<td>0.56 ± 0.03</td>
</tr>
<tr>
<td>AOT (w, 1.4)</td>
<td>6.2 ± 0.2b</td>
<td>1130 ± 25</td>
<td>-b</td>
</tr>
<tr>
<td>AOT (w, 30)</td>
<td>5.7 ± 0.2b</td>
<td>640 ± 50</td>
<td>-b</td>
</tr>
<tr>
<td>Triton X100</td>
<td>6.2 ± 0.1c</td>
<td>640 ± 30</td>
<td>0.44 ± 0.04</td>
</tr>
</tbody>
</table>

*Non-exponential decay kinetics. Average lifetimes:* Not measured because of aggregation. *Not determined.

2. Experimental details

2.1. Procedures and materials

Di-, tri- and tetrasulphonated aluminium phthalocyanines that possess a hydroxyl axial ligand were prepared by the oleum sulphonation of chloro-aluminium phthalocyanine [14], and were separated into the pure di-, tri- and tetra-components using reverse-phase, medium pressure liquid chromatography (MPLC). Analytical high pressure liquid chromatography (HPLC) of selected compounds has shown that these compounds are single peaks [15]. Mass spectrometry (plus secondary ion mass spectrometry (SIMS)) has shown that the disulphonated component used in this study contains sulphonates with an adjacent configuration. AIS_Pc was purchased from Porphyrin Products. Essentially fatty-acid-free HSA was purchased from Sigma and used as received. All the phthalocyanine solutions were prepared in 0.1 M phosphate buffered saline (PBS) at pH 7.4 and allowed to equilibrate in the dark over a period of 4 h prior to the measurements. Absorption spectra were measured before and after all the quantum yield and lifetime measurements, to ensure that photodegradation of the sensitizers had not occurred. All the samples were thoroughly oxygen outgassed in the dark prior to lifetime and quantum yield measurements, by continuous bubbling with argon or N2.

2.2. Triplet state measurements

Triplet state lifetime and quantum yield measurements were made using a laser flash photolysis apparatus which allows the simultaneous capture of the transient absorption spectrum (λ = 200–800 nm) and the growth decay kinetics at a single wavelength. The system is reported in detail elsewhere [16]. Briefly, the system uses an excimer-pumped dye laser to provide 0.05–0.5 mJ pulses of 10 ns duration as the pump and the output of a 100 W tungsten–halogen lamp as the probe. The growth–decay kinetics were measured at a single wavelength using a monochromator together with a photomultiplier (Thorn EMI 9698QB) or a photodiode detector. The intensity of the transients collected was determined using a Tetronix 2432A digital storage scope. Time–gated absorption spectra were recorded using an intensified diode array (EG and G PAR 1420) and spectrograph that provided a 500 nm spectral window and with a resolution of 5 nm. The ability to gate the diode array detector at various time delays after the laser pulse allowed full UV–vis study of the triplet state, and allowed the formation and decay of any intermediates to be observed as a function of time. The aim of this paper is to provide a quantitative rationale for the observed exponential triplet state decay kinetics of the monounsulphonated phthalocyanines in terms of a dynamic equilibrium between the site(s) of phthalocyanine–protein association and the unbound form.
3. Results

3.1. Spectra

The absorption spectra of the mono-, di- and trisulphonated aluminium phthalocyanines in solution with HSA are characteristic of the monomeric form at all the protein concentrations used. The absorption spectra of the tetrasulphonated compound, which indicates the significant presence (λmax = 646 nm) of the dimeric species in a homogeneous solution, exhibits some disaggregation on the addition of HSA. For all four compounds, the addition of HSA induces a bathochromic shift of 2-10 nm in the Q band absorption spectra, which is attributed to the overall lower dielectric constant of the protein environment.

The transient difference spectra of Al&PC (n = 1, 2, 3) in the presence of HSA closely resemble those obtained in a homogeneous solution. The triplet–triplet extinction coefficient (ετ = 3800 ± 400 mol⁻¹ dm³ cm⁻¹) measured at 490 nm (using the singlet depletion method) appears to be unaffected by the presence of the protein. As a result of the presence of aggregates, the triplet–triplet extinction coefficient for AlS₄Pc was not determined. Fig. 1 shows the transient difference spectrum of AlS₄Pc (2.5 mM) in the presence of HSA (50 μM), together with the Soret and Q band ground state bleaches.

