Available online at www.sciencedirect.com
ScienceDirect
European Journal of Medicinal Chemistry 42 (2007) 817–822
EUROPEAN JOURNAL OF MEDICINAL CHEMISTRY
http://www.elsevier.com/locate/ejmech

Original article

In vivo insulin-mimetic activity of [*N*,*N*′-1,3-propyl-bis(salicyladimine)]oxovanadium(IV)

Mingjin Xie [a,*], Guangjun Xu [b], Ling Li [c], Weiping Liu [d], Yanfen Niu [c], Shiping Yan [b,**]

[a] *Department of Chemistry, Yunnan University, Cuihu North Road No. 2, Kunming, Yunnan 650091, China*
[b] *Department of Chemistry, Nankai University, Tianjin 300071, China*
[c] *Yunnan Pharmacological Laboratories of Natural products, Kunming Medical College, Kunming 650031, China*
[d] *Kunming Institute of Precious Metals, Kunming 650221, China*

Received 19 December 2005; received in revised form 5 December 2006; accepted 5 December 2006
Available online 12 January 2007

Abstract

[*N*,*N*′-1,3-Propyl-bis(salicyladimine)]oxovanadium(IV) − an oxovanadium complex has been tested for bioactivity as an insulin-enhancing agent. Its structure reveals that the vanadium(IV) ion is hexacoordinated and situated in a distorted octahedral environment. The complex was administered intragastrically to both normal and streptozotocin (STZ)-diabetic rats for two weeks. The results showed that the complex, at a dose of 10.0 and 20.0 mg V·kg^{-1}, could lower the blood glucose level in STZ-diabetic rats and improve the response to an oral glucose challenge. This complex did not alter these parameters in normal rats. It was suggested that the complex exerted an antidiabetic effect in STZ-diabetic rats.
© 2007 Elsevier Masson SAS. All rights reserved.

Keywords: Oxovanadium; *N*,*N*′-1,3-Propyl-bis(salicyladimine); Insulinomimetic activity

1. Introduction

Interest in vanadium coordination chemistry over the past decade has been accelerated because of its biological importance [1−3]. Diabetes mellitus (DM) which is expected to be the most significant disease in the 21st century [4−6] is generally classified into two main types, namely type 1 DM and type 2 DM [7]. Type 1diabetes is insulin dependant as it is the result of absolute insulin deficiency, whereas type 2 diabetes is characterized by a relative insulin deficiency due to low insulin sensitivity and is therefore not insulin dependent for survival. Although several types of insulin preparations and synthetic therapeutics are available for clinical use in patients with diabetes, none of the current treatment strategies reinstate physiological insulin release or address all underlying cellular lesions of diabetes. Current therapies also have unwanted side effects and contra-indicatious for use [8,9]. Therefore, the development of new approaches to the treatment of DM remains necessary.

The insulin-like effect of vanadium salts on cells [10,11] and diabetic animals [12−18] has been known since 1980s. However, these compounds are poorly absorbed and required high doses that were associated with undesirable side effects. In order to achieve a better absorption and to reduce the doses of the element, it seemed to be appropriate to administer vanadium in the form of an organic matrix. A large class of compounds based on V(IV) chelate complexes [13,19] have been extensively studied mainly by the Sakurai and Orvig/McNeill groups, respectively [13,16,20,21]. [*N*,*N*′-Bis(salicylidene)-ethane-1,2-diaminato]oxovanadium(IV), VOSALEN, was prepared by first synthesizing the ligand, and then complexing the ligand to vanadium, by usual methods [22]. It was found to be air-stable and sparingly water-soluble. The coordination geometry was suggested to be octahedral in DMSO, but square pyramidal in the solid state. VOSALEN was orally effective

* Corresponding author. Tel./fax: +86 0871 5035640.
** Corresponding author. Tel.: +86 022 23509957; fax: +86 022 23502779.
E-mail addresses: xmj7193@sohu.com (M. Xie), yansp@nankai.edu.cn (S. Yan).

0223-5234/$ - see front matter © 2007 Elsevier Masson SAS. All rights reserved.
doi:10.1016/j.ejmech.2006.12.021

(7.5 mmol kg^{-1} d^{-1} for 30 days) for glucose lowering in alloxan-induced diabetic rats; however, rats tended to become hypoglycemic, and withdrawal of treatment brought an immediate return to hyperglycemia [23].

In this paper, we report the results of insulin-mimetic tests of a $V^{IV}O$ complex, [*N*,*N*′-1,3-propyl-bis(salicyladimine)]oxovanadium(IV). The evaluation of the insulin-like action of the complex was undertaken in streptozotocin (STZ)-induced diabetic rats.

