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Synthesis, DNA-binding and DNA-mediated luminescence quenching of Ru(II) polypyridine complexes

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Contents

Abstract

The aim of this paper is to report the recent laboratory work on the synthesis of novel Ru(II) polypyridine complexes and the studies of their interaction with DNA. The first part describes the syntheses and crystal structures of several novel polypyridine ligands containing

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imidazole or Schiff base moiety and their Ru(II) complexes. The second part focuses on the influences of the shape, size and substitute groups of intercalative ligands and co-ligands on the binding modes, intensities and enantioselectivities of the complexes to DNA. The interactions of Ru(II) complexes to different native DNAs are also compared. Certain regularity between the selection of a particular binding mode and the structure of complexes and/or DNA was observed. The final part of the paper reports the preliminary studies on a new DNA-mediated luminescence quenching assembly. The experimental results seem to support the notion that the π-stacked bases of DNA polymer maybe offer a pathway for electron transfer.

Keywords: Ru(II) complex; Polypyridine; DNA; Synthesis; Luminescence

1. Introduction

Considerable attention has been given to the design of small molecules that bind to DNA with site selectivity in order to develop novel therapeutics and chemical probes for nucleic acid sites and structure, as well as novel diagnostic agents targeted to double-helical DNA [1].

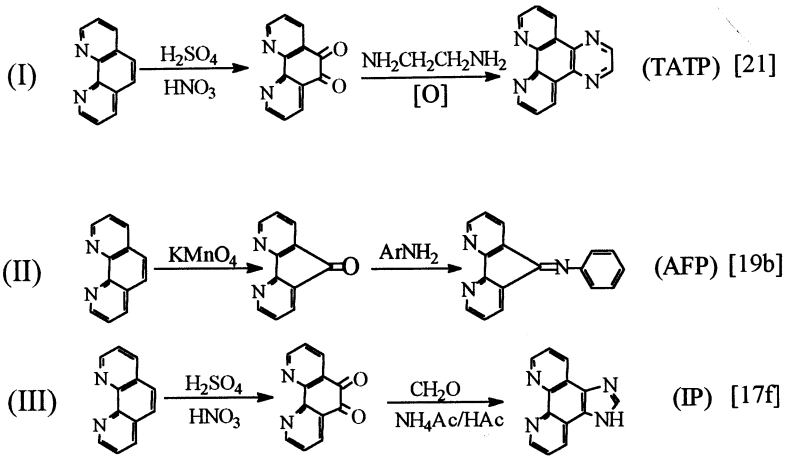
Ru(II) polypyridine complexes are ideally suited to application as sensitive non-covalent probes for polymer structure. The complexes are water-soluble, coordinatively saturated, and inert to substitution. Their metal-to-ligand charge transfer (MLCT) transition can be perturbed with binding to DNA, providing a sensitive spectroscopic probe to monitor their interactions with nucleic acids [1a]. Over the past decade, the interaction of this type of complexes with DNA has been extensively studied, and the applications of a number of such complexes have received increasing attention as DNA structure probes, DNA-dependent electron transfer (ET) probes, and DNA footprinting and sequence-specific cleaving agents, and others [2–13]. These studies have also made important contributions to the development of synthetic chemistry of Ru(II) polypyridine complexes [2a–d,i,h,3d,14,15]. However, unlike some other metal complexes that interact with DNA [16], no high resolution structural information precisely reflecting the binding nature of such complexes to nucleic acids has been reported yet. The exact association mechanism involving the prototype complex $[Ru(phen)_3]^{2+}$ remains an issue of vigorous debate [6a,b,7a,b].

The aim of this paper is not to make an exhaustive review of the work reported in the literature. Instead, we focus on the recent studies from our own laboratory on the DNA-binding behavior of new Ru(II) polypyridine complexes, most of which have either been reported or will be published soon [17–27].

2. Syntheses and structures of new polypyridine ligands and their Ru(II) complexes

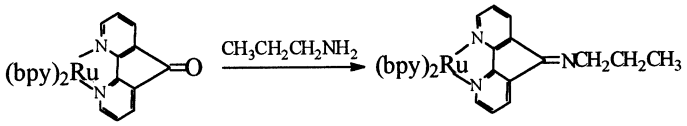
We have been interested in the design and synthesis of polypyridyl ligands with different size, geometry and electron-donor or -acceptor groups, primarily with the

aim of probing in detail the non-covalent binding nature of their octahedral Ru(II) complexes to DNA. The main synthetic routes are presented below using the synthesis of TATP, AFP and IP as examples:



Many derivatives of the above compounds were synthesized by the similar routes. Their structural formulas with abbreviations are shown at the end of this paper.

Most of the complexes derived from above ligands were synthesized by the reaction between $Ru(bpy)_2Cl_2$ or $Ru(phen)_2Cl_2$ and the respective ligand. Several complexes were however synthesized by the reaction of a mixed-ligand Ru(II) complex, i.e. a precursor, with appropriate reagents. For example [19f]:



The molecular structures of some of the ligands and their complexes were determined by X-ray single crystal diffraction. They provided valuable information about the molecular configuration and for the understanding of binding of the complexes to DNA.

The structure of AFP is presented in Fig. 1, [19c]. 4,5-Diazafluorenylidene and the phenyl ring are not coplanar, with a dihedral angle of 104.3°. Due to sp^2 hybridization of the nitrogen atom, the C=N–C is of a bent geometry with an angle of 121.5°. The structural features of AFP suggest that it would be very difficult for Ru(II)–AFP complexes to intercalate into DNA base pairs since an intercalative ligand generally requires not only an extended aromatic surface but also a special geometry that permits the overlapping between the intercalative ligand and the base pairs in DNA.

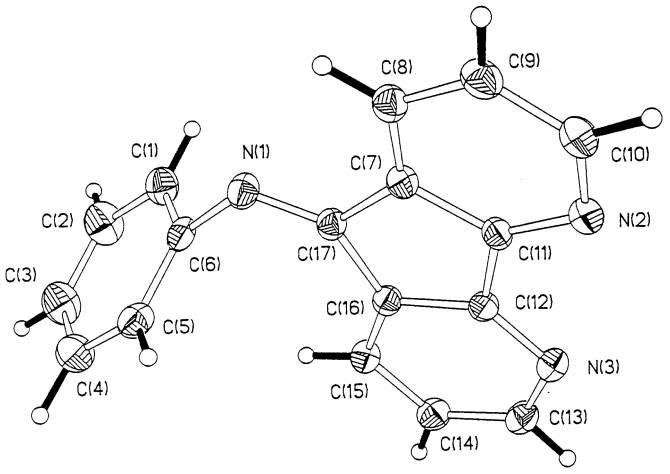


Fig. 1. Structure of AFP.

