[COMMUNICATION NO. 1139 FROM THE KODAK RESEARCH LABORATORIES]

Oxidation Processes. XX.[1] The Mechanism of Autoxidation Reactions in the Presence of Foreign Catalysts and Inhibitors

BY JAMES E. LUVALLE AND A. WEISSBERGER

The autoxidation of enediols and hydroquinones, R, to diketones and quinones, T, proceeds by way of semiquinones,[2] S, as a two-step oxidation-reduction.[3] Inasmuch as S is formed from R and T, and rapidly oxidized, T catalyzes the over-all reaction. This mechanism implies (a) that the reactions are inhibited by removal of S, and, if catalyzed by T, by removal of T,[2b,4] and (b) that substances other than T catalyze the over-all reactions provided these substances react with R forming S.[5] Examples of such inhibition and catalysis by foreign substances were investigated in earlier papers of this series.[2,4,5]

The present paper applies the more recently advanced systematic treatment of two-step oxidations[6] to inhibition and catalysis by foreign substances. To this end reactions I, II, and III are added to the list of reactions discussed in the earlier paper,[6] which compose the over-all reaction, $R + O_2 \rightarrow T + O_2^=$.

$$R + C \rightleftharpoons R{\cdot}C \rightleftharpoons S{\cdot}S_c \rightleftharpoons S + S_c \qquad I$$
$$S + I \rightleftharpoons S{\cdot}I \rightleftharpoons R{\cdot}S_i \rightleftharpoons R + S_i \qquad II$$
$$T + I \rightleftharpoons T{\cdot}I \rightleftharpoons R{\cdot}T_i \rightleftharpoons R + T_i \qquad III$$

C is a catalyst and S_c is its monovalent reduction product, I is an inhibitor, and S_i and T_i are its monovalent and divalent oxidation products, respectively. The pairs of binary complexes in each reaction differ by their electronic distributions. Inasmuch as reactions of electron transfer take place instantaneously, the rate-limiting reactions are those of complex formation and/or decomposition. Likewise, electrolytic ionizations are considered as instantaneous reactions.

Catalysis.—Catalysis takes place if the oxidant reacts faster with the binary complex or with the semiquinone than with R, and if the formation of the complex and/or S is fast. The ionic equilibria can be of great importance; for instance, the catalysis of the autoxidation of *l*-ascorbic acid by divalent copper is quantitatively accounted for by the action of Cu^{++} upon the monovalent ion.[5] The kinetics of the catalysis differ, depending on whether the dicomplex is oxidized as a molecular compound or whether the two semiquinones undergo oxidation after decomposition of the complex. Michaelis suggested that a catalyst in oxidation-reduction reactions forms a complex with the substrate which favors the semiquinone in the equilibrium between R, S, and T. The catalysis by quinone is but a special case of the general type of catalysis where R is oxidized to S by a component which is reduced to S_c and regenerated by oxidation.

I. The binary complex undergoes autoxidation as a molecular compound

$$R + C \underset{k_{-1}}{\overset{k_1}{\rightleftharpoons}} S{\cdot}S_c \qquad (1)$$

$$S{\cdot}S_c + O_2 \underset{k_{-2}}{\overset{k_2}{\rightleftharpoons}} S{\cdot}S_c{\cdot}O_2 \qquad (2)$$

$$S{\cdot}S_c{\cdot}O_2 \xrightarrow{k_3} T + C + O_2^= \qquad (3)$$

A. The concentrations of the complexes $(S{\cdot}S_c)$ and $(S{\cdot}S{\cdot}O_2)$ are not negligible relative to C

$$(C) + (S{\cdot}S_c) + (S{\cdot}S_c{\cdot}O_2) = (C_t) \qquad (4a)$$

where (C_t) is the total concentration of the catalyst.

Rate laws:—Solution of the steady-state equations for $(S{\cdot}S_c)$ and $(S.S_c.O_2)$ gives the following rate laws:

1. All rate constants are of the same order of magnitude

$$\frac{-d(O_2)}{dt} = \frac{k_1k_2k_3(R)(C_t)(O_2)}{k_{-1}(k_{-2}+k_3)+k_1(k_{-2}+k_3)(R)+k_2k_3(O_2)+k_1k_3(R)(O_2)} \qquad (5)$$

2a. $k_3 \gg k_{-2}, k_{-1}, k_2(O_2), k_1(R)$; $k_2(O_2) > k_1(R) > k_{-1}$; $k_2(O_2) > k_{-2}$

$$-d(O_2)/dt = k_1(R)(C_t) \qquad (6)$$

In this case the rate of formation of the binary complex is rate-limiting. The rate is first-order with respect to (R) and (C_t), respectively, and independent of (O_2). Practically all of the catalyst is free.

