# Vanadium Peroxide Complexes

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## Contents



## I. Introduction

With the discovery of vanadium bromoperoxidase, a vanadium(V)-containing enzyme which is isolated primarily from marine algae and catalyzes the oxidation of halides ($Br^-$, $Cl^-$, $I^-$) by hydrogen peroxide,[1,2] the reactivity of vanadium(V) peroxide complexes is receiving renewed attention. The propensity of vanadium(V) to coordinate peroxides is well-established, as illustrated by the classic spot test for vanadate, which is the formation of the red oxoperoxovanadium(V) ion.[3] Peroxovanadates are remarkably stable, which, in many cases, has permitted their structural characterization.

The reactivity of these peroxovanadium(V) complexes is rich and varied (Figure 1). Peroxovanadium-(V) complexes perform a variety of net two-electron oxidation reactions, which are presented in detail below. Alkenes and allylic alcohols can be epoxidized and hydroxylated.[4-12] Sulfides can be oxidized to sulfoxides and sulfones.[12-16] Benzene and other arenes and alkanes can be hydroxylated.[9,10,17-20] Primary and secondary alcohols are oxidized to aldehydes and ketones.[21,22] A thiolato Co(III) complex is also oxidized to the corresponding sulfenato (S=O) complex.[23] In intriguing, but poorly understood reactions, nicotinic acid undergoes an unusual C6-substitution reaction in the presence of $VO_2^+$ and $H_2O_2$ in acidic, aqueous ethanol to give 6-(1-hydroxyethyl)nicotinic acid,[24] and 2,2′-bipyridine is oxidized to picolinic acid in aqueous solution in the presence of V(V) and hydrogen peroxide.[25] Halides are oxidized to oxidized halogen species, which undergo further reaction.[26,27] Sulfur dioxide is oxidized to sulfate by a triperoxovanadium(V) complex.[28]

Some of these transformations have been modified to be enantioselective. For instance, chiral hydroxamic acids serve as ligands to vanadium(V) in the catalytic, asymmetric epoxidation of allylic alcohols by tBuOOH.[7] $VO(acac)_2$ is a catalyst precursor for the regioselective epoxidation of allylic alcohols and the stereoselective epoxidation of 4-β-hydroxycholesterol in benzene solution.[6] $H_2$sal-L-Ala forms a complex with vanadium(V) which is catalytically active in the asymmetric oxidation of methyl phenyl sulfide by tBuOOH.[13]

This review will focus on the reactivity of well-defined vanadium peroxide systems, including a summary of structural features of peroxovanadium(V) complexes. Peroxovanadium(V) species are invoked in several examples of vanadium-mediated reactions, particularly in biological systems, although these reactions will not be reviewed herein. For example, vanadate mediates the oxidation of reduced nicotinamide adenine dinucleotides (NADH, NADPH), in a reaction that may involve superoxide, hydrogen peroxide, and a free radical mediated chain reaction.[102] In addition, vanadyl(IV) ion reduces hydrogen peroxide, which leads to DNA cleavage by hydroxyl radicals,[29] halide oxidation,[30] and nuclease activity of vanadyl bleomycin.[31] The reader is also referred to a relatively recent book, *Vanadium in Biological Systems*, edited by N. D. Chasteen, for additional accounts of the reactivity of vanadium in biological systems.[32]

## II. Peroxide Complexes of Vanadium(V)

### A. Structural Characterization

Numerous oxoperoxovanadium(V) and oxodiperoxovanadium(V) complexes have been characterized at the crystallographic level (Table 1). Peroxide is triangular bidentate in all of the cases known (Figure 2). The monomeric oxoperoxo complexes all have one or two peroxides bound in the equatorial plane relative to the axial oxo ligand. In the majority of the structures the geometry is pentagonal bipyramidal. In a few cases where a pentagonal pyramidal geometry is considered, however, the oxo ligand of another molecule may well serve as the seventh ligand as a long axial bond.

The key differences in peroxide coordination between mono- and diperoxo complexes can be seen in Table 1. In general, the O–O peroxo bond is longer in diperoxo

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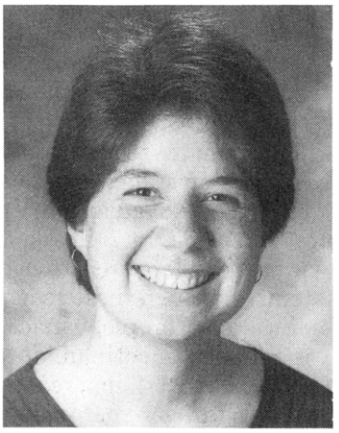
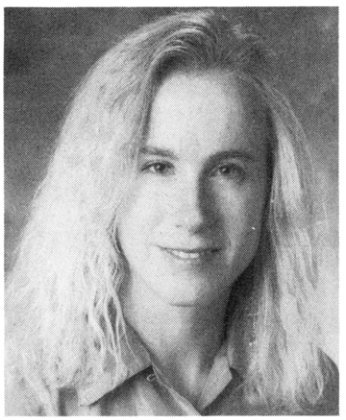


Melissa J. Clague was born in New York City. She earned a B.S. in Chemistry at Yale University, where she worked in Bob Crabtree's laboratory in organometallic chemistry. After a year of working in cell biology, she entered graduate school in Chemistry at the University of California, Santa Barbara and is presently a graduate student in the laboratory of Prof. Alison Butler. She is the recipient of a University of California Regents Special Fellowship (1989–93) and a University of California President's Dissertation Year Fellowship (1993–4). Her research interests are primarily in metal-centered redox chemistry, particularly in biological contexts.



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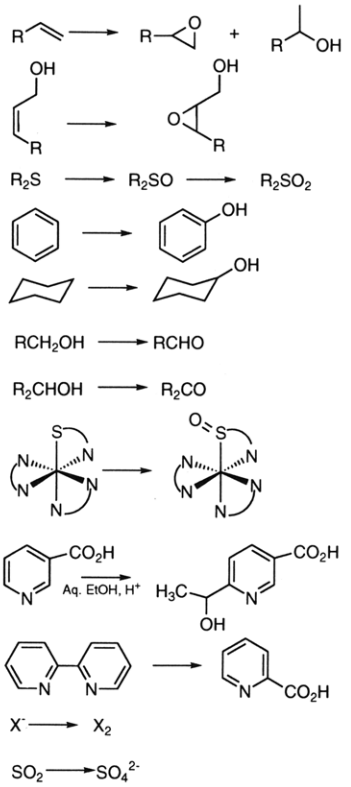



**Figure 1.** Examples of the reaction types mediated by peroxovanadium(V) complexes.

complexes than in monoperoxo complexes. In the monoperoxo complexes, the peroxide is symmetrically coordinated, while in the diperoxo complexes, the $V–O_{trans}$ bond is slightly longer than the $V–O_{cis}$ bond, where $V–O_{trans}$ and $V–O_{cis}$ are defined in Figure 2.

$VO(OOtBu)(dipic)(H_2O)$ is the first and to date only alkylperoxovanadium(V) complex to be crystallographically characterized.[10] The crystal structure reveals a pentagonal bipyramidal geometry with asymmetric, side-on coordination of *tert*-butyl hydroperoxide: one V–O bond length is 1.872 Å; the other is 1.999 Å (Table 1, Figure 3).

Infrared and Raman spectroscopy have also provided much information on the structure of vanadium(V) peroxo complexes.[33] The mode of peroxide coordination can be established by these methods. Bidentate peroxide coordination creates a local $C_{2v}$ environment which has three IR and Raman active modes: the symmetric O–O stretch, the symmetric metal–peroxo stretch, and the asymmetric metal–peroxo stretch.[33] These vibrations occur at approximately 880, 600, and 500 $cm^{-1}$, respectively, although the metal peroxo stretches are not always clearly distinguishable.[33] Representative vibrational bands of bidentate peroxide complexes are shown in Table 2. In the case of an end-on bound hydroperoxide, only one metal–peroxo oxygen stretch would be seen and an OOH deformation should be seen above 1100 $cm^{-1}$. The bridging peroxide has only a weak dipole and should be very weak in the IR,

