symmetry element, this rigidity leads to nonequivalence of all four bridge protons in ligands **2** and **3**. In a previous study of $Ru(\mathbf{2})_3^{2+}$ and $Ru(\mathbf{3})_3^{2+}$, we found that in the former case the coordinated ligand **2** was rigid while in the latter case the coordinated ligand **3** was mobile.[3e]

The complex **5** showed a long wavelength absorption in its electronic spectrum consisting of two principal bands centered at 490 and 577 nm. Figure 1 illustrates this spectrum along with the spectra of $Ru(\mathbf{1})_3^{2+}$, $Ru(\mathbf{2})_3^{2+}$, and $Ru(\mathbf{3})_3^{2+}$. These three curves are all very similar in shape, consisting of a major band and a shoulder at shorter wavelength, and are associated with a metal-to-ligand charge transfer (MLCT) state. The shift of this MLCT band to longer wavelength with increased delocalizing ability of the ligand is a well-documented phenomena.[12] The absorption curve for **5** shows essentially no component for charge transfer into the bpy ligand. We have shown that the energy for this transition for complexes of the type $Ru(bpy)_2L$ remains invariant as the electronegativity of L increases.[4a] For MLCT into the biquinoline ligand we observe the band at 490 nm, which corresponds to the shoulder of the major band for the complex $Ru(\mathbf{2})_3^{2+}$. For MLCT into the binaphthyridine ligand we observe the band at 577 nm, which corresponds to the major band of $Ru(\mathbf{3})_3^{2+}$. Since these transitions have yet to be unequivocally assigned, we cannot read too much into these results except to note that MLCT is apparently allowed only for certain transitions in these complexes.

The half-wave potentials for complex **5** as determined by cyclic voltammetry are summarized in Table I along with those of other related ruthenium complexes. The oxidation potential of +1.26 V demonstrates with surprising exactness that ligands **2** and **3** share equally in mediating the ease with which the complex loses an electron. This value represents the average oxidation potential for the complexes $Ru(\mathbf{2})_3^{2+}$ and $Ru(\mathbf{3})_3^{2+}$ as well as the average value for $Ru(\mathbf{1})_2(\mathbf{2})^{2+}$ and $Ru(\mathbf{1})_2(\mathbf{3})^{2+}$. The implication is that fine tuning of the oxidation potential of complexes of the type $Ru(bpy)LL'^{2+}$ may be accomplished in a readily predicted fashion. Since the oxidation potential is a reflection of the energy of the highest filled t_{2g} level of ruthenium, it is also apparent that the energy of this level is affected in a very regular fashion by the attached ligands. Data from additional systems is clearly needed to lend more certainty to these conclusions.

The first reduction potential of **5** is more positive than those of any of the three related symmetrical RuL_3^{2+} complexes or the mixed complexes $Ru(bpy)_2L^{2+}$, implying that the π^* orbital, which is the LUMO for this system, is unusually low in energy. The lowering of this π^* level may be associated with the lack of symmetry of complex **5** as compared with the RuL_3^{2+} complexes, which have D_3 symmetry, and the $Ru(bpy)_2L^{2+}$ complexes, which have C_2 symmetry. The second and third reduction potentials appear to be less affected.

Experimental Section

Nuclear magnetic resonance spectra were recorded on a Nicolet NT-300 WB spectrometer in CD_3CN with chemical shifts reported in parts per million downfield from Me_4Si. Electronic absorption spectra were recorded on a Perkin-Elmer 330 spectrophotometer. LC-MS measurements were performed on a Biospec mass spectrometer with a thermospray ionization interface. Elemental analyses were performed by the Canadian Microanalytical Service ltd., New Westminister, BC, Canada. The preparation of ligands **2** and **3** has been previously described.[13] The $[Ru(bpy)Cl_3]_n$ was prepared according to a procedure described by Krause.[8]

Cyclic voltammograms were recorded by using a PAR Model 174A polarographic analyzer, a PAR Model 175 universal programmer, and a Houston Instruments, Omnigraphic 2000 X-Y recorder. A three-electrode system was employed, consisting of a platinum-button working electrode, a platinum-wire auxiliary electrode, and a saturated calomel reference electrode. The reference electrode was separated from the bulk of the solution by a cracked-glass bridge filled with 0.1 M TBAP in acetonitrile. Deaeration of all solutions was performed by passing high-purity nitrogen through the solution for 5 min and maintaining a blanket of nitrogen over the solution while measurements were made. Reagent grade acetonitrile was distilled twice from P_2O_5 under nitrogen. The supporting electrolyte, tetra-*n*-butylammonium perchlorate (TBAP), was recrystallized from EtOAc/hexane, dried, and stored in a desiccator. Half-wave potentials were calculated as an average of the cathodic and anodic peak potentials. The criteria for reversibility was that the separation of the anodic and cathodic peaks is equal to less than 60 mV for a one-electron process and that the ratio of anodic to cathodic current is unity.[14]

