H_2O–KNO_3 as eluent). It was assumed to be $[Ru(tterpy)]_2(tpbp)^{2+}$ on the basis of its ^1H NMR spectrum (sharp singlet for the 4'- and 6'-protons) and FAB-MS measurements.[10]

Formation of a C–C coupling product from dpb-H is not obvious from a mechanistic viewpoint, but it is likely to involve radicals located on the 5'-position of the central ring once coordinated to the ruthenium(II) center, this position becoming relatively oxidizable after formation of the $Ru-C_{5'}$ bond.

The chemical nature of $[Ru(tterpy)]_2(tpbp)^{2+}$ was confirmed by a crystallographic study of its PF_6^- salt.[11] The X-ray structure of the complex is shown in Figure 1. The electrochemical data for both mono- and dinuclear complexes are as follows:

$Ru^{III/II}(tterpy)(dpb)^{2+/+}$: $E_{1/2}$ = +0.485 V (vs SCE in CH_3CN)

$[Ru_2^{III,III/III,II}(tterpy)_2](tpbp)^{4+/3+}$: $E_{1/2}$ = +0.50 V

$[Ru_2^{III,II/II,II}(tterpy)_2](tpbp)^{3+/2+}$: $E_{1/2}$ = +0.34 V

Each electrochemical process is reversible. The calculated comproportionation constant K_c for the reaction $Ru_2^{III,III} + Ru_2^{II,II} \rightleftharpoons 2Ru_2^{III,II}$ is ~600, i.e., comparable to that in $ClRu(bipy)_2(pz)Ru(Cl)(bipy)_2^{3+}$[12] (pz is 1,4-pyrazine) in spite of a much larger distance between the metals, 11.009 (2) Å.

The UV-visible spectrum of the $Ru_2^{II,II}$ complex was as expected. It shows very intense metal-to-ligand charge transfer (MLCT) bands in the visible region (λ_{max} = 516 and 543 nm; ϵ = 30 700 M^{-1} cm^{-1} for both maxima). Neither the $Ru_2^{II,II}$ species nor the oxidized form $Ru_2^{III,III}$ (excess Br_2 in CH_3CN) displayed any absorption in the near-infrared (near-IR) domain.

The most striking property of the mixed-valence state $[Ru_2(tterpy)_2](tpbp)]^{3+}$ is related to its intervalence transfer (IT) transition. The mixed-valence complex was generated by addition of gradual amounts of Br_2 in CH_3CN. As expected, an intense MLCT band was also present in the visible (λ_{max} = 523 nm; ϵ = 31 700 M^{-1} cm^{-1}). The near-IR spectrum shows a huge IT band centered at λ_{max} ~ 1820 nm, with an extraordinary extinction coefficient (ϵ ~ 27 000 M^{-1} cm^{-1}). The band is much narrower ($\Delta\nu_{1/2} = 2.82 \times 10^3$ cm^{-1}) than expected from calculations following Hush's theory,[13] also supporting significant delocalization for the $Ru_2^{III,II}$ state. The α value[13] found in the present case amounts to α = 0.23.

The very special coupling properties of the $tpbp^{2-}$ ligand in the present dinuclear ruthenium complex open the route to long-distance electron transfer across related anionic aromatic bridges.

Acknowledgment. We thank the CNRS for financial support and Johnson Matthey for a gift of $RuCl_3$.

(10) $Ru(tterpy)(dpb)^+(PF_6^-)$: ^1H NMR (200 MHz, CD_3CN) δ 8.98 (s, 2 H), 8.55 (d, 2 H, 8 Hz), 8.25 (d, 2 H, 8 Hz), 8.16 (m, 4 H), 7.70 (td, 2 H, 8, 2 Hz), 7.56 (m, 4 H), 7.45 (t, 1 H, 8 Hz), 7.09 (m, 4 H), 6.94 (m, 2 H), 6.65 (m, 2 H), 2.51 (s, 3 H); FAB-MS (nitrobenzyl alcohol matrix) m/z = 656.1, $Ru(tterpy)(dpb)^+$ requires 656. $[Ru(tterpy)]_2(tpbp)^{2+}(PF_6^-)_2$: ^1H NMR (200 MHz, CD_3CN) δ 9.05 (s, 4 H), 8.97 (s, 4 H), 8.62 (d, 4 H, 8 Hz), 8.48 (d, 4 H, 8 Hz), 8.14 (d, 4 H, 8 Hz), 7.78 (d, 4 H, 8 Hz), 7.70 (d, 4 H, 8 Hz), 7.57 (d, 4 H, 8 Hz), 7.30 (d, 4 H, 6 Hz), 7.18 (d, 4 H, 6 Hz), 7.04 (t, 4 H, 6 Hz), 6.73 (t, 4 H, 6 Hz), 2.53 (s, 6 H); FAB-MS (nitrobenzyl alcohol matrix) m/z = 1455.2, $[Ru_2(tterpy)_2(tpbp)(PF_6)]^+$ requires 1455.