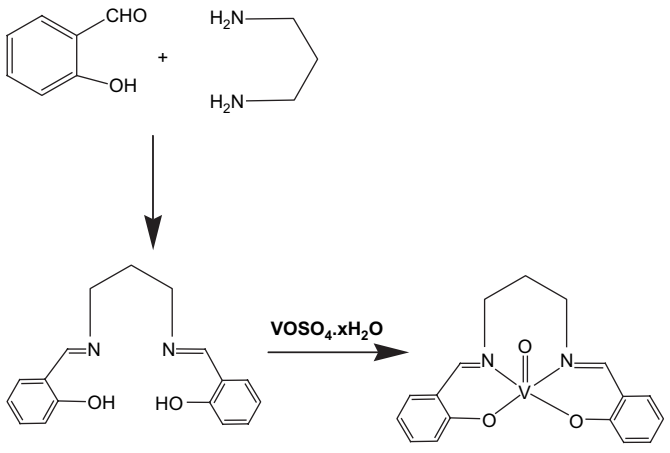
2. Results and discussion

2.1. Preparation of complex

Scheme 1 shows the reactions of salicylaldehyde and *N*,*N*′-1,3-diaminopropane with vanadyl sulfate produces a monomeric V^{IV} complex.

[*N*,*N*′-1,3-Propyl-bis(salicyladimine)]oxovanadium(IV) (VOL) was very air-stable in the solid state; it can be stored for months and it exhibits a magnetic moment at room temperature in the solid state. With an electron configuration of $[Ar]3d^1$, V(IV) has one unpaired electron for which the spin-only formula predicts a magnetic moment of 1.63 BM. VO(FA) has an effective magnetic moment (μ_{eff}) of 1.76 BM at 300 K, and this is a typical value for a V(IV) complex with S = 1/2. This confirmed that the complex is in the mononuclear vanadyl state. The ESR spectrum of VOL at room temperature exhibited the characteristic widely spaced eight-lined pattern due to coupling of the unpaired electron with large moment of ~100% abundant ^{51}V nucleus (I = 7/2) (Fig. 1) indicated that only one mononuclear vanadium(IV) species predominates in the complex solution examined.

In the visible absorption spectrum of the complex, three absorption bands at 273, 362 and 745 nm were observed. Absorption bands at 273 nm can be assigned to $\pi \rightarrow \pi^*$ transition based on the ligand. Generally, vanadyl complexes with lower symmetry than C_{4v} usually give rise to three d–d bands, due to splitting of the degenerate d_{xz} and d_{yz} [24,25]. Two or three absorption bands assigned to d-d transitions [$d_{xy} \rightarrow (d_{xz}, d_{yz})$, $d_{xy} \rightarrow d_{x^2-y^2}$, and $d_{xy} \rightarrow d_{z^2}$ in the order of decreasing wavelength] were observed in the visible region [26]. Absorption bands at 745 nm can be assigned to a $d_{xy} \rightarrow (d_{xz}, d_{yz})$ transition. The increase of the ligand field strength is responsible for a blue shift of the absorption bands. Therefore, absorption bands at 362 nm can be assigned to a $d_{xy} \rightarrow d_{z^2}$ transition (Fig. 2). The IR absorption band due to V=O stretching frequency was found at 856 cm^{-1}.



Scheme 1. Formulas of the vanadium(IV) complex and its preparation.

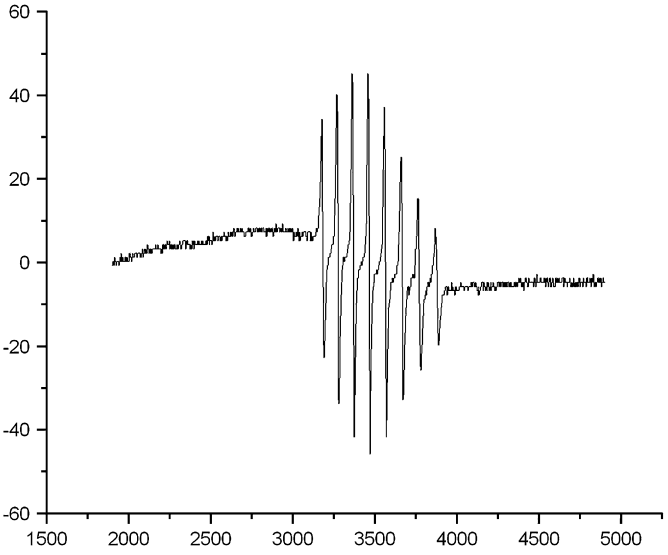


Fig. 1. X-band ESR spectra of VOL in DMSO (at room temperature).

2.2. Structural results

The structure of V(IV)O(SALPN)·DMSO (Fig. 3) is similar to that described in a previous report [27,28]. Intramolecular bond lengths and angles around the metal ion in VOL (Table 1) and an ORTEP drawing of the molecule (Fig. 3) are shown here.

