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Research Article

OXIDATION OF PHENOL BY BRIDGING SUPEROXIDE LIGAND IN A BINUCLEAR Co^{III} COMPLEX CONTAINING HETEROLEPTIC LIGANDS: A KINETIC AND MECHANISTIC STUDY

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ABSTRACT

PhOH is a good scavenger of superoxide ions as well as very good free radical scavenger (FRS). In aqueous acetate buffer (pH:3.4-4.2), PhOH reduces the bridging superoxide in $[(en)(dien)Co^{III}(O_2)Co^{III}(en)(dien)](ClO_4)_5$(**1**) to the corresponding hydroperoxo complex, $[(en)(dien)Co^{III}(\mu\text{-}O_2H)Co^{III}(en)(dien)]]^{5+}$(**2**) and itself gets oxidized, following both proton coupled electron transfer (PCET) and electron transfer(EC) path. In the presence of excess [PhOH] over [**1**], the reaction obeys first-order kinetics and rate of the reaction decreases with increase in $[H^+]$. Since de-protonation of PhOH(pK_a = 10.0) is not possible in the working pH, so protonation of **1** at equilibrium generates $[(en)(dien)Co^{III}(\mu\text{-}O_2H)Co^{III}(en)(dien)]]^{6+}$(**1**H), the conjugate acid from **1**, which appears to be a kinetic dead-end species and that accounts for the observed inverse proton dependence on rate. Reaction rate significantly decreases with increasing proportion of D_2O replacing H_2O in the solvent and an H-atom transfer (HAT) from the reducing species to the bridging superoxide in **1** seems reasonable at the rate step.

Keywords: Kinetics, Mechanisms, Phenol, Superoxide, Redox

1. INTRODUCTION

In most biological processes ubiquitous and uncontrolled concentrations of free radicals have been widely regarded as a cause of cell injury and even death [1]. Superoxide anion ($O_2^{\cdot-}$) is particularly important as the product of the one electron reduction of dioxygen O_2, which occurs widely in nature [2]. In the human body due to numerous physiological and biochemical processes may also produce this oxygen-centered free radicals *i.e.*, superoxide ions ($O_2^{\cdot-}$) [3]. It is biologically quite toxic and is deployed by the immune system to kill microorganisms. In phagocytes, superoxide is produced in large quantities by the enzyme NADPH [4] oxidase for use in oxygen-dependent killing mechanisms of invading pathogens. Superoxide is also deleterious when produced as a byproduct of mitochondrial respiration; it may contribute to the pathogenesis of many diseases [5] and aging via the oxidative damage that inflicts on cells. Because of its toxicity, nearly all organisms living in the presence of oxygen contain isoforms of the superoxide-scavenger [6]. Phenol is a good scavenger of superoxide ions as well as very good free radical scavenger (FRS) [7] at the same time phenolic compounds are good natural antioxidant in fact most of the natural antioxidant are phenolic in nature [8]. It primarily inhibit lipid oxidation through their free radical scavenger ability and generate the low energy phenolic radicals which decreases the energy of the antioxidant and prevent autoxidation of the antioxidant radical into additional free radicals [9] , which will influence the antioxidant effectiveness of a free radical scavenger (FRS). Moreover phenolic compounds are ubiquitous in plant foods, and therefore, a significant quantity is consumed in our daily diet. Because of the high bioavailability in humans the Phenolic compounds are highly beneficial [10] on human health and used in traditional medicine of several cultures [11]. But their role in human health and disease is a subject of research [12, 13]. Some phenols are germicidal and are used in formulating disinfectants. So study of reaction between metal bound superoxide and phenol is very important. The present work represents the kinetic and mechanistic study for the oxidation of phenol by a Co^{III}-bound superoxide in aqueous acetate buffer medium.