Preparation of $Ru(\mathbf{1})(\mathbf{2})(\mathbf{3})[PF_6]_2$ (5). A solution of 47.4 mg (0.118 mmol) of $[Ru(bpy)Cl_3]_n$ and 33.5 mg (0.118 mmol) of 3,3'-dimethylene-2,2'-biquinoline (**2**) in 15 mL of 1:1 $EtOH/H_2O$ was refluxed for 24 h. The solution was cooled, 34 mg (0.118 mmol) of 3,3'-dimethylene-2,2'-binaphthyridine (**3**) was added, and then reflux was continued another 48 h. After cooling, an aqueous solution of NH_4PF_6 (2 equiv) was added. The resulting blue precipitate was collected, dried, and chromatographed on 20 g of alumina eluting with 1:1 acetonitrile/toluene. Slow evaporation of acetonitrile from the eluent provided 67 mg (57%) of **5** as purple crystals. Anal. Calcd for $C_{48}H_{34}F_{12}N_8P_2Ru$: C, 51.75; H, 3.05; N, 10.06. Found: C, 51.92; H, 3.21; N, 9.74.

Acknowledgment. Financial support from the Robert A. Welch Foundation, the donors of the Petroleum Research Fund, administered by the American Chemical Society, and the National Science Foundation (Grant CHE-8607935) is gratefully acknowledged.

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^{31}P NMR Studies of $[Au_2(\mu\text{-dppe})]^{2+}$ Antitumor Complexes. Conversion into $[Au(dppe)_2]^+$ Induced by Thiols and Blood Plasma

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We have recently reported the unusual stability of tetrahedral Au(I) complexes with chelated diphosphine ligands.[1,2] The most common coordination geometry for Au(I) is linear, and in previous studies of Au(I) complexes with flexible diphosphine ligands chelation has not been considered important.[3-5] Diphosphine-bridged-digold complexes $ClAu(Ph_2P(CH_2)_nPPh_2)AuCl$ are readily converted into the bischelated species in the presence of added diphosphine ligand. Chelated complexes of the type $[Au(R_2P(CH_2)_nPR'_2)_2]Cl$ form readily when R and R' are phenyl and n = 2 or 3 and also with the more rigid ligand *cis*-$Ph_2PCH{=}CHPPh_2$.[2]

A number of diphosphine-bridged-digold complexes, $XAu(Ph_2P(CH_2)_nPPh_2)AuX$, have been reported to have good antitumor activity in animal models.[6,7] Activity was maximized for

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those ligands able to form stable five- or six-membered rings by chelation. Ring-closed species could therefore be intermediates in the metabolism of these complexes.[7] Indeed, $[Au(dppe)_2]Cl$ has recently been found to exhibit activity in several tumor models.[8]

We have therefore investigated the reactivity of two of the active diphosphine-bridged-digold complexes $[(AuCl)_2(dppe)]$ and $[(AuStg)_2(dppe)]$ with thiols and blood plasma, with the aim of elucidating whether they might undergo conversion into chelated species in vivo.

Abbreviations. dppe = $Ph_2P(CH_2)_2PPh_2$; NaStg = sodium salt of β-D-thioglucose; GSH = reduced glutathione; DMA = *N*,*N*-dimethylacetamide; HSAtg = tetraacetylthioglucose.

Experimental Section

Materials. $[(AuCl)_2(dppe)]$ and $[Au(dppe)_2]Cl$ were prepared as described previously.[1,2] $[(AuStg)_2(dppe)]$ was supplied by SK&F Laboratories (Philadelphia, PA). GSH and NaStg were purchased from Sigma. Venous blood was drawn from a healthy volunteer and placed in Sterilin plastic vials containing EDTA as anticoagulant. The plasma was separated by centrifugation and was used within a few hours. Lyophilized bovine serum was purchased from Wellcome.

