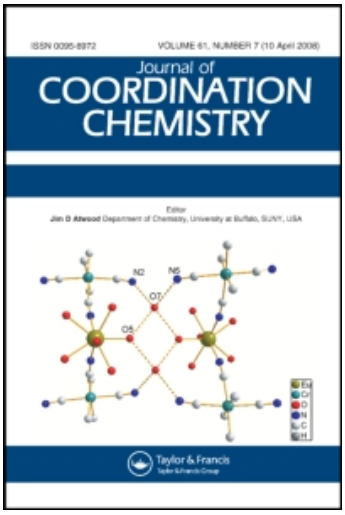

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Synthesis, crystal structure, and biological activity of a nickel(II) complex of 2-acetylpyridine N(4)-methylthiosemicarbazone

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Synthesis, crystal structure, and biological activity of a nickel(II) complex of 2-acetylpyridine N(4)-methylthiosemicarbazone

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Nickel complex formulated as $Ni(L)_2$ (L = monodeprotonated ligand corresponding to 2-acetylpyridine N(4)-methylthiosemicarbazone, HL) has been synthesized and characterized by elemental analysis, IR spectra, and single-crystal X-ray diffraction. The complex consists of discrete monomeric molecules with octahedral nickel(II) with two anionic 2-acetylpyridine N(4)-methylthiosemicarbazones as NNS tridentate ligands coordinated to nickel *via* the pyridine nitrogen, azomethine nitrogen, and sulfur. Hydrogen bonds link the different components to stabilize the crystal structure. Biological studies, carried out *in vitro* against bacteria, fungi, and the K562 leukemic cell line have shown that the free ligand and complex show distinct differences in biological activity.

Keywords: Thiosemicarbazone complex; Crystal structure; Cytotoxic activity

1. Introduction

The search for new antibacterial and antitumor agents has immense importance for drug development. Considerable attention has been focused on substituted thiosemicarbazone derivatives due to their coordination chemistry [1] and biological activity, antiparasital, antibacterial, and antitumor activities [2–7]. Triapine, a very good case in point, has been tested in phase I trial for patients with advanced solid tumors [8]. The biological activities of thiosemicarbazones often show a high dependence on their substituents. Earlier reports on N(4)-substituted thiosemicarbazones have concluded that the presence of bulky groups at N(4) of the thiosemicarbazone moiety greatly enhances the biological activity [9–11]. In addition, metal complexes of thiosemicarbazones exhibit bioactivities which differ from those of either ligands or metal ions [12–16]. In some cases, the highest biological activity is associated with a metal

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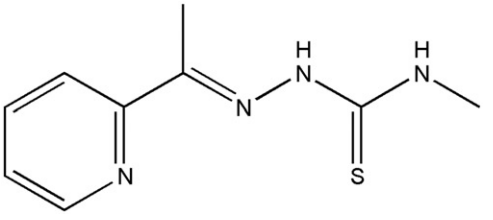

Scheme 1. 2-Acetylpyridine N(4)-methylthiosemicarbazone, HL.

and some side effects may decrease upon complexation. The mechanism of action is still controversial in many respects, but it is known that thiosemicarbazones inhibit ribonucleotide reductase, a key enzyme in the biosynthesis of DNA precursors [17].

Thiosemicarbazones and their metal complexes derived from 2-acetylpyridine have been the subject of extensive investigation [18]. However, the structural and biological studies of nickel complexes have not been examined.

Continuing our research on biological properties of thiosemicarbazones [19], in this article we have studied the antibacterial, antifungal, and antitumor properties of 2-acetylpyridine N(4)-methylthiosemicarbazone HL (scheme 1) and its nickel(II) complex against bacteria, fungi, and the K562 leukemic cell line. We also describe the synthesis, IR spectra, and single-crystal X-ray crystal structure of the nickel(II) complex with 2-acetylpyridine N(4)-methylthiosemicarbazone.

2. Experimental

2.1. *General procedures*

All solvents and reagents are commercially available and are used without purification. 2-Acetylpyridine N(4)-methylthiosemicarbazone was prepared according to the method given in the literature [20]. Elemental analysis of C, H, and N was performed with a Perkin Elmer 240 analyzer. IR spectra were recorded from KBr discs with a Nicolet 170 FT-IR spectrophotometer.