The triplet state quantum yields (Φₜ), together with the fluorescence quantum yields (Φf), average fluorescence lifetimes (τf), triplet state quantum yields (Φₜ), and triplet state lifetimes (τₜ) of AlS₄Pc in PBS (pH 7.4) are listed in Table 3. The moderately large errors reported here for the quantum yields with HSA reflect the difficulties in determining excited state triplet quantum yields with light scattering protein solutions.

Fig. 2 illustrates the triplet state decay profiles of AlS₂Pc (2.5 μM) in PBS (curve A) and HSA (100 μM) (curve B) plotted on a logarithmic (difference absorbance) scale. The decay time of the AlS₂Pc (2.5 μM) triplet state increases rapidly from a value of τₜ = 500 ± 30 μs in PBS at pH 7.4 to a 'plateau' value of τₜ = 1100 ± 50 μs at the highest HSA (150 μM) concentration employed. At all the protein concentrations, the triplet state decay kinetics remain exponential. A similar triplet state lifetime is observed for this compound in the presence of 100 μM solution of LDL, i.e. τₜ,LDL = 1000 ± 50 μs.

The increase in triplet state lifetime (or the reduction in the rate of triplet state decay kobs) does not scale according to any linear function with the increase in protein concentration. Fig. 3 illustrates this increase in the triplet state lifetimes.

| Table 2 | Summary of the fluorescence quantum yields (Φf), average fluorescence lifetimes (τf), triplet state quantum yields (Φₜ), and triplet state lifetimes (τₜ) of AlS₄Pc in solution with HSA |
|---------|---------------------------------|-----------------|-----------------|-----------------|
|         | Φf                              | τf (ns)         | Φₜ              | τₜ (ns)         |
|          |                                |                 |                 |                 |
| AlS₄Pc  | 4.9 ± 0.2                       | 5.5 ± 0.2       | 5.5 ± 0.2       | 5.2 ± 0.2       |
| AlS₂Pc  | 760.0 ± 40                      | 1100 ± 50       | 850.0 ± 50      | 750.0 ± 50      |
| AlS₁Pc  | 0.25 ± 0.04                     | 0.40 ± 0.04     | 0.40 ± 0.04     | –               |
| AlS₀Pc  | 0.10 ± 0.03                     | 0.18 ± 0.03     | 0.18 ± 0.03     | –               |

3.2. Triplet state lifetimes

Fig. 2. Logarithmic plot of triplet state decay of AlS₂Pc (2.5 μM) in PBS (curve A) and HSA (100 μM) (curve B).
of AlS₄Pc measured at λ = 490 nm as a function of the protein concentration.

The triplet state lifetimes of the mono-, tri- and tetrasulphonated compounds also increase with increasing protein concentration and remain exponential. The triplet state lifetimes as a function of the protein concentration is not as significant when compared with the case of the disulphonated compound. Fluorescence lifetimes are included for comparison.

In solutions of HSA prepared in D₂O (pD = 7.8), the triplet lifetimes of AlS₄Pc (n = 1, 2, 3, 4), monitored at λ = 490 nm, are all independent of the degree of sulphonation and of the molar ratio of phthalocyanine to protein. The triplet state lifetime τ = 1.40 ± 0.2 ms is comparable with the triplet lifetimes observed for these compounds in homogeneous solutions of D₂O and the calculated limiting lifetime of AlS₄Pc bound to HSA (vide infra).

4. Discussion

4.1. Triplet state quantum yields

In general, the in vitro photodynamic efficacy of a photosensitizer (particularly via a singlet oxygen Type II mechanism [2]) is determined by its triplet state quantum yield and triplet state lifetime. The triplet state quantum yield Φₚ is determined by the relative competition between the rate of S₁ → T₁ intersystem crossing and the rates which result in a deactivation of the excited singlet state, i.e. S₁ → S₀ internal conversion; fluorescence; and bimolecular quenching. The sulphonated aluminium phthalocyanines show a marked propensity towards aggregation, with the tendency towards aggregation being the most significant for AlS₄Pc. The dimers and higher aggregates are non-fluorescent and deactivate exclusively via internal conversion. Hence, neither the triplet state of the aggregate nor triplet-triplet annihilation induced delayed fluorescence is observed.

