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含 2-氨基吡啶及去甲基斑蝥酸根的钴(Ⅱ)配合物的晶体结构、与 DNA 和 BSA 相互作用及体外抗增殖活性

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摘要:溶液法合成了二(去甲基斑蝥酸根)合钴(Ⅱ)酸二(2-氨基吡啶鎓)配合物$(Hapy)_2[Co(DCA)_2]\cdot 6H_2O$(DCA=去甲基斑蝥酸根,$C_8H_8O_5$;Hapy=2-氨基吡啶鎓,$C_5H_7N_2$)。通过元素分析、摩尔电导、红外光谱、热重分析和 X-射线单晶衍射对配合物进行了结构表征。该配合物为三斜晶系 $P\bar{1}$ 空间群,中心离子配位数为 6。利用荧光光谱法和粘度法对配合物与 DNA 之间的相互作用进行了研究。结果表明,配合物以部分插入模式与 DNA 键合,猝灭常数 K_{sq} 为 0.18。同时,利用荧光光谱研究了配合物与牛血清白蛋白(BSA)的相互作用。结果显示,配合物能与 BSA 发生较强的键合作用,键合常数 $K_a=3.88\times10^6$ L·mol^{-1}。体外抗增值活性结果显示,配合物对人肝癌细胞(SMMC-7721)和人胃癌细胞(MGC80-3)的抑制活性均强于去甲基斑蝥酸钠。

关键词:去甲基斑蝥酸根;2-氨基吡啶;钴(Ⅱ)配合物;与 DNA 和 BSA 相互作用
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Crystal Structure, Interaction with DNA and BSA and Antiproliferative Activities of Cobalt(Ⅱ) Complex with Demethylcantharate and 2-Aminopyridine

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Abstract: A cobalt(Ⅱ) complex $(Hapy)_2[Co(DCA)_2]\cdot 6H_2O$ (DCA=demethylcantharate, 7-oxabicyclo[2.2.1]heptane-2,3-dicarboxylate, $C_8H_8O_5$; Hapy=2-aminopyridinum, $C_5H_7N_2$) was synthesized and characterized by elemental analysis, molar conductance, infrared spectra, thermogravimetric analysis and X-ray diffraction. The complex crystallized in the triclinic crystal system, $P\bar{1}$ space group, with a=0.662 23(7) nm, b=1.016 41(11) nm, c=1.194 60(12) nm, V=0.792 49(14) nm^3, Z=1, D_c=1.520 g·cm^{-3}, M_r=725.57, λ(Mo $K\alpha$)=0.071 073 nm, F(000)=381, R=0.028 3 and wR=0.082 1 ($I>2\sigma$ (I)). The coordination number of metal ion was six and DCA ions were tridentate ligand. DNA binding property of the complex was investigated by fluorescence spectra and viscosity measurements. Results indicated the complex could bind to DNA via partial intercalation mode. The value of quenching constant K_{sq} was 0.18. The interaction of the complex with bovine serum albumin (BSA) was also studied by fluorescence spectra. The results suggested that the complex could quench the fluorescence of BSA with the binding constant K_a of 3.88×10^6 L·mol^{-1}. The antiproliferative activity test revealed that complex showed stronger inhibition ratios against human hepatoma cells (SMMC-7721) lines and human gastric cancer cells (MGC80-3) lines than Na_2(DCA) in vitro. CCDC: 823705.

Key words: demethylcantharate; 2-aminopyridine; cobalt(Ⅱ) complex; interaction with DNA and BSA

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The interaction of transition metal complexes with biomacromolecules has received a great deal of attention during the past decade[1-2]. Researchers find that the bioactivities of complexes could be improved by inserting drugs molecules as ligands[3-4]. Demethylcantharidin (NCTD, 7-oxabicyclo [2,2,1]heptane-2,3-dicarbo-xylc acid anhydride), which is the derivative of cantharidin, has been used in clinical [5]. Na_2(DCA) (DCA=demethylcantharate) could inhibit protein serine /threonine phosphatases (PP1, PP2A and PP2B)[6]. Meanwhile, the metal complexes containing demethylcantharate have been widely reported for their strong anti-tumour activities[7-8]. Pyridine is an important class of *N*-containing heterocycles. The bioactivities of complexes containing pyridine ring have been reported, such as DNA binding property and antibacterial activity[9-10]. The bioinorganic chemistry of cobalt has been rapidly expanded due to the increasing number of cobalt complexes of biological interest reported in the literature[11-12].

