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Effect of the structure and concentration of cyclodextrins in the quenching process of naproxen

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Abstract

The effect of the formation of the naproxen:cyclodextrin inclusion complex on the quenching of the drug by iodide has been studied. The results show that α-cyclodextrin (α-CD), β-cyclodextrin (β-CD), and hydroxypropyl-β-cyclodextrin (HP-β-CD) provide good protection for the drug against iodide quenching. The trend of protection is α-CD > HP-β-CD ≈ β-CD > H_2O, in contrast to the binding constant values and the structure of these complexes. The effect of these cyclodextrins on the quenching process is discussed on the basis of the parameters obtained by fitting the experimental data to the modified Stern–Volmer, and finite-sink approximation models.

Keywords: Quenching; Naproxen; α-Cyclodextrin; β-Cyclodextrin; Hydroxypropyl-β-cyclodextrin

1. Introduction

Over the past two decades fluorescence quenching has been widely studied because it is of considerable interest in the field of physical chemistry as well as in those of biochemistry and biophysics. Quenching is used in protein, membrane, and nucleic acid research. A new area of sensing technology for medical and industrial applications based on fluorescence and luminescence quenching is currently expanding at a huge rate.

The fluorescence quenching of organic molecules in solution by several quenchers has been studied in depth, using steady-state and transient methods [1–9].

Several models have been developed to fit quenching data with a view to understand the mechanisms involved, although it is not yet known what the best model is. Even the physical meaning of any quenching parameter included in these models has been questioned recently [5].

The effect of cyclodextrin complex formation on the quenching process of several compounds has also been studied [5,8,10–15]. Nevertheless, there is no clear evidence that can account for how cyclodextrin modifies the quenching process of the fluorophore.

In light of the above, here we were prompted to study the effect of the complexation of the anti-inflammatory drug naproxen with different cyclodextrins on the quenching by iodide.

The cyclodextrins studied were α-cyclodextrin (α-CD), β-cyclodextrin (β-CD) and hydroxypropyl-β-cyclodextrin (HP-β-CD). These cyclodextrins were chosen because they complex the drug in different ways and with different affinities [16–22]. In the case of α-CD, the drug is included through the methoxy group, the naphthalene ring remaining protruding into the aqueous media. In the cases of both β-CDs, the naphthalene ring is included inside the cyclodextrin cavity. The difference between these two cyclodextrins is that in the natural β-CD the carboxylic group of the naproxen is in contact with the aqueous media, whereas in the naproxen:HP-β-CD complex the carboxylic group of the drug is inside the hydroxypropyl groups of the cyclodextrin [17].

Therefore, these inclusion complexes provide different environments for the drug that must undoubtedly elicit different interactions with the molecules dissolved in the aqueous media.

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In addition, we studied the effect of the cyclodextrin concentration, independently of the complexing process, on the drug quenching.

The final objective of the present work was to determine the factors that play an important role in the quenching process. For this purpose, we undertook a steady-state and time-resolved fluorescence quenching study of naproxen complexed with α-, β-, and HP-β-CD in the presence of different concentrations of α- and HP-β-CD. The data were analyzed using modified Stern–Volmer and finite-sink approximation quenching models.

2. Material and methods

2.1. Materials

Naproxen, 2-(6-methoxy-α-methyl-2-naphthyl) propionic acid, sodium salt, was purchased from Sigma Chemical Co. α-CD, β-CD, and HP-β-CD, containing an average molar substitution of 0.8 hydroxypropyl groups per glucopyranose unit, was obtained from Sigma. Potassium iodide, for analysis, was purchased from Merck (Scheme 1).

These reagents were considered to be sufficiently well-characterized by the manufacturer for use without further purification. Water was treated with a Milli-Q system from Millipore.

2.2. Methods

The naproxen concentration was held constant at 4.0×10^{-5} M. The KI concentration was varied between 0 and 0.15 M.

All measurements were carried out at 25.0 °C and at least 24 h after sample preparation to ensure that equilibrium had been reached.

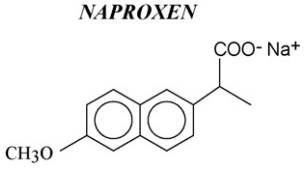
The cyclodextrin concentration was chosen in order to ensure that most of the drug would be complexed. On this basis, the concentration of each cyclodextrin was: 12×10^{-3} M for β-CD and HP-β-CD; and 100×10^{-3} M for α-CD.