2.2. *Synthesis of the title complex Ni(L)$_2$*

An ethanol solution containing $Ni(ClO_4)_2 \cdot 6H_2O$ (0.091 g, 0.25 mmol) was added dropwise to a solution of 2-acetylpyridine N(4)-methylthiosemicarbazone (0.10 g, 0.5 mmol) dissolved in 30 mL of methanol. After refluxing for 5 h, the resultant solution was filtered. Deep-red crystals suitable for X-ray studies were obtained by slow evaporation of its ethanol solution. Anal. Calcd (%) for $C_{18}H_{22}NiN_8S_2$: C, 45.64; H, 4.65; N, 23.67. Found (%): C, 45.70; H, 4.63; N, 23.44.

Caution! Although no problems were encountered in this work, perchlorates in presence of organic ligands are potentially explosive and should be handled carefully.

Table 1. Summary of crystallographic data and refinement results for the complex.

Empirical formula	$C_{18}H_{22}NiN_8S_2$
Formula weight	473.27
Temperature (K)	296(2)
Crystal system	Monoclinic
Space group	*P2(1)/c*
Unit cell dimensions (Å, °)	
a	9.844(2)
b	7.807(1)
c	27.176(5)
β	93.705(3)
Volume (Å^3), *Z*	2084.3(6), 4
Calculated density (g cm^{-3})	1.508
Absorption coefficient (mm^{-1})	1.154
F(000)	984
Crystal size (mm^3)	$0.28 \times 0.24 \times 0.18$
θ range for data collection (°)	1.50–25.25
Limiting indices	$-6 \le h \le 11$; $-9 \le k \le 9$; $-32 \le l \le 32$
Reflections collected	3770
Independent reflection	2427 [R(int) = 0.0505]
Parameters	264
Goodness-of-fit on F^2	0.808
Final R indices [$I \ge 2\sigma(I)$]	$R_1 = 0.04$, $wR_2 = 0.06$
R indices (all data)	$R_1 = 0.06$, $wR_2 = 0.07$
Largest difference peak and hole (e Å^{-3})	0.22 and −0.28

2.3. *Crystal structure determination*

Intensities of the title complex were collected on a Siemens SMART-CCD diffractometer equipped with a graphite-monochromatic Mo-Kα (λ = 0.71073 Å) radiation using the SMART and SAINT programs. The structure was solved by direct method and refined on F^2 by full-matrix least-squares with SHELXTL version 5.1 [21]. All non-hydrogen atoms were refined with anisotropic thermal displacement parameters. The hydrogens were positioned according to the theoretical models. Crystallographic data are listed in table 1, selected bond distances and angles in table 2, and hydrogen bond lengths and angles in table 3.

2.4. *Biological experiments*

2.4.1. *In vitro* antimicrobial study. The *in vitro* antibacterial activities of the ligand and complex were investigated against several Gram-positive bacteria (*Bacillus subtilis*, *Staphylococcus aureus*, *Agrobacterium tumefaciens*) and Gram-negative bacteria (*Pseudomonas aeruginosa*, *Escherichia coli*). The antifungal activity was assayed against mold (*Aspergillus niger*) and yeast (*Candida lusitaniae*). The minimal inhibitory concentrations (MIC, μg mL^{-1}) were estimated by the disk diffusion method [22]. The final concentration of all cultures in Mueller–Hinton agar (MHA) for bacteria, potato dextrose agar (PDA) for mold spores and Sabouraud dextrose agar (SDA) for yeast cells was adjusted to 10^6 cfu mL^{-1} (bacteria) or 2×10^5 cfu mL^{-1} (mold spores and yeast cells) and used for inoculation in the MIC test. Serial dilutions of the test compounds, previously dissolved in DMSO were prepared at 0–2000 μg mL^{-1}. Each plate was inoculated with 0.1 mL of the prepared bacterial and fungi cultures.

