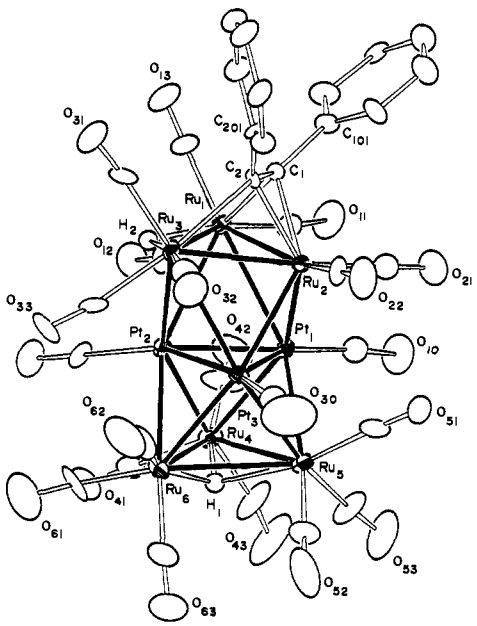
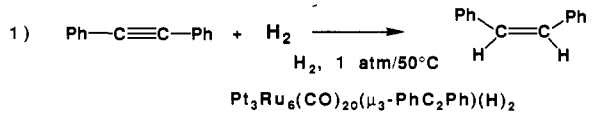


Figure 2. An ORTEP diagram of $Pt_3Ru_6(CO)_{20}(\mu_3\text{-}PhC_2Ph)(\mu_3\text{-}H)(\mu\text{-}H)$, **2**. Selected interatomic distances (Å) are Pt(1)–Pt(2) = 2.631 (1), Pt(1)–Pt(3) = 2.686 (1), Pt(2)–Pt(3) = 2.636 (1), Ru(1)–Ru(2) = 2.854 (2), Ru(1)–Ru(3) = 2.986 (2), Ru(2)–Ru(3) = 2.771 (2), Ru(4)–Ru(5) = 3.052 (2), Ru(4)–Ru(6) = 2.921 (2), Ru(5)–Ru(6) = 3.037 (2), C(1)–C(2) = 1.36 (2).

$PhC_2Ph)(\mu_3\text{-}H)(\mu\text{-}H)$, **2**, in 28% yield.[8] Compound **2** was characterized by IR, ^1H NMR, and single-crystal X-ray diffraction analyses, and an ORTEP diagram of its molecular structure is shown in Figure 2.[9] This molecule also consists of a layered structure of Pt_3 and Ru_3 triangles analogous to that of **1**, but in addition, there is a triply bridging PhC_2Ph ligand coordinated to the Ru(1)–Ru(2)–Ru(3) triangle. The alkyne has adopted the μ-$\parallel$ coordination mode that is commonly observed for these ligands in trimetallic cluster complexes.[9] Complex **2** contains only two hydride ligands, δ −17.97 (s, 1 H), −18.71 (s, 1 H) at −58 °C. These were observed crystallographically as an edge-bridging ligand across the Ru(1)–Ru(3) bond and a triply bridging ligand spanning the Ru(4)–Ru(5)–Ru(6) triangle.[10] Bridging alkyne ligands have been observed in ruthenium cluster complexes[9b] and in platinum cluster complexes.[11]

Preliminary experiments have indicated that compound **2** is also an unusually active catalyst for the selective hydrogenation of diphenylacetylene to (*Z*)-stilbene, eq 1.[12] When 8.0 mg of **2** was



allowed to react with PhC_2Ph (present in a 100-fold excess) in hexane solution (65 mL) under 1 atm of H_2 at 50 °C for 1 h, 31% of the PhC_2Ph was converted to (*Z*)-stilbene with 100% selectivity, corresponding to a turnover frequency of 31 h^{-1}. The catalytic activity for **2** is both significantly higher and more selective than that observed for catalysts derived from cluster complexes of either pure ruthenium or pure platinum.[13]

The reason for the preference of the alkyne for a triruthenium site in **2** is not clear at this time, but coordination preferences sometimes referred to as "metalloselectivity"[14] can be expected to lead to reactivity preferences and the potential for cooperative reactivity by involving the different types of metal atoms. This may provide an explanation for the high catalytic activity of **2**. It may also explain some aspects of the unique activity of certain metal alloy catalysts.[2g]

Acknowledgment. These studies were supported by the National Science Foundation under Grant No. CHE-8919786.