In association with HSA, the triplet state quantum yields for AlS₄Pc and AlS₄Pc (Φₚ = 0.18 ± 0.03) do not appear to be affected by the presence of the protein. In aqueous solution, the triplet state quantum yield is 0.17 ± 0.03. In contrast, the association of AlS₄Pc with HSA has a deleterious effect on the triplet state quantum yield. A reduction in the fluorescence quantum yield Φᵢ is also observed. These fluorescence and triplet state quantum yields are summarized in Tables 2 and 3. Because the absorption spectra of AlS₄Pc in protein solutions do not indicate the presence of aggregates, it is proposed that AlS₄Pc binds at a site where quenching of the excited state occurs.

4.2. Triplet state lifetimes

To explain the increase in the triplet state lifetimes of the sulphonated aluminium phthalocyanines with increasing protein concentration, it is worth noting the factors which determine the overall rate of triplet state deactivation.

In homogeneous solution, the observed triplet state lifetimes of the sulphonated aluminium phthalocyanines are dependent on a number of factors: (1) the solvent; (2) the ground state concentration; (3) the triplet state concentration; and (4) the presence of quenchers.

The solvent effect on the rate of triplet state deactivation [13] has been studied extensively and can be rationalized in terms of the solvent (O–H) inducing a perturbation in the coupling between the excited triplet (T₁) state and the ground (S₀) potential energy surfaces. For example, the observed rate of triplet state deactivation of AlS₄Pc (2.5 μM) in H₂O is 2000 ± 130 s⁻¹. However, in non-hydroxyl solvents such as dimethyl sulphoxide (kₗₛ = 770 ± 40 s⁻¹) or in D₂O (kₗₛ = 890 ± 30 s⁻¹), this rate is reduced significantly. This effect is useful, because it provides a means to probe the nature of the microenvironment of AlS₄Pc in model and biological systems.

In addition to the solvent effect, the rate of disappearance of the triplet (T₁) state can be given by the general expression

$$\frac{-d[T_1]}{dt} = k_p[T_1] + k_i[T_1] + k_{TR}[T_1]^2 + k_{sq}[T_1][S_0] + \sum k_{qm}[T_1][M_i]$$  

in which the third and fourth terms on the right-hand side are triplet–triplet annihilation and self-quenching terms, and the fifth term represents bimolecular quenching by intrinsic and extrinsic quenchers (Mᵢ)—especially dioxygen. The rate of disappearance of the triplet state (T₁) is so dominated by radiationless processes (i.e. the second to fifth terms on the right-hand side of Eq. (1)) that phosphorescence is not observed.

In the absence of dioxygen and other extrinsic quenchers, ground and excited state phthalocyanine concentration studies have shown that the observed rate of decay of the triplet state is linearly dependent on the self-quenching (kₗₛ) and triplet–triplet annihilation (kₚₜ) pathways of deactivation, which both occur at a bimolecular rate of approximately 10⁹ mol⁻¹ dm³ s⁻¹. Under the present conditions of low laser power intensity, the ground state concentration exceeds the...
triplet state concentration and self-quenching dominates triplet–triplet annihilation. From the ground state concentration studies at low pump power, the hypothetical triplet state lifetime of AlS3Pc in H2O at infinite dilution (τ_{infty}), i.e., in the absence of self-quenching, would be extended from 500 ± 30 μs at a ground state concentration of 2.5 μM to 600 ± 40 μs. Similarly, for AlS2Pc in D2O or methanol as the solvent, the triplet state lifetime at infinite dilution would be 1590 ± 60 μs and 820 ± 40 μs respectively. (The triplet state lifetimes of 2.5 μM solutions in D2O and methanol are 1130 ± 30 μs and 670 ± 15 μs respectively.) For AlS1Pc (which possesses a chloride axial ligand and putatively undergoes a faster rate of intersystem crossing than does the compound that possesses a hydroxyl (O–H) axial ligand), the triplet state lifetime in the absence of self-quenching would be increased from 390 ± 10 μs to approximately 440 μs in H2O, and from 880 ± 40 μs to approximately 1160 μs in D2O.