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

Ni(1)–N(3)	2.032(2)	Ni(1)–N(7)	2.033(2)
Ni(1)–N(8)	2.092(2)	Ni(1)–N(4)	2.111(2)
Ni(1)–S(2)	2.408(1)	Ni(1)–S(1)	2.423(1)
S(1)–C(2)	1.728(3)	S(2)–C(11)	1.720(3)
N(1)–C(2)	1.356(3)	N(1)–C(1)	1.454(4)
N(2)–C(2)	1.332(3)	N(2)–N(3)	1.378(3)
N(3)–C(3)	1.306(3)	N(4)–C(9)	1.335(3)
N(4)–C(5)	1.355(3)	N(5)–C(11)	1.348(4)
N(5)–C(10)	1.453(3)	N(6)–C(11)	1.343(3)
N(6)–N(7)	1.366(3)	N(7)–C(12)	1.311(3)
N(8)–C(18)	1.327(4)	N(8)–C(14)	1.356(3)
N(3)–Ni(1)–N(7)	171.6(1)	N(3)–Ni(1)–N(8)	100.4(1)
N(7)–Ni(1)–N(8)	78.48(1)	N(3)–Ni(1)–N(4)	77.97(9)
N(7)–Ni(1)–N(4)	93.71(9)	N(8)–Ni(1)–N(4)	93.27(9)
N(3)–Ni(1)–S(2)	99.20(7)	N(7)–Ni(1)–S(2)	81.50(7)
N(8)–Ni(1)–S(2)	159.9(1)	N(4)–Ni(1)–S(2)	86.66(7)
N(3)–Ni(1)–S(1)	81.26(7)	N(7)–Ni(1)–S(1)	107.0(1)
N(8)–Ni(1)–S(1)	89.91(7)	N(4)–Ni(1)–S(1)	159.2(1)
S(2)–Ni(1)–S(1)	97.25(3)		

Table 3. Hydrogen bond lengths (Å) and angles (°) for the complex.

D–H···A	d(H···A)	d(D···A)	∠(DHA)
N(5)–H(5A)–S(2)$^{\#1}$	2.78	3.393(3)	129.6

Symmetry codes: $^{\#1} -x+1,\ y+1/2,\ -z+1/2$.

Similarly, each plate carried a blank disc, with solvent DMSO only in the center to serve as a negative control. The inoculated plates were then incubated at either 37°C for 18–20 h (bacteria) or 28°C for 48–96 h (fungi). The MIC was detected as the lowest concentration of drug for which no visible growth took place by macroscopic evaluation. All determinations were performed in triplicate and confirmed by three separate experiments.

2.4.2. *In vitro* antitumor study. K562 (a human leukocythemia cancer cell line purchased from the Institute of Biochemistry and Cell Biology, Shanghai Institutes for Biological Sciences (SIBS), Chinese Academy of Sciences, CAS) was cultured in RPMI-1640 medium supplemented with 10% fetal bovine serum (FBS), 100 U mL^{-1} of penicillin, 100 μg (200 μL per well) of streptomycin at 37°C in humid air atmosphere of 5% CO_2. Cell cytotoxicity was assessed by the method of transcriptional and translational (MTT) assay. Briefly, cells were placed into a 96-well plate (5×10^3 cells per well). The next day, the compound diluted in culture medium at various concentrations was added (200 μL per well) to the wells. After 48 h, 20 μL of MTT (0.5 mg mL^{-1} MTT in phosphate buffered saline, PBS) was added and cells were incubated for a further 4 h. Two hundred microliters of DMSO was added to each culture to dissolve the MTT crystals. The MTT–formazan product dissolved in DMSO was estimated by measuring absorbance at 570 nm with a microplate reader. Then, the

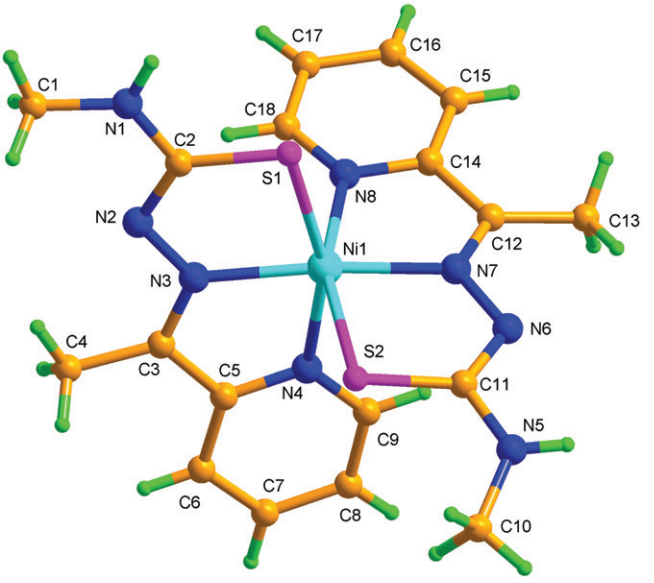

Figure 1. The molecular structure of the title complex along with the atom numbering scheme.

