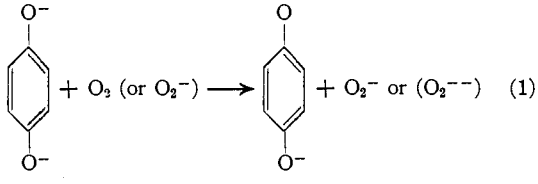
[COMMUNICATION NO. 924 FROM THE KODAK RESEARCH LABORATORIES]

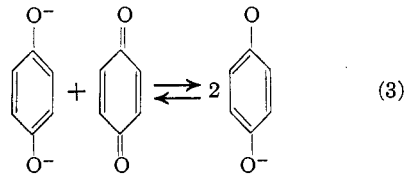
Oxidation Processes. XV.[1] The Effect of Reducing Agents on the Autoxidation of Some Photographic Developing Agents

BY A. WEISSBERGER, D. S. THOMAS, JR., AND J. E. LU VALLE

Hydroquinone and related compounds in alkaline solution form with molecular oxygen the corresponding quinones and hydrogen peroxide. This autoxidation proceeds in two steps: in the first, a divalent hydroquinone ion loses one electron and yields a semiquinone ion, (1); in the second, the semiquinone ion gives off an electron forming the quinone, (2). The semi-

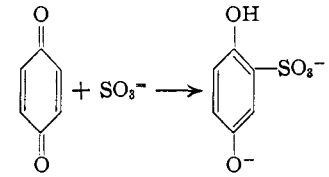


quinone ion is also formed by the reaction of hydroquinone ion with quinone (Eq.(3)). Therefore,



if the semiquinone ion is more reactive than the hydroquinone ion, the quinone will catalyze the autoxidation of a hydroquinone. This catalysis by the corresponding quinone was observed in the autoxidation of durohydroquinone and of ψ-cumohydroquinone.[2] With the durene derivatives, the acceleration is proportional to the quinone concentration. With the ψ-cumene derivatives, the acceleration is linear only at relatively low concentrations of the ψ-cumoquinone. At higher concentrations of the latter, the reaction rate approaches a constant value. This saturation of the quinone catalysis was explained by a reaction of the semiquinone ion with the quinone, which regenerates ψ-cumohydroquinone and ψ-cumoquinone.[3] No quinone catalysis was observed with xylohydroquinone, toluhydroquinone, and hydroquinone.[2c] However, the autoxidation of these compounds may only seem to be indifferent to the addition of quinone, because the catalysis reaches saturation at very low quinone concentrations.

Sulfite inhibits the autoxidation of hydroquinone and of other developing agents and the action of these compounds in photographic development.[4] A similar property was described for some organic thio compounds.[2b,c] Both sulfite[5] and the thio compounds[6] condense readily with quinone, reducing the carbonyl groups to hydroxyl



groups, and the inhibition by these compounds appears to be connected with this property, *viz.*, sulfite and the thio compounds in question may act as inhibitors because they lower the quinone concentrations beneath the saturation points of the respective catalysts. If this assumption is correct, the inhibitory action of these compounds belongs to that group of retardations in which a catalyst is eliminated.[7] It is at variance with another mechanism suggested to explain the retardation of autoxidations, *i. e.*, the breaking of reaction chains.[7]

If the elimination mechanism is correct, compounds which reduce quinones with sufficient speed will retard the autoxidation of developing agents. The action of such reducing compounds may be tested in several ways. (1) The reducing agent may be added to the developing agent and allowed to react with it, prior to the start of the autoxidation. An *inhibition* of the oxygen ab-

(1) Part XIV, Weissberger and Thomas, THIS JOURNAL, **64**, 1561 (1942).

(2) (a) James and Weissberger, *ibid.*, **60**, 98 (1938); (b) James and Weissberger, *ibid.*, **61**, 442 (1939); (c) James, Snell and Weissberger, *ibid.*, **60**, 2084 (1938); (d) Michaelis and Schubert, *Chem. Rev.*, **22**, 437 (1938).

