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Short communication

Structure and biological activity of cationic $[PtLCl(DMSO)]NO_3 \cdot DMSO$ complex containing a chelated diaminosugar: methyl-3,4-diamino-2,3,4,6-tetradeoxy-α-L-lyxopyranoside

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Abstract

Cationic platinum(II) complex $[Pt(C_7H_{16}N_2O_2)(DMSO)Cl]NO_3 \cdot DMSO$ containing one chloride anion, methyl-3,4-diamino-2,3,4,6-tetradeoxy-α-L-lyxopyranoside ($C_7H_{16}N_2O_2$) and dimethylsulfoxide (DMSO) molecules forming a square-plane has been prepared and characterised, both spectroscopically and by single-crystal X-ray diffraction. Biological tests performed using leukemia L1210 cells have shown that toxicity of the title complex is similar to that of cisplatin.

Keywords: Platinum(II); DMSO; *O*-Methyl-3,4-diamino-2,3,4,6-tetradeoxy-α-L-lyxopyranoside; Structure; Antitumor activity

1. Introduction

Cisplatin (*cis*-diamminedichloroplatinum(II), CDDP) is reported among the most widely used drugs for the treatment of cancer. Its clinical utility, however, meets numerous limitations because of the increasing toxicological and/or tumor resistance considerations. A great effort has been made to address these restrictions. The most popular strategy is the development of novel CDDP analogues, less toxic but comparatively effective. However, despite the synthesis and biological tests preformed for about 3000 platinum complexes, only four: cisplatin, carboplatin, nedaplatin and oxalilplatin[1] have been approved for clinical use until 1999 [1–5]. Another way to reduce the toxicity of platinum(II)-based drugs is the coadministration of numerous species, e.g. sulphur containing compounds: thiols, dithiocarbamates or glutathione [6–9], which act as detoxicant agents against metal-containing drugs. It has been also found that simultaneous administration of dimethylsulfoxide (DMSO) with cisplatin results in a considerable reduction in the nephrotoxicity produced by the latter [10].

At present, it is a textbook knowledge that pronounced antitumor properties of the *cis*-platinum(II)-like complexes are caused by formation the DNA inter- and intra-strand cross-links, which can result in modification of its structure or functions. So, chemical modification of the original cisplatin molecule and the replacement of at least one chloride anion by another ligand may result in an alteration in the formation of the platinum–DNA complex. For example, it has been found that the hydrolysis rate (i.e. conversion into the therapeutically active species) of sterically hindered

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[1] The last three complexes: *cis*-diammine(1,1-cyclobutanedicarboxylato)platinum(II) (CBDCA-*O*,*O*′), *cis*-diammine(glycolato-*O*1,*O*2)platinum(II) (254-S) and oxalato (*trans*-*l*-1,2-diamminocyclohexane)platinum(II) (*l*-OHP), respectively.

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platinum(II) drug ZD0473[2] is more than two times slower than that of cisplatin, and significantly different from that for the 3-picoline–Pt(II) complex. The hydrolysis rate constants, k_1, for the 2-picoline–, 3-picoline–Pt(II) complexes and cisplatin determined under similar conditions are equal 1.9, 44.7 and 75.9 ($\times 10^6$ s^{-1}), respectively [11].

Nowadays, after about 35 years of medical application of the cisplatin, the antitumor activity has been reported not only for the neutral platinum(II) compounds but also for the cationic complexes. The therapeutic properties have been presented, e.g. for the $[Pt(L)(R'R''SO)Cl]NO_3$ species, where L denotes 1,2-diaminocyclohexane and R′R″SO denotes various alkyl sulfoxides [12]. A great number of another *cis*- and *trans*-platinum cationic complexes with different ligands, including DMSO, have been examined for their activity against both fluid suspension and solid tumor standard cell lines. It has been found that some of these complexes exhibit remarkably greater cytotoxic effects than original cisplatin [13]. Among the cationic complexes, these containing the DMSO molecules as leaving ligands are of special interest. Their antitumor activity usually is of the same magnitude as that of cisplatin, but they are found only to be marginally nephrotoxic [14–16]. Because they attach to DNA with simultaneous release of the sulfoxide ligand, it can be expected that their antitumor activity significantly depends on the nature of the amine molecule, which determines directly the Pt···guanine–N(7) bond strength and geometry. The comparison of biological activity of neutral and cationic organometallic platinum(II) complexes is also of interest, since it has been announced that dinuclear platinum(II) neutral complex binds to DNA in different way than its DMSO analogue [17]. As a result, the biological activity of both dinuclear complexes significantly differs from each other.