The ORTEP views of the $[Ru(bpy)_2(IP)]^{2+}$ [17f] and $[Ru(bpy)_2(MCP)]^{2+}$ [23b] cations are illustrated in Figs. 2 and 3, respectively. The IP ligand in the former is planar with an average deviation of 0.0340 Å from the least-squares plane. In the latter, the torsional angle between the IP moiety and 3-chlorophenyl group is only 5.3°, and all the atoms of MCP basically lie on a plane with an average deviation of 0.0504 Å from the least-squares plane. This is different from $[Ru(DIP)_3]^{2+}$, in which the phenyl groups are skew to the phen moiety [28]. The longest distance

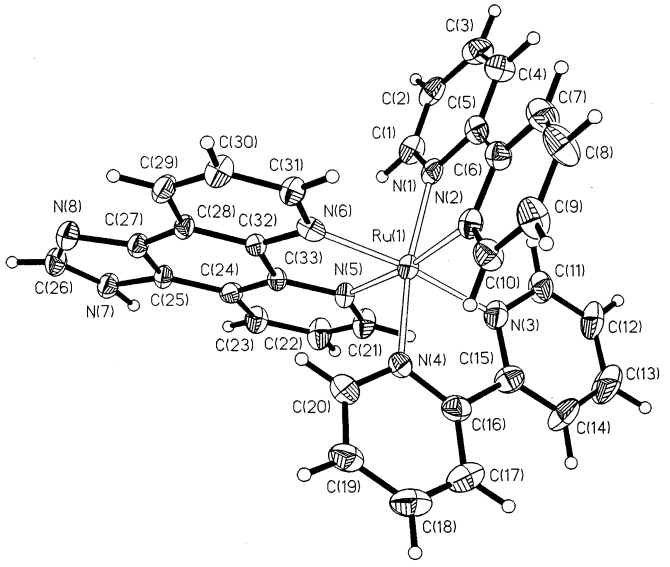


Fig. 2. An ORTEP view of $[Ru(bpy)_2(IP)]^{2+}$.

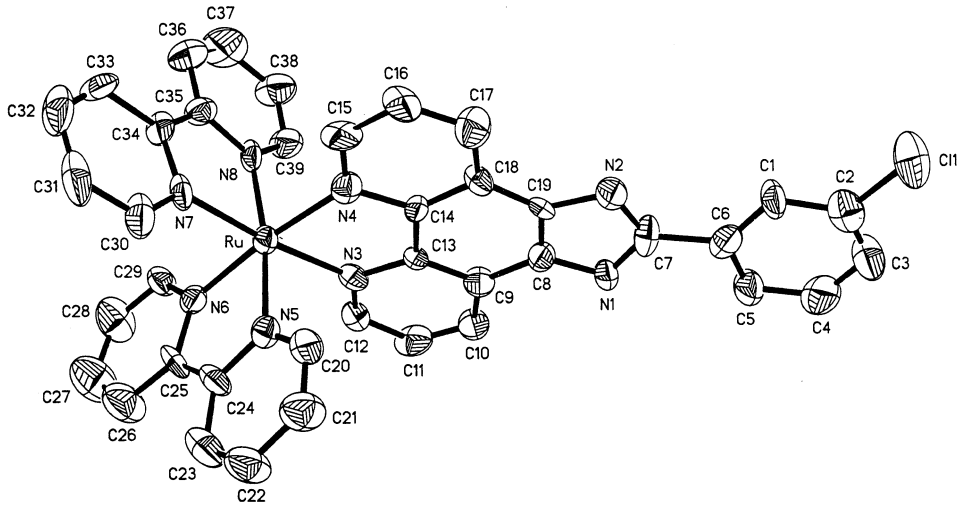


Fig. 3. An ORTEP view of $[Ru(bpy)_2(MCP)]^{2+}$.

from the edge of the MCP plane to Ru(II) center is ca. 11.49 Å, longer than that from the dppz edge to Re^{2+} center in $[Re(dppz)(CO)_3(py)]^{2+}$ (a known strong intercalative complex) (ca. 10.1 Å) [29]. To our knowledge, even though the crystal structures of polypyridyl Ru(II) complexes have been reported, the molecular structures of related complexes with such extended planar ligands have been rare in the literature. The stacking interaction between the IPs or MCPs is also observed in their crystals, respectively. Although the single crystals of $[Ru(bpy)_2(PIP)]^{2+}$ and $[Ru(bpy)_2(MOP)]^{2+}$ have not yet been obtained, the crystal structures of PIP and MOP are known [17d,g]. Their remote phenyl rings are almost coplanar with their IP moieties. These structure characters suggest that the PIP, MOP, MCP and IP of the complexes may intercalate DNA adjacent base pairs.

In addition, the crystal structures of $[Ru(dmp)_2(AFO)]^{2+}$ [19g], $[Ru(bpy)_2(AFO)]^{2+}$ [19g], $[Ru(bpy)_2(Me\text{-}phen)]^{2+}$ [22], $[Ru(phen)_2(bpy)]^{2+}$ [22] and $[Ru(bpy)_2(CIP)]^{2+}$ [23a] have also been determined. Their bond lengths and angles all fall within normal range.

3. Interaction of the complexes with DNA

3.1. Effect of ligand shape

Modification of the ligand may lead to subtle or substantial changes in the binding modes, location and affinities of the complexes to DNA. We have investigated the influence of ligand shapes on the binding mode of the complexes to DNA by gradually changing the type of heteroaromatic ligands [19]. Here, the series of complexes $[Ru(bpy)_2(AFPN_p)]^{2+}$, $[Ru(phen)_2(AFPN_p)]^{2+}$, $[Ru(bpy)_2(AFNP)]^{2+}$ and $[Ru(phen)_2(AFNP)]^{2+}$ are selected as examples [19e].

When $[Ru(phen)_2(AFPN_p)]^{2+}$ is mixed with calf thymus DNA(C.T.DNA) only very weak hypochromism and a small spectral shift can be observed, as shown in Table 1. The optical changes of the Ru(II) complexes upon binding to DNA are different from those observed for proven intercalators (for example, $[Ru(phen)_2(dppz)]^{2+}$ [7c]), but very similar to that of $[Ru(bpy)_3]^{2+}$. The absorption spectrum of $[Ru(bpy)_2(AFPN_p)]^{2+}$ was not affected by binding to DNA. But for $[Ru(phen)_2(AFNP)]^{2+}$ and $[Ru(bpy)_2(AFNP)]^{2+}$, we observed pronounced hypochromism and a large red shift. Because the magnitude of the hypochromism and red shift were found to correlate with the strength of the intercalative interaction [1a], we conclude that $[Ru(bpy)_2(AFPN_p)]^{2+}$ and $[Ru(phen)_2(AFPN_p)]^{2+}$ do not intercalate into the DNA base pairs while $[Ru(bpy)_2(AFNP)]^{2+}$ and $[Ru(phen)_2(AFNP)]^{2+}$ bind to DNA probably by intercalation.