(11) The compound $[Ru_2(tterpy)_2(tpbp)](PF_6)_2 \cdot (CH_3)_2CO$ crystallized in the monoclinic space group $P2_1/n$, a = 22.464 (3) Å, b = 17.834 (2) Å, c = 20.107 (2) Å, β = 98.72 (1)°, V = 7962 (3) Å^3, μ(Cu Kα) = 41.86 cm^{-1} and D_{calcd} = 1.38 $g \cdot cm^{-3}$ for Z = 4, $Ru_2P_2F_{12}ON_{10}C_{79}H_{60}$ MW = 1657.5. Reflections were measured on an Enraf-Nonius CAD 4 diffractometer using Cu Kα graphite-monochromated radiation ($\omega/2\theta$ scan mode, scan width (ω) 0.55 + 0.14 tan θ, 4° < θ < 59°). The compound is unstable at room temperature, due to a loss of acetone; 11 264 unique data, 7458 with $I > 3\sigma(I)$. A decay correction and an empirical absorption correction (ψ scan) were applied. The non-hydrogen atoms were located by direct methods and refined anisotropically. The hydrogen atoms except those of the acetone molecule were included as idealized contributions. Actual R = 0.067 and R_w = 0.121. All calculations were done with SDP (Structure Determination Package; B. A. Frenz and Associates Inc., College Station, TX, and Enraf-Nonius, Delft, 1983). Diffraction and calculation work was performed at Laboratoire de Cristallographie Biologique, IBMC, Strasbourg.

(12) Callahan, W. H.; Keene, F. R.; Meyer, T. J.; Salmon, D. J. *J. Am. Chem. Soc.* **1977**, *99*, 1064-1073.

(13) Hush, N. S. *Prog. Inorg. Chem.* **1967**, *8*, 391.

Supplementary Material Available: An atom-labeling scheme of $[Ru(tterpy)]_2(tpbp)^{2+}$ and tables of positional and isotropic equivalent thermal parameters, anisotropic thermal parameters, bond distances and bond angles, and least-squares planes (13 pages); listing of observed and calculated structure factors (30 pages). Ordering information is given on any current masthead page.

The First Potent Inhibitor of Squalene Synthase: A Profound Contribution of an Ether Oxygen to Inhibitor–Enzyme Interaction†

Scott A. Biller,* Michael J. Sofia, Barbara DeLange, Cornelia Forster, Eric M. Gordon, Thomas Harrity, Lois C. Rich, and Carl P. Ciosek, Jr.

The Bristol-Myers Squibb Pharmaceutical Research Institute
P.O. Box 4000, Princeton, New Jersey 08543

Received July 15, 1991

An accounting of the noncovalent forces between host and guest is key to understanding the interactions involved in molecular recognition. Such knowledge could provide predictive input for the design of new inhibitors of physiologically important enzymes. In this communication, we report the discovery of ether **6**, the first potent inhibitor of squalene synthase. This agent was designed on the basis of a proposed mechanism for the enzymatic reaction. We demonstrate herein that the ether oxygen of **6** makes a substantial contribution to the overall binding energy between enzyme and inhibitor.

Squalene synthase,[1] a key enzyme of the cholesterol biosynthetic pathway, catalyzes the reductive dimerization of farnesyl diphosphate (FPP, **1**) to squalene (**3**) via the intermediate cyclopropane, presqualene diphosphate (**2**) (Scheme I). Studies on the related prenyl transferase reaction[2] and the second step of the squalene synthase reaction[1a,2a,3] suggest that both transformations in Scheme I proceed through an initial carbocation–diphosphate ion pair. As illustrated for step 1 in Figure 1A, we envision that the donor[1] FPP is bound in the enzyme's active site via hydrophobic interactions with the isoprene portion and ionic interactions with the highly charged diphosphate moiety. We expect that the enzyme catalyzes the heterolytic cleavage of A to B by binding the two fragments more tightly as the transition-state separation is approached. Furthermore, we propose that squalene synthase utilizes an active-site acid catalyst to promote ion-pair formation.