The main objective of our previous studies was to synthesise *O*-methyl-3,4-diamino-2,3,4,6-tetradeoxy-α-L-lyxopyranoside, a diaminosugar similar to a minor groove binding carbohydrate—daunosamine—and test the neutral platinum(II) complex as a potential antitumor drug. It has appeared, that the complex indeed exhibits a biological activity against two mouse lymphoma cell lines (L5178Y) which differ in their double strand breaks (DSB) and nucleotide excision repair (NER) [18,19]. So, the question arises if the cationic complex also exhibits any antitumor activity. In the presented studies, we have prepared and structurally characterised the cationic complex $[Pt(C_7H_{16}N_2O_2)(DMSO)Cl]NO_3 \cdot DMSO$ where one DMSO molecule replaces the chloride anion in the square-planar neutral complex. The antitumor activity of the complex was tested against standard the L1210 murine leukemia cell line.

2. Results

2.1. Structural studies

The molecular structure of the cationic complex was studied using the FT-IR spectroscopic and single-crystal X-ray diffraction methods. Vibrational spectra (Fig. 1) have shown that the cationic complex retains a free molecule of DMSO even after recrystallisation from alcohol. Splitting of the S–O stretching vibration, observed in the region of 1030 cm^{-1}, confirms the existence of two differently bound DMSO molecules. The full infrared spectrum presents the characteristic ν(N–H) absorption bands which form a broad peak in the region of 3430 cm^{-1}, and two strong bands at 1565 and 1608 cm^{-1}, assigned to numerous $-NH_2$ binding modes. The C(1)–H bending vibration at 862 cm^{-1} confirms the α-anomeric configuration of L-lyxose.

Single-crystal X-ray diffraction studies of the investigated complex, $[Pt(C_7H_{16}N_2O_2)(DMSO)Cl]NO_3 \cdot DMSO$ ($M = 607.01$) have shown that the complex crystallises in the orthorombic form (space group $P2_12_12_1$; No. 19) with $a = 10.165(2)$ Å, $b = 12.756(3)$ Å, $c = 15.868$ (3) Å, $U = 2057.5(7)$ Å^3, $Z = 4$, $F(0\,0\,0) = 1184$ and calc. density = 1.960 g cm^{-3}. Data concerning

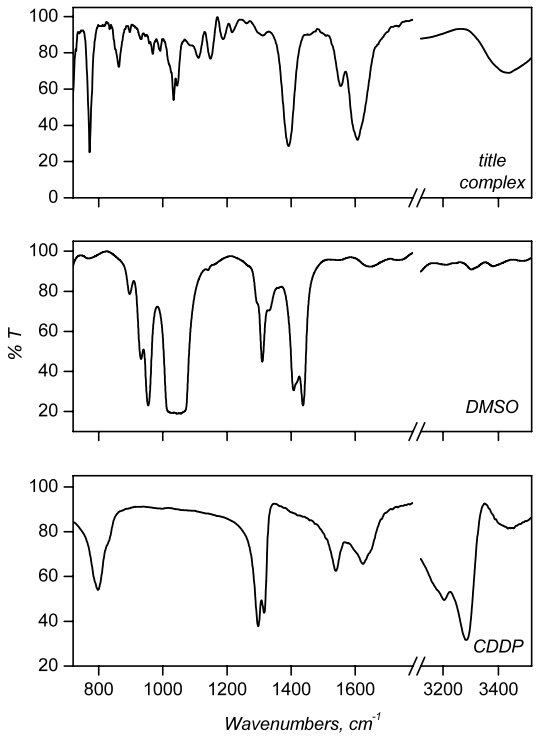

Fig. 1. Vibrational spectra of the cisplatin, DMSO and the title complex ($[Pt(C_7H_{16}N_2O_2)(DMSO)Cl]NO_3 \cdot DMSO$).

[2] *cis*-amminedichloro(2-methylpyridine)platinum(II), formerly known as JM473 and AMD473.

the detailed bond lengths and angles are presented in Table 1. An ORTEP view of the cationic $[Pt(C_7H_{16}N_2O_2)(DMSO)Cl]^+$ species is shown in Fig. 2. The results obtained have confirmed that the crystal structure of the cationic species is similar to that of the cisplatin molecule. However, it consists of the cationic $[Pt(diaminosugar)(DMSO)Cl]^+$ species, the $[NO_3]^-$ anions and free DMSO molecules present in the crystal lattice. The bidendate diaminosugar, dimethylsulfoxide and the chloride anion complete the square plane around the platinum atom. The Pt–S bond length is equal to 2.203(5) Å, which correlates well with the values obtained, e.g. by Lippard and coworkers [20] and Elding and coworkers [21]. The platinum(II)–sulphur–oxygen line (Pt–S–O) forms an angle of 115.2(6)°.