**Table 1. Bond Lengths of Peroxovanadium(V) Compounds[d]**

| | O–O | V–$O_{peroxo\text{-}cis}$ | V–$O_{peroxo\text{-}trans}$ | V=O | ref |
|---|---|---|---|---|---|
| | | Oxoperoxo Complexes | | | |
| $[VO(O_2)(pic)(bipy)]\cdot H_2O$ | 1.424(7) | 1.862(5), 1.887(5) | | 1.604(5) | 25 |
| $K_3[VO(O_2)(ox)_2]\cdot{}^1/_2H_2O$ | 1.430(10) | 1.871(6), 1.882(6) | | 1.625(6) | 76 |
| $NH_4[VO(O_2)(H_2O)(dipic)]\cdot 1.3H_2O$ | 1.441(3) | 1.870(2), 1.872(2) | | 1.579(2) | 77 |
| $NH_4[CO(O_2)IDA]$ | 1.435(5) | 1.876(3), 1.886(3) | | 1.587(3) | 78 |
| $[VO(O_2)(bipy)_2]ClO_4$ | 1.24[c] | 1.81, 1.86 | | 1.625 | 79 |
| $[VO(O_2)(phen)_2]ClO_4$ | 1.3[c] | 1.83, 1.86 | | 1.6 | 79 |
| $VO(O_2)(pic)(H_2O)_2$ | 1.435 | 1.867, 1.878 | | 1.583 | 9 |
| $VO(O_2)(PAN)(pyr)$ | 1.440(7) | 1.859(5), 1.847(5) | | 1.608(5) | 80 |
| | | Oxodiperoxo Complexes | | | |
| $(NH_4)[VO(O_2)_2(bipy)\cdot 4H_2O$ | 1.467(3)[a] | 1.875(2)[a] | 1.912(2)[a] | 1.612(2) | 41 |
| $NH_4[VO(O_2)_2(bipy)]\cdot 4H_2O$ | 1.465(4), 1.471(4) | 1.883(3), 1.880(3) | 1.911(3), 1.909(3) | 1.619(3) | 81 |
| $K_3[VO(O_2)_2CO_3]$ | 1.467(3)[a] | 1.873(2)[a] | 1.945(2)[a] | 1.617(3) | 82 |
| $K_3[VO(O_2)_2(ox)]\cdot H_2O_2$ | 1.466(4)[a] | 1.876(3)[a] | 1.923(3)[a] | 1.620(3) | 41 |
| $K_3[VO(O_2)_2(ox)]\cdot H_2O$ | 1.460(6), 1.451(6) | 1.866(4), 1.856(4) | 1.934(4), 1.911(4) | 1.622(4) | 83 |
| $K_2[VO(O_2)_2pic]\cdot 2H_2O$ | 1.464(5), 1.458(6) | 1.881(4), 1.895(4) | 1.899(4), 1.917(4) | 1.599(4) | 68 |
| $K_2[VO(O_2)_2OHpic]\cdot 3H_2O$ | 1.463(2), 1.461(2) | 1.902(2), 1.877(2) | 1.924(2), 1.908(2) | 1.606(2) | 68 |
| $(NH_4)_3[VF_2O(O_2)_2]$ | 1.466(2), 1.462(2) | 1.890(2), 1.887(2) | 1.921(2), 1.927(2) | 1.609(2) | 84 |
| $Cs_2[VFO(O_2)_2]$ | 1.44(3), 1.46(2), 1.48(3), 1.52(4)[b] | 1.85(2), 1.90(2), 1.96(2), 1.86(3)[b] | 1.90(2), 1.90(2), 1.91(2), 1.92(3)[b] | 1.62(2), 1.62(3)[b] | 85 |
| $K_2[VFO(O_2)_2]$ | 1.468(7), 1.505(6), 1.501(8), 1.474(9) | 1.882(5), 1.880(4), 1.930(5), 1.882(7) | 1.906(6), 1.901(4), 1.888(6), 1.858(7) | 1.620(5), 1.605(7) | 86 |
| $NH_4[VO(O_2)_2(NH_3)]$ | 1.472(4)[a] | 1.882(3)[a] | 1.883(3)[a] | 1.606(3) | 87 |
| $(Hbipy)[VO(O_2)_2(bipy)\cdot (3+x)H_2O_2\cdot(2-x)H_2O$ | 1.473(4), 1.474(4) | 1.862(3), 1.914(3) | 1.942(3), 1.938(3) | 1.619(3) | 88 |
| | | Oxoperoxo Dimers | | | |
| $K_2[VO(O_2)(cit)]_2\cdot H_2O$ | 1.427(7)[a] | 1.873(1), 1.888(1)[a] | | 1.661(1)[a] | 89 |
| $K_3[F(O_2)\{VO(O_2)F\}_2]$ | 1.441(5), 1.447(5), 1.467(5) (bridge) | 1.856(5), 1.872(5), 2.038(4) (bridge) | 1.873(4), 1.871(5), 1.974(4) (bridge) | | 90 |
| | | Oxodiperoxo Dimers | | | |
| $(NH_4)_3[HO\{VO(O_2)_2\}_2]\cdot H_2O$ | | 1.879(4), 1.864(3), 1.876(4), 1.879(3) | 1.893(4), 1.915(3), 1.881(3), 1.912(3) | 1.597(4)[a] | 91 |
| $(NH_4)_4[O(VO(O_2)_2)_2]$ | 1.449(26), 1.44(24), 1.447(24), 1.436(24) | 1.851(19), 1.875(18), 1.851(18), 1.876(18) | 1.870(18), 1.915(15), 1.857(18), 1.839(18) | 1.578(18)[a] | 92 |
| $(NH_4)_4[O\{VO(O_2)_2\}_2]$ | 1.469(4), 1.465(3), 1.474(4), 1.463(4) | 1.884(3), 1.875(3), 1.882(3), 1.878(3) | 1.896(3), 1.914(3), 1.895(3), 1.912(2) | 1.601(3), 1.613(3) | 93 |
| $(Hbipy)[H\{VO(O_2)_2(bipy)\}_2]\cdot XH_2O_2\cdot 6H_2O$ | 1.461(3), 1.471(3)[a] | 1.892(2), 1.869(2)[a] | 1.995(2), 1.903(2)[a] | 1.612(2)[a] | 94 |
| $[N(CH_3)_4]_2[V_2P_2(O_2)_4(H_2O)]\cdot 2H_2O$ | 1.466(5), 1.460(4), 1.458(5), 1.468(5) | 1.868(3), 1.883(4), 1.868(3), 1.919(4) | 1.865(3), 1.878(3), 1.880(4), 1.863(4) | 1.601(4), 1.591(4) | 95 |
| $K_2[V_2O_2(O_2)_4(H_2O)]\cdot 3H_2O$ | 1.465(4), 1.469(3), 1.470(5), 1.457(5) | 1.869(4), 1.891(4), 1.947(4), 1.878(3) | 1.869(4), 1.914(4), 1.894(4), 1.881(3) | 1.604(5), 1.586(3) | 96 |
| | | Triperoxo Complex | | | |
| $(NH_4)_2[V(O_2)_3F]$ | 1.47(3)[a] | 1.84(4), 1.95(3), 2.07(3)[a] | | | 38 |
| | | Alkylperoxo Complex | | | |
| $(dipic)VO(OOtBu)\cdot H_2O$ | 1.503(6) | 1.872(3), 1.999(3) | | 1.574(3) | 10 |

[a] Averages reported. [b] Duplicate structures analyzed. [c] The authors attribute these short bonds to the transfer of electron density from the peroxide ligand to the aromatic ligands. Since such an effect is not observed (or observed to an even lesser degree) in other peroxo or diperoxo complexes containing aromatic ligands, the short bonds may arise from disorder in the crystals. [d] Abbreviations: bipy, 2,2-bipyridine; cit, citrato; dipic, pyridine-2,6-dicarboxylato; GlyH, glycine; Hbipy, 2,2′-bipyridinium; IDA, iminodiacetato, OHpic, 3-hydroxypyridine-2-carboxylato, ox, oxalato; PAN, 1-(2-pyridylazo)-2-naphthol; pic, picolinato, pyridine-2-carboxylato; phen, 1,10-phenanthroline.

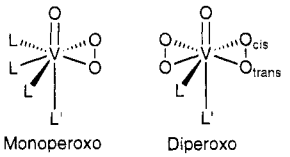



**Figure 2.** The coordination geometry of mono- and diperoxovanadium complexes.

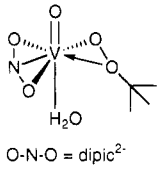



**Figure 3.** Structure of VO(dipic)(OOtBu).

and likewise, the peroxide of crystallization should be invisible in the IR.[33]

In the absence of interfering ligand vibrations, important structural distinctions can be determined from the frequencies of the symmetric and asymmetric metal–peroxo vibrations. In monoperoxo complexes, these vibrations essentially overlap at about 560 $cm^{-1}$ or are only slightly separated. A monodentate ligand on a diperoxo complex results in a splitting of roughly 100 $cm^{-1}$, with the two bands occurring at approximately 630 and 520 $cm^{-1}$, and a bidentate ligand results in a splitting of roughly 40 $cm^{-1}$, with the two bands occurring at approximately 625 and 585 $cm^{-1}$.[34] Representative examples of the vibrational frequencies detected for these different types of complexes are shown in Table 2.

Chaudhuri and co-workers have reported the preparation of complexes of the form $A[V(O_2)_3]$, where $A^+$ is $Na^+$ or $K^+$[35] and $A_2[V(O_2)_3L]$, where L = F– or Cl–[36,37] and $A^+$ is $NH_4^+$, $Na^+$, or $K^+$. The IR spectra of these complexes do not have stretching frequencies characteristic of V=O bonds, and the stretching frequencies of the O–O bands are shifted to lower frequencies than those of the oxomonoperoxo and oxodiperoxo complexes, possibly due to a reduction in bond order (see Table 2).[35–37] More recently the crystal structure of $(NH_4)_2[V(O_2)_3F]\cdot H_2O$ was reported to be a trigonal prism with bidentate peroxide ligands.[38] Like the pentagonal bipyramidal diperoxo complexes, two of the three peroxides are asymmetrically coordinated. The peroxo ligand trans to fluoride has much longer bonds, as shown in Table 1.[38] Triperoxo complexes containing bidentate heteroligands, $A_3[V(O_2)_3L]$, where L = bipy, phen, or $ox^{2-}$, have also been reported.[39] However, later IR analyses by Vuletic and Djordjevic suggested that diperoxo complexes were formed in these preparations.[40] In addition, the crystal structure of "$K_3[V(O_2)_3(ox)]H_2O$" was more recently reported and found to actually be $K_3[VO(O_2)_2(ox)]H_2O_2$.[41]

To date, no structures of tetraperoxo complexes have been reported. The complex $K_3[V(O_2)_4]$ has been analyzed by infrared[34,42,43] and Raman spectroscopies.[43] Like the triperoxo complexes, the O–O stretch is shifted to a lower frequency, which may be indicative of longer O–O bonds. In addition, modes at 620 and 565 $cm^{-1}$ indicate bidentate peroxide coordination.[34,42,43]

While many peroxovanadium(V) complexes have been characterized in the solid state, the solution conformations of these complexes are less well-established. Vibrational data show that side-on bound peroxide is still the norm in aqueous solution. Raman data recorded for the complexes $K_2[VO(O_2)_2(ox)]\cdot H_2O$[44] and $NH_4[VO(O_2)_2NH_3]$[45] in water suggest that these species have essentially the same bonding configurations in solution and the solid state.

