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KINETICS AND MECHANISM OF OXIDATION OF L-ASCORBIC ACID BY CHROMIUM (VI)

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Abstract

The kinetics of oxidation of L-ascorbic acid by potassium chromate was studied as a function of pH, L-ascorbic acid concentration and temperature using the spectrophotometric technique. The rate of reaction is first order with respect to the concentration of each reactant and increases as $[H^+]$ increases. The kinetic data indicate involvement of the monoprotonated ascorbate species (HA^-) in the redox process. A mechanism involving the formation of chromium (VI)- ascorbate intermediate is a rate-determining step followed by a redox step was suggested. The activation enthalpy and activation entropy changes for the reaction have been calculated to be $\Delta H^* = 69$ kJmol^{-1} and $\Delta S^* = -209$ JK^{-1} mol^{-1}, respectively.

Introduction

Several studies have reviewed the toxicity and carcinogenicity of chromium compounds (1-3). Epidemiological and animal studies indicate that chromium (VI) compounds cause serious dangers to biological systems whereas chromium (III) compounds are relatively nontoxic. Chromium (VI) crosses the cell membrane and oxidizes cellular components in a process which leads to cellular damage, including interference with the genetic machinery. L-ascorbic acid is very widely known and used for its reducing properties (4-16). Among the reactions studied are those involving its efficient reduction of many transition metal ions and complexes by outer- and inner-sphere mechanisms (17-18). L-ascorbic acid is oxidized by the potentially carcinogenic chromium (VI) ion at the body's physiological pH of 7.40, as well as at lower pH. This carcinogenicity is thought to be related to the oxidation of various cellular constituents. Ascorbic acid being a constituent of the cell, and a good reductant, may therefore, functions as an antichromatic agent in vivo against chromate poisoning (10-19). Previous studies observed that various intercellular metabolites, including ascorbic acid, are capable of reducing chromate to pH 8.75. We have therefore extended the study to involve a wider range of pH and ascorbic acid concentration.

Experimental

L-ascorbic acid (BDH) of biochemical grade was used as supplied. All other chemicals were Analar grade used as obtained. The chromate solution was prepared and determined spectrophotometrically. Kinetic experiments were recorded on a Unicam Helios α-spectrophotometer equipped with a water-jacketed cell holder. The ionic strength I was kept constant at 0.1 M by using $NaNO_3$ solution.

Kinetic measurements

The reduction of chromium (VI) (3.3×10^{-3} M) by ascorbic acid (3.3×10^{-2} M) at basic medium (pH 8.2 to 9.3) was monitored by following the decrease of absorbance of chromium (VI) at 406 nm as a function of time. The pH was adjusted using KOH and measured on a radiometer M62 pH meter fitted with a combined glass-calomel electrode. Apparent second order rate constants for the reaction of chromium (VI) with ascorbate were determined from the slope of the linear plots of pseudo-first order rate constants, k_{obs}, versus ascorbate concentration.

Stoichiometry

The results of experiments performed to established the stoichiometry of the chromium (VI) oxidation of L-ascorbic acid at alkaline medium are shown in Figure 1. On the basis of the limiting value in absorbance at 406 nm, the break point occurs at molar ratio of Cr(VI) : L-ascorbic acid = 1.

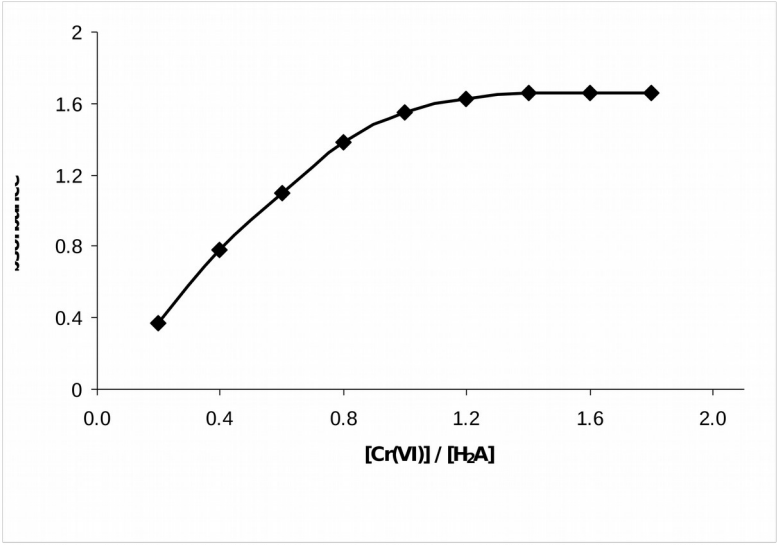

Fig. 1 Spectrophotometric redox reaction of L-ascorbic acid and chromium (VI).

