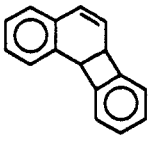
an excellent precursor of **1**. Mesylate **4**, as a ca. 5:1 mixture of the cis and trans isomers,[17] was prepared by starting from the corresponding α-(trimethylsilyl)cyclobutanone (**5**) prepared as described by Swenton et al.[18] Ketone **5** was reduced by AlH_3[19,20] to a ca. 5:1 mixture of the cis and trans isomers of the corresponding alcohol, which was converted to the mesylate by standard techniques.

The ^1H NMR spectrum of the solution formed by mixing an acetonitrile-d_3 (CD_3CN) solution of **4** (10^{-3} M) and a CD_3CN solution of tetrabutylammonium fluoride (TBAF)[16] (5×10^{-2} M) at a total flow rate of 45 mL/min[12] is shown in Figure 1a. The three strong peaks at δ 6.36, 6.26, and 5.78 are assigned to the reactive molecule benzocyclobutadiene (**1**). The spectrum shown in Figure 1b was obtained at a total flow rate of 3 mL/min and is plotted with smaller line broadening than the spectrum shown in Figure 1a. The spectrum in Figure 1b shows that the peaks at δ 6.26 and 5.78 are the AA'BB' multiplets resulting from the six-membered-ring protons of **1**. Thus the lowest field signal at δ 6.36 is assigned to the protons of the four-membered ring.

At slower flow rates, the signals for **1** are replaced with those of an intermediate, which relatively rapidly (within minutes) changes to dimer **6**, the known product of the dimerization of **1**.[21] Currently we are studying this transformation.



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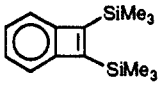
The positions of the six-membered-ring proton signals (δ 6.26 and 5.78) of **1** are very close to those reported (δ 6.30 and 5.75 in CD_3CN) for 1,2-bis(trimethylsilyl)benzocyclobutadiene (**7**).[11]



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It is interesting to note that these two signals are also similar to those of the ring protons of *o*-xylylene (**2**) which are at δ 6.3 and 6.0,[12] and to the signal of the six-membered-ring protons of 2,2-dimethylisoindene (**8**, 2,2-dimethyl-2*H*-indene) which is at δ 6.08.[22] The six membered ring proton signals of **1**, however, are significantly upfield from the benzene-ring protons of benzocyclobutene (δ 7.0)[23] and biphenylene (δ 6.72 and 6.62).[24]

The position of the four membered ring proton signal (δ 6.36) of **1** is very close to that of the olefinic-ring protons of cyclobutene (δ 6.0)[25] and 3,4-dimethylenecyclobutene (δ 6.7)[26] and to that of the five-membered-ring protons of **8** (δ 6.55).[22]

The picture of **1** that emerges from these comparisons is that it is a nonaromatic (polyolefinic) compound, not an aromatic or antiaromatic compound. The similarities of the spectra of **1**, **2**, and **8** are consistent with the view that it is an *o*-QDM, structure **9**. However, the X-ray structure of a highly substituted ben-

9

zocyclobutadiene, 1,2-di-*tert*-butyl-3,4,5,6-tetramethylbenzocyclobutadiene, indicates that the benzocyclobutadiene framework is better represented by structure **1**.[27] Of course, the large substituents could be distorting the benzocyclobutadiene framework.

Although we have not studied the kinetics of the dimerization of **1**, it is evident from our flow NMR experiments that it is slightly less reactive than *o*-xylylene (**2**).

Acknowledgment. This work was supported by the U.S. Department of Energy, Office of Basic Energy Sciences, Chemical Sciences Division, under Contract W-7405-ENG-82. The cooperation and excellent technical assistance of Robert David Scott, the operator of the NMR spectrometer, are greatly appreciated.

Registry No. 1, 4026-23-7; *cis*-**4**, 126543-57-5; *trans*-**4**, 126543-58-6.