Supplementary Material Available: Complete listings of crystal data, atomic positional parameters, bond distances and angles, and anisotropic thermal parameters for **1** and **2** (23 pages); listing of structure factor amplitudes for **1** and **2** (49 pages). Ordering information is given on any current masthead page.

(7) Mingos, D. M. P.; May, A. S. In *The Chemistry of Metal Cluster Complexes*; Shriver, D. F., Kaesz, H. D., Adams, R. D., Eds.; VCH Publishers: New York, 1990; Chapter 2.

(8) **1**, 15.0 mg (0.0084 mmol), and 3.3 mg (0.0185 mmol) of Ph_2C_2 were dissolved in 80 mL of hexane, and the solution was refluxed for 70 min. Separation by TLC using 3/1 hexane/CH_2Cl_2 solvent yielded gray-green $Ru_7Pt_3(CO)_{22}(\mu_3\text{-}H)_2$[2d] (1.2 mg, 10%), followed by gray-green $Ru_6Pt_3(CO)_{20}(Ph_2C_2)(\mu_3\text{-}H)(\mu\text{-}H)$, **2** (4.2 mg, 28%). IR for **2**: 2098 (w), 2065 (vs), 2033 (m), 2015 (w, sh), 2000 (w, sh), 1950 (vw, sh). ^1H NMR (δ in CD_2Cl_2 at −58 °C): 7.70–7.05 (m, 10 H), −17.97 (s, 1 H), −18.71 (s, 1 H). Crystal data for **2**: space group = *Pccn*, a = 10.852 (4) Å, b = 45.509 (2) Å, c = 17.172 (3) Å, Z = 8, 4441 reflections, R = 0.043. Diffraction data at 20 °C were collected on a Rigaku AFC5R diffractometer using Cu Kα radiation.

(9) (a) Raithby, P. R.; Rosales, M. J. *Adv. Inorg. Chem. Radiochem.* **1985**, *29*, 169. (b) Sappa, E.; Tiripicchio, A.; Braunstein, P. *Chem. Rev.* **1983**, *83*, 203.

(10) The two hydride ligands that were lost in the reaction were transferred to an equivalent of PhC_2Ph to yield (*Z*)-stilbene. This was confirmed by ^1H NMR spectroscopy by performing the reaction in C_6D_6 solvent.

(11) (a) Rashidi, M.; Puddephatt, R. J. *Organometallics* **1988**, *7*, 1636. (b) Manojlovic, L.-M.; Muir, K. W.; Rashidi, M.; Schoettel, G.; Puddephatt, R. J. *Organometallics* **1991**, *10*, 1719. (c) Stone, F. G. A. *Inorg. Chim. Acta* **1981**, *50*, 33.

(12) A solution of 8.0 mg of **2** (0.004 14 mmol) and 73.7 mg of PhC_2Ph (0.414 mmol) in 65 mL of hexane was heated to 50 °C and purged slowly with H_2 (1 atm) for 1 h. Workup by TLC yielded 22.6 mg of (*Z*)-stilbene (31%, 0.126 mmol), 36.9 mg of unreacted PhC_2Ph, and 1.8 mg of 1,2-diphenylethane (0.0099 mmol). **2**, 7.3 mg was recovered. The turnover number of formation of (*Z*)-stilbene is 31 at a rate of 31 h^{-1}. There was no evidence for the formation of (*E*)-stilbene.