The complex $(Hapy)_2[Co(DCA)_2]\cdot 6H_2O$ was synthesized using cobalt acetate, NCTD and 2-aminopyridine (apy). The interaction of complex with DNA was investigated by fluorescence spectra and viscosity measurements. The interaction of complex with BSA was studied by fluorescence spectra. In addition, the antiproliferative activities of the complex against human hepatoma cells (SMMC-7721) and human gastric cancer cells (MGC80-3) were tested in vitro.

1 Experimental

1.1 Materials and instruments

All reagents and chemicals were obtained from commercial sources. Demethylcantharidin (NCTD, $C_8H_8O_4$) was obtained from Nanjing Zelang Medical Technology Co. Ltd.; Ethidium bromide (EB) was obtained from Fluka Co.; 2-aminopyridine (apy, $C_5H_6N_2$) and DNA was obtained from Sinopharm Chemical Reagent Co. Ltd.; DNA (ρ=200 μg·mL^{-1}, c=3.72×10^{-4} mol·L^{-1}), which A_{260}/A_{280}=1.8~2.0, was prepared by 50 mmol·L^{-1} NaCl; Bovine Serum Albumin (BSA) was purchased from Beijing BioDee BioTech Co. Ltd. and was stored at 277 K; BSA (ρ=500 μg·mL^{-1}, c=7.47×10^{-6} mol·L^{-1}) was prepared by 5 mmol·L^{-1} NaCl; Human hepatoma cells (SMMC-7721) and human gastric cancer cells (MGC80-3) were purchased from Shanghai Institute of Cell Bank. Other chemical reagents in analytical reagent grade were used without further purification.

Elemental analyses of C, H and N were carried out in Vario EL Ⅲ elemental analyzer. The molar conductance value was obtained from Orion 150Aplus conductometer. Infrared spectra were measured using the KBr disc method by NEXUS-670 FT-IR spectrometer in the spectral range 4 000~400 cm^{-1}. The thermogravimetric analyses were monitored on TGA/SDTA851^e thermo gravimetric analyzer. Diffraction intensities of the complexes were collected at 293 K on Bruker SMART APEX Ⅱ CCD difffractometer. Fluorescence emission spectra were obtained by Perkin-Elmer LS-55 spectrofluorometer. Viscosity experiments were carried on Ubbelodhe viscometer.

1.2 Synthesis of the complex

The solution of $Co(Ac)_2\cdot 6H_2O$ (0.5 mmol) and 2-aminopyridine (0.5 mmol) was stirred at the room temperature for 2 h. The solution of NCTD (0.5 mmol) was added dropwise in the mixed solution. The pH of the solution was adjusted to 6.5 using dilute NaOH solution. The solution was then filtered after two hours. After two weeks, crystals with suitable size for single-crystal X-ray diffraction were obtained.

Anal. Calcd. for $C_{26}H_{42}N_4O_6Co$(%): C, 43.00; H, 5.79; N, 7.72. Found (%): C, 42.81; H, 5.97; N, 7.68. IR spectra (KBr, cm^{-1}): 1 687 (ν_{as}(COO$^-$)); 1 413 (ν_s(COO$^-$)); 1 266, 10 29, 985 (ν(C-O-C)). The value of molar conductance of complex is 148 S·cm^2·mol^{-1} in 10^{-3} mol·L^{-1} DMF at 20 ℃, which suggest that the complex is 2:1 type electrolyte[13].