We previously reported that **4** (Table I), a stable analogue of FPP where the reactive allylic and anhydride oxygen atoms were each replaced with CH_2, was a competitive inhibitor of rat liver microsomal squalene synthase (I_{50} = 31.5 μM, K_i = 10 μM; apparent K_m(FPP) = 12.7 μM).[4,5] Linker homologue **5**, an

† This paper is dedicated to Professor Gilbert Stork on the occasion of his 70th birthday.

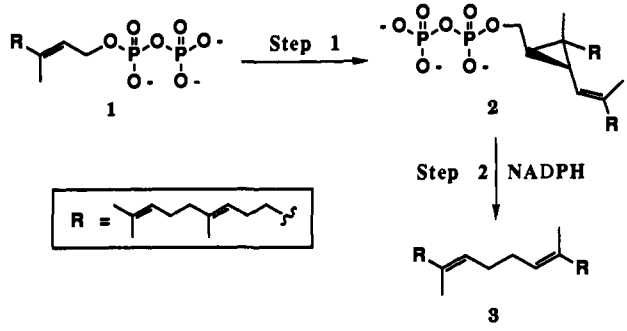
(1) (a) Review: Poulter, C. D.; Rilling, H. C. In *Biosynthesis of Isoprenoid Compounds*; Porter, J. W., Spurgeon, S. L., Eds.; Wiley: New York, 1981; Vol. 1, Chapter 8. (b) Purification from yeast: Sasiak, K.; Rilling, H. C. *Arch. Biochem. Biophys.* **1988**, *260*, 622-627. (c) Cloning from yeast: Jennings, S. M.; Tsay, Y. H.; Fisch, T. M.; Robinson, G. W. *Proc. Natl. Acad. Sci. U.S.A.* **1991**, *88*, 6038-6042. Händler, B.; Soltis, D.; Movva, R.; Kocher, H. P.; Ha, S.; Kathawala, F.; Nemecek, G. *Abstract of Papers*, 201st National Meeting of the American Chemical Society, Atlanta, GA; American Chemical Society: Washington, DC, 1991; BIOT 92.

(2) (a) Rilling, H. C. *Biochem. Soc. Trans.* **1985**, *13*, 997-1003. (b) Poulter, C. D.; Argyle, J. C.; Mash, E. A.; Laskovics, P. L.; Wiggins, P. L.; King, C. R. *Pr. Nauk. Inst. Chem. Org. Fiz. Politech. Wroclaw.* **1981**, *22*, 149-162. (c) Poulter, C. D.; Wiggins, P. L.; Le, A. T. *J. Am. Chem. Soc.* **1981**, *103*, 3926-3927. (d) Mash, E. A.; Gurria, G. M.; Poulter, C. D. *J. Am. Chem. Soc.* **1981**, *103*, 3927-3929. (e) Poulter, C. D.; Rilling, H. C. In *Biosynthesis of Isoprenoid Compounds*; Porter, J. W., Spurgeon, S. L., Eds.; Wiley: New York, 1981; Vol. 1, Chapter 4.

(3) Poulter, C. D. *Acc. Chem. Res.* **1990**, *23*, 70-77.

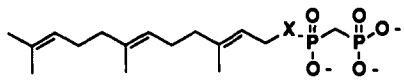
0002-7863/91/1513-8522$02.50/0

Scheme I



1 2 3

Table I. Inhibition of Rat Liver Microsomal Squalene Synthase[13]



4-12

compd	linker X	I_{50},[a] μM	compd	linker X	I_{50},[a] μM
4	$-CH_2-$	31.5	9	$-SCH_2-$	122
5	$-CH_2CH_2-$	67.0	10	$-OCH_2CH_2-$	2.3
6	$-OCH_2-$	0.05	11	$-CH_2OCH_2-$	0.15
7	$-CH_2O-$	16.0	12	$-CH_2CH_2OCH_2-$	79.3
8	$-NH_2^+CH_2-$	16.0			

[a] Reference 5.