Results and Discussion

The addition of ascorbate to a solution of chromium (VI) at pH ranged from 8.2 to 9.3 resulted in the formation of a green complex. During the reaction, the chromate peak at 406 nm gradually disappeared, while a peak at 550 nm indicates that chromium (III) species was formed. The spectrum of the green products was similar to that of a solution of hexaquochromium (III) in ascorbate under the same conditions. The green product slowly turned brown in air. This is due to the formation of products involving dehydroascorbic acid and chromium (III) (20).

The kinetics of the reaction were studied under pseudo-first order conditions with the concentration of L-ascorbic acid greatly exceeding that of the chromate solution. The conventional plots of 1n (A_t-A_∞) versus time were linear for the three half-lives or more (A_t and A_∞ are absorbancies at time t and infinity). The pseudo-first order rate constants were calculated from the slops of these plots and these are listed in Table 1. The constancy of k_{obs} at constant ionic strength, pH, temperature and ascorbate concentration is in agreement with a first-order dependence on the chromate concentration. The variation of k_{obs} with ascorbate concentration is linear passing through the origin which indicate a first-order dependence on its concentration (Figure 2), with slope (apparent second-order rate constant) equal 0.120 to 0.025 from pH ranged from 8.5 to 9.3 at 35°C (Table 2).

Table (1) Observed rate constant for reaction of L-ascorbic acid with chromium (VI) $[CrO_4^{2-}]$**= 3.3x10^{-3}** M , ionic strength I = 0.1 M.

Temp. (°C)	pH	$[H_2A]$ (M)	$10^4 k_{obs}$ (s^{-1})	Temp. (°C)	pH	$[H_2A]$ (M)	10^4 k_{obs} (s^{-1})
25	8.20	0.033	40	35	8.50	0.042	51
	8.40	0.033	25		8.80	0.042	30
	8.50	0.033	20		9.00	0.042	17
	8.80	0.033	10		9.30	0.042	10
	9.00	0.033	6		8.50	0.050	59
	9.25	0.033	4		8.80	0.050	36
30	8.20	0.033	64		9.00	0.050	22
	8.40[a]	0.033	40		9.30	0.050	12
	8.40	0.033	41		8.50	0.058	70
	8.40[b]	0.033	41		8.80	0.058	40
	8.50	0.033	30		9.00	0.058	26
	8.80	0.033	16		9.30	0.058	14
	9.00	0.033	10	40	8.40	0.033	99
35	8.20	0.033	84		8.50	0.033	74
	8.40	0.033	58		8.80	0.033	37
	8.50	0.033	42		9.00	0.033	25
	8.90	0.033	22				
	9.00	0.033	14				

(a) $[CrO_4^{2-}]$= 2.5x 10^{-3}M (b) $[CrO_4^{2-}]$=4.1x10^{-3}M

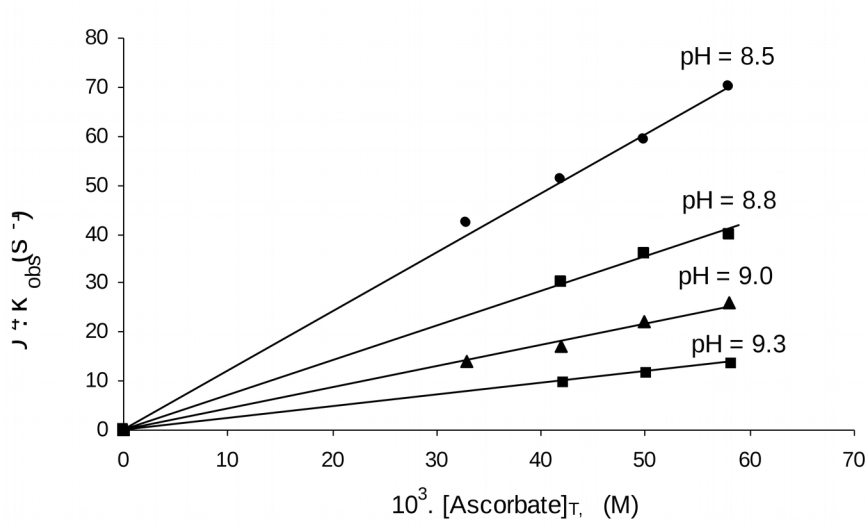

Fig. 2 Plots of k_{obs} versus $[ascorbate]_T$ for the oxidation of L-ascorbic acid by chromium (VI) at different pH values.