1.3 DNA binding

1.3.1 Fluorescence spectra

Fluorescence quenching experiment was carried out by adding complex solution (0~2.50×10^{-4} mol·L^{-1}) to the samples containing 5.00×10^{-6} mol·L^{-1} EB and 7.44×10^{-5} mol·L^{-1} DNA. The mixture were diluted by Tris-HCl buffer solution (pH=7.4). Fluorescence was read at excitation wavelength (λ_{ex}) of 252 nm and

Table 1　Crystal data and structure refinement details for the complex

Chemical formula	$CoC_{26}H_{42}N_4O_{16}$	Shape	Block
Formula weight	725.57	Color	Pink
Crystal system	Triclinic	D_c / (g·cm^{-3})	1.520
Space group	$P\bar{1}$	θ rang for data collection / (°)	1.71 to 24.99
a / nm	0.662 23(7)	Reflections collected / Unique	10 814 / 2 778
b / nm	1.016 41(11)	Rint	0.019 1
c / nm	1.194 60(14)	Absorption coefficient / mm^{-1}	0.623
α / (°)	92.902(5)	F(000)	381
β / (°)	90.281(5)	R / wR (I>2σ(I))	0.028 3 / 0.082 1
γ / (°)	99.273 (5)	Restraints / parameters	3 / 214
Volume / nm^3	0.792 49(14)	Goodness-of-fit on F^2	1.066
Z	1	Largest diff. peak and hole / (e·nm^{-3})	436, −322

emission wavelength (λ_{em}) between 500 nm and 700 nm.

1.3.2　Viscosity measurements

Viscosity measurements were conducted on an Ubbelodhe viscometer. The compounds were added to DNA solution (3.72×10^{-4} mol·L^{-1}) by microsyringe. The concentration of the compounds were controlled in 0~3.33×10^{-6} mol·L^{-1}. The relative viscosities η were calculated through equation [14]: $\eta=(t-t_0)/t_0$, where t_0 and t represent the flow time of DNA solution in the absence and presence of complex through the capillary, respectively.

1.4　Interaction with BSA

The complex (0~3.33×10^{-8} mol·L^{-1}) were added to the mixed solution, which contains 4.98×10^{-7} mol·L^{-1} BSA and Tris-HCl buffer. Fluorescence quenching spectra were obtained by recording the emission spectra (255~500 nm) corresponding to excitation wavelength at 255 nm.

1.5　Antiproliferative activity evaluation

The antiproliferative activities of Na_2(DCA) and the complex were evaluated by human hepatoma cells (SMMC-7721) and human gastric cancer cells (MGC80-3). The antiproliferative activities were measured by the MTT assay. The compounds were dissolved in DMSO as 100 mmol·L^{-1} stock solutions and diluted with culture medium before using. The final concentration of DMSO in the medium was less than 0.1% and it showed no interference with the biological activities tested[15]. Cells were seeded for 24 h, and the complex or Na_2(DCA) were added and incubated for 72 h. Then MTT (100 L, 1 mg·mL^{-1}, dissolved in culture medium) was added into each well and incubated for 4 h (37 ℃). The inhibition rate was calculated. The errors quoted were standard deviations, in which three replicates were used in the calculation[16].

1.6　Crystal structure determination

Single crystal, sized 0.167 mm×0.169 mm×0.246 mm, was analyzed by X-ray diffraction. The structure was solved by direct methods and refined by full-matrix least-squares techniques using the SHELXTL-97 program package[17-18]. All non-hydrogen atoms were refined anisotropically. Except the hydrogen atoms on oxygen atoms were located from the difference Fourier maps, the other hydrogen atoms were generated geometrically. Crystal data and experimental details for structural analyses are listed in Table 1.

CCDC: 823705.

2　Results and discussion

2.1　Characterization and crystal structure

2.1.1　Thermogravimetric analysis (TGA)

This experiment was performed in an air atmosphere with a heating rate of 10 ℃·min^{-1} and temperature range of 30~800 ℃. The TG-DTG curves of complex are shown in Fig.1. According to the TG-DTG curves, three steps of the weight loss processes were happened in this process. The first weight loss (14.03%) occurred in temperature range of 49~102 ℃, which corresponded to the departure of the six crystal