(13) (a) Süss-Fink, G.; Neumann, G. *The Chemistry of the Metal-Carbon Bond*; Hartley, F. R., Ed.; Wiley: New York, 1989; Vol. 5, Chapter 7 and references therein. (b) Fusi, A.; Ugo, R.; Psaro, R.; Braunstein, P.; Dehand, J. *J. Mol. Catal.* **1982**, *16*, 217. (c) Castiglioni, M.; Sappa, E.; Valle, M.; Lanfranchi, M.; Tiripicchio, A. *J. Organomet. Chem.* **1983**, *241*, 99. (d) Castiglioni, M.; Giordano, R.; Sappa, E. *J. Organomet. Chem.* **1989**, *369*, 419.

(14) (a) Matsuzaka, H.; Kodama, T.; Uchida, Y.; Hidai, M. *Organometallics* **1988**, *7*, 1608. (b) Hidai, M.; Matsuzaka, H. *Polyhedron* **1988**, *7*, 2369. (c) Rossi, S.; Pursiainen, J.; Pakkanen, T. A. *J. Organomet. Chem.* **1990**, *397*, 81. (d) Braunstein, P.; Mourey, L.; Rosé, J.; Granger, P.; Richert, T.; Balegroune, F.; Grandjean, D. *Organometallics* **1992**, *11*, 2628.

Site-Specific Covalent Duocarmycin A—Intramolecular DNA Triplex Complex

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DNA triple helices[1] can be either of the pyrimidine·purine·pyrimidine type, where the third pyrimidine strand is positioned in the major groove and parallel to the purine strand,[2] or of the pyrimidine·purine·purine type where the third purine strand is positioned in the major groove and antiparallel to the purine

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(1) (a) Felsenfeld, G.; Davies, D.; Rich, A. *J. Am. Chem. Soc.* **1957**, *79*, 2023–2024. (b) Arnott, S.; Selsing, E. *J. Mol. Biol.* **1974**, *88*, 509–521. (c) Wells, R. D.; Collier, D. A.; Hanvey, J. C.; Shimizu, M.; Wohlraub, F. *FASEB J.* **1988**, *2*, 2939–2949.

(2) (a) Moser, H.; Dervan, P. B. *Science* **1987**, *238*, 645–650. (b) Le Doan, T.; Perronault, L.; Praseuth, D.; Habhoub, N.; Decoult, J. L.; Thuong, N. T.; Lhomme, J.; Helene, C. *Nucleic Acids Res.* **1987**, *15*, 7749–7760. (c) Plum, G. E.; Park, Y. W.; Singleton, S. F.; Dervan, P. B.; Breslauer, K. J. *Proc. Natl. Acad. Sci. U.S.A.* **1990**, *87*, 9436–9440. (d) Roberts, R. W.; Crothers, D. M. *Proc. Natl. Acad. Sci. U.S.A.* **1991**, *88*, 9397–9401.

0002-7863/92/1514-10658$03.00/0

strand.[3] For either family of triplexes, the minor groove of the Watson–Crick duplex is unoccupied and can potentially accommodate either noncovalent or covalent binding ligands. Recent contributions have addressed antibiotic ligand binding to DNA triplexes,[4,5] to triplex–duplex junctions,[6] and to third strand crossover sites in DNA triplexes.[7]

Duocarmycin A is a DNA-alkylating agent[8] with a structure that bears similarity to a related antibiotic CC-1065,[9] which covalently binds to the N^3 of adenine in a specific sequence context in the minor groove of duplex DNA. Since duocarmycin A has one fewer substituted indole ring than CC-1065, it appeared to be the appropriate ligand for initial studies aimed at probing the minor groove of DNA triplexes. The goal was to generate a complex of duocarmycin A site-specifically and covalently bound to the minor groove N^3 atom of a single adenine of a pyrimidine·purine·pyrimidine DNA triplex and to monitor the potential groove cross-talk[5] between the duocarmycin A and the third pyrimidine strand.

