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New DNA-binding ruthenium(II) complexes as photoreagents for mononucleotides and DNA

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ABSTRACT: The spectroscopic properties of two photoprobes for DNA, $Ru(phen)_2(PHEHAT)^{2+}$ and $Ru(TAP)_2(PHEHAT)^{2+}$ (phen = 1,10-phenanthroline, TAP = 1,4,5,8-tetraazaphenanthrene, PHEHAT = 1,10-phenanthrolino[5,6-*b*]-1,4,5,8,9,12-hexaazatriphenylene), were examined and compared with those of complexes containing either an extended planar ligand (DPPZ) or π-acceptor ligands. The orbitals involved in the absorption and emission processes for $Ru(phen)_2(PHEHAT)^{2+}$ imply the PHEHAT ligand, whereas the chromophore and luminophore for $Ru(TAP)_2(PHEHAT)^{2+}$ are associated with the Ru(II) $\rightarrow$ TAP MLCT transition. The two complexes exhibit completely different behaviour in the presence of DNA. Whereas $Ru(phen)_2(PHEHAT)^{2+}$, which does not emit in water, luminesces upon intercalation between the DNA base pairs, the luminescence of $Ru(TAP)_2(PHEHAT)^{2+}$ is quenched by binding to DNA. Emission quenching is also observed in the presence of GMP, with a quenching rate constant of 1.25×10^9 l mol^{-1} s^{-1}. This strongly suggests the presence of a photo-induced electron transfer from the guanine residues of GMP or DNA to the excited complex and leads to the conclusion that this complex is a good DNA photoreagent. © 1998 John Wiley & Sons, Ltd.

KEYWORDS: DNA binding agents; ruthenium(II) complexes; photoreagents; DNA; mononucleotides

INTRODUCTION

The binding of ruthenium(II) complexes to nucleic acids has been extensively studied in the last 10 years.[1–3] The interesting absorption and emission properties of these compounds, easily tunable by varying the ligands, make them attractive candidates for probing nucleic acids.[4] We have been interested in the interactions and photoreactions of TAP (1,4,5,8-tetraazaphenanthrene) and HAT (1,4,5,8,9,12-hexaazatriphenylene) ruthenium(II) complexes with DNA[5,6]. The π-deficient character of these ligands makes the corresponding excited complexes more oxidizing than their 2,2′-bipyridyl (bpy) or 1,10-phenanthroline (phen) analogues. Different combinations of these ligands allow the preparation of a series of complexes whose excited state reduction potential $E^{\circ}_{Ru^{2+*/1+}}$ is anodically shifted by *ca* 700 mV compared with the reduction potential of $Ru(bpy)3^{2+*}$. It has been shown that the most oxidizing complexes are able to induce, under illumination, an electron transfer from the guanine of a mononucleotide or of the DNA double helix to the excited complex.[7,8] This photo-electron transfer process leads to two types of DNA reactions: single-strand cleavages and the formation of photoadducts between the complex and the DNA[7,9,10].

However, although these properties make these complexes attractive for different applications, in photochemotherapy, for example, their relatively weak binding constants to DNA represent limiting factors for their direct application in biological systems. One possibility for solving this problem consists in preparing complexes able to intercalate one of their ligands between the base pairs of the DNA double helix. This approach led to the preparation of an extended planar ligand, dipyrido [3,2a-2′,3′*c*]phenazine (DPPZ),[11] and the corresponding $Ru(bpy/phen)_2(DPPZ)^{2+}$ complexes.[12–17]. It has been demonstrated that these compounds have an excellent affinity for DNA, owing to the intercalative property of the DPPZ ligand. However, they are not sufficiently oxidant in their[3]MLCT excited states to induce an electron transfer in the presence of mono- or polynucleotides. Therefore, in order to combine, in the same complex, the intercalation property with the photooxidizing power, we have designed the PHEHAT (1,10-phenanthrolino [5,6-*b*]-1,4,5,8,9,12-hexaazatriphenylene) ligand,[18] where a hexaazatriphenylene is annelated to a phenanthroline motif.