NMR Measurements. Proton-decoupled ^{31}P NMR spectra were recorded on a JEOL FX60 instrument (24.15 MHz) in 10-mm tubes or on a Bruker WM200 instrument (80.96 MHz) in 15-mm tubes. All spectra were referenced to external 85% H_3PO_4 obtained with an external D_2O lock.[9] For the titrations, the samples were contained in 8-mm tubes, with D_2O in an external 10-mm tube. For the plasma samples ^{31}P NMR spectra at 80.96 MHz were recorded with gated broad-band decoupling, a 10-kHz spectral width, 0.82-s acquisition time, 20-μs pulse width, 1-s relaxation delay, and 16K data points.

Reactions with Thiols. (A) Aliquots (0.5 mL) of a 24 mM solution of $[(AuStg)_2(dppe)]$ in MeOH were added to solutions containing 0–10 mol equiv of GSH in H_2O (0.5 mL) at pH 7. Final solvent was H_2O–MeOH (1:1 v/v).

(B) Aliquots (1.0 mL) of a 10 mM solution of $[(AuCl)_2(dppe)]$ in DMA were mixed with solutions containing 0–4 mol equiv of NaStg in H_2O (0.2 mL). Final solvent was DMA–H_2O (5:1 v/v). All solutions containing NaStg were clear and colorless, but the solution containing only $[(AuCl)_2(dppe)]$ was slightly cloudy.

(C) A 1.5-mL quantity of an 8.7 mM solution of $[(AuCl)_2(dppe)]$ in DMA was added to a solution containing 10 mol equiv of GSH in D_2O (0.75 mL) at pH 7 (meter reading). A white precipitate started to form, but this dissolved with gentle shaking.

Isolation of $[Au(dppe)_2]^+$. $[(AuCl)_2(dppe)]$ (0.10 g, 0.116 mmol) was suspended in acetone (10 mL). A solution of NaStg (0.152 g, 0.697 mmol) in H_2O (2 mL) was added with stirring. The solid dissolved, leaving a small amount of insoluble material; this was filtered off. H_2O (ca. 20 mL) was added to the cloud point, and the solution was left at 25 °C overnight. The white precipitate was filtered, dried in vacuo, and analyzed by ^1H and ^{31}P NMR spectroscopy.

Plasma Experiments. (A) A 150-μL aliquot of a 16 mM solution of $[(AuStg)_2(dppe)]$ in Me_2SO was added to fresh human plasma (2 mL) that was diluted with D_2O–saline solution so that the final drug concentration was 1 mM. After the ^{31}P NMR spectrum had been acquired, some white precipitate was observed in the solution.

(B) A 50-μL aliquot of a 21 mM solution of $[(AuStg)_2(dppe)]$ in MeOH was added to 5 mL of bovine serum reconstituted in D_2O so that the $[(AuStg)_2(dppe)]$ concentration was 0.2 mM with 1% MeOH present.

(C) A 50-μL aliquot of a 100 mM solution of $[Au(dppe)_2]Cl$ in Me_2SO was added to fresh plasma (2 mL). A thick white precipitate formed immediately and was removed by centrifugation. The plasma was diluted with D_2O–saline solution (250 μL) so that the maximum final drug concentration was 2.2 mM.

Results

Figure 1 shows ^{31}P NMR spectra of $[(AuStg)_2(dppe)]$ in aqueous methanol, in the presence of increasing amounts of GSH at pH 7. The resonance due to $[(AuStg)_2(dppe)]$ at 31.6 ppm decreased in intensity on addition of GSH, and an additional peak appeared at 20.3 ppm at a GSH:Au ratio as low as 0.5:1. This

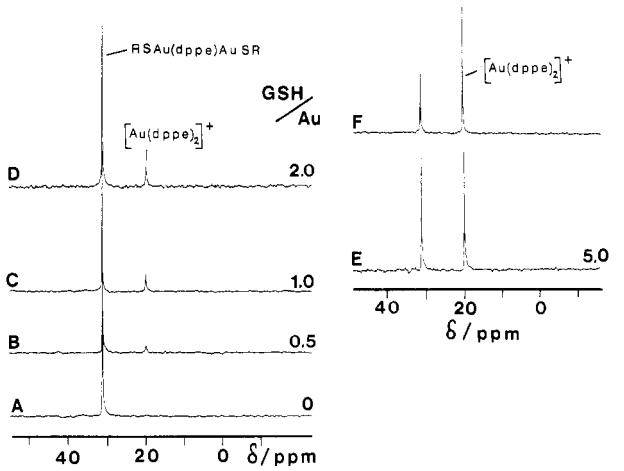

Figure 1. $^{31}P\{^1H\}$ NMR spectra (24.2 MHz) of a 12 mM solution of $[(AuStg)(dppe)]$ in MeOH–H_2O (1:1 v/v) in the presence of 0, 1, 2, 4, and 10 mol equiv of glutathione (GSH) (A–E). Spectrum F is from the same solution used for spectrum E following the addition of 3.8 μmol of $[Au(dppe)_2]Cl$.