Intramolecular pyrimidine·purine·pyrimidine triplexes linked through T_n loops can be readily generated[10] and yield excellent and readily interpretable exchangeable and nonexchangeable proton NMR spectra.[11,12] Duocarmycin A exhibits a sequence specificity for covalent binding to the 3′-terminal adenine in a 5′-A-A-A-A-3′ stretch of duplex DNA,[13] and hence studies were undertaken on an intramolecular triplex **1** containing this segment within the purine strand. We were able to generate and HPLC purify the drug–triplex complex[14] in which duocarmycin A was covalently bound to a single adenine in the triplex. The available one- and two-dimensional NMR data on the duocarmycin–triplex complex have been analyzed to identify the duocarmycin A binding site and the directionality of its alignment in the minor groove, the nature of hydrogen-bonding interactions at the modified

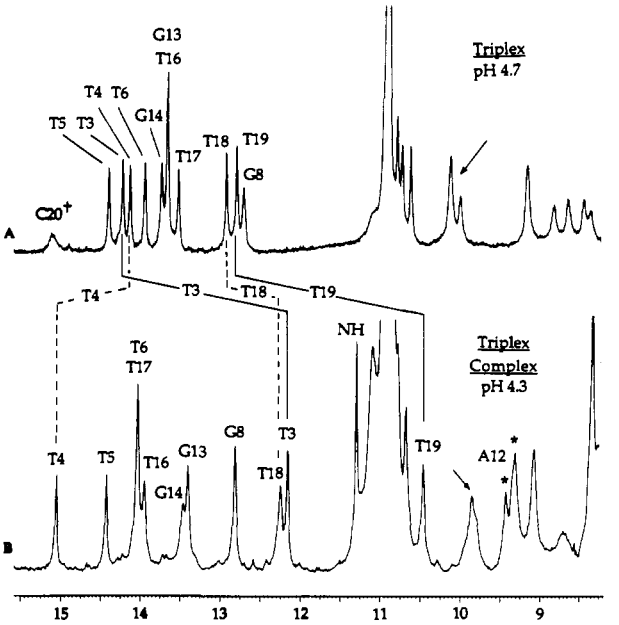
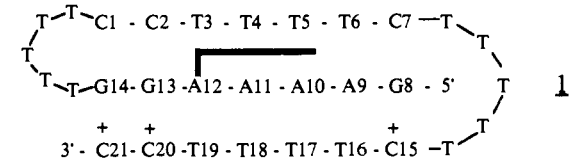


Figure 1. Exchangeable proton NMR spectra from 8.5 to 15.5 ppm of (A) the unmodified 31-mer triplex, pH 4.7, and (B) the duocarmycin–triplex complex, pH 4.3, in 100 mM NaCl, 10 mM phosphate, and H_2O at 5 °C. The thymine and guanine imino protons resonate between 12.5 and 15.5 ppm, the thymine loop imino protons resonate between 10.5 and 11.0 ppm, the hydrogen-bonded amino protons of the protonated cytidines resonate between 9.5 and 10.5 ppm (designated by arrows), and the exposed amino protons of the protonated cytidines resonate between 8.5 and 9.5 ppm in the unmodified triplex spectrum. The 6-amino protons of the duocarmycin–N^3-adenine adduct are designated by asterisks with the upfield amino proton superimposed on another exchangeable resonance in the spectrum of the complex.

T·*A·T triple, and cross-talk[5] between the covalently bound duocarmycin A in the minor groove and the third strand in the major groove.