For $[Ru(bpy)_2(AFPN_p)]^{2+}$, no CD spectral signal was observed. However CD spectra were developed for $[Ru(phen)_2(AFPN_p)]^{2+}$ and $[Ru(phen)_2(AFNP)]^{2+}$ after 48 h dialysis versus the DNA. The optical activity observed in the dialysate reflects an enrichment of the less favored enantiomer in the dialysate, suggesting that the $[Ru(phen)_2(AFPN_p)]^{2+}$ and $[Ru(phen)_2(AFNP)]^{2+}$ can bind stereoselectively to the DNA.

The effect of the complexes on the viscosity of DNA is shown in Fig. 4. Three different behaviors were observed in this experiment $[Ru(bpy)_2(AFPN_p)]^{2+}$ does not change the viscosity of DNA. The effect of $[Ru(phen)_2(AFPN_p)]^{2+}$ on DNA viscosity depends on the concentration of the complex. It decreases (increases) DNA viscosity at low (high) concentrations. The behavior of the complex is very much like that of Δ-$[Ru(phen)_3]^{2+}$ [2b]. Chaires et al. proposed that such a phenomenon could be considered as evidence for DNA kinking or bending by the compounds [7b]. For $[Ru(bpy)_2(AFNP)]^{2+}$ and $[Ru(phen)_2(AFNP)]^{2+}$, the viscosity of DNA is increased with the increase of the complex concentration, similar to those of known DNA intercalators (e.g. $[Ru(phen)_2(dppz)]^{2+}$, ethidium) [7b,c].

Table 1
The changes of UV–vis absorption of Ru(II) complexes containing various ligands upon binding to C.T.DNA

Complexes	Absorption λ_{max} (nm)			Hypochromism[c] (%)
	Free[a]	Bound[b]	$\Delta\lambda$	
$[Ru(bpy)_2(AFPN_p)]^{2+}$	446	446	0	0
$[Ru(phen)_2(AFPN_p)]^{2+}$	444	433	1	2
$[Ru(bpy)_2(AFNP)]^{2+}$	422	431	9	18
$[Ru(phen)_2(AFNP)]^{2+}$	417	428	11	21
$[Ru(bpy)_3]^{2+}$	452	452	0	0
$[Ru(phen)_2(dppz)]^{2+}$	437	440	3	8

[a] [Ru] = 10 μM.
[b] [DNA nucleotide]/[Ru] = 40.
[c] Hypochromism % = $(A_{Rn(II)complex} - A_{DNA+Rn(II)complex})/A_{Rn(II)complex}$.

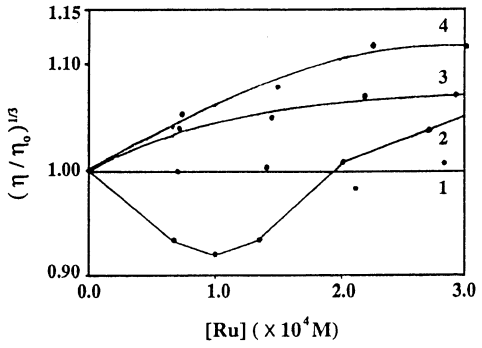


Fig. 4. Effect of the Ru(II) complexes on the viscosity of C.T.DNA. (1) $[Ru(bpy)_2(AFPN_p)]^{2+}$, (2) $[Ru(phen)_2(AFPN_p)]^{2+}$, (3) $[Ru(bpy)_2(AFNP)]^{2+}$, and (4) $[Ru(phen)_2(AFNP)]^{2+}$.

The experimental results suggest that there should be three types of binding modes for the interaction between the complexes and C.T.DNA, as discussed below:

1. Electrostatic binding mode: weak hypochromism, silent CD spectra, and viscosity measurement show that $[Ru(bpy)_2(AFPN_p)]^{2+}$ may bind the DNA by electrostatic force.
2. Groove binding mode: although $[Ru(phen)_2(AFPN_p)]^{2+}$ has weak hypochromism, it binds the DNA with some enantiomeric selectivity and can bend or kink the DNA. We infer that this complex binds to DNA neither by intercalation nor by electrostatic force. It is possible that the binding of the complex to the DNA could be described by groove binding mode. It is similar to the nonintercalative binding mode that has been used by Norden et al. to explain the experimental results of the interaction between $[Ru(phen)_3]^{2+}$ and the DNA [6b]. According to this binding mode, we could envisage that two phen of a $[Ru(phen)_2(AFPN_p)]^{2+}$ sit (not intercalate) in the minor groove of the DNA by hydrophobic interaction, while the Schiff base ligand points toward the outside.
3. Intercalative binding mode: $[Ru(bpy)_2(AFNP)]^{2+}$ and $[Ru(phen)_2(AFNP)]^{2+}$ show pronounced hypochromism and a large spectral red shift. Both complexes increase the relative viscosity of DNA in a manner consistent with the behavior expected from a classical intercalation. We conclude that the two complexes bind to DNA by intercalation.

The reason why the four complexes bind to DNA in different binding modes lies in the structures of the ligands. For $[Ru(bpy)_2(AFPN_p)]^{2+}$ and $[Ru(phen)_2(AFPN_p)]^{2+}$, although the crystal structure of $AFPN_p$ has not yet been obtained, we can speculate the role of $AFPN_p$ by referring to the structure of its analog, AFP, which shows that the nitrogen atom of C=N is sp^2 hybridized. As a result, this makes the phenyl ring incline to one-side of the ligand (see Fig. 1). In addition to the steric obstacle of the NH_2 group, the structure feature of Schiff base moiety makes their intercalation impossible [19c], which was also verified by other

analogues [19a,d,f]. Moreover, due to a larger hydrophobicity and the more extended range of π system of phen with respect to bpy, the latter seems to prefer the groove binding mode while the former prefers electrostatic binding. For $[Ru(bpy)_2(AFNP)]^{2+}$ and $[Ru(phen)_2(AFNP)]^{2+}$, the remote phenyl in the ligand, AFNP, can rotate to become co-planar with fluorenamine moiety, so that both complexes can intercalate into DNA by AFNP. As for the factors that contribute to stabilizing the metal complexes on the DNA helix, as proposed by Barton et al. [1a], the most significant factor is that of molecular shape.

3.2. Effect of intercalative ligand size

Among the discussed DNA-binding modes of Ru(II) complexes, the intercalative mode has attracted more attention since the established applications are all related to the binding mode [2,33]. DNA-intercalating Ru(II) complex generally have a larger planar ligand and two smaller ligands. The former is usually called an intercalative ligand, and the latter, co-ligands. In general, the extension of intercalative ligand planarity may increase the strength of interaction of the complex with DNA. In our laboratory, $[Ru(bpy)_2(IP)]^{2+}$ and $[Ru(bpy)_2(PIP)]^{2+}$ have been studied to illustrate this view [17f].