2b. $k_3 \gg k_{-2}, k_{-1}, k_2(O_2), k_1(R)$; $k_1(R) > k_{-1} > k_2(O_2) > k_{-2}$

$$-d(O_2)/dt = k_2(C_t)(O_2) \qquad (7)$$

The rate of formation of the ternary complex is rate-limiting. The rate is first-order with respect to (C_t) and (O_2), respectively, and independent of (R). Practically all of the catalyst is present in the binary complex.

3. $k_2(O_2) \gg k_{-2}, k_3, k_{-1}, k_1(R)$; $k_1(R) > k_3$

$$-d(O_2)/dt = k_3(C_t) \qquad (8)$$

Rate of decomposition of ternary complex into final products is rate-limiting. The rate is first-

(1) Part XIX, LuValle and Weissberger, THIS JOURNAL, **69**, 1567 (1947).

(2) James and Weissberger, *ibid.*, **60**, 98 (1938); (b) James, Snell and Weissberger, *ibid.*, **60**, 2084 (1938); (c) Weissberger, LuValle and Thomas, *ibid.*, **65**, 1934 (1943).

(3) (a) Elma, *Rec. trav. chim.*, **50**, 807 (1931); (b) Friedheim and Michaelis, *J. Biol. Chem.*, **91**, 355 (1931); (c) Michaelis, *Ann. N. Y. Acad. Sci.*, **40**, 39 (1940).

(4) (a) Weissberger, Thomas and LuValle, THIS JOURNAL, **65**, 1489 (1943); (b) James and Weissberger, *ibid.*, **61**, 442 (1939).

(5) Weissberger and LuValle, *ibid.*, **66**, 700 (1944).

(6) LuValle and Weissberger, *ibid.*, **69**, 1567 (1947).

order with respect to (C_t) and independent of (R) and (O_2), respectively. Practically all of the catalyst is present in the ternary complex.

B. The concentrations of the complexes are negligible relative to the concentration of the free catalyst.

$$(C) = (C_t) \tag{4b}$$

and the rate laws become:

1. All rate constants are of the same order of magnitude:

$$\frac{-d(O_2)}{dt} = \frac{k_1k_2k_3(R)\ (C_t)\ (O_2)}{k_{-1}(k_{-2} + k_2) + k_2k_3(O_2)} \tag{9}$$

2a. $k_3 > k_2$; $k_{-1} > k_2(O_2)$

$$-d(O_2)/dt = K_1k_3(R)\ (C_t)(O_2) \tag{10}$$

The equilibrium (1) controls the rate, and the rate is first-order with respect to (R), (C_t) and (O_2), respectively.

2b. $k_3 > k_{-2}$; $k_3(O_2) > k_{-1}$; $k_1(R) \sim k_{-1}$

The rate is given by equation (6).

3a. $k_{-2} > k_3$; $k_{-1} > k_2(O_2)$, $k_1(R) \sim k_{-1}$, $k_3(O_2) \sim k_{-2}$

$$-d(O_2)/dt = K_1K_2k_3(R)(C_t)(O_2) \tag{11}$$

The equilibria (1) and (2) poise each other, thus controlling the rate. The rate is first-order with respect to (R), (C_t), and (O_2), respectively.

II. The two semiquinones undergo autoxidation after decomposition of the complex.

$$R + C \underset{k_{-12}}{\overset{k_{12}}{\rightleftarrows}} S_c + S \tag{12}$$

$$S + O_2 \xrightarrow{k_{13}} T + O_2^- \tag{13}$$

$$S_c + O_2 \xrightarrow{k_{14}} C + O_2^- \tag{14}$$

The complexes, R·C and S·S_c, have very short half-lives and their concentrations are negligible. The fate of O_2^- in reactions 12, 13 and 14 has been discussed fully in Part XVIII of this series.[6]

A. When the concentration of S_c is not negligible relative to C

$$(S_c) + (C) = (C_t) \tag{15a}$$

Solution of the steady-state equations gives the rate law

1. $k_{12} \gtrsim k_{-12}$; $k_{13}, k_{14} > k_{-12}$:

$$\frac{-d(O_2)}{dt} = \frac{k_{12}k_{14}(R)\ (C_t)\ (O_2)}{k_{12}(R) + k_{14}(O_2)} \tag{16}$$

2a. $k_{12} \gtrsim k_{-12}$; $k_{13}, k_{14} > k_{-12}$; $k_{14}(O_2) > k_{12}(R)$

$$-d(O_2)/dt = k_{12}(R)(C_t) \tag{17}$$

Rate of formation of the semiquinones is rate-limiting. The rate is first-order with respect to (R) and (C_t), respectively, and independent of (O_2).