The packing of the diaminosugar cationic complex is illustrated in Fig. 3. The structure consists of the following layers: the cationic Pt complexes, free dimethylsulfoxide molecules and the nitrate anions. Distance of the free dimethylsulfoxide molecules from the nitrogen atoms of the diaminosugar was established to be O(4)···N(1) = 2.913 Å and O(4)···N(2) = 2.942(6) Å, while from the nitrate anions O(5)···S(2) distance 3.444(7) Å. The Pt···Pt distance in the packed unit cell of the monomeric, cationic diaminosugar platinum complex is long, >5 Å (5.833 Å), whereas in the neutral, dimeric diaminosugar complex is shortened to about 3 Å [18].

2.2. Biological tests

Biological tests were carried out using the mouse lymphoma L1210 cells. The detailed results are presented in Table 2 and Fig. 4.

L1210 cells were treated with the investigated platinum(II) complex or with the free *O*-metyl-3,4-diamine-2,3,4,6-tetradeoksy-α-L-lyxo-hexopyranoside, respectively. Cisplatin was used as a reference compound. No significant toxicity of the aminosugar was found, up to the highest concentration tested -0.1 mg cm^{-3} (the data not shown), while CDDP appeared to be highly toxic (Fig. 4). Although the ID_{50} values are similar within the experimental errors for both platinum complexes investigated (about 0.01 mg cm^{-3}), the ID_{90} values show that CDDP is about two times more toxic than the investigated complex. The interpolated values are 0.08 ± 0.03 and 0.13 ± 0.05 mg cm^{-3}, respectively.

3. Experimental

3.1. Synthesis of $[Pt(C_7H_{16}N_2O_2)(DMSO)Cl]\cdot NO_3$ complex

cis-Diaminedichloroplatinum and other chemicals, if not otherwise indicated, were purchased from Sigma-

Table 1
Bond lengths (Å) and angles (°) for $[Pt(C_7H_{16}N_2O_2)(DMSO)Cl]NO_3\cdot$ DMSO complex

Bond lengths	
Pt(1)–N(2)	2.007(16)
Pt(1)–N(1)	2.072(14)
S(1)–O(3)	1.461(13)
S(1)–C(8)	1.76(2)
N(1)–C(3)	1.50(2)
O(1)–C(5)	1.45(2)
C(1)–O(2)	1.36(2)
O(2)–C(6)	1.37(2)
C(2)–C(3)	1.54(2)
C(3)–C(4)	1.52(2)
S(2)–O(4)	1.542(15)
S(2)–C(21)	1.75(2)
N(3)–O(5)	1.18(2)
N(3)–O(6)	1.25(2)
Pt(1)–S(1)	2.203(5)
Pt(1)–Cl(1)	2.285(5)
S(1)–C(9)	1.80(2)
N(2)–C(4)	1.49(2)
O(1)–C(1)	1.46(2)
C(1)–C(2)	1.49(2)
C(4)–C(5)	1.50(3)
C(5)–C(7)	1.52(3)
S(2)–C(22)	1.78(2)
N(3)–O(7)	1.25(2)
Bond angles	
N(2)–Pt(1)–N(1)	83.4(5)
N(1)–Pt(1)–S(1)	176.4(5)
N(1)–Pt(1)–Cl(1)	90.8(4)
S(1)–Pt(1)–Cl(1)	91.87(18)
O(3)–S(1)–Pt(1)	115.2(6)
C(3)–N(1)–Pt(1)	107.7(11)
C(8)–S(1)–Pt(1)	111.0(8)
O(3)–S(1)–C(8)	108.9(11)
C(8)–S(1)–C(9)	99.7(12)
C(1)–O(1)–C(5)	113.9(15)
O(2)–C(1)–O(1)	110.4(15)
O(1)–C(1)–C(2)	109.9(15)
C(1)–C(2)–C(3)	109.0(16)
C(4)–C(3)–N(1)	110.7(15)
N(1)–C(3)–C(2)	107.0(15)
C(5)–C(4)–C(3)	110.2(16)
O(1)–C(5)–C(4)	109.7(17)
O(4)–S(2)–C(21)	108.3(10)
C(21)–S(2)–C(22)	96.3(13)
O(5)–N(3)–O(6)	122.3(19)
O(6)–N(3)–O(7)	114.4(19)
N(2)–Pt(1)–S(1)	93.9(4)
N(2)–Pt(1)–Cl(1)	174.1(4)
C(4)–N(2)–Pt(1)	113.4(11)
C(9)–S(1)–Pt(1)	110.7(8)
O(3)–S(1)–C(9)	110.4(11)
C(1)–O(2)–C(6)	114.7(17)
O(2)–C(1)–C(2)	112.9(16)
C(4)–C(3)–C(2)	111.1(13)
C(5)–C(4)–N(2)	115.8(15)
N(2)–C(4)–C(3)	105.7(15)
C(4)–C(5)–C(7)	116.2(17)
O(1)–C(5)–C(7)	104.1(18)
O(4)–S(2)–C(22)	109.8(11)
O(5)–N(3)–O(7)	123.0(2)