In this work, we examined the photophysical properties of two complexes constructed with PHEHAT, i.e. $Ru(phen)_2(PHEHAT)^{2+}$ and $Ru(TAP)_2(PHEHAT)^{2+}$, in

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Table 1. Absorption data for the ruthenium(II) complexes

Complex	λ_{max} (nm)			
	H_2O		CH_3CN	
$Ru(phen)3^{2+}$[a]		421, 423	262	446
$Ru(phen)_2(DPPZ)^{2+}$[b]	264, 278sh,[f] 318sh, 358sh, 372	440	264, 276sh, 316, 352, 360, 368	440
$Ru(phen)_2(HAT)^{2+}$[c]	262	430, 494sh	262	420, 480sh
$Ru(TAP)_2(phen)^{2+}$[d]	272	410, 466	272	412, 458
$Ru(TAP)_2(DPPZ)^{2+}$[e]	278, 366	412, 458	278, 362	412, 452
$Ru(phen)_2(PHEHAT)^{2+}$	264, 276sh, 312sh, 356, 374	440	264, 278sh, 312sh, 354sh, 370	438
$Ru(TAP)_2(PHEHAT)^{2+}$	278, 368	412, 460	276, 362	414, 450

[a] From Ref. 22.
[b] Our data. See also Ref. 15.
[c] Our data. See also Ref. 19.
[d] From Ref. 21.
[e] From Refs 23 and 25.
[f] sh = Shoulder.

the absence and presence of DNA. The properties were compared with those of complexes containing either an extended planar ligand (DPPZ) or π-acceptor ligands (TAP or HAT).

EXPERIMENTAL

Instrumentation. Absorption spectra were recorded on a Hewlett-Packard Model 8452A diode-array spectrometer and treated with a Macintosh computer. Emission spectra were recorded with a Shimadzu RF-5001 PC spectrometer equipped with a Hamamatsu R 928 photomultiplier tube.

Chemicals. High-purity reagents and solvents (analytical grade) were used without further purification. Water was purified with a Millipore Milli-Q system. Tris buffer [tris(hydroxymethyl)aminomethane] was purchased from Aldrich. Calf thymus DNA (CT-DNA, Pharmacia LKB Biotechnology) was dialysed extensively first against phosphate buffer and subsequently against water. The polynucleotide phosphate concentration was determined spectrophotometrically (for CT-DNA $\varepsilon_{260} = 6600$ l mol^{-1} cm^{-1} and for poly(d[A-T])$_2$ $\varepsilon_{262} = 6600$ l mol^{-1} cm^{-1}). Guanosine-5′-monophosphate (Aldrich) was used without further purification as the sodium salt.

Synthesis of precursors. 1,10-Phenanthroline-5,6-dione,[12] 9,10-diamino-1,4,5,8-tetraazaphenanthrene[18] and bis(1,10-phenanthroline)dichlororuthenium(II)[18] were prepared as described previously.

Synthesis of polypyridyl ruthenium(II) complexes. The syntheses of $Ru(phen)2(HAT)^{2+}$,[19] $Ru(phen)_2(DPPZ)^{2+}$,[5] $Ru(TAP)_3^{2+}$,[20] $Ru(TAP)_2(phen)^{2+}$ [21] and $Ru(phen)_2(PHEHAT)^{2+}$ [18] have already been described. The synthesis of $Ru(TAP)_2(PHEHAT)^{2+}$ was carried out from 1,10-phenanthroline-5,6-dione, 9,10-diamino-1,4,5,8-tetraazaphenanthrene and bis(1,4,5,8-tetraazaphenanthrene)dichlororuthenium(II); the procedure is similar to that already described.[18] MS (ESMS, CH_3CN, $M_W = 1139.7$): $m/z = 994.8$ (14%, $M - PF_{6^-}$), 424.9 (100%, $[M - 2(PF_6^-)]$. ^1H NMR (250 MHz, CD_3CN)(H^T refers to protons on 1,4,5,8-tetraazaphenanthrene and H^{PH} refers to protons on 1,10-phenanthrolino[5,6-*b*]-1,4,5,8,9,12-hexaazatriphenylene): 9.94 (2H, dd, H^{PH}_{γ}, $J_{\alpha\ \gamma} = 1.2$ Hz), 9.35 (4H, s, $H^{PH}_{\delta,\ \varepsilon}$), 9.0 (2H, d, H^{T}_2, $J_{23} = 2.8$ Hz), 8.98 (2H, d, H^{T}_7, $J_{67} = 2.8$ Hz), 8.63 (4H, s, $H^{T}_{9,10}$), 8.32 (2H, d, H^{T}_3), 8.28 (2H, dd, H^{PH}_{α}, $J_{\alpha\ \beta} = 5.3$ Hz), 8.26 (2H, d, H^{T}_6), 7.95 (2H, dd, H^{PH}_{β}, $J_{\beta\ \gamma} = 8.2$ Hz). For the numbering of the different protons, see Fig. 1. The different protons were assigned from a ^1H– ^1H COSY spectrum.

RESULTS AND DISCUSSION

Photophysics in solution

Absorption. Table 1 lists the absorption λ_{max} values for the PHEHAT complexes. For comparison, the data for $Ru(phen)_3^{2+}$,[22] $Ru(phen)_2(HAT)^{2+}$ and $Ru(TAP)_2(lig)^{2+}$ (with lig = bpy, phen or DPPZ)[21,23,24] are also included.