attempt to approximate the transition-state separation between the fragments, was actually a slightly weaker inhibitor than **4**. When an ether oxygen was incorporated in the linking chain to interact as an H-bond acceptor with the putative active-site acid (example **6**), a profound increase in inhibitory activity was observed. Ether **6** (I_{50} = 0.05 μM)[5] is 1340-fold more potent as an inhibitor of squalene synthase than its carbon isostere **5**! Lineweaver-Burk analysis revealed that **6** is a competitive inhibitor with respect to FPP, K_i ≈ 37 nM.[5]

The difference between the enzyme binding energies of **6** and its carbon isostere **5** in aqueous solution ($\Delta\Delta G_{H_2O}$) is 4.3 kcal/mol.[6] The differential binding energy in the absence of solvent[7] ($\Delta\Delta G_{gas}$) is a more meaningful measure of the contribution made by the ether oxygen to active-site interactions with squalene synthase. Using a thermodynamic cycle,[7,8] we can approximate $\Delta\Delta G_{gas}$ as the sum of $\Delta\Delta G_{H_2O}$ and $\Delta\Delta G_{solv}$,[9] where $\Delta\Delta G_{solv}$ is the difference between the solvation energies of **6** and **5**. According to the empirical correlations of Hine,[10] an ether oxygen contributes 4.2 kcal/mol more than a CH_2 to the solvation energy. Thus, $\Delta\Delta G_{gas}$ for **6** relative to **5** is estimated to be 8.5 kcal/mol.[11] We speculate that this large differential binding energy is due to hydrogen bonding of the ether oxygen to an active-site acid catalyst as illustrated in Figure 1C. The ether oxygen is of intermediate basicity relative to the weakly basic phosphate ester oxygen in A and the strongly basic phosphate anion in B and can be considered to be a mimic of a species along the reaction coordinate from A to B.

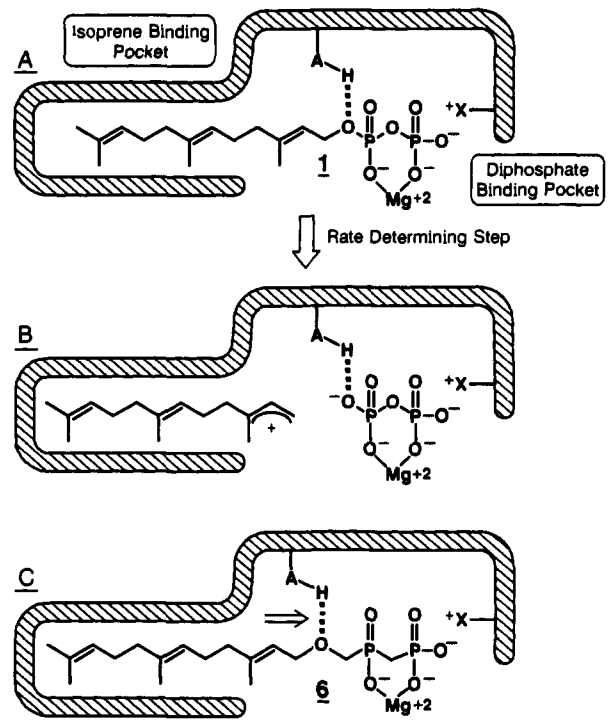
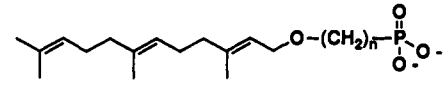


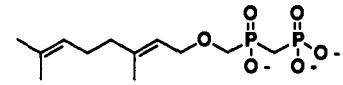
Figure 1. Hypothetical model of the donor[1] FPP binding site of squalene synthase, bound to (A) substrate FPP, (B) the allyl cation–diphosphate ion pair, and (C) ether inhibitor **6**. The active-site function A–H represents the putative acid catalyst, and X^+ represents cationic side chain(s) which ion pair with the phosphate groups.