2b. $k_{12} \gtrsim k_{-12}$; $k_{13}, k_{14} > k_{-12}$; $k_{12}(R) > k_{14}(O_2)$; $k_{13}(S) \gtrsim k_{14}(S_c)$

$$-d(O_2)/dt = k_{14}(C_t)(O_2) \tag{18}$$

The rate of autoxidation of S_c is rate-limiting. The rate is first-order with respect to (C_t) and (O_2), respectively, and independent of (R). Virtually all catalyst is present as (S_c).

If $k_{14}(S_c) > k_{13}(S)$, the rate during the first half of the reaction is given by equation (18) divided by two. After all of R is present as S, the rate is

$$-d(O_2)/dt = {}^1/_2k_{13}(S)(O_2) \tag{19}$$

If C can also react with S

$$C + S \longrightarrow S_c + T \tag{20}$$

then the rate is given by equation (18) divided by two.

B. When (S_c) is negligible relative to (C)

$$(C) = (C_t) \tag{15b}$$

and the rate law becomes

1. $k_{12} \gtrsim k_{-12}$; $k_{13}, k_{14} > k_{12}$

Rate is given by equation (17).

2. $k_{12} \gtrsim k_{-12}$; $k_{-12} > k_{13}, k_{14}$; $k_{13}(S) \sim k_{14}(S_c)$

$$\frac{-d(O_2)}{dt} = \frac{k_{12} + k_{14}k_{12}^{1/2}(R)^{1/2}(C)^{1/2}(O_2)}{2} \tag{21}$$

Equilibrium (12) controls the rate. The rate is first-order with respect to (O_2), and one-half order with respect to (R) and (C).

Reactions involving divalent reduction of the catalyst are neglected in these derivations because metal catalysts, such as cupric, manganous and ferric ion, would be precipitated in the metallic state, and quinonoid organic catalysts, such as quinones, phenosafranin, indophenol and methylene blue, form stable semiquinones. The two types of catalysis, A and B, will occur simultaneously if the rate of oxidation of the binary complex is similar to the rate of decomposition of the complex into fast-reacting S and S_c. Moreover, since reactions (1)–(3) and (11)–(13) are superimposed upon the system of reactions discussed in the earlier paper,[6] reactions involving identical molecular and ionic species poise each other. For instance, the catalytic equilibria, (1) and (12), poise with the autocatalytic equilibrium, $R+T \rightleftarrows 2S$.[6] The binary and ternary complexes may be of the type called complexes of greatest instability[7] by Marcelin, and transition-state complexes[8] by Eyring, *i.e.*, of fleeting existence and present in small amounts only, or they may accumulate to analytical concentrations. As S_c, S·S_c, or S·S_c·O_2 become more stable, the catalytic reaction becomes obviously less important. In the autoxidation of cysteine, the iron cysteine complex has been identified spectrophotometrically.[9]

If the equilibria $R \rightleftarrows S \rightleftarrows T$, and $C \rightleftarrows S_c$ are reversible, some conclusions can be drawn from the respective electrochemical oxidation-reduction potentials. Conant[10] and Dimroth[11] have shown that the rates of certain oxidations increase with

(7) Marcelin, *Ann. phys.*, **3**, 120, 185 (1915).

(8) Glasstone, Laidler and Eyring, "The Theory of Rate Processes," McGraw-Hill Book Co., New York, N. Y., 1941.

(9) Michaelis, *J. Biol. Chem.*, **84**, 777 (1929); (b) Michaelis and Barron, *ibid.*, **83**, 191 (1929); Elliot, *Biochem. J.*, **24**, 310 (1930).

(10) Conant, *Chem. Rev.*, **3**, 1 (1926); (b) Conant and Pratt, THIS JOURNAL, **48**, 3178 (1926).

(11) Dimroth, *Z. angew. Chem.*, **46**, 571 (1933).

the potentials of the oxidation–reduction systems of the oxidizing agents. Insofar as Conant and Dimroth's rule is valid, equivalent amounts of different catalysts will be the more effective, the higher their oxidation–reduction potential at the respective *p*H.

It was pointed out above that the free energy of activation of $R{\cdot}C \rightarrow S{\cdot}S_c$ is low. If the free energies of activation for the reaction of $S{\cdot}S_c$ with oxygen and for the decomposition of the ternary complex are also low, then the free energy of activation for formation of the binary complex governs the rate of reactions of type A. The same conclusion applies to reactions of type B, provided that the free energy of activation for the decomposition of the binary complex into S and S_c is low and the free energies of activation for the reaction of S and S_c with oxygen are also low. In reactions whose rate is first-order with respect to oxygen, the binary complex or the semiquinone, respectively, is in the steady state and the validity of Conant and Dimroth's rule implies that ($S{\cdot}S_c$) or (S), respectively, depend on the potential of the $C \rightleftarrows S_c$ system.