For $[Ru(bpy)_2(IP)]^{2+}$ and $[Ru(bpy)_2(PIP)]^{2+}$, the changes of their absorption spectra on binding to C.T.DNA are presented in Fig. 5. The intensities of the visible MLCT bands of $[Ru(bpy)_2(IP)]^{2+}$ and $[Ru(bpy)_2(PIP)]^{2+}$ are decreased by 15.5 and 21.9%, respectively. The extent of the hypochromism commonly parallels the intercalative binding strength [1a]. So the latter is more strongly intercalative to the DNA than the former. This result is expected on the basis that PIP possesses a larger planar area and a more extended π system than IP. Therefore, PIP would penetrate more deeply into, and stack more strongly with, the DNA base pairs. Similarly, the π–π* transition at about 280 nm of $[Ru(bpy)_2(IP)]^{2+}$ shows a slight hypochromicity (3.8%), whereas it is 20% for $[Ru(bpy)_2(PIP)]^{2+}$. The bands around 250 nm exhibit hyperchromicities of 11.5 and 28.0%, respectively. We infer that these MLCT or π–π* bands are predominantly based on bpy. The nonintercalation character of bpy leads to opposite changes, but the extent of hyperchromicity is consistent with the intercalative ability of IP and PIP.

Both complexes can emit luminescence in Tris buffer at ambient temperature in a manner similar to $[Ru(bpy)_3]^{2+}$, with maxima at 625 and 615 nm, respectively. Upon addition of DNA, enhancements in both integrated emission intensities and lifetimes of the complexes were observed and monitored by time-resolved technique at the respective emission maxima, as shown in Fig. 6. The extent of enhancement increases from $[Ru(bpy)_2(IP)]^{2+}$ to $[Ru(bpy)_2(PIP)]^{2+}$, consistent with the intercalation mode. Since PIP is expected to insert more deeply and strongly in DNA than IP, two factors are expected to be responsible for the observed changes of emission intensities and lifetime. First, the hydrophobic environment inside the DNA helix reduces the accessibility of water molecules to the complex; and secondly, the complex mobility is restricted at the binding site, leading to a decrease of the vibrational modes of relaxation.

Steady-state emission quenching experiments using $[Fe(CN)_6]^{4-}$ as the quencher, support the above proposal. As illustrated in Fig. 7, in the absence of DNA, $[Ru(bpy)_2(IP)]^{2+}$ and $[Ru(bpy)_2(PIP)]^{2+}$ were efficiently quenched by $[Fe(CN)_6]^{4-}$, resulting in two strictly linear Stern–Volmer plots. However, the presence of DNA results in drastically curved plots. This can be explained by the repulsion of the highly anionic species, $[Fe(CN)_6]^{4-}$, by the DNA polyanion which hinders quenching the emission of the bound complex [2b,c]. The slope can therefore be taken as a measurement of binding affinity, a larger value corresponding to poorer protection and lower binding. So $[Ru(bpy)_2(PIP)]^{2+}$ binds more tightly than $[Ru(bpy)_2(IP)]^{2+}$, consistent with a stronger penetrating ability of PIP than that of IP.

However, we also found that it was not necessarily true that the larger size of the intercalative ligands would lead to larger changes of photophysical properties of the complex on binding to DNA. Although the planar sizes of the intercalative ligands are in the order of dppn > dppz > TATP > phen, the hypochromicities of the complex MLCT transitions follow the order of $[Ru(bpy)_2(TATP)]^{2+}$ (12%) > $[Ru(bpy)_2(dppz)]^{2+}$ (6%) > $[Ru(bpy)_2(dppn)]^{2+}$ (5%) > $[Ru(bpy)_2(phen)]^{2+}$ (2%) [18f], in the presence of C.T.DNA. Barton et al. [2i] and Yam et al. [29] reported

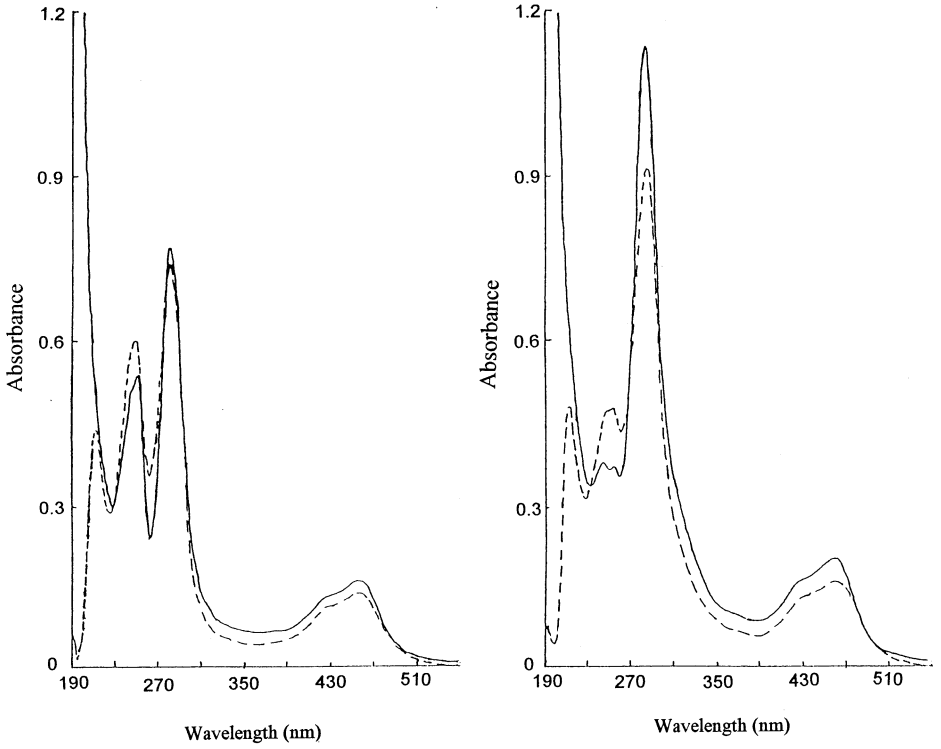


Fig. 5. Absorption spectra of $[Ru(bpy)_2(IP)]^{2+}$ (left) and $[Ru(bpy)_2(PIP)]^{2+}$ (right) with no C.T.DNA (—) and [DNA]:[Ru] = 26:1 (---). [Ru] = 10 μM.