2.3. Spectroscopy

UV–vis absorption spectra were recorded on a Hitachi UV–vis spectrophotometer, model 150-20. Steady-state fluorescence emission spectra were recorded on a Perkin–Elmer LS 50B spectrofluorimeter. The instrumental response at each wavelength was corrected by means of a curve provided by the apparatus. Emission spectra were obtained in the λ_{em} = 325–450 nm range, with excitation at λ_{exc} = 317.0 nm. The spectral slits used were 0 and 0 nm (this value corresponds to the minimum possible width, which remains constant for a given instrument and is lower than 2.5 nm). The fluorescence data were taken at the maximum emission wavelength, λ_{em} = 355 nm.

The fluorescence lifetimes of the excited state of naproxen were measured with a FL920 fluorescence lifetime spectrometer from Edinburgh Instruments using the time-correlated single-photon counting technique at 298 K. Fluorescence decays were obtained up to 10^4 counts in the peak and were analyzed by an iterative deconvolution procedure based on the Marquardt algorithm [23]. The goodness of the fit was measured by the magnitude χ^2 ($\chi^2 = \sum (F_i - f_i)^2$), where F_i is the value of the ith data point and f_i is the value obtained from the fit) and the shape of functions of the weighted residuals.

Samples were excited at 317 nm, using a nanosecond flash lamp filled with hydrogen. Emission was monitored at 372 nm. A scatter solution of aqueous Ludox (colloidal silica) was used to collect the lamp response function needed for recovery of the decay parameters.



Scheme 1. (*S*)-6-methoxy-α-methyl)-2-naphthaleneacetic acid, sodium salt.

3. Results and discussion

3.1. Steady-state and time-resolved study

The presence of iodide induces a decrease in the fluorescence intensity of naproxen, but did not result in any spectral shift at the quencher concentrations used.

The results were initially represented as Stern–Volmer plots (Fig. 1). In the solutions of naproxen with and without any CD the plot showed slight positive deviations. The phenomenon has been observed many times for fluorescence quenching by KI and other quenchers [2,5–12].

The percentage of complexed drug at the CD concentrations used are: 60, for α-, and 93% for β- and HP-β-CD, and hence in the α-CD system there was an important percentage of free drug, which would be quenched. In addition, the cavity size of this cyclodextrin is too small to include the whole of the naphthalene ring. Accordingly, in the naproxen:α-CD complex the drug must

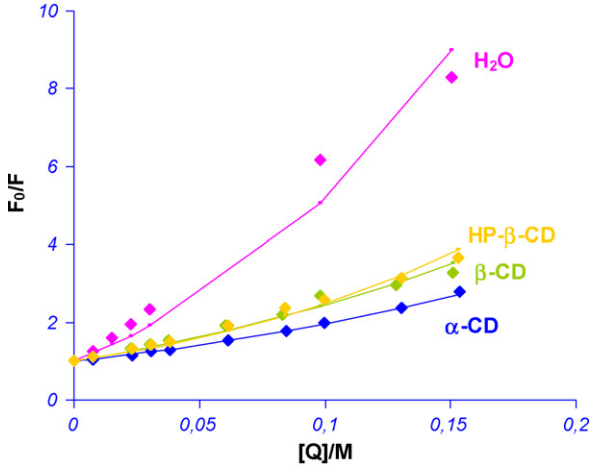


Fig. 1. Experimental quenching data (solid points) and fitting of these data to the modified Stern–Volmer, Eq. (4).

have the naphthalene ring protruding into the water. Under these conditions, drug quenching should be smaller than in water but greater than in the presence of both β-CDs, although it provided better protection for naproxen.

In order to gain further insight into the quenching process, the fluorescence decay curves of naproxen, free and complexed with these cyclodextrins, in the absence and the presence of 0.1 M of KI were measured. The data in Table 1 show that in the presence of the quencher the decays were bi-exponential, therefore in this case the average lifetime of naproxen was determined with the following equation:

$$\langle \tau_F \rangle = \sum_i A_i \tau_i \quad (1)$$

The bimolecular collisional quenching constant can be calculated from these data by using the expression:

$$k_q = (1/\tau_q - 1/\tau_0)/[Q] \quad (2)$$

where k_q is the bimolecular quenching rate constant, τ_0 is the lifetime of naproxen in the absence of the quencher, τ_q is the lifetime of naproxen in the presence of the quencher that in our case corresponds to τ_F, k_q is related to K_{SV} by means of:

$$K_{sv} = k_q^* \tau_0 \quad (3)$$

The values of K_{SV} obtained are shown in Table 1.