LaMer and Temple[12] found that the autoxidation of hydroquinone catalyzed by manganous ion in the *p*H range 5.3 to 6.3 is first-order with respect to the concentration of manganous ion and oxygen, and to the reciprocal of the hydrogen-ion concentration, respectively. The latter dependency indicates that the catalysis proceeds through the monovalent hydroquinone ion. The mechanism may be

$$Mn^{++} + RH^- \rightleftarrows Mn^+ + S$$

but the observation that the monovalent manganous ion exists only in complexes[13] makes the mechanism $Mn^{++} + RH^- \rightleftarrows Mn^+{\cdot}S$ more likely.

The autoxidation of ascorbic acid in the presence of cupric ion is first-order with respect to the concentration of monovalent ascorbate ion, copper and oxygen, respectively.[14]

The rate dependencies are thus consistent with equations (10) and (11), *i.e.*, the autoxidation proceeds through formation of the binary and ternary complexes. Both cupric and cuprous ions tend to form complexes in which more than one molecule is bound to the ion.[15] It has been assumed previously that cupric ion and *l*-ascorbic acid form a complex which disintegrates to give cuprous ion and ascorbate semiquinone.[14d,g] This mechanism, however, gives a rate law similar to that of equation (16), which does not conform with the experimental rate dependency upon (R) and (O_2).

Mapson[14i,j] has shown that halide ion in high concentration inhibits the copper-catalyzed autoxidation of ascorbic acid, while halide ion in low concentration promotes the oxidation by air and by hydrogen peroxide. He also found that a high halide ion concentration inhibits the autoxidation of cuprous ion by oxygen and that a low halide ion concentration accelerates the rate of reduction of cupric ion by ascorbic acid. The inhibition of the autoxidation at high halide ion concentrations is therefore attributed to the inhibited autoxidation of cuprous ion. No explanation is given for the promotion by halide ion. The latter might be attributed to a formation of a ternary reactive complex, $S{\cdot}S_c{\cdot}Cl^-$, while the inhibition at high halide ion concentrations is attributed to the formation of the complex $S{\cdot}S_c{\cdot}Cl_2^-$, the halide ion competing with the oxygen for the monohalide complex. The failure of halide ion in high concentration to inhibit the oxidation by hydrogen peroxide would then indicate a greater stability of $S{\cdot}S_c{\cdot}H_2O_2$ than $S{\cdot}S_c{\cdot}Cl_2^-$. The data of Mapson[14i,j] are thus explainable on the hypothesis that mechanism A applies to the cupric ion catalysis of the autoxidation of *l*-ascorbic acid.

In the oxidation of enediols, enolamines, enediamines and their vinylogs, the intermediate radicals are stabilized by resonance between equivalent structures. In other cases, the one-step oxidation products frequently dimerize, *e.g.*, in the oxidation of cysteine to cystine. The ferric or cupric ion catalysis of the autoxidation of cysteine is first-order with respect to catalyst, oxygen and substrate, respectively,[9a,b] and the complex of catalyst and substrate may be isolated in the absence of oxygen.[9c] According to Michaelis[16] the autoxidation of cysteine proceeds by mechanism A. Szent-Györgyi[17] has suggested ternary complex formation to explain heavy-metal catalysis in the oxidation of several phenols.

Inhibition of a reaction takes place if a compound: (1) decreases the concentration or reactivity of a critical intermediate, *Primary Inhibition;* (2) decreases the concentration or reactivity of a catalyst, *Secondary Inhibition.*

If the critical intermediate is a member of a reaction chain, inhibition is usually very efficient. Many examples of chain reactions have been found. However, the autoxidation of enediols and related compounds in aqueous neutral or alkaline solution does not necessitate the assumption of a chain mechanism. The inhibition of chain reactions is therefore not specifically discussed in the present paper.

If the inhibitor, I, is subject to oxidation by molecular oxygen, this autoxidation will be superimposed upon the inhibited autoxidation of the

(12) LaMer and Temple, *Proc. Natl. Acad. Sci.*, **15**, 191 (1929).

(13) Latimer, "Oxidation Potentials," Prentice-Hall Co., Inc., New York, N. Y., 1938, p. 219.

(14) (a) Barton, DeMeio and Klemperer, *J. Biol. Chem.*, **112**, 625 (1936); (b) Lyman, Schultze and King, *ibid.*, **118**, 757 (1937); (c) Borsook, Davenport, Jeffreys and Warner, *ibid.*, **117**, 237 (1937); (d) Dekker and Dickinson, THIS JOURNAL, **62**, 2165 (1940); (e) Hand and Greison, *ibid.*, **64**, 358 (1942); (f) Steinman and Dawson, *ibid.*, **64**, 1212 (1942); (g) Silverblatt, Robinson and King, *ibid.*, **65**, 137 (1943); (h) Peterson and Walton, *ibid.*, **65**, 1212 (1943); (i) Mapson, *Biochem. J.*, **35**, 1332 (1941); (j) Mapson, *ibid.*, **39**, 228 (1945).