The analysis of the UV–visible absorption data for $Ru(phen)_2(PHEHAT)^{2+}$ and $Ru(TAP)_2(PHEHAT)^{2+}$ reveals two different behaviours. For $Ru(phen)_2(PHEHAT)^{2+}$, the MLCT band is not red shifted compared with $Ru(phen)_3^{2+}$, in contrast to expectation for a complex with a π-acceptor ligand, as observed with $Ru(phen)_2(HAT)^{2+}$. Hence the annelation of a HAT fragment to the phenanthroline moiety does not significantly change the MLCT band, and the chromophore Ru(II) $\rightarrow$ PHEHAT mainly involves the phen part of the ligand (Fig. 1).[18]

In contrast, for $Ru(TAP)_2(PHEHAT)^{2+}$, the comparison of the spectra with those of the other complexes

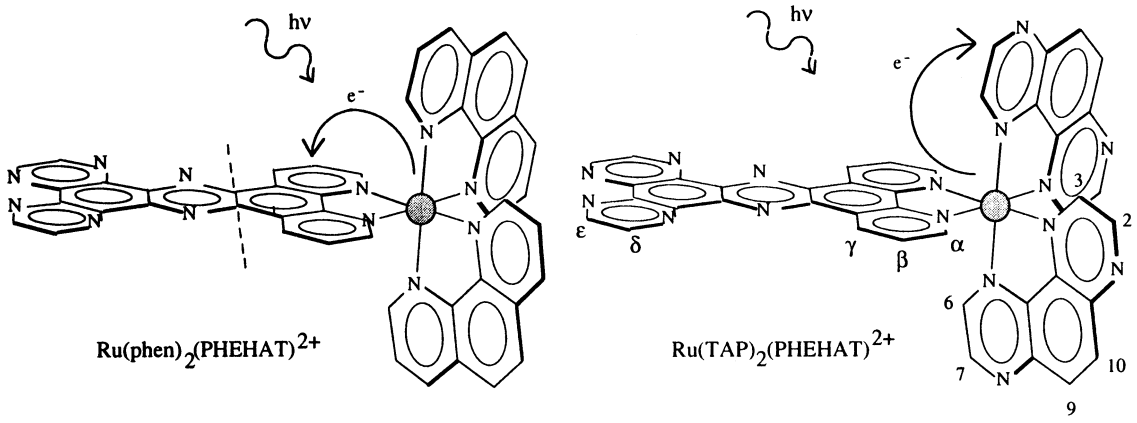

Figure 1. Lowest MLCT transition in absorption for $Ru(phen)_2(PHEHAT)^{2+}$ and $Ru(TAP)_2(PHEHAT)^{2+}$.

suggests that the lowest lying MLCT transition is not Ru(II) → PHEHAT but Ru(II) → TAP (Fig. 1). The λ_{max} values of the MLCT bands are very close to those obtained for $Ru(TAP)_2(phen/bpy)^{2+}$ and $Ru(TAP)_2(DPPZ)^{2+}$.[21,23,25]

Emission. $Ru(phen)_2(PHEHAT)^{2+}$ and $Ru(TAP)_2(PHEHAT)^{2+}$ exhibit at room temperature emission spectra without vibrational structures; however, $Ru(phen)_2(PHEHAT)^{2+}$ does not emit in water. The emission maxima recorded in water and acetonitrile are given in Table 2, along with the data for the reference compounds. Table 2 also includes the corresponding luminescence lifetimes in acetonitrile.

The comparison of the ^{3}MLCT emission maximum of $Ru(phen)_2(PHEHAT)^{2+}$ in acetonitrile with the values for the other complexes shows that the emission energy decreases in the order $Ru(phen)_3^{2+} > Ru(phen)_2(DPPZ)^{2+} > Ru(phen)_2(PHEHAT)^{2+} > Ru(phen)_2(HAT)^{2+}$. This suggests that the level of the π^* orbital involved in the emission process for $Ru(phen)_2(PHEHAT)^{2+}$ lies between the levels of the phen-type π^* orbital and the HAT-type π^* orbital, hence the transition involves the whole PHEHAT ligand.[18]

It is interesting that $Ru(phen)_2(PHEHAT)^{2+}$, similarly $Ru(bpy/phen)_2(DPPZ)^{2+}$,[11,14,15] does not luminesce in water. This behaviour can be attributed, as proposed for $Ru(bpy/phen)_2(DPPZ)^{2+}$, to the non-radiative rate constant which increases with increasing polarity of the medium[27] and with the ability of the medium to form hydrogen bonds with the phenazine nitrogens of the excited complex.[28]