The asymmetric unit of complex (I) consists of a monomeric vanadium(IV) complex and one dimethyl sulfoxide (DMSO) molecule. The V^{IV} atom is hexacoordinate in a distorted octahedral environment. The basal square plane is constituted by the *N*,*N*′-1,3-propyl-bis(salicyladimine) molecule, which acts as a tetradentate ligand through its *o*-phenylenediamine N atoms and its deprotonated phenol O atoms. The V atom is located 0.272(2) Å above the mean plane defined by atoms N1/N2/O1/O2. This distance is different to those observed for $VO(acac)_2$ (0.55 Å) [29] and [VO(acen)] [acen is *N*,*N*-ethylenebis(acetylacetoneiminate); 0.58 Å] [30]. The apical position is occupied by the oxo ligand; the $V-O_{oxo}$ bond distance of 1.596(6) Å is typical for five-coordinate vanadyl species. Different to other VO(salen) [*N*,*N*′-ethylenebis(salicylaldiminato)]oxovanadium(IV)-type compounds [31–33], the molecule forms discrete units with a DMSO oxygen atom in the sixth coordination site. One does not observe a sixth ligand in the VO(salen) structure while aqua and perchlorate molecules bind to $[VO(SALEN)]^+$. This may be the reason of $V^{IV}O$(SALPN)·DMSO in orange and $V^{IV}O$(SALEN) in blue-green.

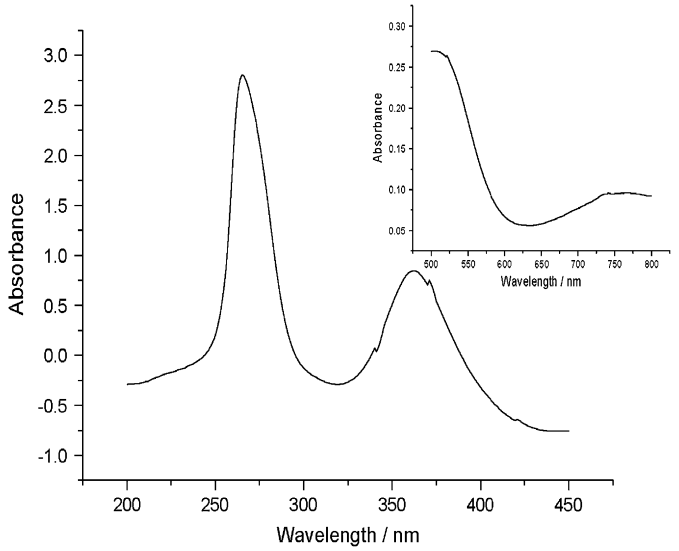


Fig. 2. Electronic absorption spectra of VOL in DMSO (at room temperature).

2.3. Effects of VOL on blood glucose in both normal and STZ-induced diabetic rats

During the experiment, the STZ-diabetic rats exhibited a significant increase in the fluid and food consumption, compared with the normal rats. After administration, VOL markedly reduced the intake of fluid in STZ-diabetic rats but not in normal rats. The food intake was transitory decreased in the VOL-treated STZ-diabetic rats, compared with the STZ-diabetic rats. The mean body weight in STZ-diabetic rats was lower than that in normal rats. After administration of VOL, the body weight, both in the VOL-treated diabetic groups and treated normal groups, was decreased in comparison with the diabetic and normal control groups, respectively (Fig. 4).

In present study, the blood glucose level in the treated normal rats (VOL 10.0 mg $V \cdot kg^{-1}$ ig for two weeks) was not altered as compared with the untreated normal rats ($P > 0.05$). The STZ-diabetic rats exhibited significant hyperglycemia.

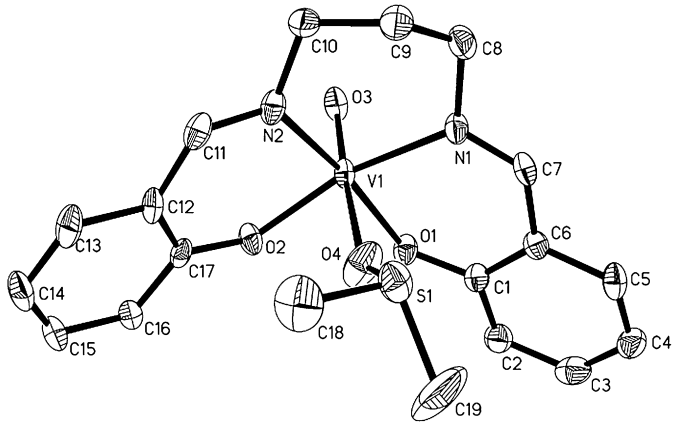


Fig. 3. [*N*,*N*′-1,3-Propyl-bis(salicyladimine)]oxovanadium(IV) complex showing the labeling scheme of the non-H atoms and their displacement ellipsoids at the 30% probability level.