(15) Latimer and Hildebrand, "Reference Book of Inorganic Chemistry," 2nd ed., Macmillan Co., New York, N. Y., 1940, pp. 105–111.

(16) Michaelis, in Green, "Currents in Biochemical Research," Interscience Publishers, Inc., New York, N. Y., 1946, chap. 14.

(17) Szent-Györgyi, *Z. physiol. Chem.*, **254**, 147 (1938).

substrate. If the rate of autoxidation of I is low relative to the rate of autoxidation of the substrate, the observed inhibited rate is practically the rate of the inhibited reaction. If the autoxidation of I to S_i is much faster than the autoxidation of the substrate to semiquinone, the observed rate is practically the rate of autoxidation of the inhibitor.

I. It is immaterial for the kinetics of primary inhibition whether the reaction of I with the critical intermediate is irreversible or reversible provided that (I) is considerably greater than (S).

A. Irreversible inhibition

$$R + O_2 \xrightarrow{k_{22}} S + O_2^- \quad (22)$$

$$I + S \xrightarrow{k_{23}} S_i \cdot R \quad (23)$$

$$O_2 + S_i \cdot R \underset{k_{-24}}{\overset{k_{24}}{\rightleftarrows}} O_2 \cdot S_i \cdot R \quad (24)$$

$$O_2 \cdot S_i \cdot R \xrightarrow{k_{25}} R + T_i + O_2^- \quad (25)$$

Solution of the steady-state equations gives the rate law

$$-d(O_2)/dt = k_{22}(R)(O_2) \quad (26)$$

i.e., the inhibited rate is independent of the inhibitor concentration.

B. Reversible inhibition. Equation (22) is followed by

$$I + S \underset{k_{-27}}{\overset{k_{27}}{\rightleftarrows}} S_i + R \quad (27)$$

$$S_i + O_2 \xrightarrow{k_{28}} T_i + O_2^- \quad (28)$$

Solution of the steady-state equations gives again the rate law of equation (26).

Cysteine and thioglycolic acid are strong primary inhibitors for the autoxidation of hydroquinones[4b] and of catechol.[18] The products of the inhibition are thiol derivatives,[19] formed in an irreversible reaction. Ascorbic acid is a strong inhibitor of the autoxidation of many developing agents.[4a] The rate in the early part of the reaction equals the rate of autoxidation of ascorbic acid, as shown in Fig. 1.

Branch and Joslyn[18] considered the inhibition of the autoxidation of catechol by cysteine and sulfite as evidence for a chain mechanism. However, the preceding discussion shows that inhibition alone is not necessarily proof of a chain mechanism. The data for the autoxidation of catechol in the absence[6] and in the presence of foreign catalysts and inhibitors can be explained by a semiquinone mechanism.

II. The kinetics of *secondary inhibition* differ, depending on whether a foreign catalyst or an autocatalyst is removed.

In the former case, the inhibitor either removes or weakens the catalyst so that in the extreme case the reaction proceeds as if neither catalyst nor inhibitor were present.

For the removal of an autocatalyst, the following cases will be considered: A. Reversible removal of T. 1. All rate constants of the same order of magnitude. Equation (22) followed by

$$S + O_2 \xrightarrow{k_{29}} T + O_2^- \quad (29)$$

$$R + T \xrightarrow{k_{30}} 2S \quad (30)$$

$$I + T \underset{k_{-31}}{\overset{k_{31}}{\rightleftarrows}} T_i + R \quad (31)$$