(3) Kornfeld and Weissberger, *ibid.*, **61**, 360 (1939).

(4) Mees, "The Theory of the Photographic Process," The Macmillan Co., New York, N. Y., 1942, p. 382.

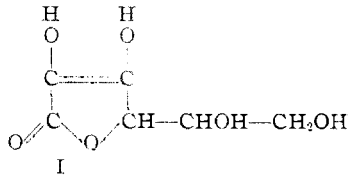
(5) Carstanjen, *J. prakt. Chem.*, **15**, 478 (1877).

(6) Snell and Weissberger, THIS JOURNAL, **61**, 450 (1939).

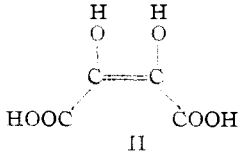
(7) Bailey, "Retardation of Chemical Reactions," Edward Arnold and Co., London, 1937.

sorption should be observed wherever a quinone catalysis occurs, provided that the inhibitor lowers the concentration of the quinone below the saturation point mentioned above, and that the rate of the oxygen absorption by the inhibitor is not greater than the retardation. (2) The reducing agent is added after the start of the autoxidation. An inhibition of the oxygen absorption should be observed if the reduction by the inhibitor is rapid enough to overtake the quinone formation and to lower the quinone concentration below the saturation value of the catalysis, provided the resultant retardation is greater than the combined oxygen absorptions of the inhibitor and of the reduction product of the quinone. In the presence of oxygen, the reduction of the quinone may not go all the way to the hydroquinone because the semiquinone, formed as an intermediate in the reduction, (5a), reacts rapidly with the oxygen according to (2). If this occurs, the reducing agent may cause an *acceleration*, *i. e.*, an oxygen absorption which is faster than that of the original and the regenerated developing agent together with the added reducing agent.

As inhibitors, ascorbic acid, I, and dihydroxymaleic acid, II, were chosen for most of the experiments.

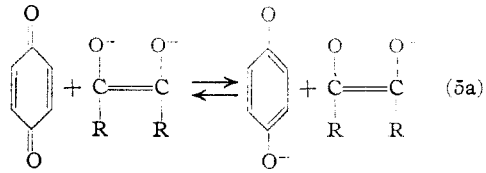


I

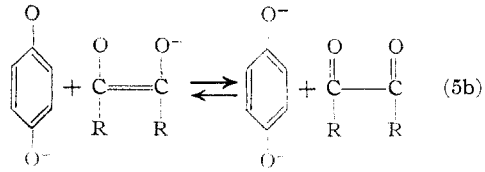


II

The reaction of these compounds with a quinone in alkaline solution may be written as



(5a)



(5b)

using arbitrarily the divalent ion of the inhibitor in (5a) and the charged radical in (5b). It is evident that the reactive semiquinone ion is formed according to (5a), while its formation according to (3) is suppressed. The semiquinone is eliminated according to (5b), which prevents the oxidation to the quinone according to (2).

Both inhibitors are reducing agents not only in alkaline, but also in neutral and acid, solutions. They can, therefore, when added to the solutions of the developing agents, react with easily reduced quinones which contaminate the latter, before the solutions are added to alkali. It is possible that the autoxidation of these reducing agents, like that of the developing agents, is subject to a catalysis by their oxidation products and to a retardation by reducing agents, *i. e.*, the developing agents. Furthermore, the reactive species of the inhibitors, as they react with the quinones, will be withdrawn from the reaction with the oxygen. Both factors will diminish the autoxidation rates of the inhibitors. To safeguard against errors caused by these effects, the oxygen absorptions by the inhibitors are, in general, neglected in the present paper. Only those cases are considered as retardations where the oxygen absorptions of the inhibitor-containing mixtures are slower than those of the corresponding solution of the developing agent alone.