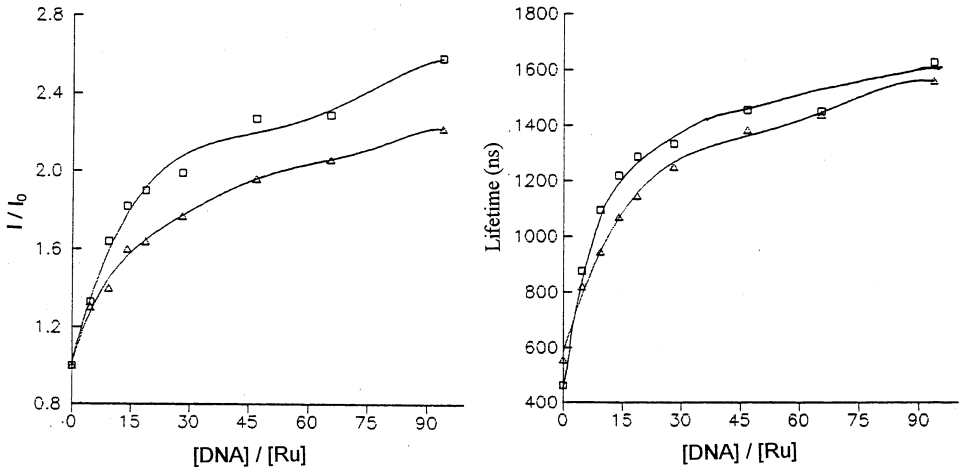


Fig. 6. Plots of relative integrated emission intensity (left) and excited state lifetime (right) versus [DNA]:[Ru] rate for $[Ru(bpy)_2(IP)]^{2+}$ (△) and $[Ru(bpy)_2(PIP)]^{2+}$ (□).

that the variations of luminescent intensities were $[Ru(phen)_2(dppz)]^{2+} \gg [Ru(phen)_2(dppn)]^{2+}$ and $[Re(dppz)(CO)_3(py)]^{2+} \gg [Re(dppn)(CO)_3(py)]^{2+}$ on binding to DNA, respectively. These results may be related to the notion that the

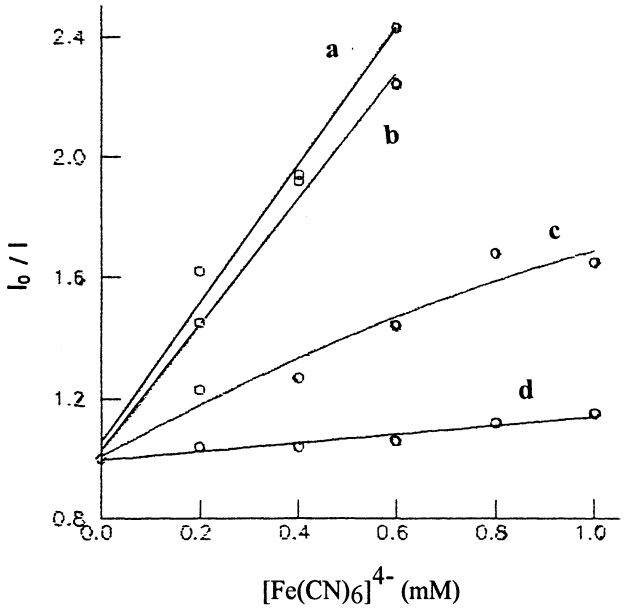


Fig. 7. Emission quenching of the complexes increasing concentrations of $[Fe(CN)_6]^{4-}$. [Ru] = 2 μM, [DNA]:[Ru] = 40:1. (a) Free $[Ru(bpy)_2(IP)]^{2+}$, (b) free $[Ru(bpy)_2(PIP)]^{2+}$, (c) $[Ru(bpy)_2(IP)]^{2+}$ + DNA, and (d) $[Ru(bpy)_2(PIP)]^{2+}$ + DNA.

Table 2
Absorption spectroscopic changes of Ru(II) complexes containing various co-ligands on binding to C.T.DNA

Complexes	Absorption λ_{max} (nm)			Hypochromism (%)
	Free[a]	Bound[b]	$\Delta\lambda$	
$[Ru(bPy)_2(pdphen)]^{2+}$	452	453	1	10
$[Ru(phen)_2(pdphen)]^{2+}$	448	433	15	11
$[Ru(dmp)_2(pdphen)]^{2+}$	461	464	3	5
$[Ru(5\text{-nphen})_2(pdphen)]^{2+}$	446	453	7	3

[a] [Ru] = 10 μM.
[b] [DNA nucleotide]/[Ru] = 30.

base overlap region has only an effective width of about 3.5 Å [30]. The issue remains to be clarified as to how many fused aromatic rings for the intercalative ligand of Ru complex are an optimum size, although an optimum organic intercalator is thought to be a planar charomophore with three fused aromatic rings [31].

3.3. Effect of co-ligand

Compared with the intercalative ligand, the influence of the co-ligand of the complex has received less attention. However, the co-ligand was also found to play an important role in governing DNA-binding of the complex [18d,25].

According to the data in Table 2, [18d] (which were obtained from the complexes containing the same intercalative ligand, i.e. pdphen, but different co-ligands), $[Ru(phen)_2(pdphen)]^{2+}$ shows the largest changes in absorption in the presence of C.T.DNA. The complex thus appear to bind strongly to the DNA. $[Ru(dmp)_2(pdphen)]^{2+}$ and $[Ru(5\text{-nphen})_2(pdphen)]^{2+}$ give a smaller hypochromic effect, and therefore bind much less strongly to DNA. These observations imply that the increase of co-ligand size increases its hydrophobicity, facilitating interaction with DNA. However, if the co-ligand is too large, its steric hindrance would interfere with the depth of intercalation in the base pair of DNA.

3.4. Enantioselectivity in binding to DNA

According to the insertion model proposed by Barton and co-workers [2a,d,h], the Δ enantiomer of polypyridyl Ru(II) complex, a right-handed propeller-like structure, displays a greater affinity than the A enantiomer for right-handed C.T.DNA helix, due to appropriate steric matching. Equilibrium dialysis experiments offer the opportunity to examine any enantiomeric selectivities associated with the binding.

The CD spectra in the UV region of $[Ru(bpy)_2(IP)]^{2+}$ and $[Ru(bpy)_2(PIP)]^{2+}$ after their racemic solutions had been dialyzed against C.T.DNA were observed [17f]. The presence of CD signals indicates enrichment of the isomer which binds

less favorably to the DNA. The spectra of dialysates of $[Ru(bpy)_2(IP)]^{2+}$ and $[Ru(bpy)_2(PIP)]^{2+}$, which are very similar to each other and to that of the analogous $[Ru(bpy)_2(pzp)]^{2+}$ dialysate [4a], proved to be Λ configuration [4b]. We therefore conclude that the absolute configurations of the dialysates of $[Ru(bpy)_2(IP)]^{2+}$ and $[Ru(bpy)_2(PIP)]^{2+}$ are also Λ isomers, and that the Δ isomers bind preferentially to the DNA, as anticipated from the intercalative binding mode.