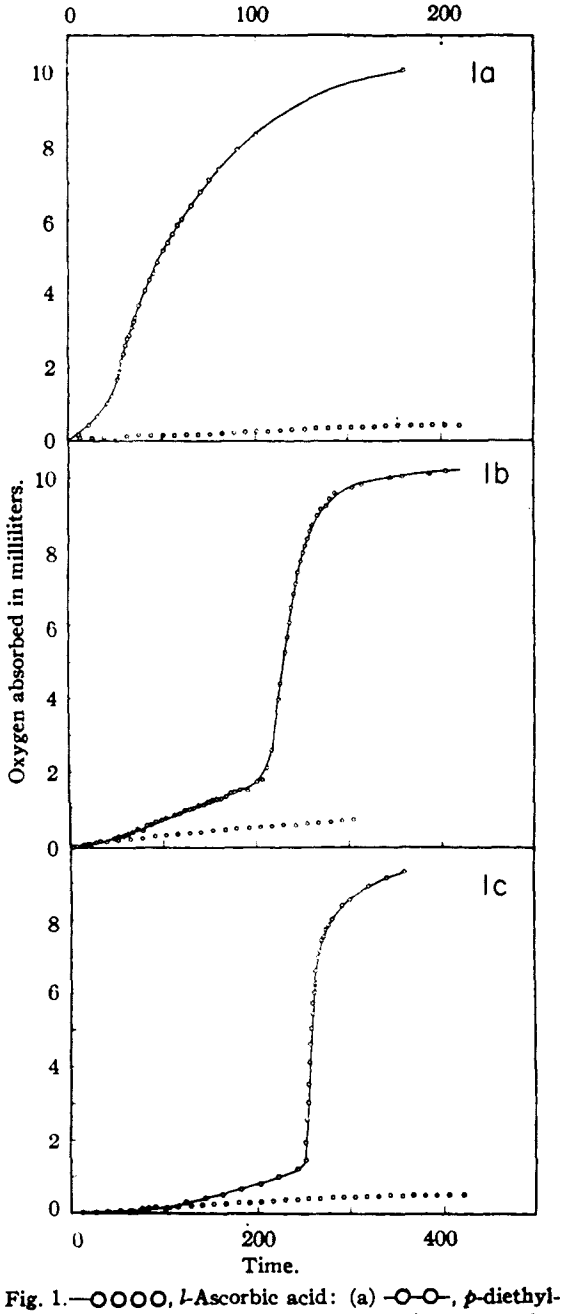

Fig. 1.—OOOO, *l*-Ascorbic acid: (a) -O-O-, *p*-diethylamino-*o*-toluidine and *l*-ascorbic acid; (b) -O-O-, elon and *l*-ascorbic acid; (c) -O-O-, hydroquinone and *l*-ascorbic acid. Time scales for 1b and 1c are identical.

(18) Branch and Joslyn, THIS JOURNAL, 57, 2388 (1935).

(19) Snell and Weissberger, *ibid.*, 61, 450 (1939); (b) Dimroth, Kraft and Aichinger, *Ann.*, 545, 124 (1940).

Solution of the steady-state equations gives the rate law

$$\frac{-d(O_2)}{dt} = {}^1/_2 k_{22}(R)(O_2) + \frac{k_{22}(R)(O_2)\{k_{21}(I) + k_{20}(R)\} + 2k_{20}k_{-21}(T_1)(R)^2}{2\{k_{21}(I) - k_{20}(R)\}} \quad (32)$$

2. $k_{21}(I) > k_{20}(R)$

$$\frac{-d(O_2)}{dt} = k_{22}(R)(O_2) + \frac{k_{20}(T_1)(R)^2}{K_{21}(I)} \quad (33)$$

When K_{21} is very large, the rate is given by the first term of equation (33).

B. Irreversible removal of T: 1. All rate constants of the same order of magnitude: equations (22), (29) and (30) followed by

$$T + I \xrightarrow{k_{24}} R - T_1 \quad (34)$$

Solution of the steady-state equations gives the rate law

$$\frac{-d(O_2)}{dt} = \frac{k_{22}k_{24}(R)(I)(O_2)}{k_{24}(I) - k_{20}(R)} \quad (35)$$

This is identical with the rate law of Class I-C of the preceding papers.[6]

2. When $k_{24}(I) > k_{20}(R)$, the rate law becomes identical with that of equation (26)

C. In many cases secondary inhibition is probably accompanied by primary inhibition. The discussion of the resulting mixed inhibition will be restricted to the case in which both S and T are removed irreversibly. Reactions (22) to (25), (29), (30) and (34) constitute the mechanism. If we assume that $k_{25} > k_{-24}$, reactions (24) and (25) may be combined

$$S_1{\cdot}R + O_2 \longrightarrow R + T_1 + O_2^- \quad (36)$$

We also assume that k_{-23} is negligible relative to k_{26}.

Solution of the steady-state equations gives the rate law. Rate constants not specifically mentioned in the following are considered to be of the same order of magnitude.