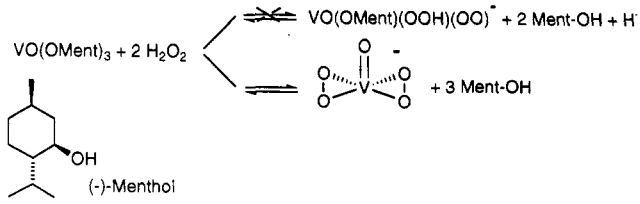
Circular dichroism (CD) was used to suggest the presence of side-on bound peroxide in organic solvents as well.[46,47] Addition of excess $H_2O_2$ to the vanadium(V) complex of the chiral ligand (–)-menthol, $VO(OMent)_3$, should give either $VO(OMent)(OOH)(OO^-)$ if peroxide is end-on bound or $VO(O_2)_2^-$ if peroxide is side-on bound (Scheme 1). The latter no longer has a

**Table 2. Stretching Frequencies of Selected Peroxovanadium(V) Compounds**[a]

| | V=O | O–O | $V–O_{peroxo}$ | ref |
|---|---|---|---|---|
| | | Oxoperoxo Complexes | | |
| $VO(O_2)(pic)(py)_2$ | 945 | 935 | 575, 560 | 9 |
| $VO(O_2)(pic)(bipy)(H_2O)$ | 945 | 935 | 575, 560 | 9 |
| $VO(O_2)(pic)(MeOH)(H_2O)$ | 975 | 940 | 580, 550 | 9 |
| $(NH_4)_2[VO(O_2)(OH)F_2]$ | 970s | 895vs | 590s, 550s | 97 |
| | | Pentagonal Bipyramidal Oxodiperoxo Complexes | | |
| $K[VO(O_2)_2(bipy)]\cdot 5H_2O$ | 930vs | 875s, 856vs | 621m, 582s | 40 |
| $K[VO(O_2)_2(phen)]\cdot 3H_2O$ | 925vs | 870s, 854vs | 635m, 590m | 40 |
| $K_3[VO(O_2)_2(ox)]\cdot 2H_2O$ | 930vs | 877s, 852w | 631s, 585s | 40 |
| $[VO(O_2)_2(ox)]^{3-}$, sodium | 949vs | 894s | 636w, 592m | 44 |
| $K_3[VO(O_2)_2CO_3]$ | 936s | 877m | 626m, 586m | 98 |
| | | Pentagonal Pyramidal Oxodiperoxo Complexes | | |
| $[VO(O_2)_2GlyH]^-$ | 960 | 850 | 605, 510 | 99 |
| $[VO(O_2)_2F]^{2-}$ | 952s | 875s | 618s, 526m | 100 |
| $(NH_4)[VO(O_2)_2NH_3]$ | 1000m, 957vs | 884s | 623m, 533vs | 45 |
| $[VO(O_2)_2NH_3]^-$, solution | 984s | 888s | 627m, 537vs | 45 |
| | | Triperoxo Complexes | | |
| $Na[V(O_2)_3]$ | | 850 | 600, 530 | 35 |
| $K_2[V(O_2)_3Cl]$ | | 855 | 620 | 37 |
| | | Tetraperoxo Complex | | |
| $K_3[VO(O_2)_4]$ | | 854s | 620s, 567vs | 43 |

[a] The vibrational modes of peroxovanadium complexes have been recently reassigned by Schwendt *et al.* from normal coordinate analysis.[101] The older analysis as described by Griffith has been used in this review.[33] This analysis assumes a $C_{2v}$ symmetry for the vanadium–peroxo triangle which has $2A_1 + B_2$ modes: $\upsilon_1 = A_1$ = O–O stretch, $\upsilon_2 = A_1$ = symmetric $V–O_{peroxide}$ stretch and $\upsilon_3 = B_2$ = asymmetric $V–O_{peroxide}$ stretch.

**Scheme 1. Formation of a Diperoxo Complex from $VO(OMent)_3$ (adapted from ref 46)**




chiral ligand and thus should not have a CD spectrum. Di Furia and Modena showed that addition of $H_2O_2$ to $VO(OMent)_3$ in DMF (where only the diperoxo species is observed) caused the $VO(OMent)_3$ CD band to disappear and no new bands appeared, thus suggesting that peroxide coordination is bidentate in solution.

## B. Formation of Peroxovanadium(V) Complexes in Aqueous and Nonaqueous Solution

### 1. Equilibria

In aqueous solution, hydrogen peroxide readily coordinates to vanadium(V), forming monoperoxo, diperoxo, triperoxo, and even tetraperoxo complexes. The equilibrium distribution of vanadium peroxide complexes is highly dependent on pH, although, in general, higher pH conditions are required to promote coordination of multiple ($\geq$2) peroxide ligands.

In acidic solution ($[H^+] \geq 0.01$ M), vanadium(V) exists as $VO_2^+$. Addition of hydrogen peroxide to $VO_2^+$ can give the red oxoperoxo $VO(O_2)^+$ and the yellow oxodiperoxo $VO(O_2)_2^-$ species, as described by the expressions for $K_1$ and $K_2$.

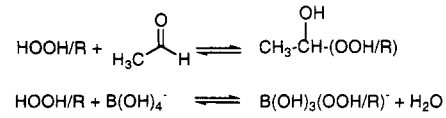
$$VO_2^+ + H_2O_2 \rightleftarrows VO(O_2)^+ + H_2O \qquad K_1$$

$$VO(O_2)^+ + H_2O_2 \rightleftarrows VO(O_2)_2^- + 2H^+ \qquad K_2$$

The equilibrium constants are $K_1 = 3.5 \times 10^4$ and $K_2 = 1.3$ at 25 °C and $I = 1.0$ M.[48] At 25 °C and $I = 0.3$ M, the same constants are $K_1 = 3.7 \times 10^4$ M and $K_2 = 0.6$ M.[26] Thus under conditions of 2:1 $H_2O_2$:V(V) and $[V(V)] = 10^{-3}$ M, as the pH is raised to 2 and above, the diperoxo species becomes favored. From $^{51}V$ NMR studies on the formation and decomposition of peroxovanadium(V) complexes in near-neutral, aqueous solution, formation constants (both pH-dependent and pH-independent) have been determined for the mono-, di-, and triperoxovanadates(V), as well as tetraperoxodivanadate(V).[49] Extensive studies on the formation of peroxo- and diperoxovanadium(V) complexes with peptide and other smaller ligands are beginning to map the general behavior of these complexes, with an emphasis on the potentially biochemically relevant configurations.[50,51]

The addition of hydrogen peroxide to $VO(2\text{-}OPr)_3$ in ethanol and dioxane/15% ethanol has also been studied.[52] A monoperoxo species, formulated as $VO(O_2)(OEt)_2$, is the primary one in solution at less than *ca.* 2 mM $H_2O_2$ and 1 mM $VO(2\text{-}OPr)_3$. At higher hydrogen peroxide concentrations, a diperoxo species is formed which appears to have lost all alkoxy moieties.[52] In dioxane or dioxane/2.5% ethanol, however, only the monoperoxo species was observed, up to hydrogen peroxide concentrations of 0.15 M. The oxodiperoxovanadium anion is strongly acidic, so its formation is suppressed in nonpolar solvents.[52]

**Scheme 2. Complexation of Hydrogen Peroxide and Alkylhydroperoxide by Acetaldehyde and Borate (adapted from ref 53)**




Prior to establishment of the first crystal structure of a vanadium alkylperoxide complex,[10] a study was done which tried to deduce from the reactivity of these species whether *tert*-butyl hydroperoxide was bound to vanadium(V) in the same manner as hydrogen peroxide. The following year, a crystal structure appeared which demonstrated that the $OOR^-$ moiety was coordinated to vanadium side-on with quite different $V\text{–}O_{peroxide}$ bond lengths (see above).[10]

Di Furia's hypothesis was that, if HOOH and ROOH have the same binding mode, they should have comparable formation constants ($K_f$).[53] If they have very different formation constants, different modes of coordination can be inferred. Indeed, reaction of $H_2O_2$ and ROOH with carbonyl compounds (*e.g.*, acetaldehyde) or with borate ion is found to occur with a difference in $K_f$ of a factor of 4 where $K_f^{H_2O_2} > K_f^{ROOH}$. The peroxides are monodentate in both cases (Scheme 2). A difference of a factor of 2 is expected for statistical reasons: $H_2O_2$ has two dissociable protons, while ROOH has one. Steric factors should be negligible and electronic influences should be small since there are two oxygen atoms between the R (or H) and the atom to which the peroxide binds.

This line of reasoning is sound for end-on complexes, but weakens for side-on complexes. For side-on complexes, it is difficult to invoke the necessary correlation between binding geometry, on the one hand, and binding properties and spectral characteristics on the other, especially since hydrogen peroxide is known to coordinate to vanadium(V) as $O_2^{2-}$. The experimental data bear out a difference in both binding constants and spectral properties, but the inference regarding binding geometry is only one possible interpretation of the data.

In the case of vanadium(V), complexation of tBuOOH by $VO_2^+$ is measurable by electronic spectroscopy only at high concentrations (0.1 M of each), indicating that coordination is either very weak or significantly different from hydrogen peroxide coordination, in which a strong charge-transfer transition is observed.[54] From $^{13}C$ NMR data, the binding constant for tBuOOH and $VO(OtBu)_3$ in $CDCl_3$ is $6 \times 10^{-2}$ at −40 °C.[53] The binding constant for $H_2O_2$ and vanadium(V) is greater than $10^3$, although the exact value depends slightly on the solvent. From this difference in binding constants of $H_2O_2$ and ROOH of a factor of $\sim 10^4$ (in favor of $H_2O_2$ complexes), Di Furia *et al.* suggest that *in solution* alkyl hydroperoxide complexes have a different structure from the symmetrical three-membered ring structure adopted by hydrogen peroxide complexes of vanadium(V) in solution and in the solid state. They propose an end-on, per-ester geometry. However, the observation that tBuOOH forms a complex with $VO_2^+$ in acid, but the more sterically hindered *tert*-amyl hydroperoxide does not,[54] suggests that the solution structure is actually an

intermediate structure, with a weak interaction between the distal oxygen and the vanadium center.