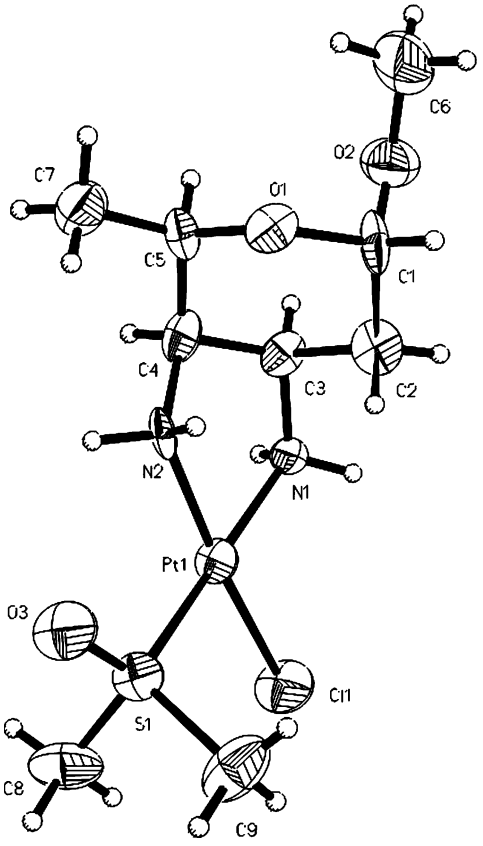

Fig. 2. Molecular structure of the cationic complex $[Pt(C_7H_{16}N_2O_2)(DMSO)Cl]^+$ together with atom labeling.

Aldrich. They were used without further purification. The carbohydrate (**1**) ligand was synthesised in several steps from 3,4-di-*O*-acetyl-L-rhamnal as described in [18] using the procedure of Kolar et al. [22]. The neutral platinum(II) complex, $Pt(C_7H_{16}N_2O_2)Cl_2$ (**2**) was obtained using procedure of Tsubomura et al. [23,24]. The details of the synthesis are described elsewhere [18].

Table 2
Survival fraction and estimated ID_{50} of L1210 cells treated with Platinum(II) cationic complex or cis-Pt, respectively

Dose (mg cm^{-3})	Surviving fraction [a]	
	Title platinum(II) cationic complex	*cis*-Pt (CDDP)
0.001	0.83 ± 0.05	0.78 ± 0.08
0.005	0.75 ± 0.14	0.58 ± 0.11
0.01	0.43 ± 0.08	0.50 ± 0.17
0.05	0.34 ± 0.12	0.12 ± 0.01
0.1	0.17 ± 0.04	0.06 ± 0.01
	ID_{50} (mg cm^{-3})	
50% inhibition dose	0.009 ± 0.002	0.009 ± 0.003

[a] Mean ± S.D.; $n = 3$.