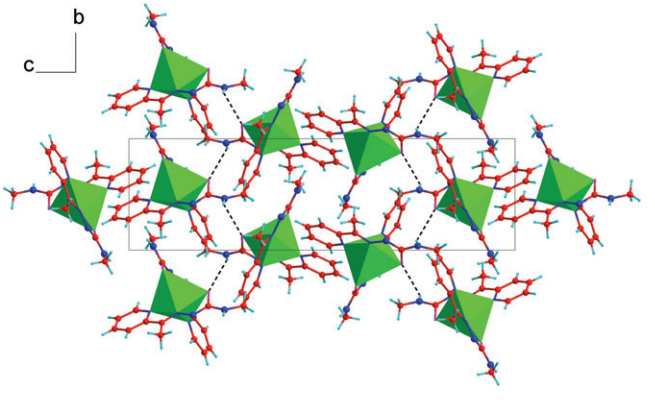

Figure 2. The molecular packing projected along the *c*-axis of the crystal.

inhibitory percentage of each compound at various concentrations was calculated, and the IC_{50} value was determined.

3. Results and discussion

3.1. *Crystal structure of the title complex*

Figures 1 and 2 represent the molecular structure of the nickel complex with the atom numbering scheme and the unit cell packing, respectively.

The complex crystallizes in monoclinic system with space group *P2(1)/c*. As shown in figure 1, the two 2-acetylpyridine N(4)-methylthiosemicarbazones which are deprotonated as anionic ligands are NNS tridentate ligands coordinated to the central nickel(II) octahedrally *via* pyridine nitrogen, imine nitrogen, and sulfur. The equatorial positions are occupied by one sulfur, one imine, and one pyridine nitrogen from one ligand and one imine nitrogen from another ligand; one sulfur and one pyridine nitrogen from different ligands occupied the two remaining axial positions in the octahedral geometry. The pseudo-macrocyclic coordination mode of each ligand affords two five-membered chelate rings, which are nearly planar. The dihedral angles between the chelate rings in both ligands are 6.4° and 4.7°, respectively.

The two pyridine rings (mean plane deviations of 0.0029 and 0.0025 Å) form a dihedral angle of 79.7°. The C(2)–S(1) and C(11)–S(2) bond lengths of 1.728(3) and 1.720(3) Å, respectively, are within the normal range of C–S single bonds, indicating that the thiosemicarbazone moieties adopt the thiol tautomeric form [23]. The C–N and N–N bond lengths in L^- are intermediate between single and double bonds, pointing to an extensive electron delocalization over the entire molecular skeleton. The two thiosemicarbazone ligands have slightly different Ni–N(pyridine) bond distances and are longer than the Ni–N(imine) distances, attributed to imine nitrogen being a stronger base than pyridine nitrogen [24].

The title complex is stabilized by intermolecular hydrogen bonds (figure 2) involving the uncoordinated nitrogen N(5) and sulfur atom S(2). The uncoordinated nitrogen N(5) is a hydrogen bond donor, while sulfur S(2) is an acceptor with the N(5)···S(2) separations of 3.393(3) Å and angle N5–H5A···S2 at 129.6° (symmetry codes: $-x+1$, $y+1/2$, $-z+1/2$).

3.2. *IR spectra*

The IR spectral bands most useful for determining the mode of coordination of the ligand are ν(C=N), ν(N−N), and ν(C=S). The ν(C=N) bands of the ligand and the title complex are at 1578 and 1541 cm^{-1}, respectively. The decrease in frequency of this band in the complex is evidence for coordination *via* the azomethine nitrogen. The increase in frequency of ν(N–N) of the thiosemicarbazone in spectra of the complex is due to the increase in bond strength, again confirming coordination *via* azomethine nitrogen [25]. The band at 836 cm^{-1} for the ligand attributed to ν(C=S) shifts to lower energies (821 cm^{-1}) in the complex, indicating coordination of sulfur. The breathing motion of the pyridine ring is shifted to higher frequency upon complexation, consistent with pyridine ring nitrogen coordination. These observations have also been confirmed by single-crystal X-ray analysis.