Materials and Methods

Dihydroxymaleic acid was prepared according to Neuberg and Schwenk.[8]

Ascorbic Acid.—Eastman Kodak Co. *l*-ascorbic acid was used.

The **developing agents** were identical with those employed in the preceding paper.

Dioxane.—To 1000 g. of Eastman Kodak Co. dioxane was added 50 g. of finely ground ferrous sulfate and 50 ml. of water, and the mixture was stirred at room temperature for two hours. The dioxane was separated by decantation and dried with three successive 100-g. portions of potassium hydroxide pellets. After decantation, it was refluxed for six hours over 10 g. of sodium, cut into small pieces, and the dioxane distilled from 10 g. of fresh sodium. It showed no formation of benzidine blue in the benzidine–ferrous sulfate test.[9]

Water was redistilled in an all-Pyrex apparatus.

It was not thought necessary for the purpose of the present investigation to take other precautions against heavy metals.

The apparatus and the method were those described previously.[10] When the inhibitor was added before the

(8) Neuberg and Schwenk, *Biochem. Z.*, **71**, 112 (1915).
(9) Hess and Frahm, *Ber.*, **71**, 2627 (1938).
(10) Weissberger, Mainz and Strasser, *ibid.*, **62**, 1942 (1929).

start of the autoxidation, the slightly acidic solutions of the developing agent and of the inhibitor were mixed in the upper chamber of the two-chamber reaction vessel, and, after standing for twenty to forty minutes, were run into the buffer, contained in the lower chamber. In the experiments where the inhibitor was added after the start of the autoxidation, the three-chamber reaction vessel[2a] was used. The inhibitor was kept in the top chamber and added to the reacting mixture after a certain time. The curves (Figs. 2 to 6) interspersed with × represent these experiments. The inhibitor was added at the point marked by the first ×. The addition of quinone to autoxidizing solutions of ascorbic acid and of dihydroxymaleic acid was made in a similar way.

The volume of the reacting solutions was 50 ml., except in the case of the dioxane solutions which had a volume of 60 ml. The *p*H values were so chosen that the rates of the oxygen absorptions could be measured conveniently. They were determined before and after each run, and the values are stated under "*p*H range." The buffer solutions were 0.1 to 1.0 molar, depending on the buffer capacity required. Borate buffers were excluded because of their reactivity with hydroxy compounds. The temperature was kept at 20.00 ± 0.02°.

Results and Discussion

Ascorbic acid is rapidly oxidized by quinone to dihydroascorbic acid.[11] The fate of the quinone is shown by the following: when 0.2 g. of ascorbic acid was added to 0.1 g. of quinone in 50 ml. of phosphate buffer, *p*H 7.4, under an atmosphere of nitrogen, the quinone color disappeared instantly. The colorless solution was extracted with ethyl acetate, the solvent evaporated, and the crystalline residue sublimed in a high vacuum. The sublimate was identified as hydroquinone. Allowing for mechanical losses, the reaction appears to be almost quantitative. The residue from the sublimation consisted of a sticky brown mass, obviously the decomposition products of dihydroascorbic acid, which are rapidly formed at a *p*H above 5.[12]

As judged by the disappearance of the yellow quinone color, the reaction of ψ-cumoquinone with ascorbic acid is less rapid but still rather fast, the color disappearing in a few seconds at *p*H 8. Solutions of *p*-aminophenols and of *p*-phenylenediamine, like those of hydroquinone, do not darken as long as ascorbic or dihydroxymaleic acid is present. This demonstrates the reduction of the primary oxidation products of these developing agents by the inhibitors.

Dihydroxymaleic and ascorbic acids react with oxygen at different rates; the *p*H dependencies of these rates are also different; hence, differences in their reactivity with other oxidants, like the quinones, may be expected. The autoxidation rates at several *p*H values are indicated in Table I, by the times necessary for 0.5 mmole of either acid to absorb 3 ml. (0.125 mmole) of oxygen.