### 2. Kinetics of Formation from Vanadium(V) Precursors

The kinetics of formation of simple peroxovanadium(V) species (eg., $VO(O_2)^+$ and $VO(O_2)_2^-$) in strongly acidic solution (eg., 0.016–3.0 M), determined by stopped-flow methods,[48] have the following forms. The formation of $VO(O_2)^+$ is governed by a rate law which has terms that have an inverse dependence on $[H^+]$, an independence from $[H^+]$, and a first-order dependence on $[H^+]$:

$$\frac{d[VO(O_2)^+]}{dt} = \frac{k_1[VO_2^+][H_2O_2]}{[H^+]} + k_2[VO_2^+][H_2O_2] + k_3[VO_2^+][H_2O_2][H^+]$$

The complexity of this rate law is proposed to derive in part from the reaction of $H_2O_2$ with $VO_2^+$ and a protonated form of V(V); the inverse acid term is suggested to be a combination of the reaction of the excellent nucleophile $HO_2^-$ with $VO_2^+$ and the reaction of $H_2O_2$ with a hydroxylated form of $VO_2^+$. On the other hand, the formation of $VO(O_2)_2^-$ from $VO(O_2)^+$ follows a simple second-order rate law:

$$\frac{d[VO(O_2)_2^-]}{dt} = k_1[VO(O_2)^+][H_2O_2]$$

It appears that an acid-dependent, hydrogen peroxide-independent path may also be relevant, but the rates were too fast for quantitative work.[48]

Tanaka and co-workers studied the kinetics of the reaction of hydrogen peroxide with $VO_2(mida)^-$,[55] $VO_2(NTA)^{2-}$,[56] and $VO_2(dipic)^-$,[56] where $H_2mida$, $H_2NTA$, and $H_2dipic$ are N-methyliminodiacetic acid, nitrilotriacetic acid, and pyridine-2,6-dicarboxylic acid, respectively. Unlike the rate law for formation of $VO(O_2)^+$, the formation of $VO(O_2)(mida)^-$ from $H_2O_2$ and $VO_2(mida)^-$ (which is the sole species under the reaction conditions pH 3–4) obeys second-order kinetics:

$$\frac{d[VO(O_2)(mida)^-]}{dt} = k[VO_2(mida)^-][H_2O_2]$$

The kinetics of formation of the other two complexes are less straightforward. The rate law for the formation of $VO(O_2)(NTA)^{2-}$ in the pH range 1.5–5 is

$$\frac{d[VO(O_2)(NTA)^{2-}]}{dt} = (k_1 + k_2[H^+])[VO_2(NTA)^{2-}][H_2O_2]$$

The rate law for the formation of $VO(O_2)(dipic)^-$ under the same conditions (pH 1.5–5) was determined[56] to be

$$\frac{d(VO(O_2)(dipic)(H_2O)^-]}{dt} = \{k_1[H^+] \times [VO_2(dipic)(H_2O)^-] + k_2[VO_2(dipic)(H_2O)^-] + k_3[VO_2(dipic)(OH^-)^{2-}]\}[H_2O_2]$$

This rate law is functionally identical to the one for formation of $VO(O_2)^+$ in that acid-dependent, acid-independent, and inverse acid terms govern the rate of formation of $VO(O_2)(dipic)(H_2O)^-$. The mechanistic significance is attributed to different protonation states of the vanadium complex, without any participation from $H_3O_2^+$ or $HO_2^-$.

A later study by Wieghardt also analyzed the formation of $VO(O_2)(dipic)^-$ over a greater range of acid concentration [$(6 \times 10^{-5})$–0.1 M] and obtained a different rate law.[57] The kinetics are consistent with $VO_2(dipic)^-$ being trimeric in solution. Hydrogen peroxide putatively adds to the trimer by an associative mechanism involving addition of both $H_2O_2$ and $HO_2^-$, depending on the acid concentration. Subsequent to peroxide addition, the trimer undergoes bridge cleavage ($\mu$-oxo or $\mu$-hydroxo) in a rate-limiting step. The fragments react rapidly with $H_2O_2$ to form two different diperoxovanadium complexes, which convert to the product $VO(O_2)(dipic)^-$ by an acid-catalyzed pathway.

The kinetics of peroxide dissociation from oxodiperoxovanadium(V) complexes to form oxoperoxovanadium(V) complexes were determined for a series of $[VO(O_2)_2L]^{n-}$ complexes, where L = oxalato, bipyridine, phenanthroline, picolinato, and others.[58] The measured reaction was

$$[VO(O_2)_2L]^{n-} + 2H^+ + H_2O \rightarrow [VO(O_2)L(H_2O)]^{(n-2)-} + H_2O_2$$

Both the formation of the red monoperoxo complex and the disappearance of the yellow diperoxo complex were measured optically, upon addition of acid to the diperoxo complex. The study was done at $[H^+] = 0.01$–0.3 M, $[H_2O_2] = 0$–0.01 M and [complex] $= 5 \times 10^{-4}$ M. Pseudo-first-order rate constants were observed to conform to the following forms:

$$k_{obs} = \frac{[H^+]}{a + b[H_2O_2]} \quad \text{or} \quad k_{obs} = c[H^+]$$

The latter form appears to be due to a special case of the former where $b[H_2O_2] \ll a$. The proposed mechanism involves a rapid protonation of one of the coordinated peroxide ligands to give a monodentate hydroperoxide ($HO_2^-$). The rate-determining step is protonation of the bound hydroperoxide to yield hydrogen peroxide and the coordinatively unsaturated $VO(L)(O_2)^-$, which then binds water.

### 3. Kinetics of Formation from Vanadium(IV) Precursors

Oxoperoxovanadium(V) complexes can also be made by oxidation of V(IV) in the presence of excess peroxide. Carmichael studied the oxidation of $V^{IV}$ using $H_2O_2$ and ROOH (e.g., *tert*-butyl hydroperoxide, *tert*-amyl hydroperoxide, cumenyl hydroperoxide) as oxidants. The reaction of $VO^{2+}$ and $H_2O_2$ in the presence of 5,5-dimethyl-1-pyrroline *N*-oxide (DMPO) results in the formation of the DMPO–OH radical, which can be detected by EPR.[59] Competition experiments between DMPO and other radical scavengers (formate and ethanol) suggest that DMPO–OH is formed by two pathways: the reaction of DMPO with free hydroxyl radicals and another mechanism which may be the

direct reaction between a vanadium(IV)–peroxide adduct ($VO(O_2)^{\bullet 2+}$) and DMPO.

A kinetic study by Espenson and co-workers of the reaction of unligated $VO^{2+}$ with alkyl hydroperoxides in acidic aqueous medium reveals that a weak equilibrium exists between vanadium(IV) and *tert*-butyl or *tert*-amyl hydroperoxide.[54] The vanadium(IV)–peroxo complex is present in steady-state concentrations, and the electron-transfer step is rate-determining. The products are $VO_2^+$ and RO•, which undergo β-scission to give mostly acetone, ethane, and methane from *tert*-butyl hydroperoxide, and ethane, ethylene, and *n*-butane from *tert*-amyl hydroperoxide.

Zamaraev *et al.* recently studied the vanadium(IV/V)-tBuOOH system by a combination of EPR and $^1H$ and $^{51}V$ NMR spectroscopies.[4,5] $VO(acac)_2$ is rapidly oxidized by *tert*-butyl hydroperoxide or cumenyl hydroperoxide in benzene solution to give $V^VO(acac)_2$-(OOR). In the presence of excess peroxide, the acetylacetonate ligands dissociate to give a species with only oxo (or hydroxo) and peroxo ligands. A small EPR signal ($g_o$ = 2.014) is observed under these conditions of excess peroxide, which is attributed to ROO• (*ca.* $10^{-5}$ M) arising from homolytic dissociation of the alkylperoxo complex. ROO• is proposed to be the oxidizing species in the conversion of cyclohexene to cyclohexene oxide, cyclohexanol, and cyclohexanone and the epoxidation of an unspecified allylic alcohol. No changes in the chemical shifts of the $^{51}V$ NMR spectrum are observed during epoxidation of cyclohexene. Upon the addition of the allylic alcohol, however, the $^{51}V$ NMR spectrum changes, suggesting that the alcohol or its anion coordinates to vanadium(V).

Tanaka and co-workers investigated the kinetics and mechanism of the reaction of hydrogen peroxide with $V^{IV}O(NTA)(H_2O)^-$ and $V^{IV}O(dipic)(H_2O)_2$ to form V(V) peroxo complexes.[60] The rate law for formation of the $V^V$ peroxo complexes is

$$\text{rate} = k[V^{IV}\text{complex}][H_2O_2]$$

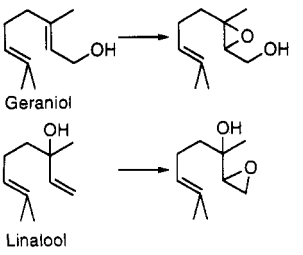
The reaction is independent of excess ligand and hydrogen ion concentrations. The rate-determining step appears to be addition of hydrogen peroxide to the V(IV) complex, which is followed by rapid electron-transfer and coordination of another molecule of peroxide. The mechanism of addition of the oxidizing peroxide is consistent with an associative interchange process. This mechanism is similar to the one observed by Espenson[54] for the reaction of $VO^{2+}$ and ROOH, but interestingly, the relative rates of peroxide coordination and electron transfer are reversed.