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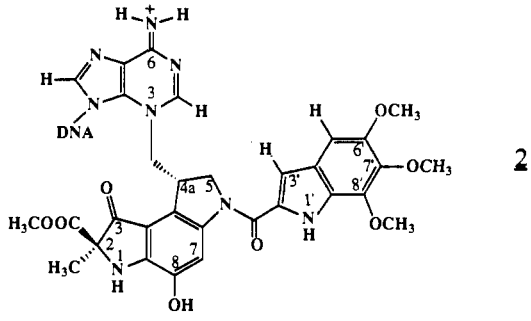

2

The exchangeable proton NMR spectra (8.0–16.0 ppm) of the unmodified triplex and the duocarmycin–triplex complex at acidic pH and low temperature are plotted in Figure 1A,B, respectively. Formation of the covalent duocarmycin–N^3-adenine adduct **2** should result in protonation of the adenine N^6 atom, resulting in downfield shifts of the adenine 6-amino protons similar to what has been previously reported for CC-1065–N^3-adenine adducts.[15] The downfield-shifted exchangeable protons at 9.35 and 9.42 ppm (designated by asterisks) in the duocarmycin–triplex complex (Figure 1B) are assigned to the 6-amino protons of A12 based

(3) (a) Letai, A. G.; Palladino, M. A.; Fromm, E.; Rizzo, V.; Fresco, J. R. *Biochemistry* **1988**, *27*, 9108–9112. (b) Kohwi, Y.; Kohwi-Shigematsu, T. *Proc. Natl. Acad. Sci. U.S.A.* **1988**, *85*, 3781–3785. (c) Beal, P. A.; Dervan, P. B. *Science* **1991**, *251*, 1360–1363. (d) Durland, R. H.; Kessler, D. J.; Gunnell, S.; Duvic, M.; Pettitt, B. M.; Hogan, M. E. *Biochemistry* **1991**, *30*, 9246–9255. (e) Radhakrishnan, I.; De los Santos, C.; Patel, D. J. *J. Mol. Biol.* **1991**, *221*, 1403–1418. (f) Pilch, D. S.; Levenson, C.; Shafer, R. H. *Biochemistry* **1991**, *30*, 6081–6087. (g) Chen, F. M. *Biochemistry* **1991**, *30*, 4472–4479. (h) Sun, J.; De Bizemont, T.; Duval-Valentin, G.; Montenay-Garestier, T.; Helene, C. *C. R. Acad. Sci. Paris* **1991**, *313*, 585–590.

(4) Mergny, J. L.; Duval-Valentin, G.; Nguyen, C. H.; Perroualt, L.; Faucon, B.; Rougee, M.; Montenay-Garestier, T.; Bisagni, E.; Helene, C. *Science* **1992**, *256*, 1681–1684.

(5) Park, Y. W.; Breslauer, K. J. *Proc. Natl. Acad. Sci. U.S.A.* **1992**, *89*, 6653–6657.

(6) Collier, D. A.; Mergny, J. L.; Thuong, N. T.; Helene, C. *Nucleic Acids Res.* **1991**, *19*, 4219–4224.

(7) Beal, P. A.; Dervan, P. B. *J. Am. Chem. Soc.* **1992**, *114*, 4976–4982.

(8) (a) Takahashi, I.; Takahashi, K. I.; Ichimura, M.; Morimoto, M.; Asano, K.; Kawamoto, I.; Tomita, F.; Nakano, H. *J. Antibiot.* **1988**, *41*, 1915–1917. (b) Sugiyama, H.; Hosoda, M.; Saito, I.; Asai, A.; Saito, H. *Tetrahedron Lett.* **1990**, *31*, 7197–7200.

(9) (a) Warpehoski, M. A.; Hurley, L. H. *Chem. Res. Toxicol.* **1988**, *1*, 315–333. (b) Coleman, R. S.; Boger, D. L. In *Studies in Natural Product Chemistry*; Atta-ur-Rahman, Ed.; Elsevier: Amsterdam, 1989; Vol. 3, pp 301.

(10) (a) Haner, R.; Dervan, P. B. *Biochemistry* **1990**, *29*, 9761–9765. (b) Sklenar, V.; Feigon, J. *Nature* **1990**, *345*, 836–838. (c) Radhakrishnan, I.; Patel, D. J.; Gao, X. *J. Am. Chem. Soc.* **1991**, *113*, 8542–8544.