Table 1
Bond lengths [Å] and angles [°] around the vanadium atom in [*N*,*N*′-1,3-propyl-bis(salicyladimine)]oxovanadium(IV), VOL

V(1)–O(3)	1.596(6)	O(3)–V(1)–O(2)	103.7(3)
V(1)–O(2)	1.953(5)	O(3)–V(1)–O(1)	101.7(3)
V(1)–O(1)	1.976(5)	O(2)–V(1)–O(1)	88.0(2)
V(1)–N(1)	2.100(6)	O(3)–V(1)–N(1)	93.6(3)
V(1)–N(2)	2.111(6)	O(2)–V(1)–N(1)	162.8(3)
V(1)–O(4)	2.229(7)	O(1)–V(1)–N(1)	88.0(2)
		O(3)–V(1)–N(2)	91.9(3)
		O(2)–V(1)–N(2)	88.1(2)
		O(1)–V(1)–N(2)	166.4(3)
		N(1)–V(1)–N(2)	92.0(2)

After two weeks administration with the complex (10.0 mg $V \cdot kg^{-1}$ and 20.0 mg $V \cdot kg^{-1}$ ig), the blood glucose level was decreased in a dose-dependent manner, compared with the diabetic control group ($P < 0.05$ and 0.01, respectively). It was suggested that the complex has hypoglycemic effect on STZ-diabetic rats (Figs. 5 and 6).

2.4. Oral glucose tolerance test (OGTT) [34]

OGTT was performed to investigate the effect of VOL on glucose tolerance. The glucose tolerance curve in the VOL-treated normal group was similar to that in the untreated normal group, suggesting that VOL did not influence the glucose tolerance in normal rats.

In the diabetic control group, the blood glucose levels showed a typical diabetic response to a glucose loading, but VOL administration for two weeks (VOL at 10 and 20 mg $V \cdot kg^{-1}$ ig) markedly lowered the blood glucose levels ($P < 0.05$ or $P < 0.01$) (Fig. 6). It showed that the complex could accelerate the glucose clearance and improve glucose tolerance in STZ-diabetic rats.

3. Conclusions

The $V^{IV}O(SALPN) \cdot DMSO$ complex is a discrete easily prepared vanadium compound which normalizes glucose and lipid values without an increase of insulin levels. Our data showed that VOL did not significantly affect the postprandial blood glucose of normal animals. In addition, our results showed that VOL improved the response to an oral glucose challenge in diabetic rats. A two-weeks administration with VOL resulted in a decrease of the blood glucose levels in STZ-diabetic rats. Furthermore, OGTT demonstrated that the impaired glucose tolerance in STZ-diabetic rats was ameliorated by VOL treatment. These findings suggested that VOL has an antidiabetic potency due to its blood glucose lowering and the abilities to improve glucose tolerance.

4. Experimental

4.1. Materials

Chemicals and solvents were of reagent grade and were used without further purification unless otherwise noted.