TABLE I

AUTOXIDATION OF ASCORBIC ACID AND DIHYDROXYMALEIC ACID (0.5 MMOLE)

*p*H	Time in minutes to absorb 3 ml. (0.125 mmole) O_2: Ascorbic acid	Dihydroxymaleic acid
10.85	..	2.1
10.68	0.5	..
7.73	59	..
7.70	..	33

To examine the possibility of an acceleration of the type mentioned above, which involves a highly reactive semiquinone, several quinones were added to autoxidizing solutions of ascorbic acid. The results are shown in Fig. 1. Addition of ψ-cumoquinone produces a considerable acceleration of the oxygen absorption. An instantaneous reduction of ψ-cumoquinone to the hydroquinone and subsequent autoxidation of the latter would have resulted in the oxygen absorption indicated by the dotted line. It is obvious that this mechanism does not account for the observations, while they are explained by the mechanism involving the semiquinone ion, which was suggested above. When benzoquinone is added to ascorbic acid, and the disturbance caused by the manipulation of the apparatus has passed, the autoxidation proceeds as if an amount of the quinone equivalent to the ascorbic acid added had been replaced by hydroquinone. In view of the rapid reaction

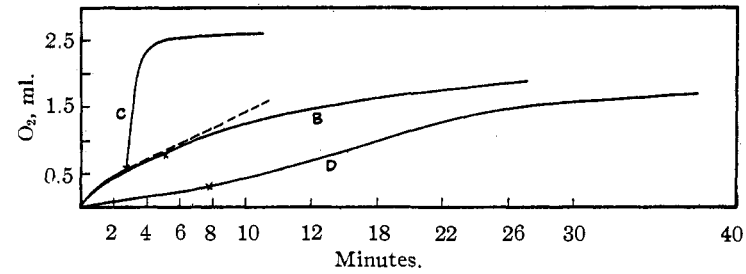


Fig. 1.—Ascorbic acid, 0.1 mmole; B, benzoquinone, 0.1 mmole, phosphate buffer, *p*H 8.05, solvent water; C, ψ-cumoquinone, 0.2 mmole, phosphate buffer, *p*H 8.28, solvent 20% ethanol; D, duroquinone, 0.2 mmole, glycine buffer, *p*H 9.11, solvent 50% dioxane; the quinones are added at the points marked ×.

(11) J. Parrod, *Bull. soc. chim.*, **5**, 938 (1938).

(12) Ball, *J. prakt. Chem.*, **118**, 219 (1937); Engelhardt and Bukin, *Chem. Abs.*, **33**, 2187 (1939).

between benzoquinone and ascorbic acid, the mechanism involving an instantaneous reduction of the quinone to hydroquinone and autoxidation of the latter agrees well with these observations.

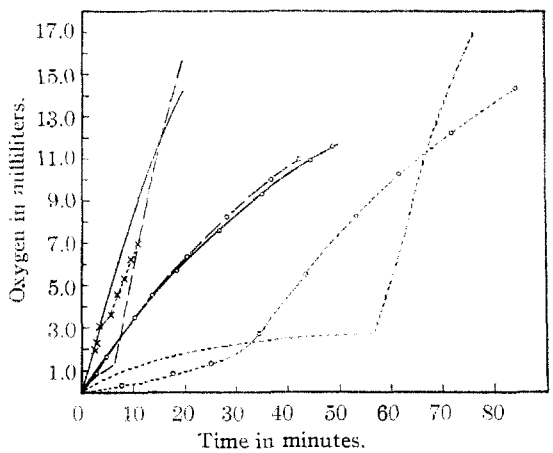


Fig. 2.—Catechol, carbonate buffer, *p*H range 9.38–9.29: catechol alone, ———; with ascorbic acid added before start, – – – – –; with ascorbic acid added after start, -x-·-x-·-x-; with dihydroxymaleic acid added before start, — — — —. Hydroquinone, pyrophosphate buffer, *p*H range 8.17–8.11: hydroquinone alone, -o-o-o-; with ascorbic acid added before start, - -o- - -o- - -o- -; with dihydroxymaleic acid added before start, - o - — — o - .