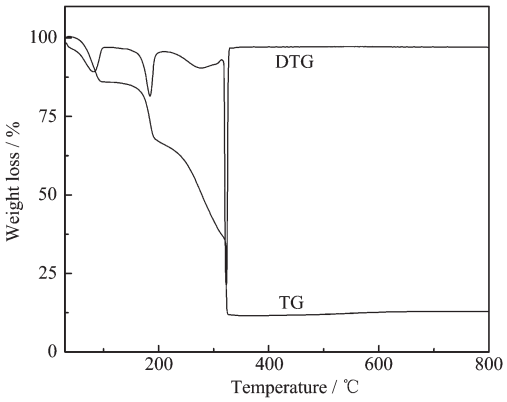


Fig.1 TG-DTG curves of the complex

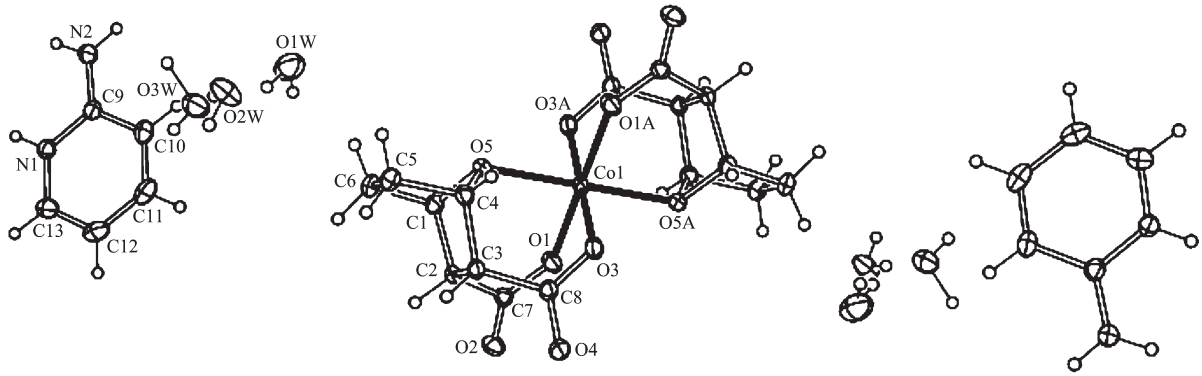
water (14.90% theoretical). The second weight loss (25.61%), which corresponded to the departure of two 2-aminopyridinum (26.22% theoretical), was occurred in the temperature range of 103~251 ℃. The complex gave an acuity weight loss peak at temperature around 252~337 ℃, which corresponds to the weigh loss of 48.02% (48.56% theoretical). It indicates the thermal decomposition of two DCA. At temperature above 338 ℃, no further weight loss occurred. The sample residue was CoO, which weigh 11.67% of the initial mass (10.33% theoretical).

These results agree with the composition of the complex determined by elemental analyses and X-ray diffraction.

2.1.2 Structural description of the complex

The X-ray structural analysis shows that the complex crystallized in the triclinic crystal system with $P\bar{1}$ space group. Molecular structure of the complex $(Hapy)_2[Co(DCA)_2]\cdot 6H_2O$ was showed in Fig. 2. Selected bond distances and angles were listed in Table 2.

The complex consisted cation part and anion part. Each Co(Ⅱ) ion was connected to two DCA ions, which formed anion part. The cation part was formed by two 2-aminopyridinum. Co (Ⅱ) ion was six-coordinated by four carboxylate oxygen atoms O1, O3, O1A and O3A in four different carboxylate groups and two bridge oxygen atoms O5 and O5A from two demethylcantharates, which formed octahedral structure. Due to the binding of the bridge oxygen atoms O5 and O5A with Co(Ⅱ), four six-membered



Symmetry transformations used to generate equivalent atoms: A: $-x+1, -y, -z$

Fig.2 Labeled ORTEP diagram of the complex with 30% thermal probability ellipsoids shown

Table 2 Selected bond lengths (nm) and angles (°) for the complex

Co1-O1	0.203 46(11)	Co1-O1A	0.203 46(11)	Co1-O3	0.210 72(11)
Co1-O3A	0.210 72(11)	Co1-O5	0.215 45(10)	Co1-O5A	0.215 45(10)
O1-Co1-O3	87.57(5)	O1-Co1-O3A	92.43(5)	O1-Co1-O5	90.03(4)
O1-Co1-O5A	89.97(4)	O3-Co1-O3A	180.0	O3-Co1-O5	89.48(4)
O3-Co1-O5A	90.52(4)	O1A-Co1-O5	89.97(4)	O3A-Co1-O5	90.52(4)
O1A-Co1-O3A	87.57(5)	O1A-Co1-O1	180.00(6)	O1A-Co1-O3	92.43(5)
O1A-Co1-O5A	90.03(4)	O3A-Co1-O5A	89.48(4)	O5A-Co1-O5	180.00(5)

Symmetry transformations used to generate equivalent atoms: A: $-x+1, -y, -z$.