3.5. Binding to different native DNAs

In the past, the studies on the interactions of Ru(II) complexes with native DNA focused on C.T.DNA. More recently, we have been interested in exploring the binding of $[Ru(phen)_3]^{2+}$ with another native DNA, herring sperm DNA [18b]. We found that the binding of the complex with herring sperm DNA was different from that with C.T.DNA although both are right-handed native DNAs.

The binding of $[Ru(phen)_3]^{2+}$ with C.T.DNA leads to a substantial perturbation in the photophysical properties of the complex, such as enhancement of emission intensity and hypochromism in the MLCT band of absorption spectrum [2a]. The binding of $[Ru(phen)_3]^{2+}$ with herring sperm DNA changes neither emission nor absorption intensities (see Table 3).

The CD spectra of $[Ru(phen)_3]^{2+}$, after its racemic solutions had been dialyzed respectively against the two DNAs, are shown in Fig. 8 [17f]. The figure indicates that the dialysis against C.T.DNA leads to the enrichment in the solution of the less-favored Λ isomer, but the dialysis against the herring sperm DNA leads to the enrichment of the less-favored Δ isomer in the solution.

According to the report by Barton [2c], $[Ru(phen)_3]^{2+}$ may bind to right-handed DNA via two modes, intercalative and groove binding, respectively. Intercalative binding through the major groove is characterized by an increase in luminescence intensity of the complex and the preferential binding to the Δ isomer. Hydrophobic groove-binding mode through the minor groove shows no enhancement in luminescence intensity of the complex and preference in binding of the Λ isomer. Combined with our results of absorption, emission and CD spectra, it is inferred that $[Ru(phen)_3]^{2+}$ binds to herring sperm DNA mainly by groove binding mode. This binding mode may be related to the structure feature of herring sperm DNA, which has a wider minor groove than C.T.DNA, similar to A-form helix [32] and therefore favored close contacts.

4. A novel DNA-mediated luminescence quenching system

An understanding of ET over large distances is essential to the characterization of fundamental redox processes in biology such as oxidative phosphorylation and photosynthesis, and to the development of efficient anticancer drugs.

After nearly two decades of painstaking research, it has gradually become agreed that proteins are not particularly good electrical conductors. Over the past few years, Barton and co-workers have focused on studies on the ET of another key

biological molecular, double-helical DNA, as the intervening medium and reported some examples of fast ET mediated by the DNA helix [2e,33]. However, the DNA-mediated long-range ET remains to be a controversial issue as intricate results have been obtained [34].

Here we report the preliminary studies on a new DNA-mediated luminescence quenching assembly, i.e. $*[Ru(bpy)_2(MCP)]^{2+}$/DNA/$[Ru(bpy)_2(MNP)]^{2+}$, to further probe the issue of DNA-mediated ET. In this system, randomly intercalated $*[Ru(bpy)_2(MCP)]^{2+}$ is regarded as a photoinduced electron donor; the NO_2 group in the intercalative ligand MNP, not the metal ion, of $[Ru(bpy)_2(MNP)]^{2+}$ is expected to serve as a center of electron acceptor and to quench luminescence of $*[Ru(bpy)_2(MCP)]^{2+}$ through 'π-way' of the DNA if the π-stacked bases of the DNA can provide an effective pathway for intermolecular ET, since it is well

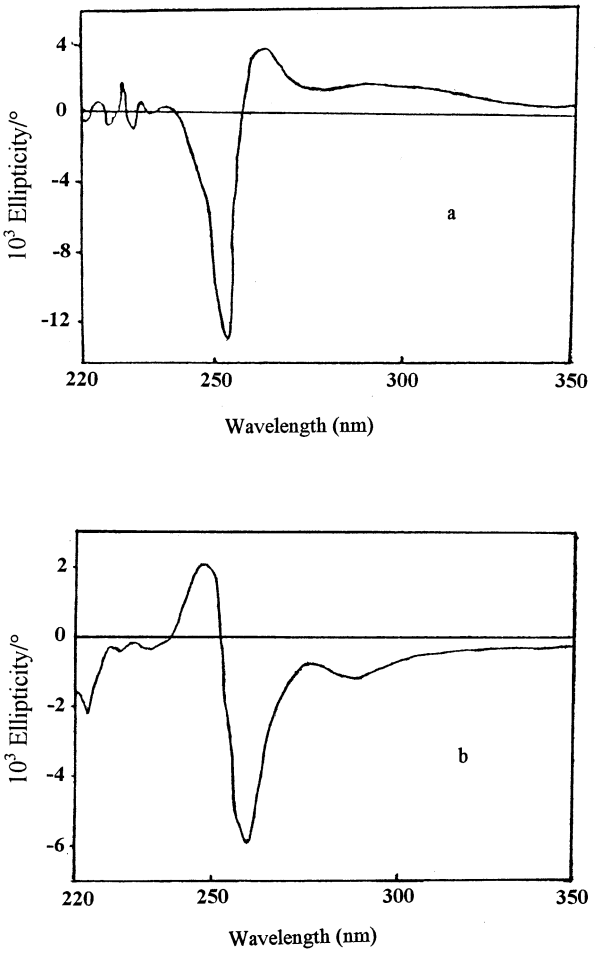


Fig. 8. CD spectra of $[Ru(phen)_3]^{2+}$ after dialysis versus C.T.DNA (a) and herring sperm DNA (b).

Table 3
Spectroscopic properties in binding $[Ru(phen)_3]^{2+}$ to C.T.DNA and herring sperm DNA

DNA	Absorption λ_{max} (nm)			Hypochromism (%)	Emission λ_{max} (nm)			Emission enhance I/I_0
	Free[a]	Bound[b]	$\Delta\lambda$		Free	Bound	$\Delta\lambda$	
C.T.DNA	445	447	2	12	599	602	3	1.92
Herring sperm DNA	445	447	2	0	599	601	2	0.99

[a] [Ru] = 10 μM.
[b] [DNA nucleotide]/[Ru] = 40.

known that nitro aromatics have an appreciable thermodynamic driving for quenching the emission of Ru(II) complexes by ET [35].

4.1. Binding mode to DNA

It is important to investigate the binding modes of donor and acceptor to DNA because the efficient ET through the DNA may occur only when the donor and acceptor are intercalated into the DNA stacked base [33h].