In contrast, $Ru(TAP)_2(PHEHAT)^{2+}$ exhibits the same emission properties as $Ru(TAP)_2(lig)^{2+}$ (with lig = phen, bpy or DPPZ). Hence the lowest lying luminophore for these complexes corresponds to the Ru(II) → TAP ^{3}MLCT excited state. It can therefore be concluded that the absorption and emission involve the same orbitals. This conclusion allows the interpretation of the different behaviour for $Ru(phen)_2(PHEHAT)^{2+}$ and $Ru(TAP)_2(PHEHAT)^{2+}$. For the former, the luminophore involves the PHEHAT ligand and, therefore, the luminescence is completely quenched in water. In contrast, for the latter, PHEHAT is not involved in the lowest lying luminophore and the luminescence properties correspond to those observed for all the other TAP complexes.

Table 2. Emission data for the ruthenium(II) complexes

Complex	H_2O: λ_{max} (nm)[a]	CH_3CN λ_{max} (nm)[a]	τ_{air} (ns)[b]
$Ru(phen)_3^{2+}$ [c]	600	596	
$Ru(phen)_2(DPPZ)^{2+}$ [d]	–[h]	630	180
$Ru(phen)_2(HAT)^{2+}$ [e]	732	696	371
$Ru(TAP)_2(phen)^{2+}$ [f]	645	629	760
$Ru(TAP)_2(DPPZ)^{2+}$ [g]	636	621	
$Ru(phen)_2(PHEHAT)^{2+}$	–[h]	662	191
$Ru(TAP)_2(PHEHAT)^{2+}$	635	623	630

[a] Corrected λ_{max} of emission.
[b] The luminescence decays correspond to strict single exponentials. Experimental errors for the lifetimes are ±5%.
[c] From Ref. 26.
[d] Our data. See also Ref. 15.
[e] Our data. See also Ref. 19.
[f] From Ref. 21.
[g] From Refs. 23 and 25.
[h] The complex does not emit in water.

Ru(phen/TAP)$_2$(PHEHAT)$^{2+}$ and nucleic acids

Absorption. The absorption changes of $Ru(TAP)_2(PHEHAT)^{2+}$ (at constant concentrations of complex) upon addition of CT-DNA are shown in Fig. 2 and Table 3 for two different NaCl and Tris buffer concentrations.

Figure 3 illustrates the absorption change at a fixed wavelength for increasing concentrations of CT-DNA and Table 3 lists the percentage of hypochromicity observed for LC and MLCT absorption bands at P/Ru ([DNA as phosphate equivalent]/[complex]) = 50.