## *III. Reactivity of Peroxovanadium(V) Complexes with Organic Compounds*

### A. Alkene Epoxidation and Hydroxylation

Vanadium-catalyzed epoxidations are stereo- and regioselective, often proceed cleanly in very high yield, and in some cases give different products from those obtained using other epoxidizing reagents. For example, the epoxidation of allylic alcohols (e.g., geraniol, linalool) by tBuOOH using $V^{IV}O(acac)_2$ as a catalyst precursor in refluxing benzene solution occurs regioselectively at the $C_2$–$C_3$ double bond near the alcohol[6] (see Scheme 3). On the other hand, peracids react solely at the more electron-rich terminal double bond.

**Scheme 3. Vanadium(V)-Catalyzed Epoxidation of Allylic Alcohols by tBuOOH**




This system was further developed for asymmetric epoxidation reactions. The use of chiral hydroxamic acids in excess over vanadium(V) gives rise *in situ* to catalysts which, while not well characterized, give significant enantiomeric excesses (up to 50%) in epoxidations of allylic alcohols.[7] The greatest asymmetric induction (ee 50%) was observed for the epoxidation of the allylic alcohol α-phenylcinnamyl alcohol ($RCH{=}C(R)CH_2OH$; R = phenyl), catalyzed by the hydroxamate ligand in Figure 4, where R = phenyl. Sharpless and co-workers propose a mechanism in which the alcohol coordinates to vanadium and oxygen-atom transfer occurs from coordinated alkyl peroxide (Figure 5).[7] A useful review of metal-catalyzed oxidations of alkenes and alkynes by *tert*-butyl hydroperoxide is available.[8,103]

Geraniol was also found to be selectively epoxidized to 2,3-epoxygeraniol by hydrogen peroxide using $VO(acac)_2$ as catalyst precursor in dioxane/2.5% EtOH.[12] The yield of the reaction is quantitative for [geraniol]/$[H_2O_2]$ = 3, although at lower ratios, the selectivity decreased. This selectivity is in marked contrast to the mixture of products obtained from cyclohexene oxidation, where the allylic alcohol and the enone are the primary products, and cyclohexene oxide is a minor product.

Efficient, stereoselective epoxidation was achieved with a series of vanadium complexes of a tridentate Schiff base ligand, hydroxyphenylsalicyclidenamine ($H_2HPS$) and an alkyl hydroperoxide.[11] Alkenes are epoxidized in polar, nondonor solvents, with reactivity that increases with increasing nucleophilicity of the alkene and is sensitive to steric hindrance of the alkene.[11]

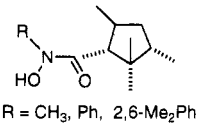



**Figure 4.** Chiral hydroxamic acids for asymmetric epoxidation.

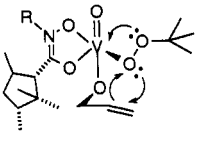
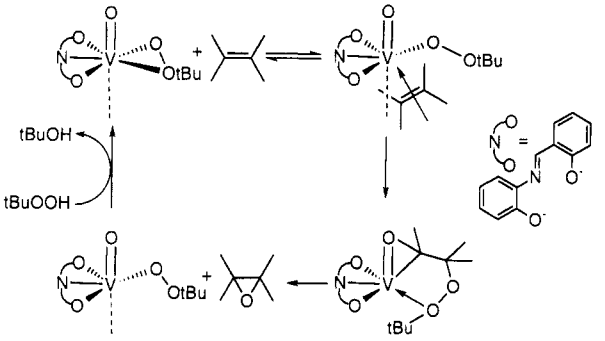
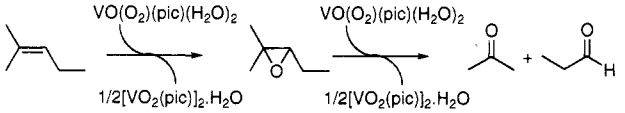



**Figure 5.** Proposed mechanism for epoxidation of an allylic alcohol by a *tert*-butylperoxo complex, adapted from ref 7.

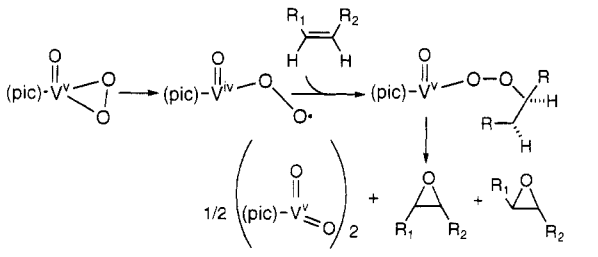
**Scheme 4. Proposed Mechanism for Epoxidation of Alkenes by VO(HPS)(OOtBu) (adapted from ref 11)**



**Scheme 5. Oxidation of Alkenes by $VO(O_2)(pic)(H_2O)$ Yield a Mixture of Products (adapted from ref 9)**



**Scheme 6. Proposed Mechanism for Epoxidation by $VO(O_2)(pic)(H_2O)_2$ (adapted from ref 9)**



The kinetics of oxidation by VO(HPS)(OOR) (R = tBu, $C(Me)_2Ph$) show saturation with respect to the alkene, which is consistent with coordination of the substrate to V(V) in a rapid pre-equilibrium step. Alkene epoxidation is proposed by Mimoun *et al.* to occur by insertion of the bound alkene into one of the metal–peroxo bonds followed by collapse of the resulting peroxometallocycle to yield the epoxide as shown in Scheme 4.[11] Values are determined for various kinetic parameters in both single-substrate and competitive experiments. The reaction is inhibited by water, alcohols, and basic ligands or solvents.

By contrast, epoxidations of alkenes by $VO(O_2)(pic)(H_2O)_2$ are not stereoselective and cleavage products are observed as well (Scheme 5). It is concluded that substrate binding does not occur.[9] The oxidations are proposed to occur via a vanadium(IV)–peroxo biradical which abstracts a hydrogen atom from the substrate. Reduction of the substrate radical by V(IV) gives products (Scheme 6).

Similarly to the picolinato complex, the alkylperoxovanadium(V) complex VO(OOtBu)(dipic)($H_2O$) and its derivatives VO(OOt$CMe_2Ph$)(dipic)($H_2O$) and VO(OOtBu)(dipic)(HMPT) are not particularly selective oxidizing reagents. In acetonitrile or dichloroethane, alkenes are slowly oxidized to a mixture of products, primarily allylic alcohols, ketones, and aldehydes; a small amount of epoxidation occurs, without stereoselectivity.[10]

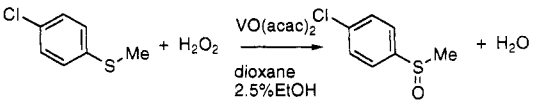


**Figure 6.** The oxidation of *p*-chlorophenyl methyl sulfide by hydrogen peroxide forming the sulfoxide.

**Scheme 7. Proposed Mechanism for the Vanadium(V)-Catalyzed Oxidation of Sulfide by Hydrogen Peroxide[12]**

$$VO(OR)_3 + H_2O_2 = VO(OR)(O_2)$$
$$VO(OR)(O_2) + R_2S \rightarrow VO_2(OR) + R_2SO$$
$$VO_2(OR) + H_2O_2 = VO(OR)(O_2) + H_2O$$

## B. Sulfide Oxidation

$VO(acac)_2$ is the catalyst precursor for the oxidation of *p*-chlorophenyl methyl sulfide by hydrogen peroxide forming the sulfoxide (Figure 6).[12] The reactions are nearly quantitative with respect to hydrogen peroxide, with 95% isolated yield of the sulfoxide. Reactions were run in dioxane/2.5% ethanol with 0.43 M $H_2O$. In this mixed solvent system, the kinetics are more tractable than in the absence of ethanol. The rate of the reaction is dependent on hydrogen peroxide concentration below $10^{-2}$ M $H_2O_2$; at higher concentrations of hydrogen peroxide the reaction is first-order in sulfide and catalyst only. Saturation with respect to $[H_2O_2]$ is observed because of the high binding constant of $H_2O_2$ to vanadium(V). An increase in the ethanol concentration from 2.5% to 15% lowers the rate of sulfide oxidation under high hydrogen peroxide conditions, a result which is consistent with the formation of a less reactive diperoxo species. The rate also depends on the nature of the alcohol: 2-propanol < ethanol < methanol. The active oxidant is $VO(OR)(O_2)$, which is formed from $VO(acac)_2$ via oxidation by $H_2O_2$, solvolysis by ROH, and coordination of $H_2O_2$ (Scheme 7).[12] $VO(OR)(O_2)$ is kinetically indistinguishable from $VO(OR)_2(OOH)$, but subsequent spectroscopic studies suggest a cyclic structure for the peroxide–vanadium moiety (see section II). $VO(OR)(O_2)$ reacts with sulfides to yield sulfoxides. Modena *et al.* rationalize the rate-dependence on the identity of the added alcohol in terms of the increasingly acidic alcohols assisting in O–O bond breaking.