Table (2) Kinetic parameters obtained from plots of k_{obs} versus $[ascorbate]_T$ for the reaction between L-ascorbic acid and chromium (VI) at different pH values at 35°C.

pH	k_f ($M^{-1}s^{-1}$)	$10^{-3}k_1$($M^{-1}s^1$)
8.5	0.120	3.54
8.8	0.075	4.43
9.0	0.045	4.20
9.3	0.025	3.24
ΔH^* = 69 kJ mol^{-1}	ΔS^*= -209 JK^{-1} mol^{-1}	

Therefore the following expression:

$$k_{obs} = a\,[ascorbate] \qquad (1)$$

The dependence of the observed rate constant on the medium pH is presented in Figure 3, where straight lines passing through the origin were obtained when k_{obs} were plotted against $[H^+]$ at different temperatures. In the pH range studied, the species are the ascorbate monoanion (HA^-) and the ascorbate dianion (A^{--}). A possible mechanism, which would account for the observed kinetics involves the reaction of ascorbate with chromium (VI) to form a chromium-ascorbate ester intermediate which then undergoes a unimolecular redox reaction. Applying the steady-state approximation for the following equations:

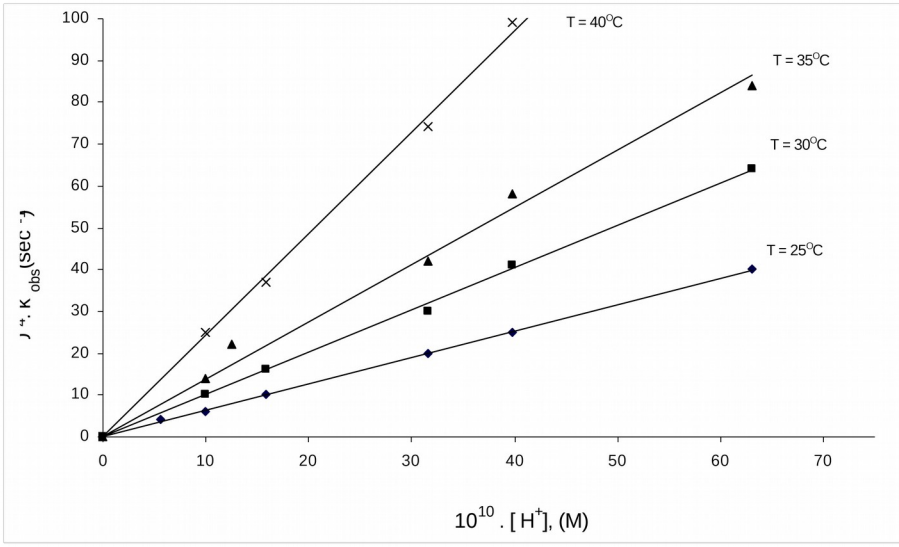

Fig. 3 Plots of kobs versus [H+] for the oxidation of L-ascorbic acid by chromium (VI) at different temperature [CrO_4^{2-}]$_T$ =3.3x10^{-3} M, [Ascorbate]$_T$ = 0.033M.

$$\text{Ascorbate} + \text{Cr (VI)} \underset{k_r}{\overset{k_f}{\rightleftharpoons}} \text{Cr (VI)- ascorbate}$$

$$\text{Cr (VI)-ascorbate} \xrightarrow{k} \text{products}$$

The rate law consistent with this mechanism is

$$-\frac{d[Cr(VI)]}{dt} = \frac{k_f.k}{k_r + k}[\text{ascorbate}][Cr(VI)]\ldots.. \qquad (2)$$

The "a" parameter of equation (1) corresponding to $\frac{k_f.k}{k_r + k}$. The estimates of the equilibrium constant K_e for the chromate –ascorbate ester formation obtained equal 740 dm^3 mol^{-1}(11). Therefore the value of k_r ($= \frac{k_f}{k_e} = \frac{0.12}{740} = 1.62x10^{-4}s^{-1}$) is small compared to k_f which means that k_r is insignificant ($k >> k_r$) and the equation (2) reduces to

$$-\frac{d[Cr(VI)]}{dt} = k_f\,[\text{ascorbate}][Cr(VI)] \ldots\ldots\ldots \qquad (3)$$

and the formation of chromium (VI) ascorbate-ester will be the rate determining step. Gould and Coworkers (6-9) involving the oxidation of L-ascorbic acid by chromium (IV) and (V) complexes highlights the rapid nature of the electron transfer reactions.