As can be seen, the lifetime and the K_{SV} of naproxen in the different media reveal that all the CDs afforded to the fluorophore good protection against collisional quenching by iodide. The trend in the collisional rate constant is α-CD ≈ HP-β-CD < β-CD « H_2O. It may be observed that the protection provided by α-CD remains being the highest. However, in this case, the behaviour of both β-CDs is clearly different.

The K_{SV} values for both drug:β-CD complexes may be interpreted on the basis of a physical protection of the drug against the collisional contact with the quencher, due to the inclusion of the fluorophore inside the CD. Moreover, the presence of the hydroxypropyl groups provides further protection to the naproxen. But, in the complex with α-CD, the naproxen is partially recovered by the cyclodextrin, the naphthlene ring being exposed to the aqueous media and therefore to the quencher, but it presents the lowest collisional rate constant.

Thus, α-CD and both β-CDs must act in different ways to protect naproxen against collisional quenching.

3.2. Static quenching study

Static quenching may be discussed using the ground-state complex [24,29] or the sphere of action static quenching models [30–32].

Ground-state complex formation was not detected in the absorption spectrum and hence, its formation is not clear.

In this situation, in most cases nonlinear quenching curves are analyzed using the Stern–Volmer modified equation to include static quenching [24–27,30,33–36].

$$F_0/F \ \ e^{(V[Q])} = 1 + K[Q] \quad (4)$$

where F_0 and F are the fluorescence intensities in the absence and presence of a quencher, respectively, and $[Q]$ is the quencher concentration. V and K are the static and collisional quenching constant, respectively.

V, as a floating parameter, was determined by a nonlinear regression method, using the S–V quenching constant values obtained from the time-resolved data K of the Table 1. A good fit to the experimental data with Eq. (4) was obtained for all systems (Fig. 1), resulting in the V, included in Table 1.

The V value obtained in the presence of β-CD is in good agreement with data previously published for naproxen complexed with β-CD and methylated-β-CD quenched by iodide and acrylamide [5].

The most general interpretation of V is that it represents an active volume element surrounding the excited fluorophore. Upon exciting the chromophore, a quencher molecule that is already present within the volume will be able to quench the fluorescence instantaneously. The probability of the quencher being in this volume at the time of excitation depends on the volume (V) and on the quencher concentration.

So, considering that the analytical quencher concentration was the same in all systems studied, the observed variations in V, must be related to different strength of quencher:naproxen interaction, or to a different concentration of the quencher in the vicinity of the drug as compared to that of the bulk solution.

Iodide, binds with α-, and β-CD with a binding constant of 12.4 and 18.0 M^{-1}, respectively [37], therefore at the concentra-

Table 1
Steady-state and time-resolved data for naproxen free and complexed with α-, β- and HP-β-CD in the absence and presence of 0.1 M KI. (K_{SV} is the collisional constant determined from time-resolved fluorescence data, respectively. V is the static quenching constant obtained from the fitting of steady-sate data to the modified Stern–Volmer equation. τ_F, is the calculated average lifetime of the drug in the presence of 0.1 M KI)

System	[KI] (M)	τ_1 (ns)	A_1	τ_2 (ns)	A_2	τ_F (ns)	K_{SV} (M^{-1})	V (M^{-1})
H_2O	0	9.28	1				24.1	4.35
	0.1	2.52	0.9542	6.80	0.0458	2.72		
α-CD	0	11.0	1				4.52	2.77
	0.1	9.44	0.6106	4.63	0.3894	7.57		
β-CD	0	10.6	1				8.84	2.66
	0.1	5.93	0.909	2.37	0.091	5.60		
HP-β-CD	0	10.5	1				4.32	5.52
	0.1	8.12	0.8224	3.66	0.1776	7.33		

tions used in this work, the free iodide able to quench the drug is 58 and 93% of the analytical iodide concentration, respectively.

So, considering that in the presence of β-CD, V is decreased by 42% from the value in water, while $[I^-]$ diminishes a 7% by complexation with the cyclodextrin, it is possible to infer that the naproxen:β-CD inclusion complex formation indeed protects the drug against the static quenching.

In the α-CD system, V is 64% lower than for the free naproxen, although the free iodide is 42% of the analytical concentration, pointing that α-CD complex formation also decreases the static quenching.