2. EXPERIMENTAL

2.1. Materials

The superoxo complex, μ_2-superoxo[bis(ethylenedi - amine)bis(diethylenetriamime)cobalt(III)] perchlorate, $[(en)(dien)Co^{III}(O_2)Co^{III}(en)(dien)](ClO_4)_5$ (**1**) was

synthesized by the literature procedure [14] and recrystallized from 0.3 M $HClO_4$. Its purity was checked by measuring absorbance at 708nm [ε^{708} mol^{-1} dm^3 cm^{-1}: found 1186; reported [14]:1210 ± 5%]. Phenol (Aldrich) was standardized spectrophotometrically [15]. 2, 6-pyridinedicarboxylic acid (dipicolinic acid, Aldrich) was used as received. $NaClO_4$ was prepared by neutralizing $HClO_4$ with $NaHCO_3$ and subsequently concentrating the neutral solution. All other solutions were of the reagent grade and used without further purification.

2.2. Physical measurements and kinetics

Absorbances and UV-VIS spectra were recorded with a Shimadzu 1800 spectrophotometer using 1.00 cm quartz cell. Reactions were conducted *in situ* in the thermostated (25 ± 0.1°C) cell housing of the spectrophotometer and in acetate buffer (pH, 3.4-4.2; T_{OAc}= 0.20 M) using excess [PhOH], at least 10 times over [**1**] at an ionic strength 0.5 M ($NaClO_4$). The kinetics was monitored at 708 nm, the visible absorption maxima of the dicobalt superoxo complex (**1**). Under these conditions, the reactions obeyed very good first-order kinetics at least up to 90% completion of reaction and the first order rate constants (k_o) were evaluated by non-linear least squares fitting of the decay of the absorbance (A_t) with time (t) data to standard first-order exponential decay equation. A pH meter (Gold-533) with electrodes calibrated with standard buffer solutions was used for pH measurements, while reporting pH values in D_2O media the relation, pD = pH + 0.4 was used [16,17] . All solutions were prepared in doubly distilled and then freshly boiled water.

2.3. Stoichiometry

The equilibrium absorbance of a mixture of PhOH with 4-5 times of **1** was measured after ~5h at 708nm and the concentration of unused **1** in such a product mixture was determined spectrophotometrically at 708 nm.

2.4. Suppression of catalytic path

Trace of metal ions usually present in the reagents or buffers used in the kinetic studies play a catalytic role. Most probably only the Cu^{2+} among all adventitious metal ion appreciably catalyze the reaction such that the direct oxidation process is inaccessible. However by using suitable chelating agents, the catalyzed path can be completely suppressed. Dipicolinic acid (pK_1 = 2:07 and pK_2 = 4:66) is known for its strong binding with Cu^{2+} and thus acts as an inhibitor for the catalytic role of Cu^{2+} [18, 19]. In this study it is observed that [Cu^{2+}] is present in the blank is ($2.2x10^{-6}$ M) and makes the reaction faster. In the experimental condition, addition of 1×10^{-3} M of dipicolinic acid is sufficient to mask the catalytic effect due to the impurity levels of Cu^{2+}ions. Therefore probed the direct reaction between (**1**) and PhOH with the use of 1.0 mM dipicolinic acid.

3. RESULT AND DISCUSSION

3.1. Stoichiometry and reaction products

Each mole of PhOH consumed very nearly 2 moles of the superoxo complex **1**. Moreover it is observed that the final spectrum is closely similar in shape and peak positions (Fig. 1) to those determined for the hydroperoxo analogues of (**1**).

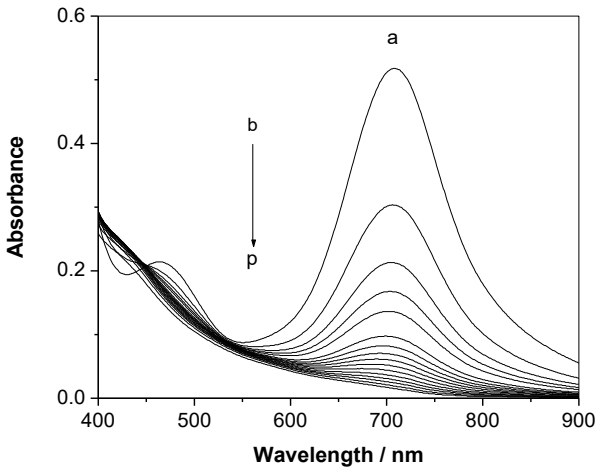


Fig. 1: Time resolved spectra of 0.50 mM of 1 reacting with 5.0 mM PhOH. pH = 4.24 in acetate buffer (T_{OAc} = 0.2 M), I = 0.5 M, T = 25.0 °C (a) Spectrum of the pure complex , (b)-(p) spectra of reaction mixture at time intervals 50, 110, 160, 210, 260, 300, 360, 420, 510, 590, 640, 700, 900, 1120 and 2250 seconds, respectively.