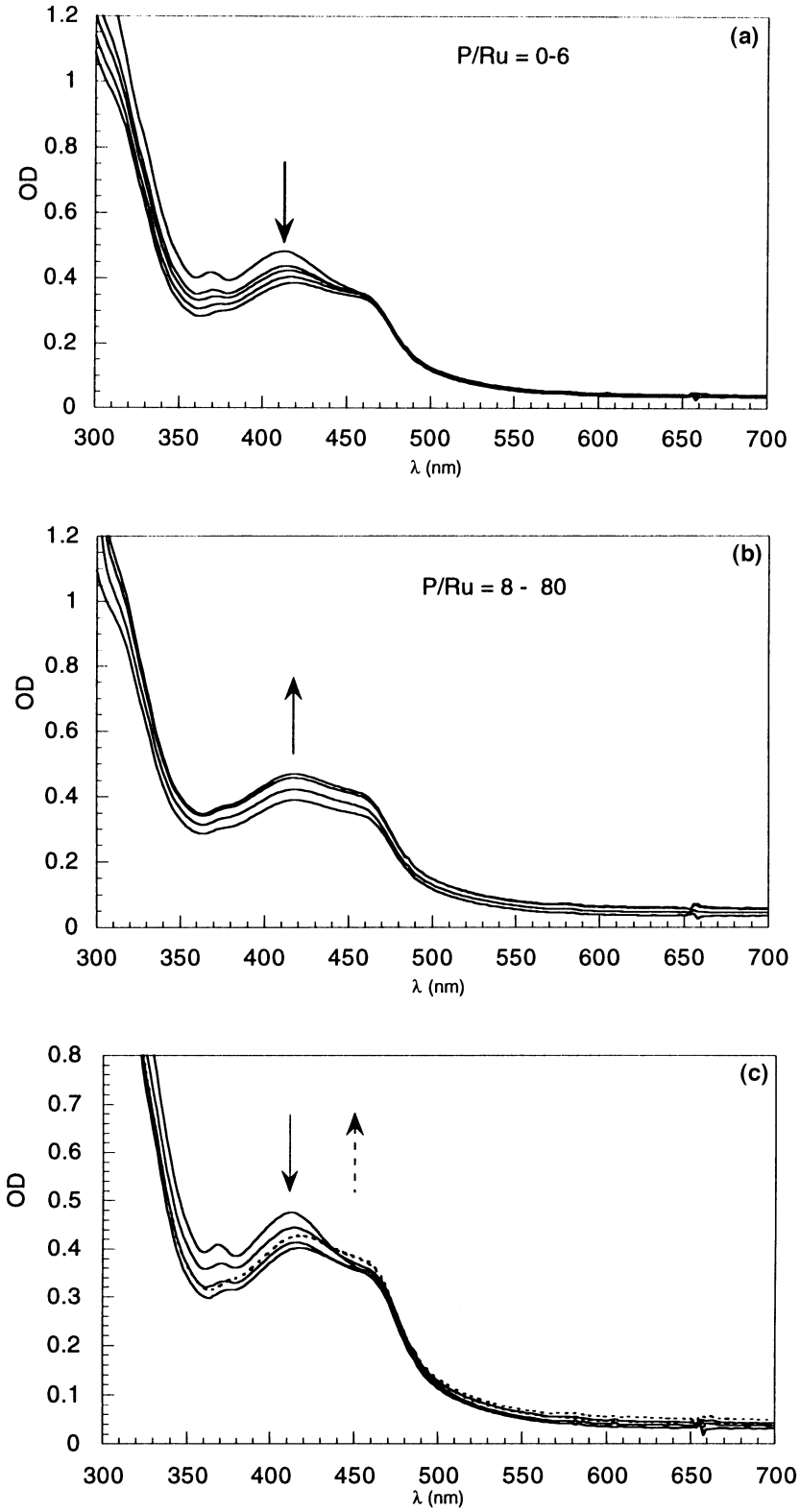

Figure 2. Absorption spectra of $[Ru(TAP)_2(PHEHAT)]Cl_2$ in the presence of calf thymus DNA ([complex] = 2×10^{-5} M). (a) and (b) [Tris buffer] = 10 mM; [NaCl] = 50 mM. The [DNA phosphate]/[complex] mixing ratios vary (a) (from top to bottom) from 0 to 6 and (b) (from bottom to top) from 8 to 80. (c) [Tris buffer] = 1 mM; [NaCl] = 10 mM. The [DNA phosphate]/[complex] mixing ratios vary (from top to bottom) from 0 to 6 (straight line) and (from bottom to top) from 8 to 80 (dotted line).

Table 3. Percentage of hypochromicity[a] observed for LC (PHEHAT) $\pi \rightarrow \pi^*$ and MLCT absorption bands for $Ru(phen)_2(PHEHAT)^{2+}$ and $Ru(TAP)_2(PHEHAT)^{2+}$

Transition involved (λ_{max})	$Ru(phen)_2(PHEHAT)^{2+}$: [NaCl] = 10 mM[b]	$Ru(TAP)_2(PHEHAT)^{2+}$	
		[NaCl] = 10 mM[b]	[NaCl] = 50 mM[c]
LC transition (368 nm)	31	24	28
MLCT Ru → PHEHAT (412 nm)	14	16	18
MLCT Ru → TAP (454 nm)	—	0	0

[a] The percentage is calculated at P/Ru ([DNA as phosphate equivalent]/[complex]) = 50, i.e. on the plateau of Fig. 4.
[b] 1 mM Tris buffer, pH 7.
[c] 10 mM Tris buffer, pH 7.