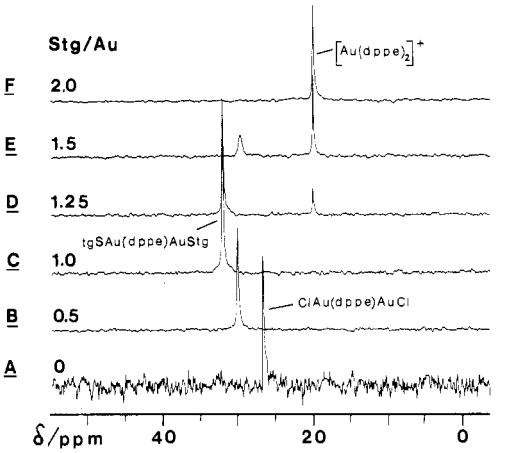

Figure 2. $^{31}P\{^1H\}$ NMR spectra (24.2 MHz) of 8 mM solutions of $[(AuCl)_2(dppe)]$ in DMA–H_2O (5:1 v/v) in the presence of 0, 1.0, 2.0, 2.5, 3.0, and 4.0 mol equiv of NaStg (A–F).

peak increased in intensity with increasing GSH concentration. Addition of $[Au(dppe)_2]Cl$ (Figure 1F) shows that the new resonance is due to $[Au(dppe)_2]^+$.

The analogous dichloro complex $[(AuCl)_2(dppe)]$ was too insoluble in DMA–D_2O (2:1 v/v) for a ^{31}P NMR spectrum to be obtained. However, it dissolved in this solvent in the presence of 10 mol equiv of GSH at pH 7. The ^{31}P NMR spectrum of the resulting solution consisted of two peaks at 32.3 and 20.7 ppm, intensity ratio 3:1.

On addition of NaStg to a solution of $[(AuCl)_2(dppe)]$ in DMA–D_2O (5:1 v/v), the ^{31}P NMR signal at 27.1 ppm shifted to high frequency (Figure 2). It reached a maximum of 32.7 ppm at a Stg:Au ratio of 1:1 (Figure 2C). This peak was assigned to $[(AuStg)_2(dppe)]$ (δ = 32.7 in this solvent). Further addition of NaStg caused the signal to shift back to low frequency, and a new signal to appear at 20.3 ppm. This increased in intensity until at a Stg:Au ratio of 2:1 it was the only peak in the spectrum (Figure 2F). Addition of $[Au(dppe)_2]Cl$ to a 2.5:1 mixture of NaStg–$[(AuCl)_2(dppe)]$ in the same solvent caused the 20.3 ppm resonance to increase in intensity.

Solutions containing Stg:Au ratios >1.25:1 were left standing for a few days at about 25 °C, and the ^{31}P NMR spectra were then rerecorded. The 20.3 ppm resonance had decreased in intensity with respect to the peak at ca. 30 ppm, which had shifted to slightly higher frequency. There was no apparent precipitate.

A white solid was obtained from the reaction of $[(AuCl)_2(dppe)]$ with 6 mol equiv of NaStg in aqueous acetone. The ^{31}P NMR spectrum of the reaction solution consisted of one resonance at

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(9) This reference occurs 2.2 ppm to low frequency (upfield) of the reference we have used previously (e.g., ref 1 and 2): 85% H_3PO_4 in D_2O (85:15 v/v).