Because the magnitude of the increase in the triplet state lifetimes of AlS3Pc on binding to HSA is significantly greater than the effect solely incurred by a diminution of the self-quenching, the possibility that the increase in the triplet state lifetimes is induced by photobleaching of the sensitizer can be eliminated. Therefore, it is concluded that the increase in the triplet state lifetimes of AlS3Pc and AlS2Pc (and, to a lesser extent (vide infra), for AlS1Pc and AlS0Pc) as a function of increasing protein concentration results from a progressive reduction in the exposure of the sensitizer to the bulk aqueous phase. A study of the excited state decay kinetics of AlS3Pc as a function of the concentration of co-solubilized water when encapsulated within the water pool core of reverse micelles mediated by the surfactant Aerosol OT dispersed in n-heptane supports this conclusion [12].

4.3. Exponential decay kinetics

The excited triplet state decay of the AlS3Pc–HSA system is well described by a single exponential function. In this respect, the kinetic behaviours of the triplet states of the protein-based phthalocyanines are very similar to the behaviours displayed by a number of porphyrins on binding to HSA [20], the phosphorescence of tryptophan residues in proteins [21] and polycyclic aromatic ligands bound to DNA [22]. The exponential triplet state decay kinetics of AlS3Pc as a function of the concentration of co-solubilized water when encapsulated within the water pool core of reverse micelles mediated by the surfactant Aerosol OT dispersed in n-heptane supports this conclusion [12].

In Fig. 4, the symbols [Pc*] and [Pc–HSA*] refer to the concentrations of unbound and protein-bound triplet state phthalocyanine at any time t. The rates of triplet state deactivation of the protein-bound and unbound phthalocyanine species are denoted by k_c and k_a respectively.

From micelle studies, the rate of dissociation (k^\text{diss}_c) in many cases is proportional to the solubility of the solute in the bulk aqueous phase [23]. The observation of exponential decay kinetics indicates that the rate of dissociation (k^\text{diss}_c) of the phthalocyanine–HSA complex competes with the unimolecular rate of decay k_a (which is of the order of 10^7 s^{-1}). In other words, there is a dynamic exchange between protein-bound and free dye molecules during the lifetime of the excited triplet state. If a dynamic equilibrium did not occur, then, at time t = 0, the fraction of free molecules decaying at a rate k_a would be equal to (1 + K^*[{\text{HSA}}])^{-1}; the fraction of bound molecules decaying with a rate k_c would be given by K^*[{\text{HSA}}]/(1 + K^*[{\text{HSA}}]); and the relative weights of the slow and fast components would change as the HSA concentration varied. The observed rate of decay would then be given by

\[ k_{\text{obs}} = \frac{k_a + K^*[{\text{HSA}}]k_c}{1 + K^*[{\text{HSA}}]} \quad \text{(2)} \]

However, an analysis of the triplet state lifetime dependence of AlS3Pc and AlS2Pc as a function of [HSA] can only be described by Eq. (2) at high protein concentrations when k_a < K^*[{\text{HSA}}]k_c.

Considering Fig. 4, the rate expressions for the free (Pc*) and bound (Pc–HSA*) forms are respectively as follows:

\[ \frac{d[Pc^*]}{dt} = k_a[Pc^*] - k^\text{diss}_c[Pc - HSA^*] + k^\text{ass}_c[Pc^*][HSA] \quad \text{(3)} \]

\[ \frac{d[Pc - HSA^*]}{dt} = k_a[Pc - HSA^*] + k^\text{diss}_c[Pc - HSA^*] - k^\text{diss}_c[Pc - HSA^*][HSA] \quad \text{(4)} \]