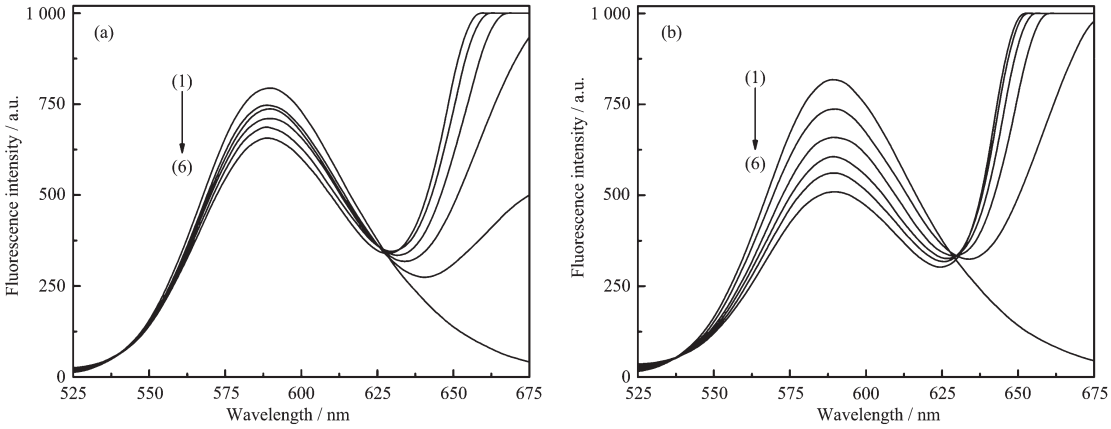
rings (Co1-O1-C7-C2-C1-O5), (Co1-O3-C8-C3-C4-O5), (Co1-O1A-C7A-C2A-C1A-O5A) and (Co1-O3A-C8A-C3A-C4A-O5A) were created. Two seven-membered rings (Co1-O1-C7-C2-C3-C8-O3) and (Co1-O1A-C7A-C2A-C3A-C8A-O3A) were formed, which could have stabilized the anion part of the complex.

2.2 DNA bingding studies

2.2.1 Fluorescence spectral studies

To study the mode and intensity of the interaction of complexes with DNA, EB was used as fluorescence probe. Fig.3 shows the emission spectra of EB bounded to DNA with 2-aminopyridine (Fig.3a) and the complex (Fig.3b) with similar peak shapes. Fig.3 suggested that, as the concentrations of the compounds increase, the emission intensity at 589 nm of EB-DNA system decrease in different degrees.

According to the Stern-Volmer equation[19]: $F_0/F = 1+K_{sq}r$, where F_0 and F represented the fluorescence intensities in the absence or presence of complexes, r stands for the concentration ratio of the complexes to DNA. The quenching constant K_{sq} was obtained as the slope of F_0/F versus r linear plot, which were 0.058 (apy) and 0.18 (complex). Results indicated that the complex could insert into the DNA base pairs, and release free EB from EB-DNA system. However, NCTD and Na_2 (DCA) can not quench the emission intensities of EB-DNA system significantly[20].

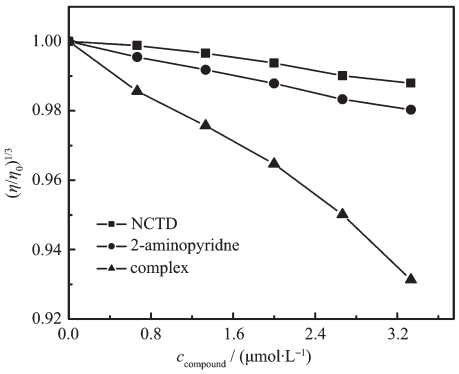


c_{EB}=5.00 μmol·L^{-1}; c_{DNA}=74.4 μmol·L^{-1}; $r=c_{complex}/c_{DNA}$=0, 0.67, 1.34, 2.02, 2.69 and 3.36, from (1) to (6), respectively; (a): 2-aminopyridine; (b): complex, λ_{ex}=252 nm