A clean conversion of the superoxo complex **1** to the hydroperoxo complex **2** is therefore anticipated (Eqns. 1& 2). The observed stoichiometric ratios also establish the generation of phenoxide radicals ($PhO^{\cdot}$) and this generated phenoxide radical immediately react with another mole of superoxide radical. Again for monophenols both the proton-transfer and radical-transfer pathway reactions are already established [20]. Therefore the equation 1 & 2 is very much reasonable.

$$2[(en)(dien)Co^{III}(O_2)Co^{III}(en)(dien)]^{5+} + PhOH \rightarrow 2[(en)(dien)Co^{III}(\mu O_2H)Co^{III}(en)(dien)]]^{5+} + PhO^{+} \quad (1)$$

$$PhO^{+} + H_2O \rightarrow Products \quad (2)$$

Hence the proposed reaction scheme in the abbreviated form can be represented as:

$$\mathbf{1} + H^+ \underset{}{\overset{K}{\rightleftharpoons}} \mathbf{1}H \quad \text{i.e, } [\mathbf{1}H] = K[\mathbf{1}][H^+]$$

Since, total concentration of the complex,

$$T_{\mathbf{1}} = [\mathbf{1}] + [\mathbf{1}H]$$

$$\text{or, } [\mathbf{1}] = \frac{T_{\mathbf{1}}}{1 + K[H^+]}$$

$$\text{Again, } \mathbf{1} + PhOH \xrightarrow{k} \text{product}$$

$$\text{So, rate} = k\,[\mathbf{1}]\,[PhOH] = k\,[PhOH] \times \frac{T_{\mathbf{1}}}{1 + K\,[H^+]}$$

$$k_{obs} = \frac{k\,[PhOH]}{1 + K\,[H]}$$

$$\text{And, } 1/k_o = \frac{1}{k\,[PhOH]} + \frac{K[H^+]}{k\,[PhOH]}$$

3.2. Kinetics

In aqueous acetate buffer media (pH: 3.4-4.2) **1** suffers no appreciable loss in absorbance over a long period of time indicating its stability against self-decomposition. Excess PhOH, however consumes **1** and the peak of absorbance (at 708 nm) drops gradually essentially to zero. The process followed good first-order kinetics (Fig. 2).

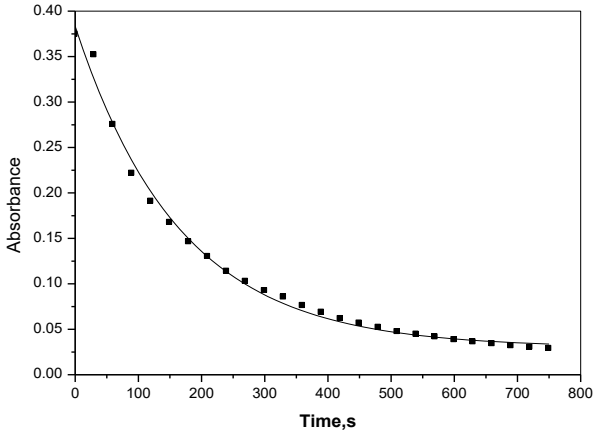


Fig. 2: Decrease in absorbance (points shown in black circles) of 1 with time at 708 nm in its reaction with PhOH gives an excellent fit (solid line) to the first-order exponential decay equation. [1] = 0.50 mM, [PhOH] = 6.0mM, pH = 4.24, *I* = 0.5M ($NaClO_4$), T_{OAc} = 0.2 M, T = 25.0 °C.

The first-order rate constants (k_o) increased linearly with [PhOH] (Fig. 3) and Table 1. The rates of reaction were found to be strongly influenced by the media of acidity and a plot of $1/k_o$ *vs* $[H^+]$ is linear with a small but significant intercept, (Fig. 4, and Table 2). Hence *k* and *K* are calculated from the plot are~ 4.16×10^{-6} s^{-1} and~ 6.24×10^{-4} respectively. But the media of ionic strength have no effect on the rate of reaction.