Further studies were aimed at revealing structural features that are important for inhibition (Table I). Placement of the crucial oxygen adjacent to the phosphorus to generate phosphonate ester **7** results in 320-fold loss in potency. This is consistent with the H-bond hypothesis, since the weakly basic ester oxygen of **7**, like that of FPP itself, is a poor H-bond acceptor. Also consistent is the unimpressive activity of ammonium ion **8**, which is an H-bond donor, and the thioether **9**, which is a weak H-bond acceptor. The position of the ether oxygen relative to the phosphinic acid is critical. Insertion of an additional CH_2 between the ether and phosphinic acid results in a 46-fold loss in activity (example **10**), whereas homologation on the other side of the ether (example **11**) leads to just a 3-fold loss. Further homologation of **11** to **12**, however, is highly deleterious. The strong interaction offered by the ether oxygen cannot compensate for the loss of either phosphorus moiety (examples **13** and **14**) or the deletion of an isoprene unit (example **15**). The latter result is consistent with the observation that geranyl diphosphate is not a substrate[12a] and is a poor inhibitor[12b] for squalene synthase.



13, n = 1, I_{50} = 92 μM[5]

14, n = 3, inactive at 300 μM[5]



15, 36 % inhibition at 300 μM[5]

(4) (a) Biller, S. A.; Forster, C.; Gordon, E. M.; Harrity, T.; Scott, W. A.; Ciosek, C. P., Jr. *J. Med. Chem.* **1988**, *31*, 1869–1871. (b) Biller, S. A.; Forster, C. *Tetrahedron* **1990**, *46*, 6645–6658. (c) Related studies: McClard, R. W.; Fujita, T. S.; Stremler, K. E.; Poulter, C. D. *J. Am. Chem. Soc.* **1987**, *109*, 5544–5545.

(5) The squalene synthase assay utilized for this study is described in ref 4a. See also: Biller, S. A.; Forster, C.; Gordon, E. M.; Harrity, T.; Rich, L. C.; Marretta, J.; Scott, W. A.; Ciosek, C. P., Jr. In *Drugs Affecting Lipid Metabolism, X: Proceedings of the Xth International Symposium*; Gotto, A. M., Jr., Smith, L. C., Eds.; Elsevier: Amsterdam, 1990; pp 213–216.

(6) This value for $\Delta\Delta G_{H_2O}$ was estimated from $-RT \ln [I_{50}(\mathbf{6})/I_{50}(\mathbf{5})]$.

(7) (a) Bartlett, P. A.; Marlowe, C. K. *Science* **1987**, *235*, 569–571. (b) Morgan, B. P.; Schlotz, J. M.; Ballinger, M. D.; Zipkin, I. D.; Bartlett, P. A. *J. Am. Chem. Soc.* **1991**, *113*, 297–307.

(8) Kati, W. M.; Wolfenden, R. *Biochemistry* **1989**, *28*, 7919–7927.

(9) This approximation follows the assumption that the solvation energies of enzyme–inhibitor (E–I) complexes of **5** and **6** are similar, since it is likely that water is excluded from the binding site.[7] If anything, this assumption should lead to an underestimation of $\Delta\Delta G_{gas}$, since the solvation energy of E–**5** has the potential to be larger than that of E–**6** in the event that water is accessible to the active-site functionality (e.g., the putative acid catalyst) of E–**5** that interacts with the ether in the case of E–**6**.

(10) Hine, J.; Mookerjee, P. K. *J. Org. Chem.* **1975**, *40*, 292–298.

(11) Large $\Delta\Delta G$'s have been observed for isosteric functional group replacements in inhibitors of thermolysin[7] and adenosine deaminase.[8]

(12) (a) Koyama, T.; Ogura, K.; Seto, S. *Biochim. Biophys. Acta* **1980**, *617*, 218–224. (b) Ortiz de Montellano, P. R.; Wei, J. S.; Castillo, R.; Hsu, C. K.; Bopari, A. *J. Med. Chem.* **1977**, *20*, 243–249.

In summary, the first potent inhibitor of squalene synthase, ether **6**, was rationally designed on the basis of a proposal for the mechanism of the enzymatic reaction. This study revealed a surprisingly large enhancement in differential binding energy resulting from the replacement of a CH_2 with an ether oxygen. We speculate that the tight binding of **6** to squalene synthase is due to H bonding of the ether oxygen with a key active-site acid catalyst.[11,13]

Acknowledgment. We thank the Bristol-Myers Squibb Analytical Chemistry Department for obtaining elemental analyses, mass spectra, IR spectra, and certain NMR spectra.

Supplementary Material Available: Detailed procedure for the synthesis of **6** including spectral data and Lineweaver-Burk plot for the inhibition of rat liver microsomal squalene synthase by **6** (8 pages). Ordering information is given on any current masthead page.