(11) (a) De los Santos, C.; Rosen, M.; Patel, D. J. *Biochemistry* **1989**, *28*, 7282–7289. (b) Radhakrishnan, I.; Gao, X.; De los Santos, C.; Live, D.; Patel, D. J. *Biochemistry* **1991**, *30*, 9022–9030. (c) Radhakrishnan, I.; Patel, D. J.; Gao, X. *Biochemistry* **1992**, *31*, 2514–2523. (d) Radhakrishnan, I.; Patel, D. J.; Veal, J. M.; Gao, X. *J. Am. Chem. Soc.* **1992**, *114*, 6913–6915.

(12) (a) Rajajopal, P.; Feigon, J. *Biochemistry* **1989**, *28*, 7559–7570. (b) Mooren, M. M.; Pulleyblank, D. E.; Wizmenga, S. S.; Bloomers, M. J.; Hilbers, C. W. *Nucleic Acids Res.* **1990**, *18*, 6523–6529. (c) Macaya, R. F.; Gilbert, D. E.; Malek, S.; Sinsheimer, J. S.; Feigon, J. *Science* **1991**, *254*, 270–274. (d) Macaya, R. F.; Wang, E.; Schultze, P.; Sklenar, V.; Feigon, J. *J. Mol. Biol.* **1992**, *225*, 755–773.

(13) Boger, D. L.; Ishizaki, T.; Zarrinmayeh, H.; Munk, S. A.; Kitos, P. A.; Suntornwat, O. *J. Am. Chem. Soc.* **1990**, *112*, 8961–8971.

(14) The adduct was prepared by adding 2 equiv of duocarmycin A to 1000 A_{260} units of triplex in 5 mL of a 200 mM NaCl, 20 mM phosphate, aqueous solution containing 20% methanol, pH 5.0, 4 °C for 12 h in the dark. The major adduct (>80% of product) was purified by three successive preparative HPLC treatments on a C_{18} column using a 20 mM phosphate/methanol gradient. Three minor adducts (<20% of product) were resolved on HPLC but were not characterized further.

(15) (a) Lin, C. H.; Hurley, L. H. *Biochemistry* **1990**, *29*, 9503–9507. (b) Lin, C. H.; Sun, D.; Hurley, L. H. *Chem. Res. Toxicol.* **1991**, *4*, 21–26. (c) Lin, C. H.; Beale, J. M.; Hurley, L. H. *Biochemistry* **1991**, *30*, 3597–3602.

on observed NOEs to the imino protons of T3 and T19 within the T3·*A12·T19 triple (supplementary Figure 1). These results establish that the duocarmycin–triplex complex under study corresponds to site-specific covalent modification of the N^3 of A12 in the triplex.

The observation of hydrogen-bonded amino protons of protonated cytidines from the pyrimidine third strand between 9.75 and 10.25 ppm (designated by arrows) in both the unmodified triplex (Figure 1A) and the duocarmycin–triplex complex (Figure 1B) establishes that the covalently bound duocarmycin A in the minor groove does not expel the pyrimidine third strand from the major groove of the triplex at low temperature and acidic pH.

However, the covalently bound duocarmycin A in the minor groove affects the pH-dependent triplex–duplex equilibrium which reflects protonation of the third strand cytidines involved in C^+·GC triple formation.[16] The transition midpoint for this equilibrium for the unmodified triplex has a pK_a of 6.8,[17] while the value drops to a pK_a of 5.0 for the duocarmycin–triplex complex[18] (supplementary Figure 2). This result establishes that the covalently bound duocarmycin A in the minor groove lowers the pK_a for protonation of third strand cytidines in the major groove by 1.8 pH units in the complex.