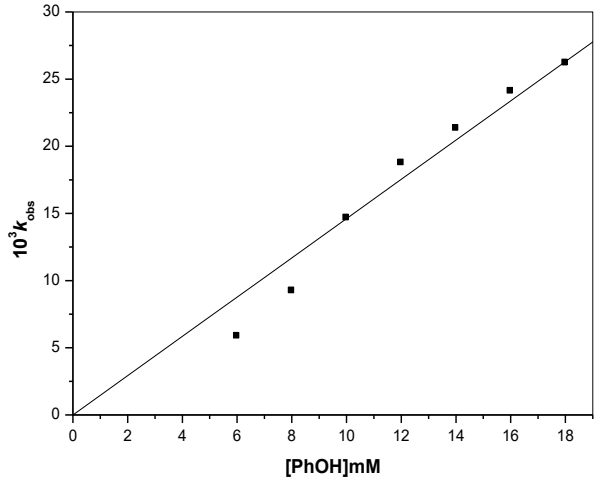


Fig. 3: Linear variation of k_o for the reaction of [PhOH] with 1 (0.50 mM), at pH = 4.01, T_{OAc} = 0.2 M, [dipicolinic acid] = 2.0 mM, *I* = 0.5M ($NaClO_4$), T = 25.0°C.

Table 1: Variation of k_o with [PhOH], with 1 (0.50 mM), at pH = 4.01, T_{OAc} = 0.2 M, [dipicolinic acid] = 2.0 mM, *I* = 0.5M ($NaClO_4$), T = 25.0°C.

[PhOH] /mM	10^3k_o / s^{-1}
6.0	5.86
8.0	9.23
10.0	14.66
12.0	18.77
14.0	21.33
16.0	24.1
18.0	26.2

The rate-enhancement with pH seems not accountable from deprotonation of PhOH as the PhOH is a weak acids (pK_a= 10.0) [21] and pH of the reaction medium 3.4 4.2. Rather, a mechanism transferring hydrogen atom (or H^++ e, proton-coupled electron transfer mechanism, PCET) to the coordinated superoxide (hydrogen atom transfer, HAT) seems reasonable as superoxide is well-known to be a fairly strong base [22].