3.3. *Cytotoxic activity*

3.3.1. Antibacterial and antifungal activity. In view of the antimicrobial activity of thiosemicarbazones [25–27], we tested the ability of the ligand and complex against bacteria and fungi. Based on the MIC (figure 3), generally, the tested compounds display more inhibitory properties against bacteria than against fungi, and ligand has maximum and broad activities compared to its nickel complex. Both the ligand and the title

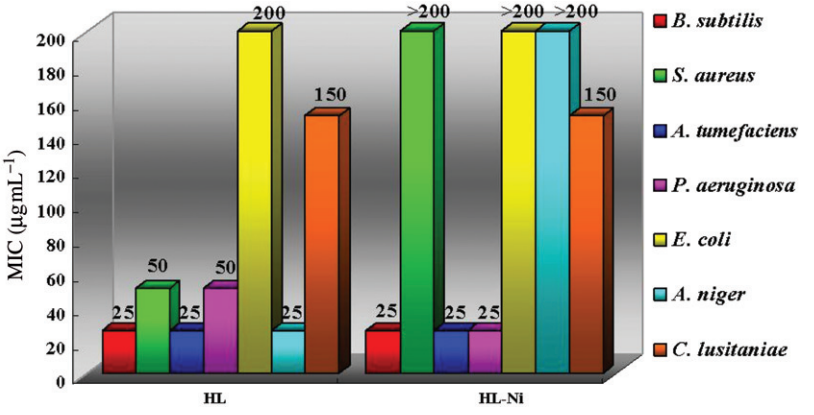

Figure 3. The antimicrobial activities of HL and the title complex against bacteria and fungi.

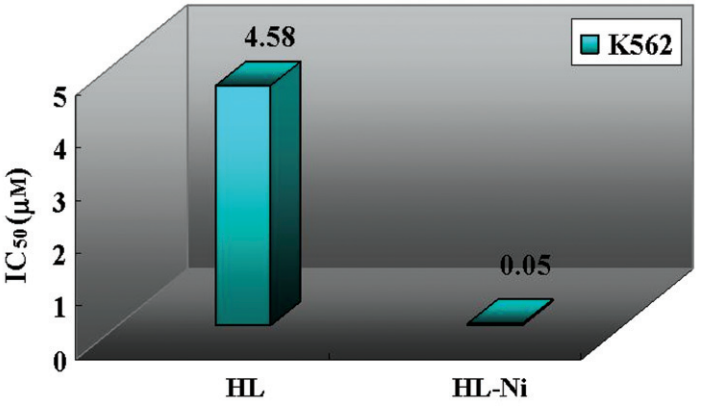

Figure 4. The antitumor activities of HL and the title complex against K562 leukocythemia cancer cell line.

complex exhibit a remarkable antimicrobial activity against the Gram-positive bacteria *B. subtilis* and *A. tumefaciens* with an MIC value of $25\,\mu g\,mL^{-1}$. The title complex showed an enhanced activity against the Gram-negative bacteria *P. aeruginosa* (MIC $25\,\mu g\,mL^{-1}$). However, both the ligand and the title complex are inactive against Gram-negative bacteria *E. coli*. The remarkable antifungal activity is observed for the ligand against mold *A. niger* with an MIC value of $25\,\mu g\,mL^{-1}$. The free ligand and the title complex exert a poor growth inhibition against yeast *C. lusitaniae* (MIC $150\,\mu g\,mL^{-1}$).

3.3.2. *In vitro* antitumor activity. In terms of the cytotoxic activity of thiosemicarbazones [28], we have tested the ability of the free ligand and the title complex to inhibit tumor cell growth. In our experiments, IC_{50} values (compound concentration that produces 50% of cell death) in micro molar units were calculated against K562 leukocythemia cell line.

As shown in figure 4, the free ligand and the title complex show a significant antitumor activity against K562 leukocythemia cell line due to the NNS tridentate

system [29]. Nickel complex shows a lower IC_{50} value (0.05 μmol) than its parent ligand (4.58 μmol). Obviously, coupling of 2-acetylpyridine N(4)-methylthiosemicarbazone to nickel(II) results in enhanced antitumor activity, similar to that observed in previously reported nickel cases [30]. This confirms the conclusion that the antitumor activities of thiosemicarbazone can be increased by coordinating the ligand to metal cations [31].

4. Conclusions

Free ligand and complex show a distinct difference in the biological activities. Both display more inhibitory properties against bacteria than against fungi, and ligand has the maximum and broad activities compared to its nickel complex; coupling of 2-acetylpyridine N(4)-methylthiosemicarbazone to nickel(II) results in enhanced antitumor activity against K562 leukocythemia cell line. These promising results are encouraging further research in this field for future applications. Our continuing and detailed studies of the toxicity of these compounds as well as mechanism of action are in process and could be helpful in designing more potent antimicrobial and antitumor agents for therapeutic use.

Supplementary material

Crystallographic data for the structural analyses reported in this article have been deposited with the Cambridge Crystallographic Data Centre with CCDC no 759410. 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).

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