Fig.3　Fluorescence spectra of EB-DNA in the absence and presence of increasing the amount of compounds

2.2.2 Viscosity measurements

To further investigate the binding mode and binding intensity of the complexes with DNA, the DNA viscosity changing at 25 ℃ was studied (Fig.4). The result showed that the relative viscosity of DNA didn't change obviously after adding 2-aminopyridine and NCTD, while steadily decreased after adding complex, which suggested that the complex may partially insert into DNA[21]. Structurally, the steric hindrance of the complex was accrescent due to the non-planar of DCA. Thus, the complex can only partially inserted into DNA.

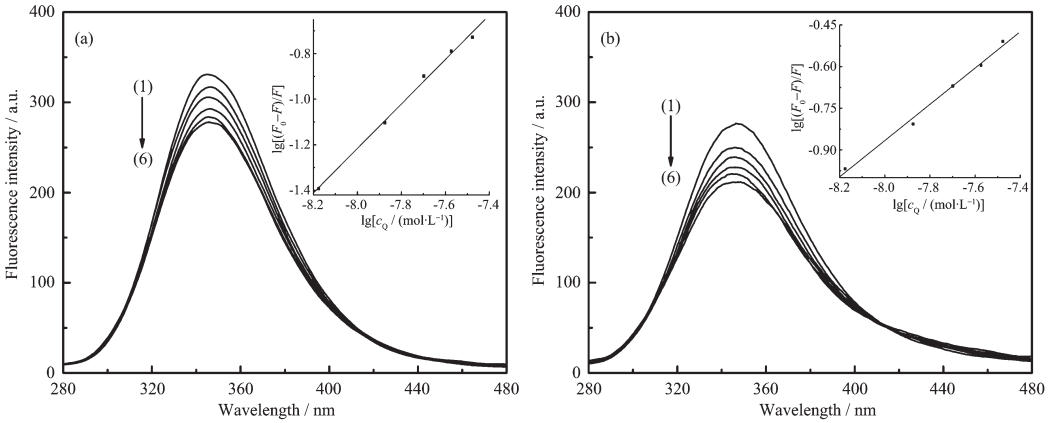


c_{DNA}=0.372 mmol·L^{-1}; $c_{compound}$=0, 0.67, 1.33, 2.00, 2.67 and 3.33 μmol·L^{-1}, respectively

Fig.4　Effect of increasing amounts of the compounds on the relative viscosity of DNA at 25 ℃

2.3 Interaction with BSA

The fluorescence quenching of BSA by the complex was showed in Fig.5 ((a): complex; (b): NCTD). The results showed that BSA has strong fluorescence emission at 345 nm. The peak intensity



c_{BSA}=0.498 μmol·L^{-1}; $c_{compound}$=0, 6.7, 13.3, 20.0, 26.7 and 33.3 nmol·L^{-1}, from (1) to (6), respectively; λ_{ex}=255 nm; (a): complex; (b): NCTD

Fig.5 Fluorescence spectra of BSA in the absence and the presence of complex Inset: Stern-Volmer plots of the fluorescence titration data of the complexes

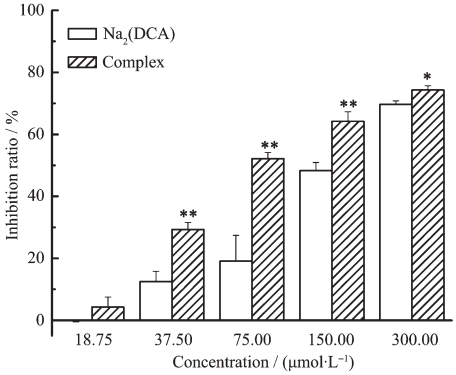
decreased with the increasing concentration of complex, which inferred that strong interactions and energy transfer between complex and BSA existed[22].