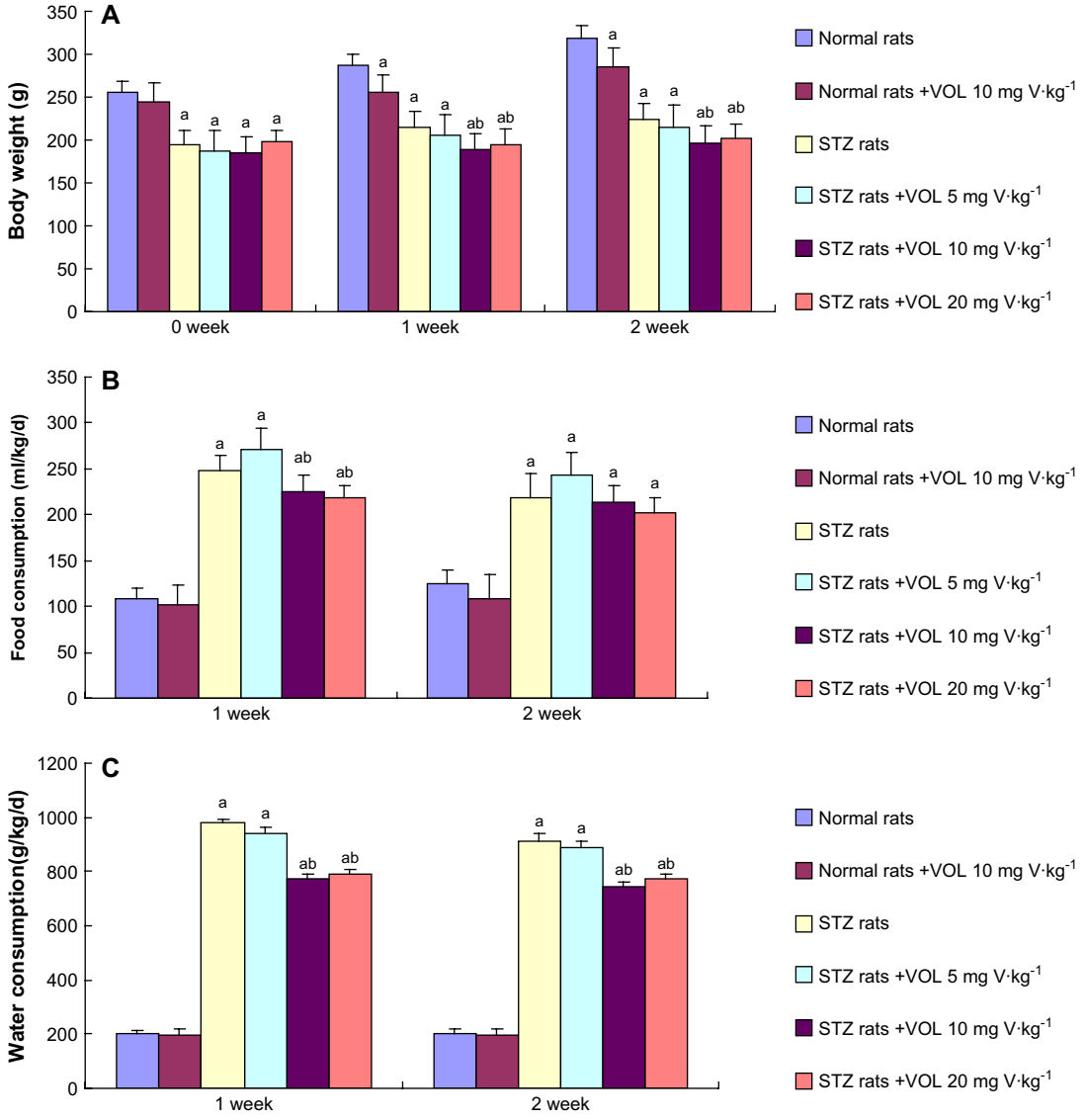


Fig. 4. Effects of VOL on body weight (A), food (B) and water (C) intake in normal and STZ-induced diabetic rats ($n = 9–10$). The diabetic rats were induced by a single intraperitoneal injection of STZ 50 mg kg^{-1}. CON: normal control group, 0.9% NaCl solution ig; CON + VOL: treated normal group, VOL 10 mg V·kg^{-1} ig; DM: diabetic group, 0.9% NaCl solution ig; DM + VOL L, M, H: treated diabetic group, VOL 5, 10, and 20 mg V·kg^{-1} ig, respectively. Values are mean ± SD. $^{a}P < 0.05$, $^{b}P < 0.01$, vs normal control (Student's *t*-test).

Vanadyl sulfate hydrate ($VOSO_4 \cdot xH_2O$, $x = 3–5$) was purchased from Aldrich. Salicylaldehyde and *N*,*N*′-1,3-diaminopropane were purchased from Shanghai Chemicals Reagents Ltd. Streptozotocin (STZ) was purchased from Sigma. Glucose reagent kit was a product of Shanghai institute of Biological Products.

4.2. Physical measurements

Elemental analyses were carried out using a Carlo-Erba 1106 elemental analyzer. IR spectra were recorded on a Nicolet 170SXFT-IR using a KBr disk technique. UV–vis spectra were recorded on a Shimadzu UV-2000 at room temperature. Electron spin resonance (ESR) spectra were recorded on a JEOL JES-RE1X spectrometer operated at a modulation amplitude width of 0.63 mT and microwave power of 5 mW at room temperature (298 K). Magnetic susceptibility (μ_{eff}) at room temperature was obtained by the Gouy method. The diamagnetic corrections were calculated from tables of Pascal's constants. The magnetic susceptibility (χ) was measured on a Quantum Design LDJ-9600 susceptometer operating at a magnetic field of 0.5 T between 80 and 300 K. All data were fitted to the Curie–Weiss equation. Biological activity experiments were measured on CL-770 Shimadzu Clinic Spectrometer, BS Semi-Automation Biochemistry analyzer which was purchased from Beijing Biochem Analyze Ltd. MP4R and OM8464 US-IEC freezing centrifugation instrument.