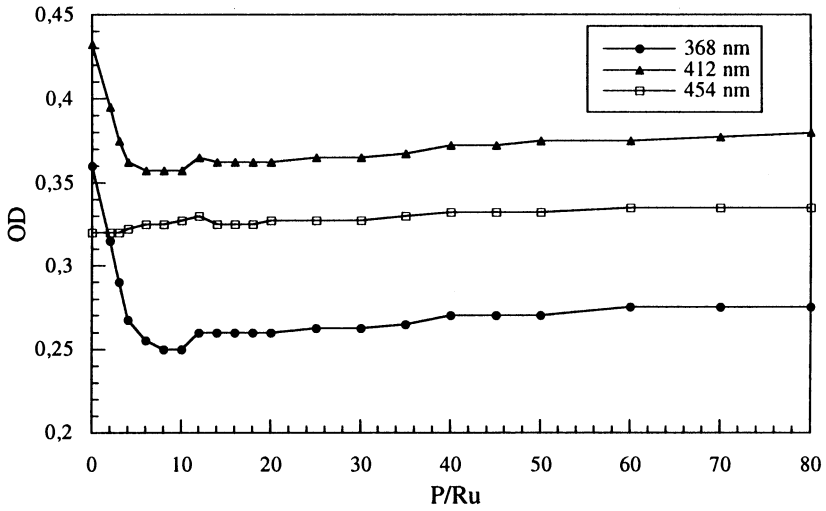

Figure 3. Absorbance of $[Ru(TAP)_2(PHEHAT)]Cl_2$ at constant concentration (2×10^{-5} M) in 10 mM NaCl and 1 mM Tris buffer, at 368, 412 and 454 nm, versus increasing [DNA phosphate]/[complex] ratio.

Increasing the DNA concentration, and thus the P/Ru ratio, results in two phases of events for these two complexes. The first corresponds to a hypochromic effect as the absorption decreases linearly when the concentration increase from zero up to P/Ru ≈ 7. The second phase corresponds to an absorption increase to a plateau value at P/Ru ≈ 20. These absorption changes obviously indicate the binding of $Ru(phen)_2(PHEHAT)^{2+}$ and $Ru(TAP)_2(PHEHAT)^{2+}$ to the DNA; the important hypochromic and the bathochromic effects would indicate intercalation of the PHEHAT ligand between the base pairs of the DNA double helix. The extra hypochromicity (P/Ru = 2–10) is attributed to closely bound metal complexes.[12] Interestingly, Table 3 suggests that the hypochromicity is the most important for the LC PHEHAT $\pi \rightarrow \pi^*$ transitions. This is in agreement with the intercalation of the ligand between the stacking of bases. A rather important hypochromicity, although less than the previous one, is also observed for the MLCT transition involving the phen part of the PHEHAT ligand. This is also in agreement with intercalative binding to DNA. In contrast, no hypochromicity is observed on the lowest energy MLCT band of $Ru(TAP)_2(PHEHAT)^{2+}$. This corroborates the assignment of this band to the Ru(II) → TAP MLCT transition. As this chromophore does not correspond to the intercalated part of the complex, this absorption band is unchanged on increasing the DNA concentration. Finally, it should be noted that the salt concentration does not seem to have an influence on the hypochromicity of the three absorption bands (the observed differences are not significant).

Emission. For many Ru(II) complexes, the interaction with a polynucleotide is accompanied by a luminescence intensity increase, as observed for $Ru(phen)_3^{2+}$, $Ru(phen/bpy)_2(TAP/HAT)^{2+}$ and $Ru(phen/bpy)_2(DPPZ)^{2+}$[14,15,29,30]. For these complexes, the lowest lying excited states are not sufficiently oxidizing to be quenched by abstraction of an electron from a nucleotidic base. As $Ru(phen)_2(PHEHAT)^{2+}$ is able to photooxidize the guanine of GMP,[18] its behaviour was examined with DNA. In that case, no photoinduced electron transfer was detected and the complex acts as a 'light-switch' for DNA. In contrast, $Ru(TAP)_2(PHEHAT)^{2+}$ exhibits a completely different behaviour (Fig. 4).

This complex emits in aqueous solution. Moreover, when its luminescence is measured at a constant complex concentration as a function of increasing amount of CT-

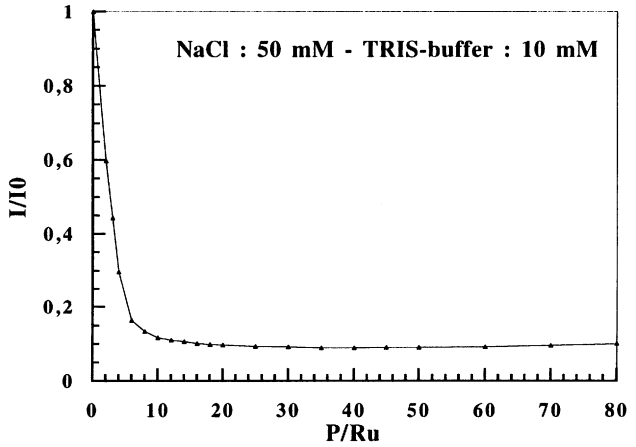

Figure 4. Emission of $[Ru(TAP)_2(PHEHAT)]Cl_2$ at constant concentration (2×10^{-5} M) in 10 mM Tris buffer and 50 mM NaCl, at 635 nm, versus increasing [DNA phosphate]/[complex] ratio.