(17) **4** (5:1 mixture of cis and trans isomers): mp 38-47 °C; IR (KBr) 3024, 1362, 1173, 843 cm^{-1}; ^1H NMR (of major isomer) ($CDCl_3$) δ 7.31-7.01 (m, 4 H), 5.94 (d, J = 5.1 Hz, 1 H), 3.41 (d, J = 5.1 Hz, 1 H), 3.07 (s, 3 H), 0.07 (s, 9 H); ^{13}C NMR (of major isomer) ($CDCl_3$) δ 144.55, 141.52, 130.66, 126.43, 123.40, 121.44, 76.79, 42.26, 38.57, -2.02; HRMS calcd for $C_{12}H_{18}O_3SSi$ 270.0746, found 270.0743.

(18) (a) Chenard, B. L.; Slapak, C.; Anderson, D. K.; Swenton, J. S. *J. Chem. Soc., Chem. Commun.* **1981**, 179. (b) Spangler, L. A. Ph.D. Dissertation, The Ohio State University, Columbus, OH, 1984.

(19) Hart, H.; Hartlage, J. A.; Fish, R. W.; Rafos, R. R. *J. Org. Chem.* **1966**, *31*, 2244.

(20) Jorgenson, M. J. *Tetrahedron Lett.* **1962**, 559.

(21) (a) Cava, M. P.; Napier, D. R. *J. Am. Chem. Soc.* **1957**, *79*, 1701. (b) Cava, M. P.; Napier, D. R. *J. Am. Chem. Soc.* **1956**, *78*, 500. (c) Nenitzescu, C. D.; Avram, M.; Dinu, D. *Chem. Ber.* **1957**, *90*, 2541.

(22) Dolbier, W. R., Jr.; Kazumasa, M.; Michl, J.; Horák, D. V. *J. Am. Chem. Soc.* **1977**, *99*, 3876.

(23) Thummel, R. P.; Nutakul, W. *J. Org. Chem.* **1977**, *42*, 300.

(24) Barton, J. W.; Rowe, D. J. *Tetrahedron* **1985**, *41*, 1323.

(25) Salaün, J.; Fadel, A. *Org. Synth.* **1986**, *64*, 50.

(26) Huntsman, W. D.; Wristers, H. J. *J. Am. Chem. Soc.* **1967**, *89*, 342.

(27) Winter, W.; Straub, H. *Angew. Chem., Int. Ed. Engl.* **1978**, *17*, 127.

Anomeric Specificity of 3-Deoxy-D-*manno*-2-octulosonate 8-Phosphate Phosphatase from *Escherichia coli*

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3-Deoxy-D-*manno*-2-octulosonate (KDO) is a specific constituent of the lipopolysaccharide (LPS) of most Gram-negative bacteria, and it provides the link between lipid A and the growing polysaccharide chain.[1] The synthesis and activation of KDO is a vital part of the assembly process of lipopolysaccharides in Gram-negative bacteria.[2] Indeed, it has been shown that an interruption of the production or utilization of KDO leads to a buildup of LPS precursors, and growth stasis.[3] There are at least four enzymes involved in the synthesis and utilization of KDO.[1-3] The KDO 8-phosphate produced from D-arabinose 5-phosphate is dephosphorylated by KDO 8-phosphate phosphatase, and the resulting KDO is then converted into cytidine monophosphate KDO by CTP:CMP-KDO cytidylyl transferase (CMP-KDO synthetase). This synthetase is considered to be the rate-limiting step in the biosynthetic incorporation of KDO into LPS.[2a] Recent studies have revealed that the β-pyranoside form of KDO, a minor form in solution, is the actual substrate of this enzyme.[4] Since the interconversion rates of the different anomers of KDO are slow, it has been suggested that the rate of KDO incorporation into lipopolysaccharides may be limited by the rate of formation of the β-pyranose form of KDO.[5] We have therefore investigated

(1) (a) Unger, F. M. *Adv. Carbohydr. Chem. Biochem.* **1981**, *38*, 323. (b) Inouye, M. *Bacterial Outer Membranes: Biogenesis and Functions*; Wiley: New York, 1979.