At last, in the case of the HP-β-CD complex, the V value is the same order of magnitude than for the free drug. No data about the binding of iodide and this cyclodextrin was found in the literature. Therefore, the static quenching may be increased or not as respect to the water, depending whether the iodide is or is not bound to the cyclodextrin.

So, after considering the effect of the iodide complexation with the cyclodextrins on the free concentration, it is confirmed that the static quenching constant value changes with a trend: HP-β-CD > H_2O > β-CD ≈ α-CD.

These changes show that in the complexes, the drug interacts with the quencher with different strength, or the quencher concentration in the vicinity of the complexed drug is different of that of the bulk solution.

3.3. Effect of the cyclodextrin concentration

Taking into account the iodide complex formation with the CDs, it is necessary to know the role of the CD concentration on the quenching process to rationalizate the parameters obtained. So, the same steady-state, time-resolved and static quenching study of the drug by iodide in the presence of 12, 40 and 100 mM of α-CD and HP-β-CD was carried out.

These two cyclodextrins were chosen because of their solubilities. Besides, in the case of α-CD, the amount of complexed drug increased with the CD concentration; while, at a 12 mM concentration of HP-β-CD most of the drug was included inside the CD cavity. Thus, the increase in the cyclodextrin concentration does not increase the amount of complexed drug. These two systems allow us to study the effect of complexation, and the concentration of CD in the quenching parameters.

At all CD concentrations studied, for both CDs the change in the fluorescence intensity with the iodide concentration was initially measured. The lifetime of naproxen at different α-CD and HP-β-CD concentrations was also measured in the absence and the presence of 0.1 M of KI and from these data the K_{SV} values were determined. All the time-resolved fluorescence data and the parameters obtained from them are included in Table 2.

As can be observed, in the absence of iodide in the system containing HP-β-CD, τ_0 remained constant as the cyclodextrin concentration increased, in good agreement with the fact that at the lowest CD concentration most of the drug was complexed. By contrast, the τ_0 of naproxen increased with the α-CD concentration, showing that complexing was taking place.

In addition, the average fluorescence lifetime in the presence of the iodide and the S–V constant (K_{SV}) of the drug complexed with HP-β-CD remained unmodified with the cyclodextrin concentration. Accordingly, it may be concluded that the physical presence of the cyclodextrin has no effect on the S–V constant value.

Despite the above, K_{SV} decreased and the naproxen lifetime increased with the α-CD concentration in the absence and in the presence of 0.1 M of iodide.

So taking altogether the results obtained for HP-β-CD and α-CD, it is possible to conclude that indeed the inclusion complex with α-CD affords protection to the drug against collisional quenching by iodide, and it is not an effect of the cyclodextrin concentration.

The fitting of the steady-state data for the different α-CD and HP-β-CD to the modified Stern–Volmer equation gives the V values included in Table 2. It can be observed that in both cases V decreases with the cyclodextrin concentration. Since in the case of HP-β-CD, K_{SV} did not change with the cyclodextrin concentration above that of the complexation, it can be concluded that the decrease in V must be due to the lowering in the effective quencher concentration. So, the data shows that iodide binds with HP-β-CD. If we consider the change in the V value

Table 2
Stern–Volmer constant for the quenching of naproxen, free and in the presence of different α-CD and HP-β-CD concentrations, by 0.1 M of iodide, determined from time-resolved (K_{SV}). (Fluorescence lifetime of the drug in the absence τ_0 and the presence τ_1 and τ_2 of 0.1 M KI and different amounts of cyclodextrins)

[α-CD] (mM)	τ_0 (ns)	τ_1 (ns)	A_1	τ_2 (ns)	A_2	τ_F (ns)	K_{SV} (M^{-1})	V (M^{-1})
0	9.28	6.8	0.0458	2.52	0.9542	2.72	24.1	4.35
12	9.49	7.01	0.2382	2.49	0.7618	3.56	10.6	7.47
40	10.1	7.81	0.4847	2.92	0.5153	5.29	5.70	6.69
70	10.4	8.32	0.5828	3.23	0.4172	6.19	4.37	4.32
100	11.0	9.45	0.6106	4.63	0.3894	7.57	3.24	3.88
[HPβCD] (mM)	τ_0 (ns)	τ_1 (ns)	A_1	τ_2 (ns)	A_2	τ_F (ns)	K_{SV} (M^{-1})	V (M^{-1})
0	9.28	6.8	0.0458	2.52	0.9542	2.72	24.1	4.35
12	10.5	8.12	0.8224	3.38	0.1716	7.33	4.32	5.52
40	10.9	7.66	0.8989	4.30	0.1011	7.23	5.04	4.15
70	10.8	8.22	0.8845	4.30	0.1155	7.76	3.91	1.92
100	10.8	8.51	0.8731	4.70	0.1269	8.02	3.45	1.55

as indicative of the extension of the binding, it is possible to observe that V decreases linearly with α-CD and HP-β-CD concentration the slope being 0.026 and 0.045, respectively. So in the less favourable situation the binding constant value of the iodide to HP-β-CD is the same order of magnitude than the β-CD, confirming that the static quenching increases when drug is complexed with the HP-β-CD.