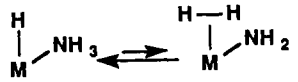
(13) All inhibitors described herein exhibited ^1H NMR, ^{13}C NMR, infrared, and mass spectra, as well as microanalyses (C, H, P), which were consistent with the assigned structures. The final target salts were purified by chromatography on CHP20P gel, as described in ref 4b.

Evidence for an Unprecedented Ir(H)(NH_3) ⇌ Ir(H_2)(NH_2) Equilibrium and Hydrogen Exchange between NH and CH Bonds

Robert Koelliker and David Milstein*

Department of Organic Chemistry
The Weizmann Institute of Science, Rehovot 76100, Israel
Received June 10, 1991

During our studies on ammonia activation, we have obtained complexes **1** and **2** by reaction of ammonia with $[Ir(C_8H_{14})_2Cl]_2$ (C_8H_{14} = cyclooctene) followed by anion exchange.[1] Complexes **3–5** are obtained by substitution reactions of **1** and **2** with PEt_3.[2,3] Our evidence suggests that these complexes are involved in an unprecedented, albeit slow, equilibrium (eq 1). When combined with a parallel equilibrium between alkyl and olefin-hydride configurations in the same complex, the result is C-H/N-H exchange.

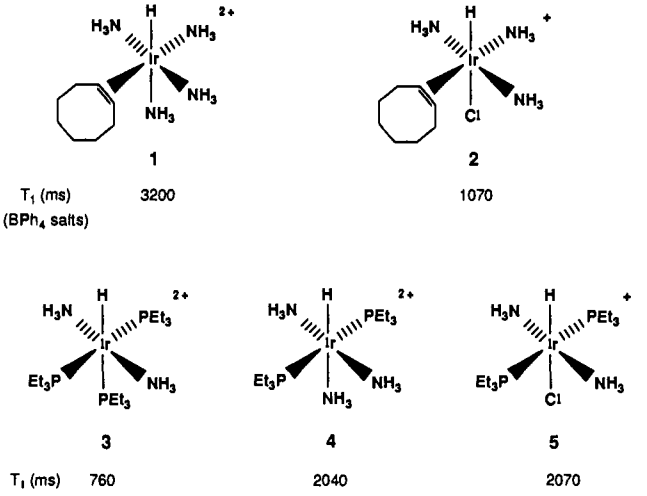


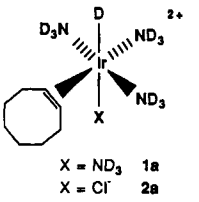
(1)

T_1 measurements[4] (Scheme I) indicate that complexes **1–5** are present in solution as classical hydrides.[5,6] Compatible with this, ^{15}N NMR measurement of the ^{15}N-enriched complexes indicates only ammine (NH_3) and no amide (NH_2) ligands.

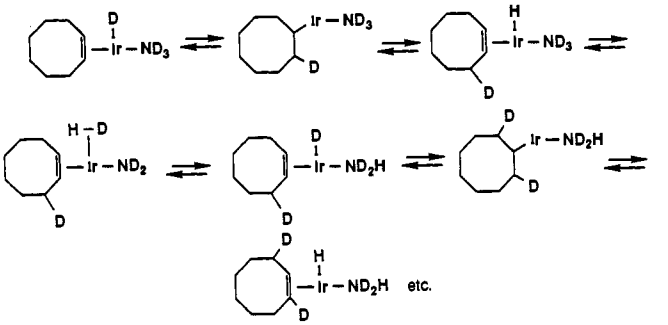
Surprisingly, when the deuterido complexes **1a** and **2a**, obtained from reaction of $[Ir(C_8H_{14})_2Cl]_2$ with ND_3, are left standing at room temperature in THF under nitrogen, Ir-H and N-H bonds

Scheme I





Scheme II



are formed. ^2D NMR measurements reveal formation of C-D bonds. This process is quite slow: when a THF solution of complexes **1a** and **2a** in a 1:1 ratio was utilized, 25% deuteration of the cyclooctene ligand (ca. 4 deuterium atoms/ligand) was observed after 20 days at room temperature. After 1 day at 65 °C, 20% olefin deuteration was obtained.[7] Interestingly, the ammine ligands differ in their degree of exchange. The NH_3 trans to the cyclooctene ligand undergoes this process most readily, and the NH_3 cis to both hydride and cyclooctene ligands exchanges to a lesser extent, whereas almost no hydrogen incorporation is observed in the ammine trans to the hydride.