1. All rate constants are of the same order of magnitude

$$\frac{-d(O_2)}{dt} = {}^1/_2 k_{22}(R)(O_2) + \frac{k_{22}(R)(O_2)\{k_{24}(I) + k_{20}(R)\}\{k_{29}(O_2) + k_{23}(I)\}}{2k_{29}(O_2)\{k_{24}(I) - k_{20}(R)\} + 2k_{23}(I)\{k_{24}(I) + k_{20}(R)\}} \quad (37)$$

2. $k_{20}(R) > k_{24}(I)$

$$\frac{-d(O_2)}{dt} = {}^1/_2 k_{22}(R)(O_2) + \frac{k_{22}(R)(O_2)\{k_{29}(O_2) + k_{23}(I)\}}{2\{k_{23}(I) - k_{29}(O_2)\}} \quad (38)$$

3a. $k_{24}(I) \sim k_{20}(R)$; $k_{24}(I), k_{20}(R) > \{k_{24}(I) - k_{20}(R)\}$

$$\frac{-d(O_2)}{dt} = {}^1/_2 k_{22}(R)(O_2) + \frac{k_{22}(R)(O_2)\{k_{29}(O_2) + k_{23}(I)\}}{2k_{23}(I)} \quad (39)$$

3b. $k_{24}(I) \sim k_{20}(R)$; $k_{24}(I), k_{20}(R) > \{k_{24}(I) - k_{20}(R)\}$; $k_{29}(O_2) > k_{23}(I)$

$$\frac{-d(O_2)}{dt} = {}^1/_2 k_{22}(R)(O_2) + \frac{k_{22}k_{29}(R)(O_2)^2}{2k_{23}(I)} \quad (40)$$

4a-b. $k_{24}(I) > k_{20}(R)$ and/or $k_{23}(I) > k_{29}(O_2)$.

Rate is given by equation (26).

The rate laws for mixed inhibition are therefore complex unless the inhibitor concentration is so high that equation (26) gives the rate law, *i.e.*, that the rate becomes independent of the inhibitor concentration.

If the rate of reaction of I and T is so great that competing reactions are negligible, (T) may be obtained by the mass action law. Since equation (34) is irreversible

$$K'_{34} = (I)(T) \quad (41)$$

and the rate law is given by

$$-d(O_2)/dt = k_{22}(R)(O_2) + K'_{34}k_{20}(R)/(I) \quad (42)$$

i.e., the autocatalytic rate, given by the second term of equation (42), is first-order with respect to (R), and to 1/(I) and independent of (O_2).

Sulfite is less efficient as an inhibitor than cysteine or thioglycolic acid.[4b,19] Figures 2 and 3 were constructed from the recalculated data of

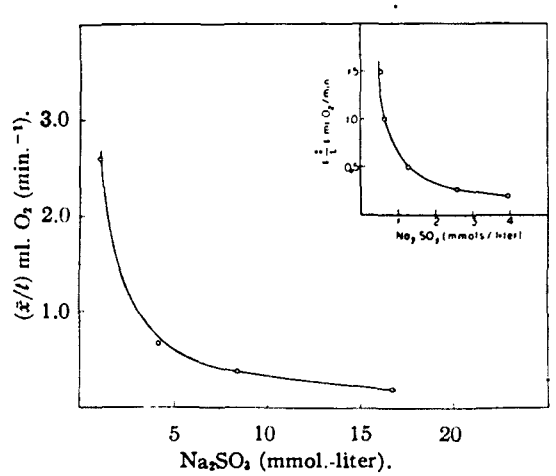

Fig. 2.—Rate against sulfite concentration. Main figure: potassium cyanide inhibits the autoxidation of the sulfite. Inset figure: Ethanol inhibits the autoxidation of the sulfite.

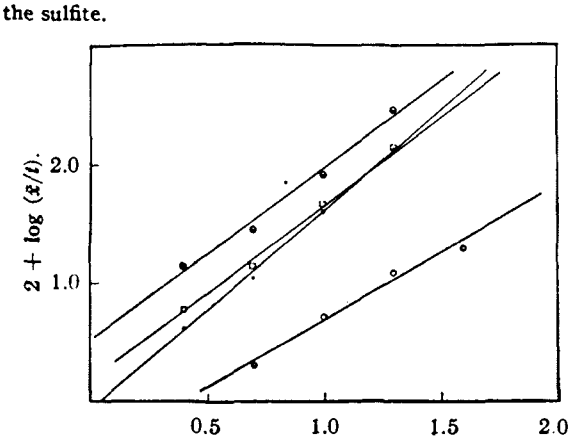

Fig. 3.—Air: -O-O-O-, *p*H 9.2, inhibitor is potassium cyanide. Oxygen: -□-□-□-, *p*H 8.9, inhibitor is potassium cyanide. -⊖-⊖-⊖-, *p*H 9.2, potassium cyanide is inhibitor. -●-●-●-, *p*H 9.0, ethanol is inhibitor.

James and Weissberger[4b] for the sulfite inhibition of the autoxidation of hydroquinone. In the absence of foreign substances the latter reacts according to Class I-E.[6] Figure 2 shows that the rate approaches independence of the sulfite concentration with increasing concentration of the inhibitor. Figure 3 shows that the dependence of the rate upon the concentration of hydroquinone is approximately first-order in air, and about three-halves order in oxygen. Equations (26) and (37) to (40) indicate that the rate for mixed inhibition becomes independent of the inhibitor concentration when the latter becomes sufficiently high. Equations (37) to (40) also indicate a rate dependency between first- and second-order with respect to hydroquinone and oxygen, respectively. Both dependencies conform with the experimental observations.[4b] The inverse proportionality of rate to inhibitor concentration reported by James and Weissberger[4b] follows from specific conditions, particularly from those leading to equation (40).