(2) (a) Ray, P. H.; Kelsey, J. E.; Bigham, E. C.; Benedict, C. D.; Miller, T. A. *Bacterial Lipopolysaccharides*; Anderson L., Unger, F. M., Eds.; ACS Symp. Series 231; American Chemical Society: Washington, DC, 1983; pp 141-170. (b) Allen, N. E. *Annu. Rep. Med. Chem.* **1985**, *20*, 155.

(3) (a) Ghalambor, M. A.; Levine, E. M.; Heath, E. C. *J. Biol. Chem.* **1966**, *241*, 3207. (b) Mauson, R. S.; Rassmusen, N. S.; Osborn, M. J. *J. Biol. Chem.* **1978**, *253*, 1503. (c) Rick, P. D.; Osborn, M. J. *J. Biol. Chem.* **1977**, *252*, 4895.

(4) Kohlbrenner, W. E.; Fesik, S. W. *J. Biol. Chem.* **1985**, *260*, 14695.

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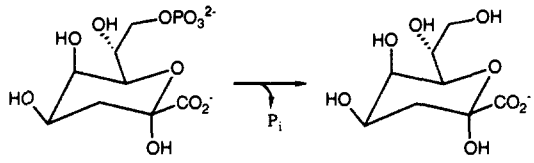
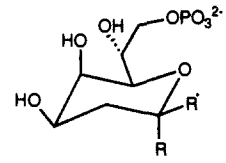
Scheme I. The Reaction Catalyzed by KDO 8-Phosphate Phosphatase



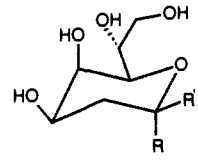
the anomeric specificity of the *preceding* enzyme, KDO 8-phosphate phosphatase (Scheme I), by the use of substrate analogues, to address the following question. If CMP-KDO synthetase utilizes only the β-pyranose form of KDO, does KDO 8-phosphate phosphatase prepare for the synthetase by producing only the β-anomer of KDO?

As a first step to defining the anomeric specificity of KDO 8-phosphate phosphatase we determined the anomeric and ring form composition of KDO 8-phosphate in solution. For this purpose we purified KDO 8-phosphate synthetase from wild type *E. coli* B[6] and used this enzyme for the enzymatic synthesis of KDO 8-phosphate.[7] By analysis of ^1H, ^{13}C, and ^{31}P NMR spectra of the purified product we found that the KDO 8-phosphate, as well as KDO,[8] exists in solution as a mixture of four forms:[9] α- and β-pyranose (65.8% and 3.1%, respectively) and α- and β-furanose (19.1% and 12.0%, respectively). Standard methods[10] for determining anomeric specificity such as fast kinetics or the direct spectroscopic demonstration[4] of the preferred KDO 8-phosphate substrate are made difficult by the small amounts of KDO 8-phosphate phosphatase available[11] and by problems in the interpretation of the spectra, since KDO, the product of the phosphatase reaction, also exists in solution as a mixture of four forms. We therefore adopted an indirect method, using the 2-deoxy substrate analogues **1** and **2** that possess the stereochemistry of either the α- (compound **1**) or the β-anomer (compound **2**) of the substrate, locked into a given configuration.[12]

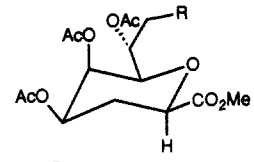
The synthesis of the analogues **1** and **2** has been accomplished from the 2-deoxy analogues of KDO[13] (compounds **3** and **4**,