In methanolic solution, $VO(O_2)(OCH_3)$ oxidizes di-*n*-butyl sulfide and methyl phenyl sulfide rapidly and quantitatively in a bimolecular, electrophilic reaction.[16] The rate law for the oxidation reaction is

$$\text{rate} = k[\text{sulfide}][VO(O_2)(OCH_3)]$$

The V(V)-catalyzed oxidation of a series of dialkyl and alkylaryl sulfides by both hydrogen peroxide and *tert*-butyl hydroperoxide in ethanol was studied to investigate the question of substrate binding to the vanadium(V)–peroxo complexes.[15] Both the monoperoxo and the diperoxo vanadium(V) complexes are present in solution, in amounts governed by the equilibrium $VO(OR)(O_2) + H_2O_2 = VO(O_2)_2^- + ROH_2^+$. As was observed for reactions in dioxane (see above), at lower peroxide concentrations, the kinetics of oxidation are consistent with a simple, bimolecular reaction:

$$\text{rate} = k[\text{sulfide}][\text{catalyst}]$$

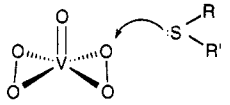
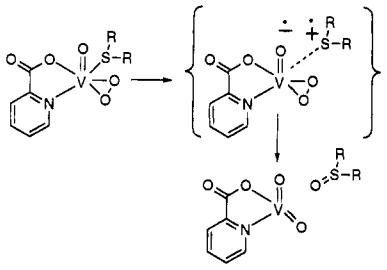


**Figure 7.** Nucleophilic attack by sulfide on bound peroxide.

**Scheme 8. Proposed Mechanism for Sulfide Oxidation by $VO(O_2)(pic)$ (adapted from ref 16)**



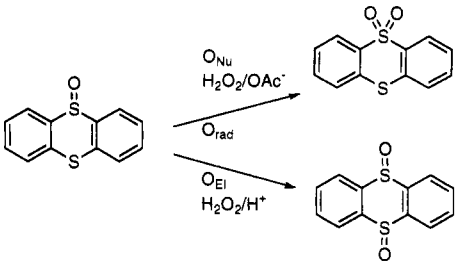
Again, the independence of peroxide concentration at these low peroxide concentrations is a consequence of the high binding constant of peroxide to the metal center. At high peroxide concentration, the formation of $VO(O_2)_2^-$ is favored. Since the neutral $VO(OR)(O_2)$ species is the better oxidant, the rate decreases. Saturation with increasing sulfide is not observed. Furthermore, like the rate of oxidation for the acid-catalyzed oxidation by hydrogen peroxide, these rates are only slightly sensitive to the different steric bulk of the sulfides, suggesting that the sulfide does not coordinate to the metal center but undergoes nucleophilic addition to the peroxide oxygen (Figure 7).

A kinetic study of the oxidation of sulfides by $VO(O_2)(pic)(H_2O)_2$ in methanol reveals more mechanistic detail (Scheme 8).[16] The yield of sulfoxide is substoichiometric with respect to the peroxo complex. The reaction is proposed to occur via a one-electron transfer from the sulfide, forming a radical cation–radical anion pair. This pair frequently collapses to the observed products, but escape from the radical cage gives rise to the other minor products (formaldehyde and its acetal, which are presumably derived from oxidation of the solvent, methanol). The rate of oxidation by $VO(O_2)(pic)(H_2O)_2$ shows saturation in substrate concentration, suggesting that the electron transfer from sulfide to vanadium occurs from bound substrate. Substrate binding would produce pentagonal coordination in the equatorial plane, which is a well-established geometry for vanadium–peroxo complexes.

Modena and co-workers attribute the different reactivity of the methoxy (see above) and picolinato complexes to the greater stability of the radical anion in the case of the picolinato complex, which shows both radical and nonradical behavior. The absence of a $\pi$-acceptor ligand in the methoxy complex, it is proposed, restricts the chemistry to nonradical oxidation. (The hydroxylation of benzene and the oxidation of alcohols by the methoxy complex, however, do appear to proceed by a radical mechanism. A fuller discussion may be found below.)

One way to assess the electrophilic character of a peroxide is to measure its preference to oxidize sulfides over sulfoxides. A study was done to determine the nucleophilicity of the peroxide in $VO(O_2)(pic)(H_2O)_2$.[14] A probe containing both a sulfide and a sulfoxide, thianthrene 5-oxide, was used. This substrate reacts with hydrogen peroxide in acidic solution to give only the bis(sulfoxide), while in basic solution the sulfone is the sole product (Scheme 9). Interestingly, $VO(O_2)(pic)(H_2O)_2$ in acetonitrile gives about equal amounts of the two products. This result is rationalized by recognizing the ability of the picolinato complex to be reduced to the radical anion. Reaction of both the neutral and anionic vanadium complexes with the substrate and its radical cation give rise to the observed products. This mechanism is supported by a more classical competition between a sulfide and a sulfoxide. The reaction of $VO(O_2)(pic)(H_2O)_2$ with *p*-chlorophenyl methyl sulfide and methyl phenyl sulfoxide in $CHCl_3$ again gives nearly equal amounts of the sulfoxide and the sulfone. The proposed mechanism of oxidation of the sulfide to the sulfoxide is electrophilic oxygen atom transfer. The oxidation of the sulfoxide to the sulfone appears to proceed via nucleophilic oxygen atom transfer, but is more consistent with a single-electron transfer step and radical recombination.[14]

**Scheme 9. Reactions of Thianthrene 5-Oxide with Nucleophilic and Electrophilic Peroxide and with Oxygen Radicals (adapted from ref 14)**



Several vanadium peroxo complexes have been shown to oxidize a Co(III)–thiolato complex to give the corresponding Co(III)–sulfenato complex.[23]

$$(en)_2Co^{III}(SCH_2CH_2NH_2) \rightarrow (en)_2Co^{III}(S(O)CH_2CH_2NH_2)$$

Kinetic studies show an acid-dependent and an acid-independent pathway. The deprotonation constant, $K_a$, was determined potentiometrically for the equilibrium $HVO(O_2)_2(pic) = VO(O_2)_2(pic)^{2-} + H^+$. The $pK_a$ was found to be 4.41. Although the site of protonation is not established from these kinetic results, it is not unreasonable to infer that protonation occurs at one of the peroxide ligands and, therefore, that both $VO(O_2)_2(pic)^{2-}$ and $VO(O_2)(OOH)(pic)^-$ oxidize the cobalt complex. By contrast, $VO(O_2)(dipic)^-$ reacts very slowly; the reaction is actually slower than that of free hydrogen peroxide. Thompson *et al.* rationalize this difference in reactivity as a difference in the inherent reactivity of mono and diperoxo vanadium complexes.[23] Yet, in ethanol, the formation of $VO(O_2)_2^-$ slows oxidation reactions in which $VO(O_2)(OR)$ is the primary oxidizing species (see above).[53] Another rationale for the greater reactivity of $VO(O_2)_2(pic)^{2-}$ is that the $dipic^{2-}$ ligand is inherently deactivating. However, in acetonitrile, $VO(dipic)(OOBu)(H_2O)$ is an excellent catalyst for the oxidation of methyl phenyl sulfide by tBuOOH.[13]

Vanadium(V) complexes can be made using the Schiff-base ligands formed from the condensation of salicylaldehyde and the L-amino acids alanine, phenylalanine, valine, and leucine. These complexes

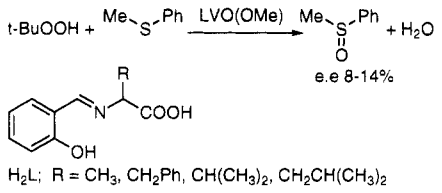



**Figure 8.** Vanadium complexes of chiral ligands catalyze asymmetric oxidations.

catalyze the asymmetric oxidation of methyl phenyl sulfide by *tert*-butyl hydroperoxide in dichloromethane to give small (8–14%) enantiomeric excesses of (*R*)-methyl phenyl sulfide (Figure 8).[13]

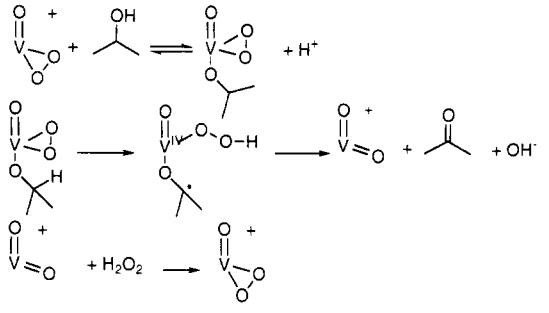
## C. Aromatic, Alkane, and Alcohol Oxidation

The mechanism of the oxidation of arenes, alkanes, and alcohols is universally consistent with single electron transfer steps. The radical chemistry varies with the identity of the vanadium catalyst.

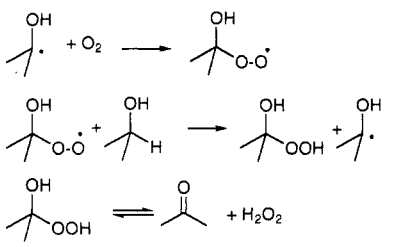
Although $VO(O_2)(OR)$ is preferentially a two-electron oxidant (e.g., when oxidizing sulfides; see III.B, above), it can function as a one-electron oxidant in cases where no two-electron pathway is accessible.[21,22] For example in catalytic reactions under argon, 2-propanol is oxidized to acetone stoichiometrically with respect to $H_2O_2$ consumption (Scheme 10). Similarly, ethanol is oxidized to acetaldehyde stoichiometrically with respect to $H_2O_2$.

In the presence of air, the oxidation of 2-propanol to acetone is accompanied by an *increase* in hydrogen peroxide concentration. Use of $^{18}O_2$ confirms that the additional hydrogen peroxide is formed by reduction of dioxygen. The proposed mechanism involves hydrogen atom abstraction from the alcohol by the vanadium–peroxo complex, followed by radical trapping by dioxygen. The organic peroxy radical decomposes to products and regenerates the one-electron oxidized substrate (Scheme 11).[21,22]

**Scheme 10. Proposed Mechanism for the Anaerobic Oxidation of 2-Propanol to Acetone by Hydrogen Peroxide**




**Scheme 11. Proposed Mechanism for the Production of Acetone and Hydrogen Peroxide from the Oxidation of 2-Propanol under Aerobic Conditions (adapted from ref 21)**



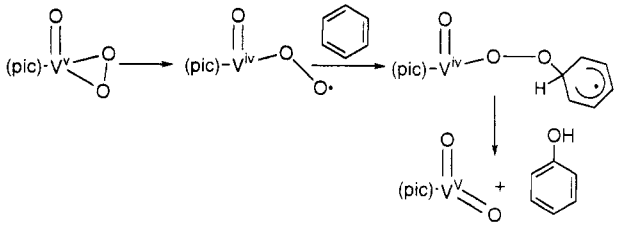

The radical chain steps are depicted here for free radicals. No evidence currently exists regarding the role of vanadium in addition of $O_2$ to the first RO•. Modena speculates on the chain termination steps. Additional work[22] under basic conditions suggests that alcohol coordination to the oxoperoxovanadium(V) complex is required for the initial one-electron oxidation. $VO(O_2)$(2-OPr) is also competent in benzene hydroxylation.[19]

Oxo–peroxo oxygen atom exchange was invoked in a previous study in which $H_2{}^{18}O_2$ was apparently diluted during incubation with $VO(OEt)_3$ and $H_2O_2$.[61] The discovery of the radical pathway, confirmed with $^{18}O_2$ experiments, enabled Modena *et al.* to reinterpret their earlier results. At this point there is no evidence for scrambling of the terminal oxide and peroxide oxygen atoms (See footnote 13 in ref 21).