The narrow imino protons in the 10.5–15.5 ppm region for both the triplex and the complex have been assigned following analysis of NOESY data sets using procedures reported previously.[11,12] These assignments are listed over the spectra and establish large upfield shifts at the imino protons of T3 ($\Delta\delta$ = 2.04 ppm) and T19 ($\Delta\delta$ = 2.31 ppm) on complex formation. This suggests weakening of the hydrogen bonds involving these imino protons at the modified T3·*A12·T19 triple induced by the covalently bound duocarmycin A.

The directionality of the duocarmycin A covalently linked to the N^3 atom of A12 in the minor groove of the triplex has been determined by monitoring intermolecular NOEs in the duocarmycin–triplex complex. We observe NOEs between the duocarmycin A indole OCH_3-6′ protons and the imino protons of T5 and T6 in the triplex (supplementary Figure 1), establishing that the duocarmycin A indole ring is directed toward the central T·A·T triple rich segment of the triplex. Consistent with this conclusion are the complexation shifts observed at the thymine imino protons of the T4-A11-T18 triple which flanks the T3·*A12·T19 triple modification site (Figure 1). Further characterization of the intermolecular contacts in the duocarmycin–triplex complex must await analysis of nonexchangeable proton data.

Our research complements the contributions of Park and Breslauer[5] who reported on the temperature-dependent optical and calorimetric measurements of the binding of netropsin to the poly(dT·dA·dT) triplex. They established that noncovalent netropsin binding in the minor groove of the triplex occurs without disruption of the third strand in the major groove and profoundly influences the melting energetics and cooperativity of the triplex–duplex transition.[5]

Acknowledgment. This research was supported by NIH Grants GM-34504 and CA-46778 to D.J.P. We thank Dr. Hiromitsu Saito of the Kyowa Hakko Kogyo Co. for a generous supply of duocarmycin A and Dr. Hiroshimi Suziyama of Kyoto University for advice and encouragement.

Supplementary Material Available: A NOESY plot of the duocarmycin–triplex complex and plots of pH dependence of the triplex–duplex equilibrium for the unmodified 31-mer triplex and the complex (4 pages). Ordering information is given on any current masthead page.

(16) (a) Lee, J. S.; Johnson, D. A.; Morgan, A. R. *Nucleic Acids Res.* **1979**, *6*, 3073–3091. (b) Povsic, T. J.; Dervan, P. B. *J. Am. Chem. Soc.* **1989**, *111*, 3059–3061.

(17) The imino protons of the duplex and triplex are in slow exchange in the unmodified 31-mer, and the pH-dependent transition midpoint can be monitored by changes in the area of the assigned imino protons in both states during the transition.

(18) The imino protons of the duplex and triplex are in intermediate exchange in the complex, and the pH-dependent transition midpoint can be monitored by the average chemical shift changes during the transition.

Highly Efficient Oxidation of Alkanes and Alkyl Alcohols with Heteroaromatic *N*-Oxides Catalyzed by Ruthenium Porphyrins

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Many transition metal-catalyzed oxidation systems for alkenes or other relatively unreactive compounds have been developed,[1,2] however, it still remains, intellectually and practically, a challenging objective to oxidize such compounds efficiently by using chemically stable and tractable oxidants. We studied the reactivity of ruthenium porphyrin complexes as oxidation catalysts because of their relation to cytochrome P-450[3] and have reported the unique reactivity of these complexes to catalyze the efficient epoxidation of olefins by heteroaromatic *N*-oxides.[4] These *N*-oxides, represented by pyridine *N*-oxide, are highly stable and have never been used as effective oxidants for the catalytic oxidation of olefins or alkanes except in our work.[5,6] We have now found that the catalytic ability of ruthenium porphyrins is enhanced by the presence of a small amount of HCl or HBr and that, in the presence of these acids, the oxidation of alkanes or alkyl alcohols with pyridine *N*-oxides is also catalyzed by ruthenium porphyrins with high efficiency.