DNA, instead of observing an emission increase, the ratio I/I_0 (I and I_0 = the intensity in the presence and absence of polynucleotide, respectively) decreases until a plateau value is reached. This luminescence quenching could be attributed to a photoinduced electron transfer from the guanine bases of the DNA to the excited complex. This hypothesis has already been confirmed for all the complexes containing at least two TAP ligands, whose luminescence is also quenched in the presence of DNA.[31]

The curve pattern is explained as follows: at low P/Ru ratios, the luminescence decreases linearly with increasing DNA concentration until P/Ru ≈ 6 (or three base pairs per ruthenium). Supplementary additions of DNA do not inhibit the luminescence further but modify the distribution of the complex on the double helix; this corresponds to the plateau situation. From this luminescence titration curve, the binding constant was calculated according to McGhee and Von Hippel's model.[32] The value obtained corresponds to 8.5×10^5 l mol^{-1} (with a site-size parameter of 2) in 50 mM NaCl and 10 mM Tris buffer solution, and is thus of the same order of magnitude as the value for $Ru(phen)_2(DPPZ)^{2+}$.[13]

In order to support the hypothesis of a photoreaction between the excited $Ru(TAP)_2(PHEHAT)^{2+}$ and the guanine bases of the DNA, luminescence quenching experiments were performed in the presence of guanosine-5′-monophosphate (GMP). Figure 5 shows the Stern–Volmer plot obtained from the luminescence intensity measurements as a function of increasing concentration of GMP in aqueous buffered solutions (0.1 M Tris buffer, pH 7).

The measurements were performed in the presence of a high buffer concentration in order to avoid variations of ionic strength with increasing nucleotide concentration. The quenching rate constant calculated from this plot (with $\tau_0 = 988$ ns in 0.1 M Tris buffer, pH 7) corresponds to 1.25×10^9 l mol^{-1} s^{-1} (error: 10%), and lies between the values obtained for $Ru(TAP)_2(phen)^{2+}$ (0.98×10^9 l mol^{-1} s^{-1}) and $Ru(TAP)_3^{2+}$ (2.2×10^9 l mol^{-1} s^{-1}.[7,31] $Ru(TAP)_2(PHEHAT)^{2+}$ thus behaves similarly to the other complexes containing at least two TAP ligands and acting as good photooxidizing agents.

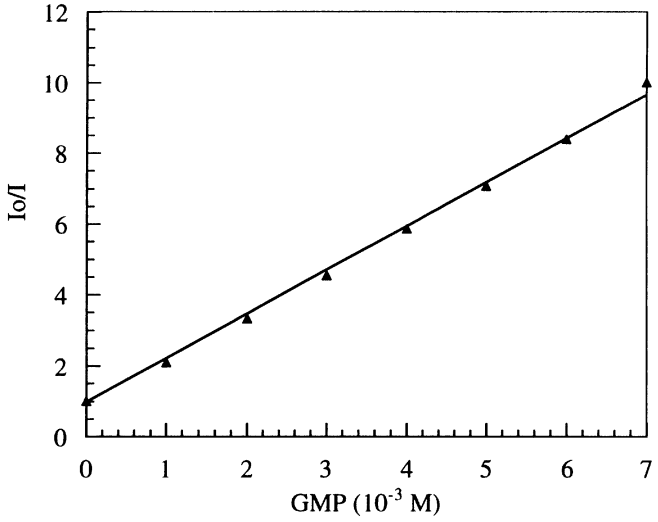

Figure 5. Stern–Volmer plot obtained from the luminescence intensities of $[Ru(TAP)_2(PHEHAT)]Cl_2$ in the presence of GMP, in water, with 0.1 M Tris buffer (pH 7).

CONCLUSION

$Ru(TAP)_2(PHEHAT)^{2+}$ is a very good candidate as a photoreagent for DNA. It combines the photooxidizing power of complexes able to induce a photo-electron transfer with a good interaction with DNA, owing to the intercalative property of the PHEHAT ligand. An extensive study of the photophysics and photochemistry of $Ru(TAP)_2(PHEHAT)^{2+}$ in the presence of DNA is in progress.