1 R=H; R'=CO_2^-
2 R=CO_2^-; R'=H



3 R=H; R'=CO_2Me
4 R=CO_2Me; R'=H



5 R=OSi(Me)$_2$*tert*-Bu
6 R=OP(O)(OPh)$_2$

respectively). Treatment of compound **3** with *t*-Bu(Me)$_2$SiCl in pyridine gave the primary silylated product, which was isolated as its triacetate (**5**) in 80% yield. Deprotection [H_2SO_4 in MeOH:THF (2:3 v/v)] and phosphorylation of the primary hydroxyl group by diphenylphosphochloridate provided compound **6** in 74% yield. Hydrogenolysis (in MeOH, over PtO_2) and saponification (LiOH, 0 °C) then provided the target compound **1** in 73% isolated yield.[14a] With use of a similar sequence of reactions, compound **4** was converted into its phosphate analogue **2**.[14b]

Incubation of **1** with KDO 8-phosphate phosphatase[15] resulted in the formation of inorganic phosphate at about 50% of the rate observed for the natural substrate KDO 8-phosphate.[16] The K_m value of compound **1** was calculated as 0.8 ± 0.15 mM, which is similar to that of natural substrate determined in the same conditions.[16] In contrast, no release of phosphate was detected when the β-anomer analogue (compound **2**) was examined as a substrate, and only weak inhibition (K_i > 5 mM) was observed when compound **2** was tested with KDO 8-phosphate as substrate. The results clearly demonstrate that KDO 8-phosphate phosphatase only catalyzes the hydrolysis of the α-anomer analogue. It is noteworthy that V_{max} for the α-anomer of the 2-deoxy analogue **1** is about 50% that for KDO 8-phosphate, which argues against any requirement for the hydroxyl group at C-2 in the phosphatase-catalyzed reaction. Although we did not examine the furanose analogues, it seems very unlikely that the phosphatase (for which no other of 18 common sugar phosphates tested were alternative substrates or inhibitors[11]), can accommodate in its binding site both pyranose and furanose forms of the substrate.

We can conclude from these results with 2-deoxy substrate analogues that KDO 8-phosphate phosphatase is absolutely specific for the α-pyranose anomer. In addition, since the phosphatase does not utilize the β-pyranose form of KDO 8-phosphate, the steady-state level of the β-pyranose form of KDO in vivo must be the result of mutarotation of other KDO forms. As has been pointed out previously,[5] these interconversion rates are particularly slow. Since the CMP-KDO synthetase, the next enzyme in the biosynthetic pathway, utilizes only the β-pyranose form of KDO,[4] our results support the recent suggestion of Fesick et al.[5] that the rate of formation of β-pyranose KDO may be what limits the incorporation of KDO into lipopolysaccharides. Finally, the

(5) Fesik, S. W.; Kohlbrenner, W. E.; Gampe, R. T., Jr.; Olejniczak, E. T. *Carbohydr. Res.* **1986**, *153*, 136.

(6) The enzyme was isolated from *Escherichia coli* B cells, grown in phosphate-containing minimal medium, following the procedure of Ray (Ray, P. H. *J. Bacteriol.* **1980**, *141*, 635) with two modifications: a French Press was used for cell disruption and the enzyme fractions pooled after the DEAE Sephadex chromatography step (step 4) were stabilized with bovine serum albumin (10 mg/mL) and concentrated by lyophilization.

(7) Bednarski, M. D.; Crans, D. C.; DiCosimo, R.; Simon, E. S.; Stein, P. D.; Whitesides, J. M.; Schneider, M. J. *Tetrahedron Lett.* **1988**, *29*, 427.

(8) (a) Cherniak, R.; Jones, R. G.; Gupta, D. S. *Carbohydr. Res.* **1979**, *75*, 39. (b) Brade, H.; Zahringer, U.; Rietschel, E. T. *Carbohydr. Res.* **1984**, *134*, 157.