We used 2,6-dichloropyridine *N*-oxide as the oxidant and $RuTMP(O)_2$[7,8] as the catalyst for the oxidation of alkanes or alkyl alcohols, because they were the reagents of choice for the epoxidation reported in a previous paper.[4a] To a mixture of adamantane (1.0 mmol), 2,6-dichloropyridine *N*-oxide (1.3 mmol), 4A molecular sieves (5.0 g)[9], and benzene (5.0 mL) were added 2–3 drops of concentrated aqueous HCl (0.1–0.2 mL, 1–2 mmol) and $RuTMP(O)_2$ (5 μmol). The mixture was stirred under Ar for 24 h at room temperature; almost all of the adamantane was consumed to afford adamantan-1-ol, adamantane-1,3-diol, and adamantan-2-one in yields of 68%, 25%, and 1% based on adamantane, respectively (run 1).

The results for the oxidation of adamantane under several conditions are summarized in Table I. Adamantane was also efficiently oxidized in the presence of HBr[10] instead of HCl (run 2). In contrast, without the addition of these acids, only a 4% yield of oxidation products was obtained and most of the adamantane remained intact after the reaction for 24 h (run 3). RuTMP(CO)[7] was also an effective catalyst (run 5). The reaction

(1) Meunier, B. *Bull. Soc. Chim. Fr.* **1986**, 578.

(2) (a) Shilov, A. E. In *Activation and Functionalization of Alkanes*; Hill, C. L., Ed.; John Wiley & Sons: New York, 1989; Chapter I. (b) Mansuy, D.; Battioni, P. *Ibid.*, Chapter VI. (c) Suslick, K. S. *Ibid.*, Chapter VII. (d) Hill, C. L. *Ibid.*, Chapter VIII. (e) Barton, D. H. R.; Ozbalik, N. *Ibid.*, Chapter IX. (f) Tolman, C. A.; Druliner, J. D.; Nappa, M. J.; Herron, N. *Ibid.*, Chapter X.

(3) (a) McMurry, T. J.; Groves, J. T. In *Cytochrome P-450: Structure, Mechanism, and Biochemistry*; Ortiz de Montellano, P., Ed.; Plenum: New York, 1986; Chapter I. (b) Mansuy, D. *Pure Appl. Chem.* **1987**, *59*, 759.

(4) (a) Higuchi, T.; Ohtake, H.; Hirobe, M. *Tetrahedron Lett.* **1989**, *30*, 6545. (b) Higuchi, T.; Ohtake, H.; Hirobe, M. *Ibid.* **1991**, *32*, 7435. (c) Ohtake, H.; Higuchi, T.; Hirobe, M. *Ibid.* **1992**, *33*, 2521.

(5) (a) Ochiai, E. *Aromatic Amine Oxides*; Elsevier: Amsterdam, 1967. (b) Katritzky, A. R.; Lagowski, J. M. In *Chemistry of The Heterocyclic N-Oxides*; Academic: London, 1971; Chapter III-2.

(6) Heteroaromatic *N*-oxides are less reactive as oxidants than aliphatic amine *N*-oxides. Only photochemical oxygen atom transfer reactions from heteroaromatic *N*-oxides to olefins or alkanes have been reported. However, these reactions did not proceed efficiently and selectively [Tsuchiya, T.; Arai, H.; Igeta, H. *Tetrahedron Lett.* **1969**, 2747].

(7) TPP: tetraphenylporphyrinato. TMP: tetramesitylporphyrinato. TDCPP: tetrakis(2,6-dichlorophenyl)porphyrinato.

(8) Groves, J. T.; Quinn, R. *Inorg. Chem.* **1984**, *23*, 2844. $RuTDCPP(O)_2$ was also prepared according to the method of this report.

(9) The reaction did not proceed efficiently without the addition of molecular sieves. Water may inhibit the reaction. As shown in Figure 1b, anhydrous acids are effective in the absence of these sieves.

(10) 1-Bromoadamantane was not detected under this condition.

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