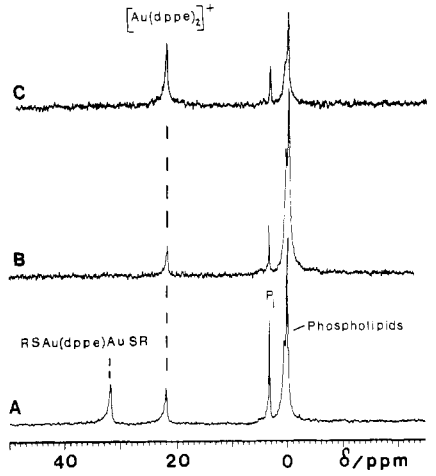

Figure 3. $^{31}P\{^{1}H\}$ NMR spectra at 80.96 MHz: (A) human plasma treated with 1 mM $[(AuStg)_2(dppe)]$, total accumulation period 14 h (28 000 transients); (B) bovine serum treated with 0.2 mM $[(AuStg)_2(dppe)]$, total accumulation period 16.5 h (33 141 transients); (C) human plasma treated with 2.2 mM $[Au(dppe)_2]Cl$ (2200 transients).

20.3 ppm, indicating that $[Au(dppe)_2]^+$ was the only phosphine species present. The white solid was only sparingly soluble in $CDCl_3$. A ^{31}P NMR spectrum of the $CDCl_3$ solution contained a minor broad peak at 26.5 ppm and a major peak at 18.7 ppm, the chemical shift of $[Au(dppe)_2]^+$ in this solvent. The ^{1}H NMR spectrum confirmed that $[Au(dppe)_2]^+$ was the major species present. Resonances attributable to thioglucose were also observed, together with two additional broadened resonances, which were not assigned.

Figure 3A shows the ^{31}P NMR spectrum acquired during 14 h of incubation of human plasma with 1 mM $[(AuStg)_2(dppe)]$. In addition to resonances due to plasma phospholipids (−0.1 and 0.5 ppm) and phosphate (3.2 ppm),[10] two new peaks were observed at 31.7 and 21.9 ppm. Both were clearly visible within 1 h of adding the complex. The peak at 31.7 ppm may correspond to intact $[(AuStg)_2(dppe)]$ (31.6 ppm in MeOH–H_2O (1:1 v/v) or to other RSAu(dppe)AuSR′ species formed by the displacement of Stg^- by plasma thiols. We assigned the peak at 21.9 ppm to $[Au(dppe)_2]^+$, as this is the only Au–dppe species with a chemical shift in the region of 20 ppm.[1,2] This assignment was substantiated by adding $[Au(dppe)_2]Cl$ (resonance at 21.9 ppm; see Figure 3C).

At a more biologically relevant concentration of 0.2 mM $[(AuStg)_2(dppe)]$, incubated with bovine serum, the ^{31}P NMR spectrum shows only one resonance at 21.9 ppm (Figure 3B). The resonance at 31.7 ppm due to dppe-bridged-digold dithiolate species is no longer visible.

Discussion

The titrations described here show that thiols can induce the conversion of diphosphine-bridged-digold complexes XAu(dppe)AuX into the bischelated cation $[Au(dppe)_2]^+$ in polar solvents.

Addition of 1 equiv of GSH to $[(AuSR)_2(dppe)]$ (a GSH:Au ratio of 0.5) is sufficient for formation of some four-coordinate Au(I) (Figure 1B), suggesting the stoichiometry

$$2[(AuSR)_2(dppe)] + 2RS^- \rightleftharpoons [Au(dppe)_2]^+ + 3[Au(SR)_2]^- \quad (1)$$

It is an equilibrium reaction, as even when the thiol is in excess the reaction does not go to completion (Figure 1C–E). It may be driven by the high thermodynamic and kinetic stability of $[Au(dppe)_2]^+$.[1,2] Reaction 1 is not a disproportionation because additional thiol is required for the reaction to proceed. However, disproportionation reactions reported by Shaw and co-workers[11,12] indicate that phosphines and other ligands of Au(I) may "unscramble" in solution:

$$2Et_3PAuCN \rightleftharpoons Au(PEt_3)_2^+ + Au(CN)_2^- \quad (2)$$

$$2RSAuCN^- \rightleftharpoons Au(RS)_2^- + Au(CN)_2^- \quad (3)$$

Unscrambling induced by addition of ligand has previously been observed by Cariati et al.[13] in $CHCl_3$:

$$2Ph_3PAuCN + Ph_3P \rightleftharpoons [(Ph_3P)_3Au]^+ + [Au(CN)_2]^- \quad (4)$$

We have used ^{31}P NMR spectroscopy to show that $[Au(dppe)_2]^+$ is the only phosphine-containing product of reaction 1. This technique did not provide characterization of thiol-containing products. However, 1:1 Au:thiolate oligomers have been shown to react with excess thiol to form $[Au(SR)_2]^-$[14,15] and these would seem likely products.