The title complex $[Pt(C_7H_{16}N_2O_2)(DMSO)Cl]\cdot NO_3$ (**3**) has been prepared according to the reaction:

$$Pt(C_7H_{16}N_2O_2)Cl_2 + DMSO + AgNO_3 \rightarrow [Pt(C_7H_{16}N_2O_2)(DMSO)Cl]NO_3 + AgCl$$

by mixing the neutral complex (**2**) with an equimolar amount of $AgNO_3$ in the methanol–DMSO solution. After filtering off the AgCl precipitated, the resulting solution was diluted with acetone until a solid reaction product appeared. The white crystals obtained remain stable in the solution unless a competing ligand has been added. Chloride ions reduce their stability. The chloride ion re-enters the coordination sphere of the cation

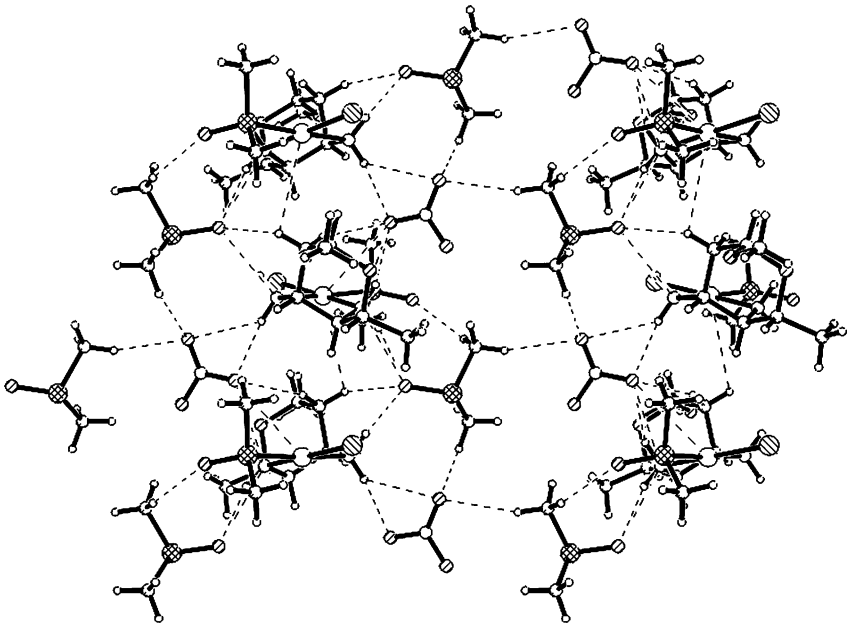

Fig. 3. Network of the title complex, $[Pt(C_7H_{16}N_2O_2)(DMSO)Cl]NO_3\cdot DMSO$, formed by the hydrogen–oxygen interactions.

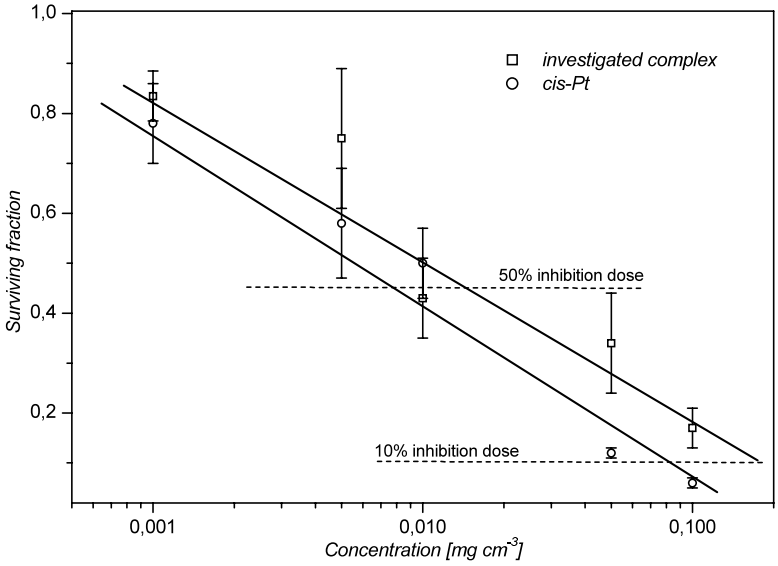

Fig. 4. Relative cell numbers in L1210 cell cultures treated with cisplatin or the tested drug.

giving the neutral dichlorocomplex. Such lability of the coordinated sulfoxide molecule has already been observed elsewhere [25].

3.2. Structural studies

IR spectra of the title complex (**3**) were recorded using a Bruker Equinox 55 FT-IR spectrometer within the 4.000–400 cm^{-1} range with the resolution of 2 cm^{-1}. Spectra of the solid samples were registered after the pelletisation in KBr (about 1% of the investigated species) and using the ATR technique. No significant differences in both series of spectra were detected.

Vibrational spectra of DMSO were registered in the cell equipped with the KBr windows.

A detailed assignment of the peaks were made according to [26] and the references cited therein. The main FT-IR bands of the title complex ($\bar{\nu}$, cm^{-1}) are: 3434, 2353, 2326, 1608, 1565, 1392, 1045, 1033, 862, 771, 713 and 678.