(9) The relative ratio of different anomers was determined by ^1H NMR of the purified KDO 8-phosphate [ion-exchange chromatography on AG 1X8 (100–200 mesh, HCO_3^- form) eluted with a linear gradient (100–600 mM) of triethylammonium bicarbonate, pH 7.5)] due to integration of axial and equatorial protons at C-3. ^1H NMR (lithium salt, pH 7.0, 25 °C, D_2O, 400 MHz, referenced to HOD at 4.63 ppm): δ α-pyranose, 1.76 (dd, 1 H, J = 13.4 and 12.9 Hz, H_{ax}), 1.70 (dd, 1 H, J = 13.4 and 6.6 Hz, H_{eq}); δ β-pyranose, 1.58 (dd, 1 H, J = 13.4 and 12.4 Hz, H_{ax}), 2.15 (dd, 1 H, J = 13.4 and 5.6 Hz, H_{eq}); δ α-furanose, 2.40 (dd, 1 H, J = 14.2 and 7.2 Hz, H_{ax}), 1.89 (dd, 1 H, J = 14.2 and 3.16 Hz, H_{eq}); δ β-furanose, 2.12 (dd, 1 H, J = 13.32 and 7.0 Hz, H_{ax}), 2.20 (dd, 1 H, J = 13.38 and 7.0 Hz, H_{eq}).

(10) (a) Schray, K. J.; Benkovic, S. J. *Acc. Chem. Res.* **1978**, 136. (b) Benkovic, S. J. *Methods Enzymol.* **1979**, *63*, 370.

(11) Ray, P. H.; Benedict, C. D. *J. Bacteriol.* **1980**, *142*, 60.

(12) The designations α and β refer to the axial and equatorial orientation, respectively, of the anomeric hydroxyl in the KDO 8-phosphate. If R = OH, then structure **1** refers to α-KDO 8-phosphate, and if R' = OH structure **2** refers to β-KDO 8-phosphate. In order to retain the similarity in definition of α and β of the 2-deoxy analogues, we called the 2-deoxy analogue of α-KDO 8-phosphate α-2-deoxy-KDO 8-phosphate (compound **1**) and the 2-deoxy analogue of β-KDO 8-phosphate β-2-deoxy-KDO 8-phosphate (compound **2**).

(13) Compounds **3** and **4** were prepared from the ammonium salt of KDO (Hershberger, C. S.; Davis, M.; Binkley, S. B. *J. Biol. Chem.* **1968**, *243*, 1585) by a slight modification of the published method: Lutham, K.; Orbe, M.; Waglund, T.; Claesson, A. *J. Org. Chem.* **1987**, *52*, 3777.

(14) (a) ^1H NMR for the lithium salt of **1**, pH 6.0 (D_2O, 400 MHz, referenced to HOD at 4.63 ppm) δ 1.49 (ddd, 1 H, $J_{3ax,3eq} = J_{3ax,2} = J_{3ax,4}$ = 12.4 Hz, C_3-H_{ax}), 1.82 (bd, 1 H, J = 12.0 Hz, C_3-H_{eq}), 3.28 (d, 1 H, J = 8.21 Hz, C_6-H), 3.60–3.86 (m, 6 H). (b) ^1H NMR for the lithium salt of **2**, pH 6.0 (D_2O, 400 MHz) δ 1.90 (ddd, 1 H, J = 12.8, 12.6, and 6.4 Hz, C_3-H_{ax}), 2.02 (dd, 1 H, J = 13.3 and 4.9 Hz, C_3-H_{eq}), 3.46 (d, 1 H, J = 8.4 Hz, C_6-H), 3.65 (ddd, 1 H, J = 12.6, 4.9, and 2.9 Hz, C_4-H), 3.84 (d, 1 H, J = 2.3 Hz, C_5-H), 3.76–3.81 (m, 2 H, C_7-H and C_8-H), 3.99 (dd, 1 H, J = 11.4 and 4.4 Hz, C_8-H), 4.48 (d, 1 H, J = 6.3 Hz, C_2-H).