4.3. Synthesis of complex

The ligand L and complex were prepared using the published procedure [35,27,28]. The complex was synthesized

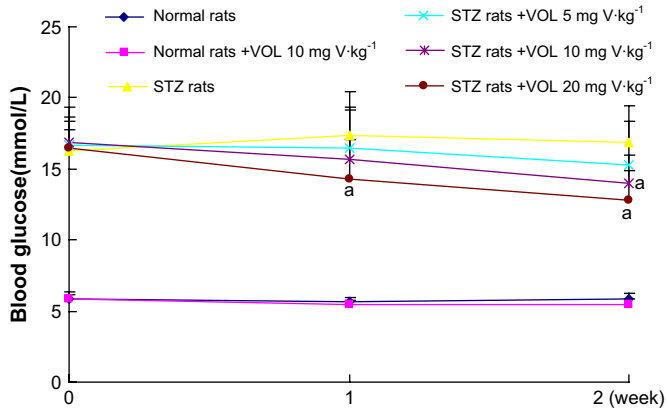


Fig. 5. Effects of intragastric VOL on blood glucose level in normal and STZ-diabetic rats ($n = 9$ or 10). The diabetic rats were induced by a single intraperitoneal injection of STZ 50 mg kg^{-1}. CON: normal control group, 0.9% NaCl solution ig; CON + VOL: treated normal group, VOL 10 mg $V \cdot kg^{-1}$ ig; DM: diabetic group, 0.9% NaCl solution ig; DM + VOL L, M, H: treated diabetic group, VOL 5, 10, and 20 mg $V \cdot kg^{-1}$ ig, respectively. Values are mean ± SD. $^{a}P < 0.05$, $^{b}P < 0.01$, vs normal control (Student's-t test).

by adding the ligand L (3 mmol) to methanol (10 ml) and added to an aqueous solution of $VOSO_4 \cdot xH_2O$ (3 mmol), then to an Et_3N (6 mmol) solution, and refluxed for 2 h. The green precipitate was collected by filtration. Crystals of suitable quality for X-ray analysis were obtained by slow evaporation of a dimethyl sulfoxide solution. $C_{19}H_{22}N_2O_4SV$, $M = 425.39$. Anal. Found: C, 53.42; H, 5.57; N, 6.16. Calc.: C, 53.60; H, 5.17; N, 6.58%. UV–vis (DMSO) $\lambda_{max} = 273$ ($\varepsilon = 2036\ M^{-1}\ cm^{-1}$), 362 ($\varepsilon = 1633\ M^{-1}\ cm^{-1}$), 767 ($\varepsilon = 33\ M^{-1}\ cm^{-1}$) nm. IR (KBr disk): ν (cm^{-1}) = 856($\nu_{v=o}$), 527 (ν_{v-o}). ESR (RT, DMSO): 8-line pattern, $g_0 = 2.03 \pm 0.001$, $A_0 = 102.6 \pm 0.1 \times 10^{-4}\ cm^{-1}$. Magnetic moment (solid): 1.63 BM (one unpaired electron).

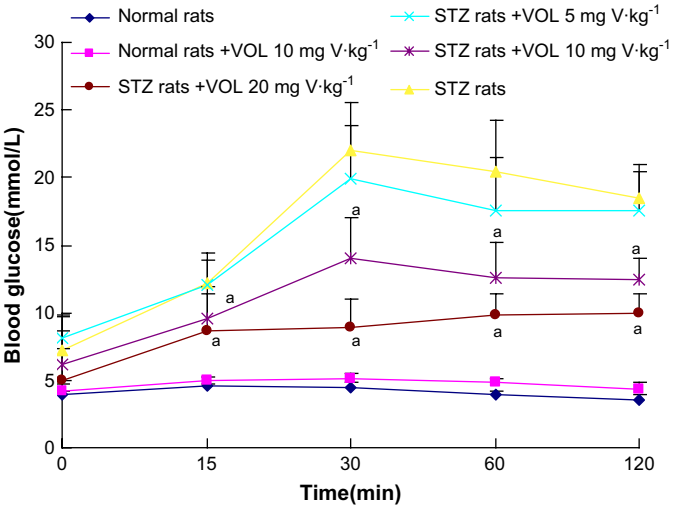


Fig. 6. Effect of VOL on glucose tolerance in normal rats and STZ-induced diabetic rats ($n = 9$ or 10). CON: normal control group, 0.9% NaCl solution ig; CON + VOL: treated normal group, VOL 10 mg $V \cdot kg^{-1}$ ig; DM: diabetic group, 0.9% NaCl solution ig; DM + VOL L, M, H: treated diabetic group, VOL 5, 10, and 20 mg $V \cdot kg^{-1}$ ig, respectively. Values are mean ± SD. $^{a}P < 0.05$, $^{b}P < 0.01$, vs normal control (Student's-t test).

Intensity data were collected at 293 K on a Bruker SMART 1000 CCD diffractometer for a rhombic block 0.18 × 0.14 × 0.08 mm^3.