Acknowledgments

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REFERENCES

1. J. K. Barton. *Science* **233,** 727–734 (1986).
2. L. A. Basile and J. K. Barton. in *Metal Ions in Biological Systems*, edited by A. Sigel and H. Sigel, Vol. 33, pp. 269–296. Marcel Dekker, New York (1989).
3. B. Nordén, P. Lincoln, B. Akerman and E. Tuite. in *Metal Ions in*

Biological Systems, edited by A. Sigel and H. Sigel, Vol. 33, pp. 177–252. Marcel Dekker, New York (1996).
4. C. Moucheron, A. Kirsch-De Mesmaeker and J. M. Kelly. in *Structure and Bonding*, Vol. 92. Less common metals in proteins and nucleic acid probes, edited by J. P. Richmond. Springer, Heidelberg.
5. A. Kirsch-De Mesmaeker, J. P. Lecomte and J. M. Kelly. *Top. Curr. Chem.* **177,** 25–76 (1996).
6. C. Moucheron and A. Kirsch-De Mesmaeker. *J. Photchem. Photobiol. B* **40,** 21–106 (1997).
7. J. P. Lecomte, A. Kirsch-De Mesmaeker, J. M. Kelly, A. B. Tossi and H. Görner. *Photochem. Photobiol.* **55,** 681–689 (1992).
8. A. Kirsch-De Mesmaeker, G. Orellana, J. K. Barton and N. J. Turro. *Photochem. Photobiol.* **52,** 461–472 (1990).
9. J. M. Kelly, M. M. Feeney, A. B. Tossi, J. P. Lecomte and A. Kirsch-De Mesmaeker. *Anti-Cancer Drug Des.* **5,** 69–75 (1990).
10. M. M. Feeney, J. M. Kelly, A. B. Tossi, A. Kirsch-De Mesmaeker and J. P. Lecomte. *J. Photochem. Photobiol. B* **23,** 69–78 (1994).
11. J. C. Chambron, J. P. Sauvage, E. Amouyal and P. Koffi. *Nouv. J. Chim.* **9,** 527–529 (1985).
12. C. Hiort, P. Lincoln and B. Norden. *J. Am. Chem. Soc.* **115,** 3448–3454 (1993).
13. I. Haq, P. Lincoln, D. Suh, B. Norden, B. Z. Chowdhry and J. B. Chaires. *J. Am. Chem. Soc.* **117,** 4788–4796 (1995).
14. A. E. Friedman, J. C. Chambron, J. P. Sauvage, N. J. Turro and J. K. Barton. *J. Am. Chem. Soc.* **112,** 4960–4962 (1990).
15. R. M. Hartshorn and J. K. Barton. *J. Am. Chem. Soc.* **114,** 5919–5925 (1992).
16. P. Lincoln, A. Broo and B. Norden. *J. Am. Chem. Soc.* **118,** 2644–2653 (1996).
17. C. M. Dupureur and J. K. Barton. *J. Am. Chem. Soc.* **116,** 10286–10287 (1994).
18. C. Moucheron, A. Kirsch-De Mesmaeker and S. Choua. *Inorg. Chem.* **36,** 584–592 (1997).
19. L. Jacquet and A. Kirsch-De Mesmaeker. *J. Chem. Soc., Faraday Trans.* **88,** 2471–2480 (1992).
20. A. Kirsch-De Mesmaeker, R. Nasielski-Hinkens, D. Maetens, D. Pauwels and J. Nasielski. *Inorg. Chem.* **23,** 377–379 (1984).
21. J. P. Lecomte, A. Kirsch-De Mesmaeker, M. Demeunynck and J. L'homme. *J. Chem. Soc., Faraday Trans.* **89,** 3261–3269 (1993).
22. C.-T Lin, W. Böttcher, M. Chou, C. Creutz and N. Sutin. *J. Am. Chem. Soc.* **98,** 6536–6544 (1976).
23. I. Ortmans. PhD Thesis, Université Libre de Bruxelles, Brussels (1996).
24. A. Masschelein, A. Kirsch-De Mesmaeker, C. J. Willsher and F. Wilkinson. *J. Chem. Soc., Faraday Trans.* **87,** 259–267 (1991).
25. I. Ortmans, A. Kirsch-De Mesmaeker, J. C. Chambron and J. P. Sauvage. to be published.
26. L. Jacquet. PhD Thesis Université Libre de Bruxelles, Brussels (1990).
27. R. B. Nair, B. M. Cullum and C. J. Murphy. *Inorg. Chem.* **36,** 962–965 (1997).
28. C. Turro, S. H. Bossmann, Y. Jenkins, J. K. Barton and N. J. Turro. *J. Am. Chem. Soc.* **117,** 9026–9032 (1995).
29. F. de Buyl, A. Kirsch-De Mesmaeker, A. Tossi and J. M. Kelly. *J. Photochem. Photobiol. A* **60,** 27–45 (1991).
30. A. B. Tossi and J. M. Kelly. *Photochem. Photobiol.* **49,** 545–556 (1989).
31. J. P. Lecomte, A. Kirsch-De Mesmaeker, M. M. Feeney and J. M. Kelly. *Inorg. Chem.* **34,** 6481–6491 (1995).
32. J. D. McGhee and P. H. Von Hippel. *J. Mol. Biol.* **86,** 469–489 (1974).