(15) The enzyme (specific activity 5 units/mg) was purified from crude extracts of *Escherichia coli* B cells, grown in phosphate-containing minimal medium, following the procedure of Ray et al.[11]

(16) The rate of P_i release was followed at saturated substrate levels, 37 °C, in 0.10 M Tris–acetate buffer, pH 7.0, containing Co^{2+} (1.0 mM).[11] Under these conditions, the K_m of **1** is 0.8 mM, while the K_m for KDO 8-phosphate is 0.5 mM.

elucidation of the anomeric specificity of this specific phosphatase helps in the design of stereochemically defined inhibitors for this enzyme, which may serve as antibiotics acting on lipopolysaccharide biosynthesis.[17]

Acknowledgment. We gratefully acknowledge the financial support of the Technion V.P.R. fund-Henri Gutwirth fund for the promotion of research.

(17) (a) Hammond, S. M.; Claesson, A.; Jannson, A. M.; Larsson, L. G.; Brian, G. P.; Town, C. M.; Ekstrom, B. *Nature* **1987**, *327*, 730. (b) Goldman, R.; Kohlbrenner, W.; Lartey, P.; Pernet, A. *Nature* **1987**, *329*, 162.

Involvement of 19-Electron Species in Oxidatively Induced Homolytic Metal–Carbon Bond Cleavage Reactions: Decomposition of 17-Electron Cyclopentadienylruthenium Methyl Cations

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Despite the vigorous current interest in the chemistry of 17- and 19-electron organotransition-metal species,[2] little is known about the mode of decomposition of 17-electron complexes to even-electron products via substitution of one-electron donors by two-electron ligands.[3] The oxidation of transition-metal alkyls and other compounds containing σ-bound ligands has been observed to lead to solvent substitution in donor solvents.[4] Accumulated evidence suggests that 17e → 19e → 17e cycles are operational when entering and leaving ligands are both two-electron donors.[5] Oxidation of $(\eta^5\text{-}C_5H_5)Fe(CO)(L)R$ compounds induces catalytic CO insertion processes[6,7] believed to proceed by similar 17e → 19e → 17e sequences. The oxidative behavior of analogous ruthenium complexes remains less thoroughly studied, although decomposition products indicative of the formation of metal-centered radicals have been reported.[8] In this communication, we describe the results of an investigation of the oxidation of ruthenium methyl compounds $(\eta^5\text{-}C_5H_5)Ru(CO)(PR_3)CH_3$ [R = Cy (cyclohexyl) (**1a**), Ph (**1b**)]. Our data suggest that Ru–CH_3 bond homolysis may take place, *after prior solvent coordination to the cation radicals*, upon oxidation of **1a** and **1b**. We present (1) large solvent effects on the rate of decomposition, indicating that the reactions occur via 19-electron species; (2) quantitative kinetic and mechanistic data showing that cations $\mathbf{1}^{\bullet+}$ react via competing processes that are of first and second order in $\mathbf{1}^{\bullet+}$, and (3) kinetic isotope effects suggestive of agostic interactions in cations $\mathbf{1}^{\bullet+}$.

The first half of the derivative cyclic voltammetry[9] (DCV) response for the oxidation of methyl compound **1a** (90:10 CH_3CN/CH_2Cl_2,[10] 0.1 M $Bu_4N^+PF_6^-$) is shown in Figure 1. Peak a (+0.19 V vs Ag/Ag^+) corresponds to the oxidation of **1a**, while peaks b (+0.64 V) and c (+1.33 V) arise from oxidation of decomposition products $(\eta^5\text{-}C_5H_5)Ru(PCy_3)(NCCH_3)_2^+$ (**2a**) and $(\eta^5\text{-}C_5H_5)Ru(CO)(PCy_3)(NCCH_3)^+$ (**3a**), respectively, verified by comparison with authentic samples. Oxidation of **1a** takes place at +0.11 V vs the ferrocene/ferricinium (FC) couple, consuming 1.1 ± 0.1 faraday/mol (constant-current coulometry with linear sweep voltammetry monitoring of substrate disappearance[11]). A 1:3 to 1:4 mixture of **2a** and **3a** was isolated after preparative-scale one-electron exhaustive electrolysis of **1a** (80% combined yield).