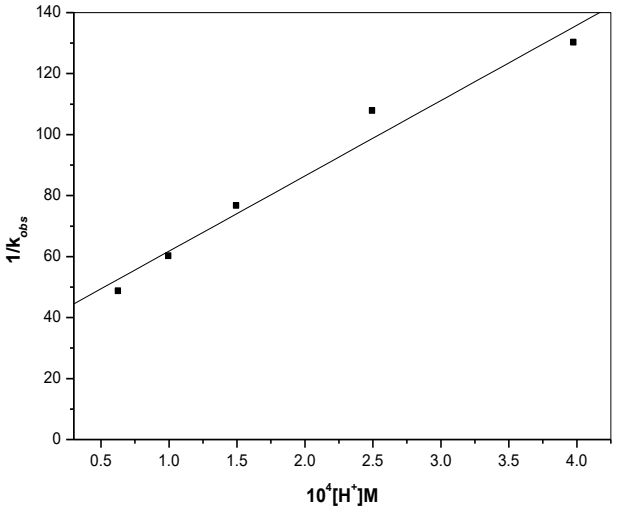


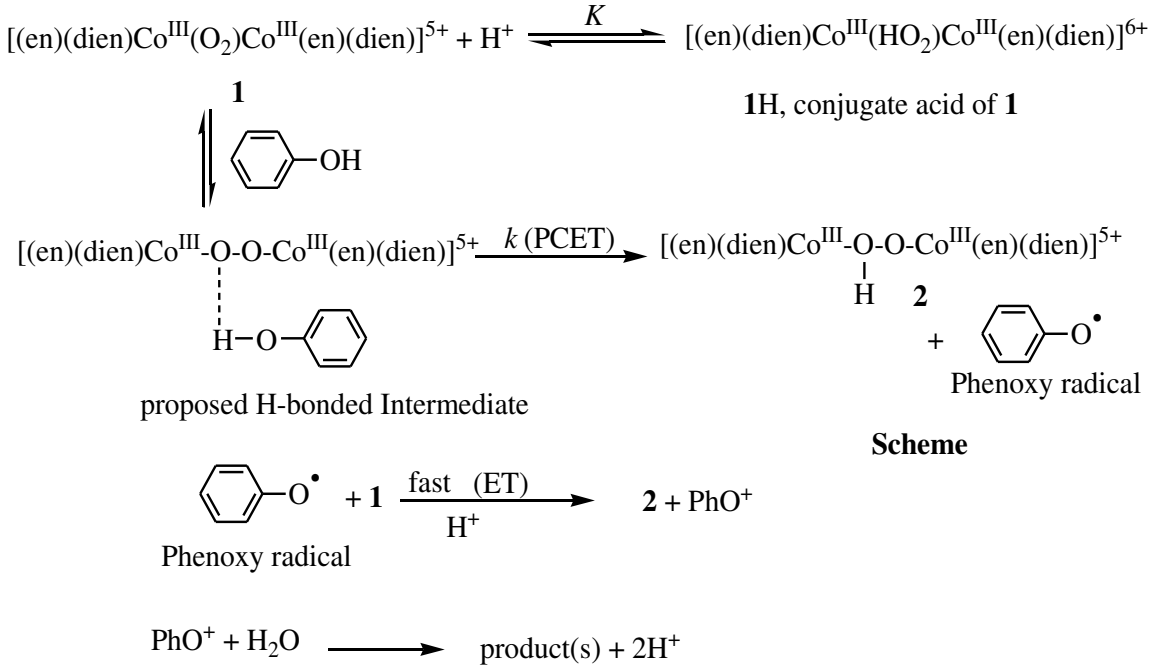
Fig. 4: Plot of $1/k_o$ *vs* $[H^+]$. [1] = 0.50 mM, [PhOH] = 6.0 mM.T_{OAc} = 0.2 M, *I* = 0.5 M ($NaClO_4$), T = 25.0 °C.

The observed proton-dependence on rate clearly establishes **1**H as a kinetically dead-end species. Increased proton concentration consumes more **1** from the solution forming more **1**H and consequently reaction rate falls.

Table 2: Variation of $1/k_o$ with pH, [1] = 0.50 mM, T_{OAc} = 0.2 M, [dipicolinic acid] = 2.0 mM, *I* = 0.5M ($NaClO_4$), T = 25.0°C.

pH	$1/k_o$ / s
3.4	130.0
3.6	107.6
3.8	76.5
4.0	60.0
4.2	48.52

1H, being already protonated species of **1** is a redox dead-end as it has no more room to accommodate a further proton following a HAT from the reducing species. In the rate determining step, **1** is reduced to its corresponding hydroperoxo complex (**2**). Compared to the superoxo, the peroxo ligand is a much stronger proton acceptor and is likely to encourage the H-transfer. Hence the following scheme can be proposed:



We observed a significant retardation in k_o values when solvent H_2O is enriched with D_2O. This supports an electroprotic HAT mechanism (H^+ + e) [23]. Again to verify the above scheme the superoxo complex **1** was reacted with hydrogen peroxide and tert-butyl hydroperoxide, they reduces **1** at a rate= (5.02×10^{-3} s^{-1}) and (4.1×10^{-3} s^{-1}) (at pH = 4.0, *I* = 0.5 M, T = 25.0 °C), respectively [24] but neither di-*tert*-butyl peroxide nor perdisulfate reacts with **1** under similar conditions. These observations strongly suggest the HAT mechanism (H^+ + e). However, being quite reactive molecules, hydrogen peroxide and *tert*-butyl hydroperoxide may be

involved in a number of side reactions. To verify the proposed mechanism, **1** was farther reacted with hydroxylamine. Hydroxylamine reacted with **1** but neither O-methyl hydroxylamine, anisole (methoxybenzene) nor phenyl methyl ether reacted under comparable conditions and this clearly substantiates the mechanistic proposal.

4. ACKNOWLEDGEMENT

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