4.4. X-ray diffraction data and crystal structure determination and refinement

Crystal data, the data collection procedure, structure determination methods and refinement results for complex 1 are summarized in Table 2. The hydrogen atoms were included in the molecular model at stereochemical positions and refined with the riding model. CCDC-232621 contains the supplementary crystallographic data for this paper. These data can be obtained free of charge at www.ccdc.cam.ac.uk/conts/retrieving.html [or from Cambridge Crystallographic Data Centre, 12 Union Road, Cambridge CB2 1EZ, UK; fax: (internat.)+44-1223/336-033; E-mail: deposit@ccdc.cam.ac.uk].

4.5. Glucose-lowering studies

Male Sprague–Dawley rats weighing 200–250 g were obtained from Experimental Animal Center, Kunming Medical Collage (Grade II, Certificate number 2005018). Rats were allowed free access to standard solid food for laboratory animals and tap water. Diabetes was induced by a single intraperitoneal injection of STZ 50 mg kg^{-1} in 0.1 mol L^{-1} citrate buffer (pH 4.4). Seven days after STZ injection, blood samples for analysis of blood glucose were obtained from the tail vein of the rats and the blood glucose level was measured by the glucose

Table 2
Crystal data and structural refinements for the complex

Empirical formula	$C_{19}H_{22}N_2O_4SV$
F_w	425.39
Temperature/K	293(2)
Wavelength/nm	0.71073
Crystal system	Monoclinic
Space group	P2(1)
Unit cell dimensions (nm, °)	$a = 11.463(5)$ $\alpha = 90$
	$b = 7.785(3)$ $\beta = 109.551(7)$
	$c = 11.668(5)$ $\gamma = 90$
Volume/nm^3	981.2(7)
z	2
Density (calc.)/g cm^{-3}	1.440
μ/mm^{-1}	0.639
$F(000)$	442
Crystal size/mm^3	0.18 × 0.14 × 0.08
θ range/°	1.85–25.01
Limiting indices	$-13 \le h \le 13$
	$-9 \le k \le 4$
	$-13 \le l \le 13$
Reflection collected	5020
Independent reflection	2341 [R(int) = 0.0557]
Max. and min. Transmission	0.950 and 0.889
Refinement method	Full-matrix least-squares on F^2
Data/restraints/parameters	2341/1/254
Goodness-of-fit on F^2	1.074
Final R indices [$I > 2\sigma(I)$]	$R1 = 0.0561$, $wR2 = 0.1247$
R indices (all data)	$R1 = 0.0842$, $wR2 = 0.1585$
Largest diff. peak and hole/e·Å^{-3}	0.358 and −0.469

oxidase method. The STZ-rats with the blood glucose level $\geqq 11.1\ \mathrm{mmol\,L^{-1}}$ were considered as diabetic rats. Normal rats were injected with 0.1 M citrate buffer alone.

The experimental animals were randomly divided into six groups of ten rats according to the blood glucose level. Normal and diabetic control groups were treated with 0.9% saline; treated normal rats received VOL $10\ \mathrm{mg\,V\cdot kg^{-1}}$ and treated STZ-diabetic rats were given VOL 5, 10, $20\ \mathrm{mg\,V\cdot kg^{-1}}$ ig. The substances above were dissolved in water (solution was prepared fresh daily) and administered intragastrically once a day at the volume of $10\ \mathrm{ml\,kg^{-1}}$ for two weeks. During experimental period, the blood glucose level and body weight were monitored weekly; food and water intakes were checked daily.

4.6. Oral glucose tolerance test [34]

Normal and STZ-diabetic rats were prepared and divided into six groups as described in Section 4.5. After the drugs were administered intragastrically in both normal and STZ-induced diabetic rats for two weeks, an oral glucose tolerance test (OGTT) was undertaken. The experimental rats were fasted for 14 h and given an oral glucose challenge at a dose of 2.0 g/kg body weight [34]. Blood glucose levels were measured at 0, 15, 30, 60, 120 min after glucose loading.

4.7. Statistics

Values were presented as means ± standard deviations. Differences between drugs and control were analyzed by Student's *t*-test.

Acknowledgements

This work was supported by National Natural Science Foundation of China (30260118), Yunnan Natural Science Foundation (2002C0019R) and Yunnan University Science Foundation (2005Q002A).