Reaction-order analysis by DCV[9b] showed $\mathbf{1a}^{\bullet+}$ to decompose slowly, exhibiting first-order behavior at substrate concentrations ranging from 0.5 to 2.0 mM. The rate of disappearance of $\mathbf{1a}^{\bullet+}$ was measured in the temperature range −20 to +20 °C, giving a first-order rate constant k (20 °C) = 0.26 ± 0.02 s^{-1}, $\Delta H^{\ddagger}$ = 10.6 ± 0.3 kcal/mol, and $\Delta S^{\ddagger}$ = −25 ± 1 eu. An inverse k_H/k_D isotope effect (0 °C) of 0.89 ± 0.02 was found when $(\eta^5\text{-}C_5H_5)Ru(CO)(PCy_3)CD_3$ (**1a-d_3**) was employed. Finally, a DCV analysis carried out in CH_2Cl_2/0.1 M $Bu_4N^+PF_6^-$ showed $\mathbf{1a}^{\bullet+}$ to undergo no reaction on the time scale of the measurement (voltage sweep rate ν = 0.1 V/s). Comparison with theoretical data for a first-order EC mechanism yields a factor of 50 as a *lower limit* for the rate enhancement upon changing the solvent from CH_2Cl_2 to CH_3CN. DCV reaction-order analysis indicated an apparent CH_3CN reaction order of 0.8 ± 0.05 in the concentration range 0–20% CH_3CN (by volume) in CH_2Cl_2.

Oxidation of **1a** with 1 equiv of $(\eta^5\text{-}C_5H_5)_2Fe^+PF_6^-$ (**4**) in CD_3CN yielded a 44:56 mixture of $(\eta^5\text{-}C_5H_5)Ru(PCy_3)(NCCD_3)_2^+$ (**2a-d_6**) and $(\eta^5\text{-}C_5H_5)Ru(CO)(PCy_3)(NCCD_3)^+$ (**3a-d_3**) (82% combined yield; ^1H NMR, internal standard). Methane was detected by ^1H NMR (δ 0.18) and GLC analysis (94 ± 8% yield). Mass spectrometry indicated a CH_4:CH_3D ratio of 93:7. Conversely, ferricinium oxidation of **1a-d_3** in CH_3CN gave a 98:2 CH_3:CD_4 ratio.

Methyl compound **1b** underwent a one-electron (constant-current coulometry), chemically irreversible (DCV) oxidation at +0.32 V vs FC. Reaction-order analysis of the decomposition of $\mathbf{1b}^{\bullet+}$ provided a strikingly different mechanistic picture from that observed for $\mathbf{1a}^{\bullet+}$. In CH_3CN, the decomposition was second order in cation $\mathbf{1b}^{\bullet+}$ in the concentration range 1–4 mM and approached first order at concentrations lower than 0.5 mM. For the second-order process, kinetic data acquired from −14 to +20 °C (2 mM) gave k (20 °C) = (1.2 ± 0.07) × 10^5 M^{-1} s^{-1}, $\Delta H^{\ddagger}$ = −0.7 ± 0.2 kcal/mol, and $\Delta S^{\ddagger}$ = −38 ± 2 eu. An inverse isotope effect of 0.87 ± 0.04 was observed. Under first-order conditions (0.25 mM), the kinetic parameters were k (20 °C) = 29 ± 2 s^{-1}, $\Delta H^{\ddagger}$ = 8.2 ± 0.6 kcal/mol, $\Delta S^{\ddagger}$ = −23 ± 2 eu, and k_H/k_D = 0.85 ± 0.08.

Oxidation of **1b** under second-order conditions (one-electron constant-current electrolysis, 2.0 mM substrate in CH_3CN/0.1

(1) (a) University of Oslo. (b) Utah State University.