The electronic absorption spectra of $[Ru(bpy)_2(MCP)]^{2+}$, in the presence of increasing amounts of C.T.DNA, show strong decreases in the peak intensities, and its visible-region MLCT absorption band displays bathochromic shifts. In addition to the changes in the absorption spectra, substantial increases in emission intensity and excited state lifetime were also observed. These spectral changes are listed in Table 4. The combination of these data provides evidence that the preferential binding of the complex to the DNA is likely intercalative in nature, as has demonstrated for other tri-chelated, mixed-ligand Ru(II) complexes such as $[Ru(bpy)_2(pzp)]^{2+}$ [4a] and $[Ru(phen)_2(dppz)]^{2+}$ [2h,i]. The hypochromicity and red shift observed specifically for MLCT transition, which is assigned as terminating on the MCP ligand, indicates that it is reasonable to assume that the complex intercalates between base pairs accessible through MCP ligand because bpy has been previously demonstrated to be a non-intercalative ligand [1a,2b,h,3a,b].

The binding constant of $[Ru(bpy)_2(MCP)]^{2+}$ to the DNA was obtained by steady-state luminescence titration method [27a]. Fixed amounts of 10 μM $[Ru(bpy)_2(MCP)]^{2+}$ were titrated with increasing amounts of DNA, over a range of the DNA concentrations from 10^{-4} to 2×10^{-3} M. An excitation wavelength of 462 nm was used, and total emission intensity was monitored at 613 nm. The concentration of the bound Ru complex was calculated using Eq. (1):

$$c_b = c[(I - I_0)/(I_{max} - I_0)] \quad (1)$$

where c is the total Ru(II) complex concentration, I and I_0 are the emission intensities in the presence and absence of DAN, and I_{max} is the fluorescence of the totally bound complex. The concentration of the free complex, c_f, is equal to $c - c_f$. A plot of r/c_f vs. r, where r is c_b/[DNA], was constructed according to the McGhee and von Hippel equation:

$$2r/c_f = K_b(1 - 2nr)[(1 - 2nr)/\{1 - 2(n-1)r\}]^{n-1} \quad (2)$$

where K_b represents the intrinsic binding constant of the complex with DNA and n is the size of a binding site in base pairs. The binding data were fitted using Eq. (2), and a high intrinsic binding constant K_b, of 1.8×10^5 M^{-1} was obtained, which is comparable to that of the known intercalator, $Ru(bpy)_2(phi)Cl_2$ [1a]. The binding site size, n, is 8–9 base pairs, which is similar to those of many Ru polypyridine complexes such as $Ru(bpy)_3^{2+}$, $Ru(bpy)_2(phen)^{2+}$, $Ru(phen)_3^{2+}$ and $Ru(phen)_2(flone)^{2+}$ [1a].

For $[Ru(bpy)_2(MNP)]^{2+}$, we detected no luminescence in either the absence or the presence of DNA. Barton et al. discovered a 'light switch' complex

Table 4
Spectroscopic properties of $Ru(bpy)_2(MCP)^{2+}$ and $Ru(bpy)_2(MNP)^{2+}$ on binding to DNA

Complex[a]	Absorption λ_{max} (nm)			Hypochromism (%)	Emission λ_{max} (nm)			I/I_0[b]	Emission lifetime (ns)		τ/τ_0[b]
	Free	Bound	$\Delta\lambda$		Free	Bound	$\Delta\lambda$		Free	Bound	
$RU(bpy)_2(MCP)^{2+}$	457.2	462.8	5.6	25	613	617	+4	3.2	641	1794	2.8
$Ru(bpy)_2(MNP)^{2+}$	458.5	462.0	3.5	21	–	–	–	–	–	–	–

[a] [Ru] = 10 μM.
[b] I_0 and I, τ and τ_0, denote the luminescence intensity and lifetime of the Ru complexes with DNA ([nucleotide]/[Ru] = 200) and without DNA, respectively.

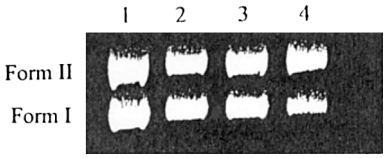


Fig. 9. Gel electrophotogram of $[Ru(bpy)_2(MNP)]^{2+}$-photosensitized cleavage of pBR322-DNA. Lane 1: a control; Lanes 2, 3, 4: irradiation for 30, 60, 90 min, respectively. I and II denote the form I and II DNA, respectively.

$[Ru(bpy)_2(dppz)]^{2+}$ [32]. It displays no detectable luminescence in aqueous solution, but intense luminescence in the presence of DNA to which it binds, with a binding constant of $> 10^6$ M^{-1}, by intercalation. This is due to the extreme sensitivity of the excited state to quenching by water; in DNA, the intercalated complex in the helix is protected from the aqueous solvent, thereby preserving the luminescence [36]. For $[Ru(bpy)_2(MNP)]^{2+}$, the 'quenching' of emission is caused by the electron acceptor NO_2 group in the complex structure itself, so it is not sensitive to the environment. Therefore, the non-emissive behavior of $[Ru(bpy)_2(MNP)]^{2+}$ does not imply that it cannot interact with DNA by intercalation. In fact, the similar changes of absorption spectra to $[Ru(bpy)_2(MCP)]^{2+}$ can be observed when $[Ru(bpy)_2(MNP)]^{2+}$ binds DNA (see Table 4). Further, the gel electrophorogram (Fig. 9) of $[Ru(bpy)_2(MNP)]^{2+}$-photoactivated cleavage of pBR322-DNA displays that $[Ru(bpy)_2(MNP)]^{2+}$ can effectively convert the covalently-closed circular form I of pBR322 plasmid DNA to nicked form II by non-covalent binding after radiation, implying a direct ET of its excited state with DNA bases [37]. Put together, the absorption spectral changes and the direct strand photocleavage suggest the assignment of binding of the complexes to double-helical DNA through intercalation of the ligand MNP [33h,38].

4.2. Luminescence quenching

The results of quenching experiments using $*[Ru(bpy)_2(MCP)]^{2+}$ as a donor and $[Ru(bpy)_2(MNP)]^{2+}$ as an acceptor are presented in Fig. 10 in Stern–Volmer plots of I_0/I and τ_0/τ vs. quencher concentration. As can be seen from the curves c and d, both I_0/I and τ_0/τ have slopes of nearly zero , just like quenching of $[Ru(phen)_2(dppz)]^{2+}$ by $[Rh(phi)_2(bpy)]^{2+}$ [33e] and $[Ru(phen)_3]^{2+}$ by $[Co(phen)3]^{2+}$ [39]. It indicates that the quenching of $[Ru(bpy)_2(MCP)]^{2+}$ emission by $[Ru(bpy)_2(MNP)]^{2+}$ is too weak to be measured in DNA-free solution. This weakness of quenching may be due to the repulsion between the two like-charges on the complexes [19f].