References

[1] S.S. Amin, K. Cryer, B. Zhang, S.K. Dutta, S.S. Eaton, O.P. Anderson, S.M. Miller, B.A. Reul, S.M. Brichard, D.C. Crans, Inorg. Chem. 39 (2000) 406–416.
[2] M. Kaliva, T. Giannadaki, A. Salifoglou, C.P. Raptopoulou, A. Terzis, V. Tangoulis, Inorg. Chem. 40 (2001) 3711–3718.
[3] S. Ghosh, K.K. Nanda, A.W. Addison, R.J. Butcher, Inorg. Chem. 41 (2002) 2243–2248.
[4] R.A. DeFronzo, R.C. Bonadonna, E. Ferrannini, Diabetes Care 15 (1992) 318–368.
[5] H. Yki-Jarvinen, Endocr. Rev. 13 (1992) 415–431.
[6] A. Vinik, G. Pittenger, R. Rafaeloff, L. Rosenberg, W. Duguid, Diabetes Rev. 4 (1996) 235–263.
[7] WHO. Diabetes mellitus; Report of a WHO study group. WHO Technical Report Series 727 (1985) 91–113.
[8] L.J. Wagenaar, E.M. Kuck, J.B. Hoekstra, Neth. J. Med. 55 (1999) 4–12.
[9] D. Luft, R.M. Schmulling, M. Eggstein, Diabetologia 30 (1978) 75–87.
[10] Y. Shechter, S.J.D. Karlish, Nature 284 (1980) 556–558.
[11] J. Meyerovitch, Z. Farfel, J. Sack, Y. Shechter, J. Biol. Chem. 262 (1987) 6658–6662.
[12] C.E. Heyliger, A.G. Tahiliani, J.H. McNeill, Science 227 (1985) 1474–1477.
[13] H. Sakurai, K. Tsuchiya, M. Nukatsuka, M. Sofue, J. Kawada, J. Endocrinol. 126 (1990) 451–459.
[14] J.H. McNeil, V.G. Yuen, H.R. Hoveyda, C. Orvig, J. Med. Chem. 35 (1992) 1489–1491.
[15] V.G. Yuen, P. Caravan, L. Gelmini, N. Glover, J.H. McNeill, I.A. Setyawati, Y. Zhou, C. Orvig, J. Inorg. Biochem. 68 (1997) 109–116.
[16] K. Kawabe, M. Tadokoro, Y. Kojima, Y. Fujisawa, H. Sakurai, Chem. Lett. (1998) 9–10.
[17] B.A. Reul, S.S. Amin, J.-P. Buchet, L.N. Ongemba, D.C. Crans, S.M. Brichard, Br. J. Pharmacol. 126 (1999) 467–477.
[18] D.C. Crans, L. Yang, J.A. Alfano, L.-H. Chi, W. Jin, M. Mahroof-Tahir, K. Robbins, M.M. Toloue, L.K. Chan, A.J. Plante, R.Z. Grayson, G.R. Willsky, Coord. Chem. Rev. 237 (2003) 13–22.
[19] H. Sakurai, K. Fujii, H. Watanabe, H. Tamura, Biochem. Biophys. Res. Commun. 214 (1995) 1095–1101.
[20] K.H. Thompson, J.H. McNeill, C. Orvig, Chem. Rev. 99 (1999) 2561–2572.
[21] H. Yasui, A. Tamura, T. Takino, H. Sakurai, J. Inorg. Biochem. 91 (2002) 327–328.
[22] J.A. Bonadies, C.J. Carrano, J. Am. Chem. Soc. 108 (1986) 4088–4095.
[23] N. Durai, G. Saminathan, J. Clin. Biochem. Nutr. 22 (1997) 31–35.
[24] J.E. Drake, J.E. Vekris, J.S. Wood, J. Chem. Soc. A 90 (1968) 1000–1005.
[25] N.D. Chasteen, R.L. Belford, I.C. Paul, Inorg. Chem. 8 (1969) 408–418.
[26] C.J. Ballhausen, H.B. Gray, Inorg. Chem. 1 (1962) 111–122.
[27] M. Mathaw, A.J. Carty, G.J. Palenik, J. Am. Chem. Soc. 92 (1970) 3197–3198.
[28] A.R. Charles, D.H. James, R.C. Charles, W.K. Jeff, L.P. Vincent, Inorg. Chem. 32 (1993) 3855–3861.
[29] R.P. Dodge, D.H. Templeton, A. Zalkin, J. Chem. Phys. 35 (1961) 55–67.
[30] D. Bruins, D.L. Weaver, Inorg. Chem. 9 (1970) 130–135.
[31] M. Pasquali, F. Marchetti, C. Floriani, M. Cesari, Inorg. Chem. 19 (1980) 1198–1202.
[32] P.E. Riley, V.L. Pecoraro, C.J. Carrano, J.A. Bonadies, K.N. Raymond, Inorg. Chem. 25 (1986) 154–160.
[33] X. Wang, X.M. Zhang, H.X. Liu, Transit. Met. Chem. 19 (1994) 611–613.
[34] P. Basnet, S. Kadota, M. Shimizu, Y. Takata, M. Kobayashi, T. Namba, Planta Med. 61 (1995) 402–405.
[35] Y. Elderman, I. Sovboda, H. Feuss, Z. Kristallogr. 196 (1991) 309–310.