(2) For recent reviews of the chemistry of 17- and 19-electron complexes, see: (a) Astruc, D. *Chem. Rev.* **1988**, *88*, 1189. (b) Baird, M. C. *Chem. Rev.* **1988**, *88*, 1217. (c) Astruc, D. *Angew. Chem., Int. Ed. Engl.* **1988**, *27*, 643. (d) *Paramagnetic Organometallic Species in Activation/Selectivity, Catalysis*; Chanon, M., Julliard, M., Poite, J. C., Eds.; Kluwer Academic: Dordrecht, 1989. (e) Tyler, D. R. *Prog. Inorg. Chem.* **1988**, *36*, 125. (f) Connelly, N. G. *Chem. Soc. Rev.* **1989**, *18*, 153.

(3) Goldberg, K. I.; Bergman, R. G. *J. Am. Chem. Soc.* **1989**, *111*, 1285.

(4) (a) Chen, J. Y.; Kochi, J. K. *J. Am. Chem. Soc.* **1977**, *99*, 1450. (b) Kotz, J. C.; Vining, W.; Coco, W.; Rosen, R.; Dias, A. R.; Garcia, M. H. *Organometallics* **1983**, *2*, 68. (c) Jordan, R. F.; LaPointe, R. E.; Bajgur, C. S.; Echols, S. F.; Willett, R. *J. Am. Chem. Soc.* **1987**, *109*, 4111.

(5) For leading entries to the extensive literature covering ligand substitution in 17-electron complexes, see references quoted in the above reviews (refs 2a–f).

(6) (a) Magnuson, R. H.; Zulu, S.; T'sai, W.-M.; Giering, W. P. *J. Am. Chem. Soc.* **1980**, *102*, 6887. (b) Magnuson, R. H.; Meirowitz, R.; Zulu, S.; Giering, W. P. *J. Am. Chem. Soc.* **1982**, *104*, 5790. (c) Magnuson, R. H.; Meirowitz, R.; Zulu, S. J.; Giering, W. P. *Organometallics* **1983**, *2*, 460. (d) Bly, R. S.; Silverman, G. S.; Bly, R. K. *Organometallics* **1985**, *4*, 374. (e) Trogler, W. C.; Therien, M. *J. Am. Chem. Soc.* **1987**, *109*, 5127. (f) Golovin, M. N.; Meirowitz, R.; Rahman, M.; Liu, H. Y.; Prock, A.; Giering, W. P. *Organometallics* **1987**, *6*, 2285. (g) Liu, H.-Y.; Golovin, M. N.; Fertal, D. A.; Tracey, A.; Eriks, K.; Giering, W. P.; Prock, A. *Organometallics* **1989**, *8*, 1454.

(7) (a) Reger, D. L.; Mintz, E. *Organometallics* **1984**, *3*, 1759. (b) Reger, D. L.; Mintz, E.; Lebioda, L. *J. Am. Chem. Soc.* **1986**, *108*, 1940. (c) Reger, D. L.; Klaeren, S. A.; Babin, J. E.; Adams, R. D. *Organometallics* **1988**, *7*, 181.

(8) Joseph, M. F.; Page, J. A.; Baird, M. C. *Organometallics* **1984**, *3*, 1749.

(9) (a) Ahlberg, E.; Parker, V. D. *J. Electroanal. Chem.* **1981**, *121*, 73. (b) Parker, V. D. *Electroanal. Chem.* **1986**, *14*, 1.

(10) CH_2Cl_2 was added as a cosolvent in order to increase the solubility of **1a**.

(11) (a) Bethell, D.; Parker, V. D. *J. Am. Chem. Soc.* **1986**, *108*, 895. (b) Tilset, M.; Bodner, G. S.; Senn, D. R.; Gladysz, J. A.; Parker, V. D. *J. Am. Chem. Soc.* **1987**, *109*, 7551.

0002-7863/90/1512-4974$02.50/0