The H/D exchange process between the ammine and olefin ligands most probably proceeds via an η^2-H_2 intermediate[8] formed as a result of interaction of the electronegative hydride with the more acidic N-D. Indications for $H^{\delta+}\cdots H^{\delta-}$ interaction were observed in *cis*-Ir(H)(OH)$(PMe_3)_4^+$.[9,10] The reverse of this

(1) Koelliker, R.; Milstein, D. *Angew. Chem., Int. Ed. Engl.* **1991**, *30*, 707.
(2) Complexes **3** and **4** are obtained by dropwise addition of 3 or 2 equiv of PEt_3, respectively, to a THF solution of **1** (PF_6 salt) at room temperature under nitrogen. Addition of 2 equiv of PEt_3 to **2** (PF_6 salt) under the same conditions leads to **5**.
(3) Spectral characterization of **3**: ^1H NMR (acetone-d_6) δ −12.15 (dt, 1 H, $J_{PH,trans}$ = 145.4 Hz, $J_{PH,cis}$ = 18.0 Hz); $^{31}P\{^1H\}$ NMR (THF) δ −18.3 (d, 2 P, J = 15.6 Hz), −33.1 (t, 1 P, J = 15.6 Hz); ^{15}N NMR (THF/THF-d_8) δ −473.0 (q, 2 N, J_{NH} = 71.3 Hz). **4**: ^{15}N NMR (THF/THF-d_8) δ −425.3 (dq, 1 N, J_{NH} = 68.5 Hz, J_{NIrH} = 170 Hz), −466.1 (q, 2 N, J_{NH} = 70.7 Hz); $^{31}P\{^1H\}$ NMR (THF) δ −9.6 (s); ^1H NMR (acetone-d_6) δ −21.64 (t, 1 H, J = 15.6 Hz; with $^{15}NH_3$: dt, J_{NH} = 17 Hz, J_{PH} = 15.6 Hz). **5**: ^1H NMR (acetone-d_6) δ −23.14 (t, 1 H, J = 14.6 Hz); $^{31}P\{^1H\}$ NMR (THF) δ −10.1 (s); ^{15}N NMR (THF/THF-d_8) δ −461.2 (q, 2 N, $^1J_{NH}$ = 70.6 Hz).
(4) T_1 experiments were performed at 20 °C using a Bruker WH-270 NMR spectrometer at 270 MHz. The BPh_4 salts of **1** and **2** and PF_6 salts of **3–5** were used in THF-d_8.
(5) Crabtree, R. H.; Lavin, M.; Bonneviot, L. *J. Am. Chem. Soc.* **1986**, *108*, 4032.
(6) (a) Crabtree, R. H. *Acc. Chem. Res.* **1990**, *23*, 95. (b) Hamilton, D. G.; Crabtree, R. H. *J. Am. Chem. Soc.* **1988**, *110*, 4126. (c) Ammann, C.; Isaia, F.; Pregosin, P. S. *Magn. Reson. Chem.* **1988**, *26*, 236.
(7) At equilibrium, equal scrambling between 10 D and 14 C-H atoms would lead to 41.6% D incorporation into the cyclooctene ligand.
(8) (a) Kubas, G. J. *Acc. Chem. Res.* **1988**, *21*, 120. (b) Crabtree, R. H. *Acc. Chem. Res.* **1990**, *23*, 95.
(9) (a) Milstein, D.; Calabrese, J. C.; Williams, I. D. *J. Am. Chem. Soc.* **1986**, *108*, 6387. (b) Stevens, R. C.; Bau, R.; Milstein, D.; Blum, O.; Koetzle, T. F. *J. Chem. Soc., Dalton Trans.* **1990**, 1429.
(10) Although less relevant, participation of metals in hydrogen-bonding interactions was recently addressed: (a) Brammer, L.; Charnock, J. H.; Goggin, P. L.; Goodfellow, R. J.; Orpen, A. G.; Koetzle, T. F. *J. Chem. Soc., Dalton Trans.* **1991**, 1789. (b) Kristjánsdóttir, S. S.; Norton, J. R.; Moroz, A.; Sweany, R. L.; Whittenburg, S. L. *Organometallics* **1991**, *10*, 2357.

0002-7863/91/1513-8524$02.50/0