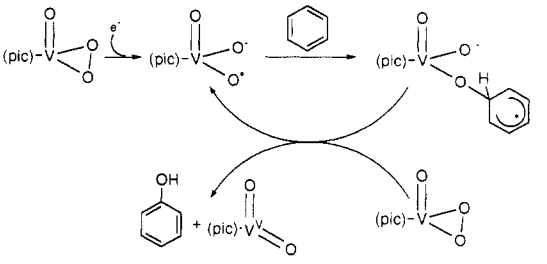
$VO(O_2)(pic)(H_2O)_2$ also functions as a radical hydroxylation catalyst, although the subtleties of the mechanistic studies of $VO(O_2)(pic)(H_2O)_2$ catalysis of the hydroxylation of benzene are contradictory. On the basis of the absence of a primary deuterium isotope effect and a high (70%) NIH shift value, Mimoun and co-workers suggest a mechanism in which the vanadium(V)–peroxo complex undergoes intramolecular electron transfer to a give $V^{IV}$–($O_2^{\bullet-}$) biradical which inserts into aromatic or aliphatic C–H bonds (Scheme 12).[9] On the other hand, Modena suggests that the reaction occurs via the radical anion of the vanadium complex (Scheme 13) on the basis of kinetic studies, solvent dependence, and the effects of one-electron donors and radical traps.[19]

Mimoun also reports that $VO(O_2)(O\text{-}N)L_2$, where O-N is pyrazine-2-carboxylate and L is $H_2O$ or a basic ligand (e.g., pyridine *N*-oxide), can oxidize benzene.[9] This system was recently reported to be catalytic in acetonitrile.[17] Cyclohexane and other alkanes are oxidized at 20–70 °C to the corresponding alkyl hydroperoxides. Depending on the workup, either a mixture of the

**Scheme 12. Proposed Mechanism for the Hydroxylation of Benzene by $VO(O_2)(pic)$ (adapted from ref 9)**




**Scheme 13. An Alternate Mechanistic Proposal for the Hydroxylation of Benzene by $VO(O_2)(pic)$ (adapted from ref 19)**



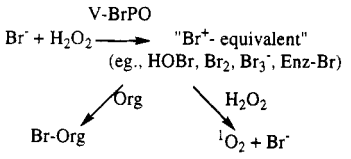
alcohol and the ketone or the alcohol alone is obtained. Total yields based on peroxide are 46% with turnover numbers around 1000. Benzene and other arenes are hydroxylated by $VO(O_2)(O\text{-}N)L_2$ at the ring and aliphatic side chain positions. On the basis of product analysis and some preliminary kinetics, including radical inhibitors, a radical mechanism is proposed. The precise nature of the radical species is unknown, but the oxidation appears not to be a chain process.[17] Irradiation decreases the induction period and increases the yield of oxidized products.[18]

## *IV. Reactivity of Peroxovanadium(V) Complexes with Inorganic Compounds*

### A. Halide Oxidation

Vanadium bromoperoxidase (V-BrPO),[1,2] a vanadium(V)-containing enzyme found primarily in marine organisms, catalyzes peroxidative halogenation reactions and the halide-assisted disproportionation of hydrogen peroxide (Scheme 14). V-BrPO functions by coordinating hydrogen peroxide and then oxidizing the halide; the nature of the oxidized halide species as enzyme-retained or enzyme-released seems to depend on the nature of the organic substrate.[62]

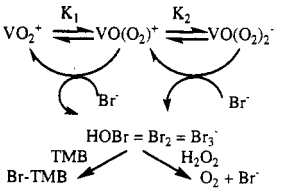
**Scheme 14. Peroxidative Bromination and Bromide-Assisted Disproportionation of Hydrogen Peroxide Catalyzed by V-BrPO by V-BrPO**



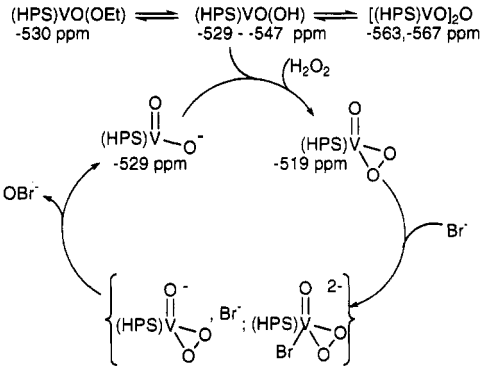

Prior to the discovery of V-BrPO, iodide oxidation by the peroxovanadium(V) species $VO(O_2)^+$ and $VO(O_2)_2^-$ had been investigated.[26] Addition of iodide to acidic solutions of vanadium(V) and hydrogen peroxide resulted in the production of iodine and, more slowly, vanadium(IV).[26] The rate of the oxidation reaction is quite sensitive to acid, vanadium(V), and hydrogen peroxide concentrations as a result of the ability of three monoperoxo- and two diperoxovanadium(V) species in different protonation states to contribute to the oxidation reaction. The reaction was first-order in iodide over the range of concentrations studied [i.e., $(2 \times 10^{-4})$–$(8 \times 10^{-3})$ M]. Iodide is proposed to undergo nucleophilic attack on the vanadium(V) center. Such a mechanism might show saturation with respect to iodide concentration, but as the highest iodide concentration was only 8 mM, the requisite concentration to observe saturation may not have been achieved.

Bromide, which is significantly harder to oxidize than iodide, is also oxidized by peroxovanadium(V) species in acidic aqueous solution.[27] The formation of the oxidized bromine species is quantitative with hydrogen peroxide consumption, as determined by the bromination of 1,3,5-trimethoxybenzene (TMB). Under the catalytic turnover conditions investigated for the bromination of TMB (e.g., 0.05 M $HClO_4$, 0.5 M KBr, 10 mM $H_2O_2$, 1 mM $VO_2^+$), both $VO(O_2)^+$ and $VO(O_2)_2^-$

**Scheme 15. $VO_2^+$-Catalyzed Oxidation of Bromide by Hydrogen Peroxide**




**Scheme 16. $(HPS)VO_8^-$-Catalyzed Oxidation of Bromide by Hydrogen Peroxide**




are formed as established by $^{51}V$ NMR and can oxidize bromide as shown in Scheme 15. Addition of bromide to the equilibrium mixture of $VO(O_2)^+$ and $VO(O_2)_2^-$ formed from 10:1 $H_2O_2$:$VO_2^+$ in 0.05 M $HClO_4$ results in the rapid disappearance of the oxodiperoxovanadium(V) $^{51}V$ NMR resonance, with a corresponding increase in the signal area of the monoperoxo complex, followed by the slower disappearance of the monoperoxo $^{51}V$ NMR resonance, indicating regeneration of $VO_2^+$. Unlike iodide, bromide does not reduce $VO_2^+$. Chloride is also oxidized, but more slowly than bromide.

Because the V-BrPO functions fastest at pH 5.5–7, while $VO_2^+$ only functions in relatively acidic solution and at a much lower turnover rate than V-BrPO, we turned to vanadium(V) complexes to further pursue the biomimetic chemistry of V-BrPO. The $HPS^{2-}$ complex of VO(OH) coordinates $H_2O_2$, forming the monoperoxo species $(HPS)VO(O_2)^-$.[63] This peroxo complex oxidizes bromide in DMF (Scheme 16).

The catalytic reaction requires acid for bromide oxidation. Even if excess $H_2O_2$ is present, only a single equivalent of bromide is oxidized in the absence of added acid. When acid is added, an additional turnover is observed for each equivalent of acid. The acid dependence is rationalized in terms of the production of base, since bromide oxidation by hydrogen peroxide in aqueous solution only occurs at near neutral pH or lower. Provided the acid concentration is sufficient, bromide oxidation is quantitative with hydrogen peroxide concentration. Interestingly, although the reaction is inhibited by alcohol, due to the formation of (HPS)VO(OR), the hydroxo complex effectively catalyzes the oxidation of bromide by hydrogen peroxide in DMF, a solvent in which epoxidation by the HPS complex of vanadium(V) is completely retarded (See section III.A, above). Other vanadium(V) complexes have also been investigated.[64] The *N*-(2-hydroxyethyl)iminodiacetic acid complex of oxoperoxovanadium(V) also oxidizes bromide.[104]

Vanadium(V)–peroxo complexes have also been shown to oxidize the pseudohalide thiocyanate.[65] This

result is not unexpected because the oxidation potential of $NCS^-$ is between that of $I^-$ and $Br^-$. At pH 7–8, vanadium pentoxide, alkali thiocyanate, and hydrogen peroxide react to give oxodiperoxovanadate complexes and sulfate. Although sulfate is present in the crystal lattice of the product, it is not coordinated to the vanadium center.[65]

### B. $SO_2$ Oxidation

Sulfate is formed from the reaction of $SO_2$ with triperoxovanadate $V(O_2)_3^-$.[28] Insertion of $SO_2$ into one of the peroxo O–O bonds gives the putative intermediate $V(O_2)_2(SO_4)^-$, which hydrolyzes to $VO(O_2)_2(H_2O)^-$ and $H_2SO_4$. Bubbling of additional $SO_2$ results in a blue-green solution (pH *ca.* 2), from which $A_2[V^{IV}O(SO_4)_2(H_2O)_3]\cdot H_2O$ ($A^+$ is $Na^+$ or $K^+$) is isolated. IR and Raman spectroscopic results suggest that sulfate coordination is monodentate to vanadium(IV).