Assuming there were n identical and independent binding sites in protein, the binding constant can be calculated using equation[23]: $\lg[(F_0-F)/F]=\lg K_a+n\lg c_Q$, where F_0 and F represented the fluorescence intensities of BSA in the absence or presence of complex, c_Q stands for the concentration of the complex. K_a and n were calculated (see the slope and intercept of the line in illustration). The values of K_a were 2.78×10^4 (NCTD) and 3.88×10^6 L·mol^{-1} (complex). The values of n were 0.66 (NCTD) and 0.98 (complex). The results suggested that strong binding force existed between the complex and the BSA with complex intensity stronger than NCTD. The binding site was one.

2.4 Antiproliferative activity evaluation

The antiproliferative activities of the Na_2(DCA) and the complex against human hepatoma cells (SMMC-7721) (Fig.6) and human gastric cancer cells (MGC80-3) (Fig.7) were investigated *in vitro*. And the values of IC_{50} for these compounds were listed in Table 3. The results showed complex had strong antiproliferative effect against two kinds of cells in the range of tested concentration. The inhibition effect was enhanced by increasing the concentration of complex.

As shown in Fig.6, the inhibition rates of complex were significantly higher than Na_2(DCA) (P <



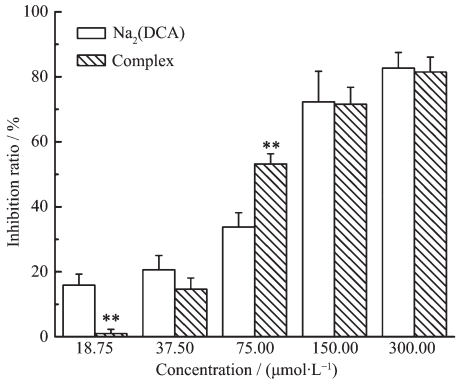
Data represent mean+S.D. and all assays were performed in triplicate for three independent experiments. *P<0.05, **P<0.01, ***P<0.001 vs Na_2(DCA) in the same concentration, t-test

Fig.6 Inhibition effects of complex and Na_2(DCA) on SMMC-7721 cell growth

Table 3 IC_{50} values (μmol·L^{-1}) (72 h) of complex and Na_2(DCA) on SMMC-7721 and MGC80-3 cells

	SMMC-7721	MGC80-3
Na_2(DCA)	152.8±15.6	93.2±13.5
Complex	87.48±0.39	88.34±9.08

0.01) against human hepatoma cells. Especially at the concentration of 75.00 μmol·L^{-1}, the inhibition intensity of complex (52.2±1.9) was much stronger than Na_2(DCA) (19.1±8.3). At concentration higher than 75.00 μmol·L^{-1}, the complex and Na_2(DCA) exhibited similar efficiency. The values of IC_{50} of complex ((87.48±0.39) μmol·L^{-1}) and Na_2(DCA) ((152.8 ± 15.6) μmol·L^{-1}) inferred that the complex had stronger



Data represent mean+S.D. and all assays were performed in triplicate for three independent experiments. *P<0.05, **P<0.01, ***P<0.001 vs Na_2(DCA) in the same concentration, t-test

Fig.7　Inhibition effects of complex and Na_2(DCA) on MGC80-3 cell growth

a ctivities against human hepatoma cells, even more intense than NCTD (IC_{50}=(115.5±9.5) μmol·L^{-1})[24], which had been used in clinical.

As shown in Fig.7, the inhibition rate of complex were higher than the rate of Na_2(DCA) (P < 0.01) against human gastric cancer cells at the concentration of 75.00 μmol·L^{-1}. At other tested concentrations, the inhibition rates of complex were close to Na_2(DCA).

3　Conclusions

A novel cobalt(Ⅱ) complex was synthesized. The chemical formula of the complex was $(Hapy)_2[Co(DCA)_2]\cdot 6H_2O$. The structure of the complex was determined by X-ray diffraction. The complex crystallized in the triclinic crystal system with $P\bar{1}$ space group.

All the tested results suggested that the complex could bind DNA via partial intercalation mode. Meanwhile, the complex could quench the fluorescence of BSA with the binding constant K_a of 3.88×10^6 L·mol^{-1}.

The antiproliferative activities testing revealed that the complex showed moderate inhibition ratios against human hepatoma cells and human gastric cancer cells in vitro.

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