3.4. Finite-sink approximation model

In order to obtain further information about the quenching process, the steady-state data were fitted to the finite-sink approximation model.

According to this model [38–41], the following modified S–V relationship is obtained:

$$K_{SV}^{-1} = (K_{SV}^{0})^{-1} - [(2\pi N)^{1/3}/4\pi N D\tau_0][Q]^{1/3} \quad (5)$$

where,

$$K_{SV}^{0} = 4\pi N D R \tau_0 K_a/(4\pi N D R + K_a) = 4\pi N D R' \tau_0 \quad (6)$$

with:

$$R' = R(1 + 4\pi N D R/K_a)^{-1} \quad (7)$$

where N, is Avogadro's number, D is the sum of the diffusion coefficients (mutual diffusion coefficient) of the reactants, and R is the distance at which the reaction proceeds, with intrinsic rate constant, K_a.

As can be observed, Fig. 2, a linear dependence of K_{SV}^{-1} on the one-third power of the quencher concentration within the error limits is confirmed. Thus, the values of D and R' can be obtained from the slope and the intercept of the plot, respectively, and are shown in Table 3. The mutual diffusion coefficients, for both the free drug and that complexed with β-CD, are lower than those previously reported [5].

But if we compare, the diffusion coefficient of the naproxen in the different systems, it can be observed that in the complexed forms it is lower than that of the free drug. Also the trend of the diffusion coefficient in the complexes is in good agreement with the increase in their size. So, the low value of the diffusion of the naproxen:α-CD complex is consistent with the partial inclusion of the drug in the cyclodextrin, while there is no difference between the diffusion of the drug complexed with both β-CDs. Hence, the method is good to determinate this parameters as pointed out previously by other authors [4].

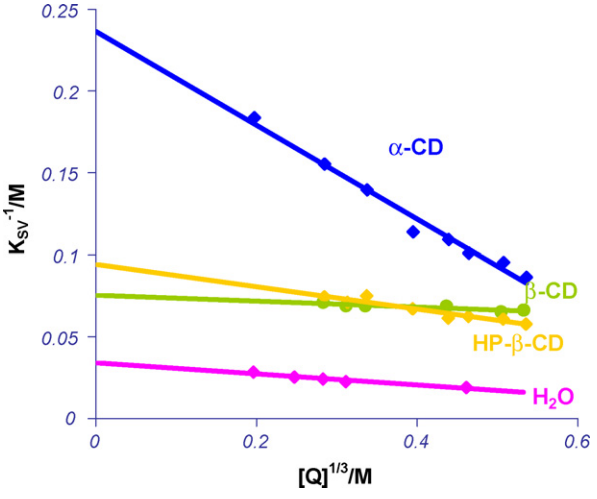


Fig. 2. Fitting of the experimental data (solid points) to the finite-sink approximation model, Eq. (5).

The lowest value of the diffusion of the naproxen:α-CD complex allows to justify the strong decrease in the collisional rate constant observed when the drug is complexed with this cyclodextrin. In addition, the decrease in the diffusion coefficient must also contribute to the decrease in the collisional rate constant value of the in both β-CDs complexes, provided that quenching process is diffusion-controlled. But this parameter does not explain the different behaviour observed for both β-CD.

From these data, and if one assumes the "reactive distance", R, as being equal to the contact separation R_c, then K_a may be recovered from Eq. (7).

The encounter distance (R_c) is the sum of the radii of the fluorophore (R_Y) and the quencher molecule (R_Q). The radii of the fluorophore (R_Y) and the quencher molecule (R_Q) had been previously reported as 3.85 and 2.8 A, respectively [5]. The corresponding values for the naproxen:α-CD, naproxen:β-CD and naproxen:HP-β-CD complexes were calculated using Edward's theory [42] and were 7.03, 7.02 and, 8.014 A. From these data, the encounter distances R_c were obtained and used to determine the K_a values, both included in Table 3.