## *V. Other Reactivity*

### A. Insulin Mimics

Vanadate and peroxide have been shown to act synergistically to mimic insulin activity with maximal effects at least as great as insulin.[66] Evidence suggests that peroxovanadates formed under physiological conditions enhance translocation of the insulin growth factor II (IGF-II) receptor to the plasma membranes of rat adipocytes. In addition, peroxovanadates activate the tyrosine kinase receptor and stimulate tyrosine phosphorylation of the receptor, both of which are believed to be necessary for insulin activity.[67]

Shaver *et al.* have determined that several discrete, stable peroxo complexes of vanadium are also effective insulin mimics.[68] These complexes have the general formula $A_n[VO(O_2)_xL\text{-}L']\cdot yH_2O$ where $A^+$ is $NH_4^+$ or $K^+$, $n$ is 0–3, $x$ is 1 or 2, and L-L′ is usually a bidentate ligand.[68,105] The mechanism by which peroxo complexes of vanadium mimic insulin is not fully understood. Unlike insulin, peroxovanadates do not appear to stimulate autophosphorylation of the receptor; these complexes do not enhance phosphorylation of purified receptors *in vitro*. It has been proposed that vanadium–peroxo complexes stimulate tyrosine phosphorylation by the inhibition of protein tyrosine phosphatases (PTPase) possibly via oxidative coupling of cysteine residues.[67] Stimulation of T cell activation by peroxovanadates, which also involves tyrosine phosphorylation of intracellular proteins, has been shown to be accompanied by PTPase inhibition.[69,70] The insulin mimetic complexes $K_2[VO(O_2)_2pic]\cdot 2H_2O$ and $K_2[VO(O_2)_2(OHpic)]\cdot 3H_2O$ do oxidize cysteine to cystine; however, this process is less efficient with these complexes than with the much poorer insulin mimic $H_2O_2$. Thus, it seems likely that the site specificity achieved by peroxovanadates may also play a role in their activity.[68]

### B. Photochemistry of $H_2O_2$ Disproportionation

The photochemistry of $VO(O_2)^+$ in acidic aqueous solution was investigated by Shinohara and Nakamura.[71] Irradiation of $VO(O_2)^+$ at 313, 366, or 436 nm into the tail of the 285 nm absorption maximum of $VO(O_2)^+$ resulted in the photoreduction of the complex with the following overall reaction:

$$4VO(O_2)^+ + 4H^+ \rightarrow 4VO^{2+} + 3O_2 + 2H_2O$$

Photolysis is proposed to generate a vanadium(IV)–superoxo complex. A plausible scheme is presented below:

$$2(VO(O_2)^+ \rightarrow [O{=}V(IV)OO^{\bullet+}]^*) \quad (a)$$

$$2([O{=}V(IV)OO^{\bullet+}]^* + H^+ \rightarrow VO^{2+} + HO_2) \quad (b)$$

$$3(VO(O_2)^+ + HO_2 + H^+ \rightarrow VO^{2+} + O_2 + H_2O_2) \quad (c)$$

$$VO^{2+} + H_2O_2 \rightarrow VO_2^+ + OH^\bullet + H^+ \quad (d)$$

$$VO(O_2)^+ + OH^\bullet \rightarrow VO_2^+ + HO_2 \quad (e)$$

$$2(VO_2^+ + H_2O_2 \rightarrow VO(O_2)^+ + H_2O) \quad (f)$$

The reaction of the complex with $HO_2$ in step c was demonstrated previously by Rush and Bielski.[72] In addition, Samuni and Czapski showed that the oxidation of $VO^{2+}$ by $H_2O_2$ is accompanied by $VO(O_2)^+$ production.[73] Addition of excess 2-propanol to the irradiation solution resulted in the formation of acetone, which supports the existence of $OH^\bullet$ radicals; a mechanism is proposed:

$$(CH_3)_2CHOH + OH^\bullet \rightarrow (CH_3)_2COH^{\bullet+}\ H_2O$$

$$(CH_3)_2COH^\bullet + VO_2^+ + H^+ \rightarrow VO^{2+} + (CH_3)_2CO + H_2O$$

### C. Oxidation of Peroxovanadium(V)

In addition to the oxidative reactivity of oxoperoxovanadium(V), Thompson demonstrated that $VO(O_2)^+$ itself can also be oxidized.[74,75] This work is important in part because it demonstrates that a peroxovanadium(V) complex, like hydrogen peroxide, can act as both an oxidant and a reductant. Furthermore, this work establishes an important link between vanadium(V)–peroxo complexes and dioxygen chemistry, invoking as it does the $VO(O_2)^{\bullet 2+}$ radical cation. This radical, formally a vanadium(V)–superoxide complex, may be the initial product of reduction of dioxygen by vanadium(IV).

The oxidation of $VO(O_2)^+$ by peroxysulfate, $HSO_5^-$, is catalyzed by $VO^{2+}$ in an overall reaction: $HSO_5^- + VO(O_2)^+ \rightarrow HSO_4^- + VO_2^+ + O_2$.[74] The reaction is proposed to proceed via the $VO(O_2)^{\bullet 2+}$ radical cation, according to the following mechanism:

$$HSO_5^- + VO^{2+} \rightarrow SO_4^{\bullet-} + VO_2^+ + H^+ \quad (k_1)$$

$$SO_4^{\bullet-} + VO(O_2)^+ + H^+ \rightarrow HSO_4^- + VO(O_2)^{\bullet 2+} \quad (k_2)$$

$$VO(O_2)^{\bullet 2+} \rightarrow VO^{2+} + O_2 \quad (k_3)$$

$$SO_4^{\bullet-} + VO^{2+} + H_2O \rightarrow HSO_4^- + VO_2^+ + H^+ \quad (k_4)$$

Intramolecular electron transfer gives the observed $VO^{2+}$ and $O_2$ products. A kinetic analysis reveals that $k_2$ is 43 times larger than $k_4$, suggesting that $SO_4^{\bullet-}$ is a selective oxidant. $VO(O_2)^+$ appears to be a one-electron reductant even where a two-electron pathway is conceivable.[74] $SO_4F^-$ shows analogous reactivity.[75]

Cobalt(III) oxidizes oxoperoxovanadium(V) according to the limiting stoichiometry[74]

$$Co^{3+} + VO(O_2)^+ \rightarrow Co^{2+} + VO^{2+} + O_2$$

The mechanism of this reaction also invokes the $VO(O_2)^{\bullet 2+}$ radical. The mechanism is fairly simple to write, but difficult to analyze because of competitive, consecutive second-order reactions.

$$Co^{3+} + VO(O_2)^+ \rightarrow Co^{2+} + VO(O_2)^{\bullet 2+}$$

$$VO(O_2)^{\bullet 2+} \rightarrow VO^{2+} + O_2$$

$Co^{3+}$ can also react with $VO^{2+}$:

$$Co^{3+} + VO^{2+} + H_2O \rightarrow Co^{2+} + VO_2^+ + 2\,H^+$$

The reaction of $Cl_2$ and $VO(O_2)^+$ was also investigated. Surprisingly, the only observed oxidation is the reaction of $Cl_2$ with free $H_2O_2$, even when measures are taken to reduce free $H_2O_2$ to trace levels.[75] HOCl oxidizes $VO(O_2)^+$ in a kinetically complicated reaction. The mechanism is tentatively proposed to involve direct reaction of HOCl and $VO(O_2)^+$ to give products, but several observations lead Thompson to suggest that a complete analysis might complicate this preliminary understanding.

$S_2O_8^{2-}$ oxidizes $VO(O_2)^+$ in a reaction catalyzed by $Ag^+$.[75]

$$S_2O_8^{2-} + VO(O_2)^+ + 2H^+ \rightarrow 2HSO_4^- + 2VO^{2+} + 2O_2$$

The mechanism of this reaction invokes one of the same intermediates found in the $HSO_5^-$ and Co(III) oxidations described above, $VO(O_2)^{\bullet 2+}$, as well as $Ag^{2+}$

$$S_2O_8^{2-} + Ag^+ \rightarrow SO_4^{\bullet-} + SO_4^{2-} + Ag^{2+}$$

$$SO_4^{\bullet-} + Ag^+ \rightarrow SO_4^{2-} + Ag^{2+}$$

$$Ag^{2+} + VO(O_2)^+ \rightarrow Ag^+ + VO(O_2)^{\bullet 2+}$$

$$VO(O_2)^{\bullet 2+} \rightarrow VO^{2+} + O_2$$

The vanadium(IV) product is also subject to oxidation:

$$Ag^{2+} + VO^{2+} + H_2O \rightarrow Ag^+ + VO_2^+ + 2\,H^+$$

Finally $VO(O_2)^+$ was found to react very slowly with $XeO_3$ and even to inhibit the normally rapid reaction between free $H_2O_2$ and $XeO_3$.

## VI. Summary

In conclusion, peroxovanadium(V) complexes have been found to carry out a variety of oxidation reactions, including alkene epoxidation and hydroxylation, sulfide oxidation, aromatic, alkane and alcohol oxidation, halide oxidation, $SO_2$ oxidation, etc. While the selectivity of these reactions varies widely, the regioselectivity and stereoselectivity can be achieved with the appropriate choice of coordinating organic ligand. In fact, peroxovanadium(V) complexes have been developed into synthetically useful catalysts. The reactivity of the peroxovanadium(V) moiety is also of biological importance as in the catalytic cyclic of vanadium bromoperoxidase. Other biologically important peroxovanadium(V) species function as insulin mimics. Developments in the elucidation of the mechanism of oxidation of NAD(P)H by vanadate and the role of superoxide and hydrogen peroxide in this process will also be interesting to follow in the coming years. In summary, peroxovanadium(V) chemistry is rich, varied, and interesting.

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