Studies on the crystal structure of the studied complex (**3**) were performed on a KUMA KM4CCD κ-axis diffractometer, applying a graphite-monochromated MoKα radiation. The data were collected at a room temperature, using the ϖ-2θ scan technique. The intensity of the control reflections varied by less than 3%, and the linear correction factor was applied to account for the effect. All the data were corrected for Lorentz and polarisation effects. No absorption correction was applied. Data reduction and analysis were carried out using the KUMA diffraction (Wroclaw) programs. The structure of the investigated crystals was solved by the direct methods [27] and refined using SHELXL computer program [28]. The refinement was based on F^2 for all reflections, except for those with the strongly negative F^2 values. Weighted R factors, wR, and all goodness-of-fit S values are based on the F^2 parameters. Conventional R factors are based on F with F set to zero for negative F^2. The $F_o^2 > 2s(F_o^2)$ criterion was used only for calculation of the R factors and is not relevant to choice of reflections for the refinement. The R factors based on F^2 were about twice as large as those based on F. All hydrogen atoms placed in the calculated positions and their thermal parameters were refined isotropically. Scattering factors were taken from the literature (Tables 6.1.1.4 and 4.2.4.2 in Ref. [29]).

Summary of the experimental details is presented in Table 3.

3.3. Biological tests

All chemicals used for biological tests were also purchased from Sigma.

Cytotoxicity of the studied complex was estimated in vitro by means of the relative growth test with 1 h exposure to the drug, as described earlier [30]. Studies were performed with mouse lymphoma cell line L1210. The cells investigated were centrifuged and re-suspended in fresh culture medium (RPMI 1640 medium supplemented with 10% of the foetal calf serum and antibiotics). The number of viable and dead cells was determined 48 h after treatment, using a Buerker haemocytometer and nigrosine exclusion test. The relative cell number was determined as a ratio of the number of viable cells in the test culture to that in the control culture. The 50% inhibition dose (ID_{50}) was determined by extrapolation of a steep part of survival curve (drug concentrations of 0–0.01 mg cm^{-3}), whereas the 90% growth inhibition dose (ID_{90}) was determined by extrapolation of a plateau part of

Table 3
Summary of the structure refinement parameters

Temperature (K)	293(2)
Wavelength (A)	0.71073
Absorption coefficient (mm^{-1})	7.187
Crystal size (mm^3)	$0.1 \times 0.07 \times 0.04$
Theta range for data collection (°)	3.26–22.49
Index ranges	$-8 \le h \le 13$, $-17 \le k \le 16$, $-21 \le l \le 20$
Reflections collected/unique	9441/2679 [$R_{int} = 0.0602$]
Refinement method	Full-matrix least-squares on F^2
Data/restraints/parameters	2665/0/253
Goodness-of-fit on F^2	1.061
Final R indices [$I > 2\sigma(I)$]	$R_1 = 0.0620$, $wR_2 = 0.1324$
R indices (all data)	$R_1 = 0.0723$, $wR_2 = 0.1390$
Largest difference peak and hole ($e\,Å^{-3}$)	0.953 and −0.856

survival curve (drug concentrations of 0.01–0.1 mg cm^{-3}). Precision of the calculated ID_{50} and ID_{10} values was estimated using propagation of measurement uncertainty technique, and the error limits of the estimates for the slope and intercept of survival curves.

4. Supplementary material

Crystallographic data for the structural analysis have been deposited at the Cambridge Crystallographic Data Centre, CCDC No. 166399. Copies of this information may be obtained free of charge from The Director, CCDC, 12 Union Road, Cambridge CB2 1EZ, UK (Fax: +44-1223-336033; e-mail: deposit@ccdc.cam.ac.uk or www: http://www.ccdc.cam.ac.uk).

Acknowledgements

Methyl-3,4-diamino-2,3,4,6-tetradeoxy-α-L-lyxopyranose ($C_7H_{14}N_2O_2$) has been synthesised in the Texas University, M.D. Anderson Cancer Center (Houston, TX). Pt (II) complex has been obtained and X-ray measurements have been undertaken at the Chemistry Department of the Warsaw University. FT-IR spectra have been recorded and biological tests performed in the Institute of Nuclear Chemistry and Technology. This research was supported by the State Committee for Scientific Research (KBN; Poland) in the frame of the Research Contract No. 4-PO5F-037-15.

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