As can be observed, K_a decreased considerably when the drug was complexed with the cyclodextrins, in good agreement with the protection provided by the cyclodextrin to the quenching of the drug.

The low K_a in the complex naproxen:α-CD, shows clearly that this cyclodextrin decrease in an important way the efficiency of the quenching process, what besides the low diffusion coefficient explain the low collisional rate constant value.

For both β-CDs complexes, K_a are different, it was higher when drug is complexed with β-CD than in the HP-β-CD complex.

The lower K_a value for the complexed drug than for the free one, pointed out that the differences observed in the V value are not due only to a different ability in the drug:quencher interaction. Since, if at a distance, R_c, the interaction rate constant is K_a, it can be expected that its value decreases with the distance, then as K_a decreases V must also decrease except if the effective concentration is different.

So, the K_a value for naproxen complexed with β-CD is in good agreement with a decrease in the V value. By contrast, taken together the K_a and V values for α-CD and HP-β-CD complex, it is possible to infer that, in these cases, the quencher concentration around the fluorophore must be higher than in the bulk solvent. This fact, seems to indicate the possibility of a ternary complex formation drug:cyclodextrin:quencher.

According to Andre et al. [25] and Zeng and Durocher [4], if "r", determined from the sphere of action model, is higher than the encounter distances (R_c) the static effect will take place, irrespective of the formation of the ground-state complex, provided that the reactions are limited by diffusion.

Table 3
Diffusion coefficient and energy of activation rate constant obtained by the finite-sink approximation and diffusion rate constant of these systems

System	$D \times 10^8$ (dm^2/s)	$R' \times 10^9$ (dm)	R_c (A)	$K_a \times 10^{-9}$ (dm^3/s mol)	r (A)	$K_d \times 10^{-9}$ (dm^3/s mol)
H_2O	6.58	6.38	6.65	78.2	12.0	3.30
α-CD	0.65	7.79	9.83	1.86	10.3	0.49
β-CD	2.59	6.42	9.82	3.63	10.2	1.92
HP-β-CD	2.87	4.66	10.81	1.78	13.0	2.35

Therefore, from the V value, the radius, "r", of the sphere of action for the naproxen-iodide in these systems was determined, considering that $V/N = 4\pi r^3/3$, where N is Avogadro's number. The results are shown in Table 3. $r > R_c$, in water and in the HP-β-CD, whereas in the complexes of α- and β-CD, $r \approx R_c$.

The diffusion rate constants of the drug-iodide couple when the drug was present in the different systems were determined from the following expression:

$$K_d = 4\pi N D R_c \quad (8)$$

Upon comparing K_d, Table 3, with K_a, it may be seen that the quenching process, was diffusion-controlled for the free drug and when it is complexed with α-CD and β-CD, however, for naproxen:HP-β-CD the quenching was diffusion-influenced. Therefore, in water as $r > R_c$ and diffusion-controlled the static effect takes place regardless of ground-state complex formation. But in the case of HP-β-CD the process is not diffusion-controlled so the static quenching does not take place independently of ground-state complex formation. So, this fact reinforces the idea of a ground-state naproxen:HP-β-CD:I^- inclusion complex.

4. Conclusions

The formation of naproxen:cyclodextrin complexes protect the drug against the quenching due to iodide; the trend of protection being: α-CD > HP-β-CD ≈ β-CD.

Cyclodextrins produce their protective effects in different ways. α-CD decreases the collisional rate constant by means of a decrease in the diffusion of the complexed drug and the decrease in the K_a value. In addition a decrease in the static quenching constant observed may be due to a lower drug-quencher interaction capacity, in the α-CD complex than in water.

β-CD and HP-β-CD provide the drug physical protection against quencher collision, the latter (HP-β-CD) being more effective in this protection due to the presence of hydroxypropyl groups. The formation of complexes with both cyclodextrins also decreases the drug-quencher interaction. In the case of the β-CD, a decrease in the static quenching rate constant is observed in comparison with the value in water. This seems to be due to the decrease in the drug-quencher interaction, as observed for the α-CD. However, in the case of HP-β-CD, V increases but K_a does not. This fact, points out that the increase in the static quenching is due to the existence of a higher local quencher concentration around the complexed fluorophore than in the bulk solution. In addition $r > R_c$ but the reaction is not diffusion-controlled. All the data suggests a ternary complex naproxen:HP-β-CD-iodide formation.

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