In the range of low intercalator/DNA loadings ($[Ru(bpy)_2(MCP)]^{2+}$:$[Ru(bpy)_2(MNP)]^{2+}$:DNA $\leq$ 1:7:200), $[Ru(bpy)_2(MCP)]^{2+}$ emission is remarkably quenched (max. quenching 64%) by $[Ru(bpy)_2(MNP)]^{2+}$, resulting in an upward-curving Stern–Volmer plot (Fig. 10a) with increasing $[Ru(bpy)_2(MNP)]^{2+}$ concentration. The slope of the lifetime quenching curve is much less than that of the

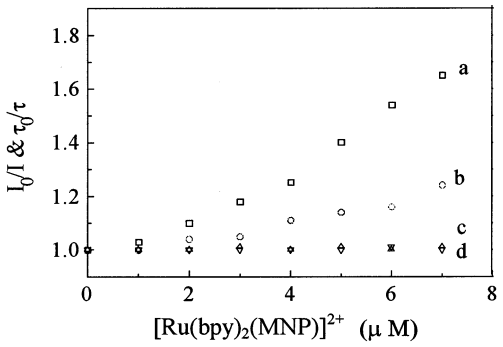


Fig. 10. Stern–Volmer plot describing luminescence quenching of $[Ru(bpy)_2(MCP)]^{2+}$ by $[Ru(bpy)_2(MNP)]^{2+}$. a and b,c and d: Quenching of integrated intensity and lifetime of 2 μM $[Ru(bpy)_2(MCP)]^{2+}$ by $[Ru(bpy)_2(MNP)]^{2+}$ in the presence of the DNA (200 μM nucleotide) and without the DNA, respectively.

intensity quenching curve under the same experimental conditions. The positive deviations from linearity of the quenching curve and the large discrepancies between intensity I_0/I and lifetime τ_0/τ quenching curves indicates that the quenching may be controlled primarily by a static mechanism, instead of a simple diffusion one [33e, 34a, 40].

The time-resolved decay profiles of $[Ru(bpy)_2(MCP)]^{2+}$ in the presence of fixed DNA concentration (20 μM) and increasing concentration of $[Ru(bpy)_2(MNP)]^{2+}$ are presented in part in Fig. 11. Fig. 11 shows the initial intensity drops remarkably, accompanied by only minimal changes in the lifetime. The quenching feature is consistent with those for the majority of the ET quenching occurring on a time scale faster than the resolution (10 ns) of instrument [33e].

In short, these observations indicate that the NO_2 group quenches the luminescence of Ru(II) complexes possibly by the 'π-way' of DNA. Although the theoretical interpretation of the character of NO_2 group is not always unambiguous [41], the quenching is suggested to be probably based on the following mechanism [35a]:

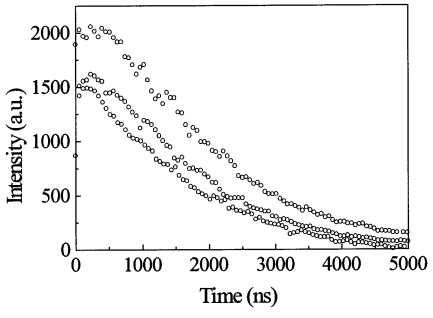
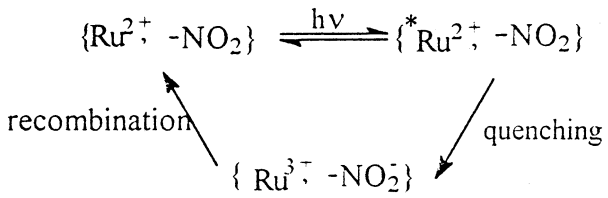


Fig. 11. The time-resolved emission decays of $[Ru(bpy)_2(MCP)]^{2+}$ (2 μM) in the DNA (200 μM nucleotide) as a function of added quencher (0, 6 and 7 μM $[Ru(bpy)_2(MNP)]^{2+}$ from top to bottom).



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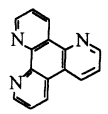
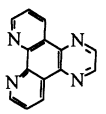
Abbreviations

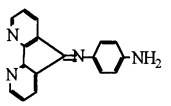
py	pyridine
bpy	1,2-bipyridine
phen	1,10-phenanthroline
DIP	4,7-diphenyl-1,10-phenanthroline
dmp	2,9-dimethyl-phenanthroline
5-nphen	5-nitrophenanthroline
Me-phen	5-methyl-1,10-phenanthroline
dppz	dipyrido[3,2-a:2′,3′-c]phenazine
dppn	benzo[*i*]dipyrido[3,2-a:2′,3′-c]phenazine
pzp	pyrazino[2,3-f][4,7]phenanthroline
AFO	4,5-diazafluoren-9-one
phi	9,10-phenanthrenequinonediimine



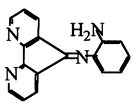
pdphen [18c]



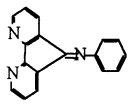
TATP [21]



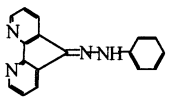
AFPNp [19e]



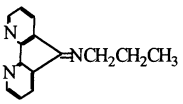
AFPNo [19d]



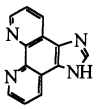
AFP [19b]



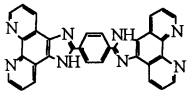
AFNP [19e]



AFPr [19f]



IP [17f]



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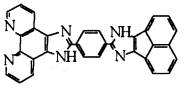
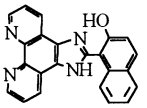
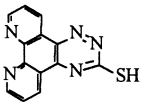


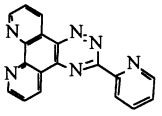
PIBIA [21]



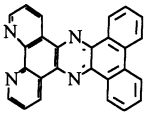
HNAIP [24]



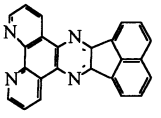
MTP [26]



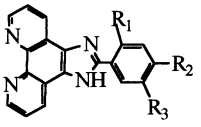
PDTP [26]



DDP [27]



ADQ [27]



PIP [17f]	$R_1 = R_2 = R_3 = H$
NOP [17e]	$R_1 = R_3 = H,\ R_2 = NO_2$
CLP [17e]	$R_1 = R_3 = H,\ R_2 = Cl$
NIP [23a]	$R_2 = R_3 = H,\ R_1 = NO_2$
CIP [23a]	$R_2 = R_3 = H,\ R_1 = Cl$
HIP [24]	$R_2 = R_3 = H,\ R_1 = OH$
HOP [17e]	$R_1 = R_3 = H,\ R_2 = OH$
MOP [17g]	$R_1 = R_3 = H,\ R_2 = OCH_3$
DMNP [17g]	$R_1 = R_3 = H,\ R_2 = N(CH_3)_2$
MCP [23b]	$R_1 = R_2 = H,\ R_3 = Cl$
MNP [23c]	$R_1 = R_2 = H,\ R_3 = NO_2$

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