During the titration of $[(AuStg)_2(dppe)]$ with NaStg, no $[Au(dppe)_2]^+$ was observed until the Stg:Au ratio exceeded 1:1. This indicates that ring closure requires prior formation of $[(AuStg)_2(dppe)]$. The intermediate species, formed at Stg:Au ratios <1:1, undergo fast exchange on the ^{31}P NMR time scale: only a single averaged resonance is observed in the ^{31}P NMR spectrum (Figure 2A–C). This is consistent with the facile ligand exchange usually observed for two- or three-coordinate Au(I) phosphine species.[16–18] The bischelated complex $[Au(dppe)_2]^+$ is kinetically stable and undergoes slow exchange (Figure 2D–E). The changes in spectra of aged solutions showed that further reactions are also involved.

Formation of $[Au(dppe)_2]^+$ from $[(AuX)_2(dppe)]$ clearly involves transfer of dppe from one bridged digold molecule to another. This could be via annular complexes formed in solution. In the crystalline state, pairs of $[(AuCl)_2(dppe)]$ are held together by short intermolecular Au–Au interactions.[19,20] If similar interactions exist in solution, they could facilitate dppe transfer. A third possibility is the involvement of thiolate-bridged species. A number of Au(I) thiolates have been shown to form oligomers with Au–S–Au linkages, including AuStg.[21–23] This could explain why ring closure requires prior formation of $[(AuSR)_2(dppe)]$.

Equilibria such as (1) are likely to be highly solvent dependent. Direct investigation of coordination chemistry in biological fluids may be more relevant to molecular pharmacology than are model reactions. We have shown that $[(AuStg)_2(dppe)]$ is converted to $[Au(dppe)_2]^+$ in fresh human plasma and bovine serum.

Shaw has recently studied the reaction of $Et_3PAuSAtg$ (auranofin) with bovine serum albumin[24] and shown that it binds at the free SH residue, Cys-34, with displacement of HSAtg. It has been proposed that the cellular association of auranofin involves ligand exchange with membrane-localized thiols[25] and Et_3PAuCl reacted with whole blood gives P–Au–S species.[26,27] Thus, it is

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known that Au(I) phosphine complexes can interact with biological thiols. We have shown that thiols in model systems induce chelation of the bridged compounds; therefore, it seems very likely that the conversion in plasma and serum is thiol-induced.

Reactive thiols are present in many in vivo sites apart from blood plasma.[28] Therefore, $[Au(dppe)_2]^+$ is likely to be a major metabolite of the bridged-digold complexes in cells. $[Au(dppe)_2]^+$ itself does not react significantly with thiols.

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Registry No. GSH, 70-18-8; NaStg, 105177-81-9; $[(AuCl)_2(dppe)]$, 18024-34-5; $[Au(dppe)_2]Cl$, 19624-67-0; $[(AuStg)_2(dppe)]$, 10593-29-0.

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Proton Site Exchange in $(\mu\text{-H})_3M_3(CO)_9(\mu_3\text{-CH})$ (M = Ru, Os)

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Recent work in this laboratory indicated that the CH and μ-H protons in $(\mu\text{-H})_3Os_3(CO)_9(\mu_3\text{-CH})$ undergo site exchange, as evidenced by scrambling of a deuterium label.[1] At room temperature, this exchange is slow, but at temperatures above 80 °C, it can be observed by spin saturation transfer.[2] In this note we present a complete description of the variable-temperature NMR experiments performed on this compound. We have also briefly examined the ruthenium analogue in order to form a more complete comparison with recent data reported for the corresponding iron system.[3]

Saturation transfer methods probe the competition between chemical exchange and spin–lattice relaxation; determination of relaxation rates, by separate inversion–recovery experiments, allows quantitation of exchange rates.[4] For an intramolecular exchange, where cross relaxation (as evidenced by a nuclear Overhauser enhancement) may be present, the modified Bloch equations describing the return to equilibrium for homonuclear spins I and S after a perturbation are[5]

$$dI_z/dt = -\rho_I(I_z - I_\infty) - \sigma(S_z - S_\infty) - k_I I_z + k_S S_z \quad (1)$$

$$dS_z/dt = -\rho_S(S_z - S_\infty) - \sigma(I_z - I_\infty) - k_S S_z + k_I I_z \quad (2)$$

where ρ_I and ρ_S = relaxation rate constants, σ = cross relaxation rate constant, k_I and k_S = exchange rate constants, I_z and S_z = instantaneous values of magnetization during recovery, and I_∞ and S_∞ = final values of magnetization after recovery.