The autoxidation of an inhibitor is occasionally catalyzed by the substrate. The mechanism is similar to that outlined in the catalysis section of this paper where C is replaced by S or T, and R by I.

When *l*-ascorbic acid is added to solutions of N-methyl-*p*-aminophenol prior to the start of autoxidation, the reaction is inhibited, but if it is added after the start of the autoxidation, the reaction is accelerated.[4a] When *l*-ascorbic acid is added to solutions of pseudocumohydroquinone or 2,4-diaminophenol, the autoxidation is catalyzed when the ascorbic acid is added prior to or after the start of the reaction.[4a] When pseudocumoquinone is added to a solution of ascorbic acid after autoxidation has commenced, a very efficient catalysis of the autoxidation takes place.[4a]

These phenomena were explained[4a] with the rapid reduction of the respective quinonoid compound to the semiquinone by the ascorbic acid. If the reduction of the quinone by ascorbic acid is too slow, for instance, when duroquinone is added to an oxidizing solution of ascorbic acid, no catalysis is observed. On the other hand, if the reduction of the quinone to the hydroquinoid state is so fast that the reduction of the semiquinone prevails over its autoxidation, the rate behaves not as though the quinone had been added but as though ascorbic acid had been replaced by an equivalent amount of the hydroquinone. This is observed when benzoquinone is added to an autoxidizing solution of ascorbic acid. Let H_2A, S_A, and A represent *l*-ascorbic acid, its semiquinone, and dehydroascorbic acid, respectively. Similarly, let H_2Q, S_Q, and Q represent the given hydroquinone, its semiquinone, and the quinone, respectively

$$H_2A + Q \rightleftharpoons S_A + S_Q \quad (a)$$

$$S_A + S_Q \rightleftharpoons A + H_2Q \quad (b)$$

$$S_A + O_2 \longrightarrow A + O_2^- \quad (c)$$

$$S_Q + O_2 \longrightarrow Q + O_2^- \quad (d)$$

$k_a \gtrsim k_b > k_c \gtrsim k_d$: The quinone will be reduced to the hydroquinone, no catalysis will take place (benzoquinone and *l*-ascorbic acid).

$k_a > k_b$, $k_d \gtrsim k_c > k_b$: The reduction will stop at the semiquinone and the quinone will act as a catalyst (pseudocumohydroquinone and *l*-ascorbic acid).

$k_a \gtrsim k_b$, k_c, $k_d > k_b$: Inappreciable catalysis, slow reduction of the quinone (duroquinone and *l*-ascorbic acid).

"Induced oxidation" ("oxygen activation")[20,21] will be observed if the hydrogen peroxide, the perhydroxyl ion or semiquinone oxidizes a compound which has been added to the reaction mixture.

Summary

The rate laws of the oxidation of enediols, hydroquinones, enolamines and their vinyl analogs in the presence of foreign catalysts and inhibitors are derived and discussed.

(20) Jorissen, *Rec. trav. chim.*, **64**, 147 (1945).

(21) Jorissen and VanCalcar, *ibid.*, **64**, 284 (1945).

ROCHESTER, N. Y. RECEIVED MARCH 17, 1947

NOTES

On the Addition of Formic Acid to Dicyclopentadiene

BY FELIX BERGMANN AND HELENE JAPHE

Several examples, reported in the literature,[1,2] demonstrate the outstanding ability of one double bond in dicyclopentadiene (I) to add various reagents. Those additions, which proceed under the influence of strong electrophilic catalysts (*e. g.*, sulfuric acid, boron trifluoride, etc.), involve a molecular rearrangement,[3] which, according to Bartlett and Schneider,[4] follows the ionic mechanism of the Wagner–Meerwein reaction and leads to the "exo"-derivatives of dicyclopentadiene. Bruson, in a recent patent,[5] states that organic acids with "dissociation constants of about 1.5×10^{-3} or more" promote the addition reaction by their own acidity. We have now observed

(1) Alder and Stein, *Ann.*, **485**, 211 (1931).

(2) Bruson and Riener, THIS JOURNAL, **67**, 1178 (1945); **68**, 8 (1946).

(3) Bruson and Riener, *ibid.*, **67**, 723 (1945).

(4) Bartlett and Schneider, *ibid.*, **68**, 6 (1946).

(5) U. S. Patent 2,395,452; *C. A.*, **40**, 3188 (1946).