The rate of reduction of the chromium (V) 2- ethyl-2-hydroxybuttyrate chelate to the chromium (IV) species is of the order $2x10^2$ $dm^3mol^{-1}s^{-1}$, while the internal electron transfer from chromium (IV) to chromium (III) ascorbates chelate is $2x10^3s^{-1}$. These rate constants for the one-electron reductions are in accord with the assumption that the formation of the ester intermediate is the rate determining step.

The pH dependence of the rate constants suggested that CrO_4^{2-}, HA^- and A^{--} are the reactive species present. Therefore, the following reactions occurs.

$$CrO_4^{2-} + HA^- \xrightarrow{k_1} \text{products}$$

$$CrO_4^{2-} + HA^{--} \xrightarrow{k_2} \text{products}$$

and the rate law consistent with the above mechanism is

$$-\frac{d[Cr(VI)]}{dt} = \left\{ \frac{k_1[H^+][A]_T}{K_2 + [H^+]} + \frac{k_2 K_2 [A]_T}{K_2 + [H^+]} \right\} [Cr(VI)] \ldots\ldots\ldots \quad (4)$$

Where K_2 corresponds to the following equilibrium

$$HA^- \rightleftharpoons H^+ + A^{--} \quad pK_2 = 11.93 \quad (12)$$

The absence of intercept in Figure 3, suggests that the second term in the above equation $\left(\frac{k_2 K_2 [A]_T}{K_2 + [H^+]} \right)$ equal zero and the reaction of chromate with A^{--} is insignificant (in our pH range, the ratio of $\frac{HA^{--}}{A^{--}}$ is greater than 430), and the above equation reduces to:

$$-\frac{d[Cr(VI)]}{dt}=\frac{k_1[H^+][A]_T}{K_2+[H^+]}[Cr(VI)]\ldots\ldots\ldots\ldots \quad (5)$$

This fact was confirmed that the reaction is completely stopped at pH=11 where A^{--} is the major species of ascorbic acid. In a previous study of reduction of chromate by thiols, it was found that the positiviely charged thiols had the largest values of k_1, whereas the thiols containing two negative charges had the lowest values. The electrostatic interaction between the thiol and chromate would be expected to affect the rate. Therefore, the rate of reaction of Cr(VI) with cysteamine at pH 7.4 (25°C) which has +1 charge, equal 1.18 $M^{-1}s^{-1}$ (10) and equal to 0.05 $M^{-1}s^{-1}$ under the same experimental conditions for thiolactate (-1 charge) (10) which is in agreement with our experimental result (k_f = 0.12 $M^{-1}s^{-1}$ at pH = 8.2, 25°C).

Therefore, the CrO_4^{2-} and HA^- are the reactive species in the rate determining step producing the ester. It is likely that under our basic conditions the acid catalyzed reaction pathway was due to the attack of chromate by the protonated ascorbate and the hydroxide ion is the leaving group. The following mechanism is proposed under basic conditions:

$$HA^- + CrO_4^{2-} \rightarrow A^{-}\cdots\cdots CrO_3^{2-}$$

(H and O)

$$A^{-}\cdots\cdots CrO_3^{2-} \rightarrow A\text{—}CrO_3^{3-}$$

(H, O, OH)

$$A\text{—}CrO_3^{3-} \rightarrow ACrO_3^{2-} + CrO_3^{2-} + OH^-$$

(OH)

This intermediate which formed in a rate-determining step undergoes rapid electron transfer, via an inner-sphere mechanism, to produce chromium (III) and dehydroascorbate as final products. The magnitude of the entropy of activation suggests some degree of organization in the transition states. This is consistent with the associative nature of the mechanism. The high activation parameters obtained (Table 2) relative to that obtained by Banas (21) for the oxidative of ascorbic acid by chromic acid (ΔS=-69.5 JK^{-1} mol^{-1} and ΔH= 28.5 kJ mol^{-1}) confirms that the reaction is thermodynamically controlled which reflects the high rate constants obtained by

Banas than those obtained in our study. From all the above observations, the following mechanism can be suggested for the reaction.