Eqs. (3) and (4) can be solved [17] under conditions where k_a < k_c, by assuming steady state conditions for [Pc*]. Under these conditions, Eq. (3) can be expressed as
The observed lifetime then depends primarily on the rate of dissociation of the phthalocyanine from the protein, i.e. \( k_{d} \), and on the rate constant for triplet state deactivation in the bound form, i.e. \( k_{c} \). We will assume the following: (a) the rate of triplet state decay of the unbound species is approximately the same as the rate observed in a homogeneous aqueous solution, i.e. \( k_{c} = \frac{2000 \pm 150}{s^{-1}} \); (b) the rate of decay of the species in the bound form (\( k_{c} \)) is slower than the rate observed at the highest protein concentration used, where the rate of change of the observed triplet state deactivation as a function of the protein concentration proves unsatisfactory. Therefore, it is proposed that AlS:\text{Pc} binds or partitions to a site (or sites) where quenching of the excited state occurs; hence, the measured triplet lifetimes originate predominantly from the unbound or unquenched bound forms. In addition, the mode by which AlS:\text{Pc} associates with the protein—whether it is via partitioning or binding—involves two separate thermodynamic processes [24]. Thus, under conditions where AlS:\text{Pc} partitions rather than binds with the protein, Eqs. 2 and 8 no longer apply.

5. Conclusions

Previous studies of solvent and model systems [11–14] have shown that the excited state decay kinetics of AlS:\text{Pc} are dependent on the ability of solvent \( \text{H}_{2}\text{O} \) or, in more general terms, hydroxyl groups to interact with the macrocycle. When AlS:\text{Pc} is encapsulated within the inner water pool of reverse micelles (mediated by the surfactant AOT dispersed in \( \text{n-heptane} \)), both the fluorescence and triplet state decay kinetics are non-exponential [12]. Within the inner water pool, there are two limiting \( \text{H}_{2}\text{O} \) types: \( \text{H}_{2}\text{O} \) molecules which preferentially solvate the surfactant head groups and ions, and "free" \( \text{H}_{2}\text{O} \) molecules the properties of which tend towards those of bulk water. In particular, the heterogeneity
in the triplet state decay kinetics (which changes as the concentration of water increases) arises from the inability of the AlS₂Pc to exchange between the different H₂O environments (at least) on the time-scale of the triplet state decay.

We have shown that the triplet state decay kinetics of AlS₃Pc (n = 2, 3), which are exponential at all the protein concentrations studied, may be rationalized in terms of a dynamic exchange between the protein-bound and free phthalocyanine photosensitizers on the time-scale of the triplet excited state. For AlS₃Pc, significant quenching of the excited state occurs and a simple one-step binding model cannot be used to interpret fully the triplet state decay kinetics as a function of the protein concentration. At all the protein concentrations, however, the triplet state decay kinetics appear exponential. It is proposed that the observed triplet state decay then originates from the unbound or unquenched bound form. These conclusions are consistent with results obtained from quenching and singlet state studies [13].

The amphiphilic AlS₂Pc and, to a lesser extent, AlS₃Pc are bound to HSA at sites which offer some protection from the aqueous phase. The magnitude of the increase in the triplet state lifetimes on binding to HSA indicates that the bound site(s) are protected from the bulk aqueous phase and/or that any surrounding H₂O molecules are preferentially involved in solvating the polar amino acids of the protein.

The increase in the triplet state lifetime of the hydrophilic AlS₃Pc—despite not being as significant as that for AlS₂Pc or AlS₄Pc—indicates that the protein matrix does attenuate the ability of the bound photosensitizer from interacting with the bulk aqueous phase.

The triplet state quantum yield and triplet-triplet extinction coefficient of AlS₃Pc (n = 1, 2, 3) are (within experimental error) identical to the values obtained in PBS. As a result of the presence of aggregates, these parameters could not be determined for AlS₄Pc.

The calculated triplet state association constants K⁺ are in reasonable agreement with the values determined from the changes in the absorption spectra where the ground state association constant (K) is approximately 10³ M⁻¹. The triplet state association constants K⁺ calculated in this study and the triplet state lifetimes in the 'bound' form for AlS₂Pc, AlS₃Pc and AlS₄Pc are listed in Table 4. These parameters could not be determined for AlS₄Pc (vide supra).

To conclude, the photophysical properties of the sulphonated aluminium phthalocyanines are perturbed by their association with HSA. When localized in a non-H₂O microenvironment, the triplet state lifetime of AlS₂Pc is significantly longer than it is in a bulk H₂O environment. Because the photodynamic activity of the sulphonated aluminium phthalocyanines is dependent on their triplet state quantum yield and lifetime, it is expected that the photochemotherapeutic efficacy of AlS₃Pc will be enhanced in vivo in non-H₂O environments.

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References