The solutions to these equations are double exponential in form, unlike the single exponential obtained in the absence of exchange or cross relaxation. Except for certain special cases, such as exchange between sites with equivalent values of T_1 ($=1/\rho$), NOE ($=\sigma/\rho$), and population[5] or the apparent absence of NOE,[6] accurate determination of exchange rates requires extensive computer fitting of data from selective inverstion–recovery experiments to the solutions to eq 1 and 2[7] or an analogous procedure on data from NOESY 2D-NMR experiments.[8] The spin system in $(\mu\text{-H})_3M_3(CO)_9(\mu_3\text{-CH})$ (M = Ru, Os) also represents a special case. Because of the large difference in relaxation rates between the sites, nearly single-exponential recovery curves are obtained from selective inversion–recovery experiments at temperatures below the onset of exchange.[9] This allows relaxation rate determination by using only data fitting routines commonly found in commercial NMR spectrometer software packages, thereby simplifying the exchange rate calculation.

Experimental Section

All NMR experiments were done on a Nicolet Magnetics NT-360 spectrometer with an 1180 computer and a 293B pulse programmer. All T_1 experiments were performed with the fast inversion–recovery sequence,[10] a symmetric five-element composite 180° pulse,[11] alternating phase of the 90° pulse, and a nonlinear three-parameter least-squares fit.[12] The NOE values were determined by difference techniques.[13] All experimental measurements were assumed to be subject to errors of ±20%. Sample temperatures were checked by replacement with a tube containing a thermocouple junction and solvent.

The compounds $(\mu\text{-H})_3Os_3(CO)_9(\mu_3\text{-CH})$[14] and $(\mu\text{-H})_3Ru_3(CO)_9(\mu_3\text{-CH})$[15] were prepared by literature methods. Solutions for the NMR experiments were prepared in perdeuteriated solvents (Aldrich, *o*-xylene (M = Os) or toluene (M = Ru)), degassed by freeze–pump–thaw sequences, and sealed in tubes under a partial atmosphere of nitrogen. A separate sample of the osmium compound was prepared in the same manner and sealed under a partial atmosphere of carbon monoxide. Although this sample was not examined as extensively as the sample sealed under nitrogen, no differences in relaxation rates or exchange rates were observed.

Results and Discussion

Nonselective inversion–recovery measurements of the "T_1's" for the CH and μ-H sites in the osmium compound at ambient temperature gave 11.3 and 2.02 s, respectively, for single-exponential data fits. The respective nuclear Overhauser enhancements were 32% and 2%. Because of the cross relaxation present, the inversion–recovery curve of the methylidyne site was expected to be biexponential. This was demonstrated by selective inversion–recovery experiments, which gave 13.0 and 2.08 s for the CH and μ-H sites, respectively. These values were taken to be the "true" values of T_1, i.e., the inverse of the desired quantity ρ. The different values of T_1 reflect the dipole–dipole relaxation mechanism of the protons, a process that is very sensitive to internuclear distances, and the μ-H sites are over 1 Å closer to each other than to the CH site.[16]

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(9) This conclusion is based on an analysis of the solutions to eq 1 and 2. The solution to eq 1 takes the form

$$I_z = C_1 \exp(\lambda_1 t) + C_2 \exp(\lambda_2 t) + I_\infty$$

where $\lambda_{1(2)} = 0.5(-(\rho_I + k_I + \rho_S + k_S) + (-)\{[(\rho_I + k_I) - (\rho_S + k_S)]^2 - 4(k_S - \sigma)(\sigma - k_I)\}^{1/2})$ and C_1 and C_2 are dependent on the initial values of the magnetization I_0 and S_0. Equation 2 has a similar solution. When $\rho_I \gg \rho_S$ and $k = 0$, then $-\lambda_1 \cong \rho_S$ and $-\lambda_2 \cong \rho_I$. For the 6-fold difference in ρ_I and ρ_S in this system, these approximations are good to within 1%. For the selective inversion–recovery sequence, C_1 is forced to zero, permitting calculation of λ_2 (and thus ρ_I) from a single exponential data analysis.

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