$H_2A \rightleftharpoons HA^- + H+$ pKa = 4.03 (12)

$HA^- + Cr^{VI} \xrightarrow[\text{slow}]{k_1} HA - Cr^{VI}$

$HA^- + Cr^{VI} \longrightarrow Cr^{V} - HA^{\bullet}$

$Cr^{V} - HA^{\bullet} \longrightarrow Cr^{VI} + H^+ + A^{\backslash}$

$Cr^{IV} + HA^{\bullet} \longrightarrow Cr^{III} + H^+ + A^{\backslash}$

$H^+ + OH^- \longrightarrow H_2O$

Where $A^{\backslash}$ is hydroascorbic acid and from the above mechanism, and considering the HA^- is only the reactive species, the observed rate constant will be:

$$k_{obs} = \frac{k_1[H^+][A]_T}{K_a + [H^+]} \quad \ldots\ldots\ldots\ldots\ldots\ldots \quad (6)$$

Under our experimental conditions, $K_a >> [H^+]$, the above equation reduces to:

$$k_{obs} = \frac{K_1}{K_a}[H^+][A]_T \quad (7)$$

Which agrees with our results, where straight lines passing through the origin were obtained when k_{obs} is plotted versus $[H^+]$ as shown in Figure 3.

References

1.S.K. TANDON, "Biological and Environmental aspect of chromium", S. Langard, Elsevier Biomedical Press. Amsterdam, 1982; pp. 209-220.

2.R.B. HAYES, ibid, 221-247.

3.T. NORSETH, Environ. Health Perspect. 1981, **40**, 121-130.

4.D. MACARTNEY and A. MCAUELY, *Can. J Chem*., 1981, **59,** 1321.

5.D. MACARTNEY and N. SUTIN, *Inorg. Chem. Acta*, 1983**, 74,** 221.

6.M.J. AKHTAR and A. HAIM, *Inorg. Chem.*, 1988, **27,** 1608.

7.S.K. GHOSH and E.S GOULD, *Inorg. Chem.*, 1989, **28,** 1538.

8.S.K. GHOSH and E.S. GOULD, *Inorg. Chem.*, 1989, **28,** 1948.

9.S.K. GHOSH, R.N. BOSE AND E.S. GOULD, *Inorg. Chem.*, 1987, **26,** 2684**.**

10.P.H. CONNETT AND K.E. and WETTERHAHN, *J. Am. Chem . Soc.*, 1985, **107,** 4282.

11.D.A. DIXON, N.P. SADLER and T.P. DASGUPTA, *J. Chem. Soc. Dalton Trans;* 1993, 3489.

12.F.A. BECKFORD, T.P. DASGUPTA and G. STEDMAN, *J. Chem.. Soc. Dalton Trans.*, 1995, 2561.

13.K. ABDUR-RASHID, T.P. DASGUPTA and J. BURGESS, *J. Chem. Soc. Dalton Trans.*, 1995, 2561.

14.ALVIN A. HOLDER, TARA P. DASGUPTA and SONG CHONLIM. *Transition Met. Chem.* 1997, **22**, 135-140.

15.M. NASIRUDDIN KHAN, ANILA SARWAR, *Turk. J. Chem.* 2001, **25**, 433-440.

16.PATRICIA C. WILKINS, MICHAEL D. JOHNSON, Alvin A. Holder, and Debbie C. Crans. *Inorg. Chem.*, 2006, **45(4),** pp. 1471-1479.

17.R.F. JAMESON and N.J. BLACKBUM, *J. Inorg. Nucl. Chem.*, 1975, **37**, 809.

18.M.M. TAQUI-KHAN and R.S. SHUKLA, *Inorg. Chem. Acta*, 1988, **149,** 89.

19.P.H. CONNETT and K.E. WTTERHAHN, *J. Am. Chem Soc.*, 1986, **108,** 1842.

20.B.M. TALBENT and J.B. WARD, Ascorbic Acid: Chemistry, Metabolism and Uses,, *Am. Chem. Soc., Washington. 1982, Ch. 5, P. 102.*

21.B. BANAS, *Inorg. Chem.*, 1981, **53,** L13.