The experiments with the *developing agents* are presented in Figs. 2 to 6. They include, besides aromatic dihydroxy compounds, hydroxyamino and diamino compounds of suitable structure.[4,13] The developing agents were used in amounts of 0.5 mmole, ascorbic acid and dihydroxymaleic acid in amounts of 0.1 mmole. Thus, in each experiment, the total amount of the inhibitor is equivalent to only one-fifth of the developing agent. If the inhibitor is added before the start of the autoxidation, the amount of the quinone to be eliminated in order to cause an initial inhibition is, of course, small. In the absence of an inhibitor, the primary oxidation products accumulate only in rare cases as they are formed, *e. g.*, with durohydroquinone.[2a] In general, these products undergo disproportionations and polymerizations and a very rapid reaction with the hydrogen peroxide formed in the autoxidation. During the course of the reaction, the inhibitor will, therefore, only have to take care of the remaining quinonoid oxidation products in order to cause a retardation.

When ascorbic acid is added to *catechol*, *p-aminophenol*, and *N-methyl-p-aminophenol (metol)*

(13) Kenner, *Nature*, **147**, 482 (1941).

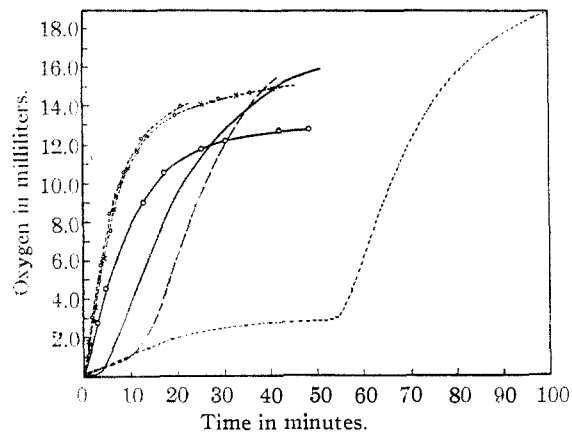


Fig. 3.—*p*-Aminophenol, carbonate buffer, *p*H range 9.33–9.23: *p*-aminophenol alone, ———; with ascorbic acid added before start, – – – – –; with dihydroxymaleic acid added before start, — — — —. 2,4-Diaminophenol, phthalate buffer, *p*H range 5.59–5.52: 2,4-diaminophenol alone, -o-o-o-; with ascorbic acid added before start, - -o- - -o- -, with ascorbic acid added after start, -o-x-o-.

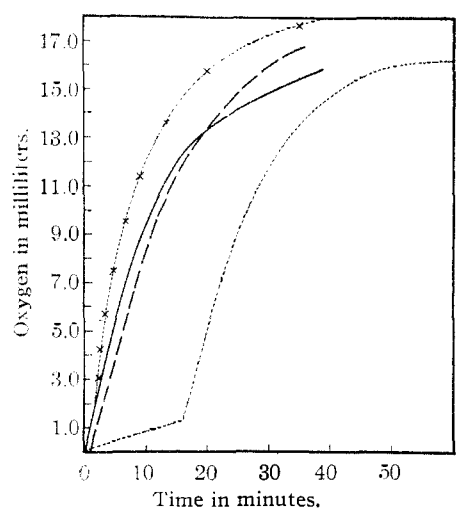


Fig. 4.—N-Methyl-*p*-aminophenol, phosphate buffer, *p*H range 7.62–7.46: N-methyl-*p*-aminophenol alone, ———; with ascorbic acid added before start, – – – –; with ascorbic acid added after start, -x-x-x-; with dihydroxymaleic acid added before start, — — — .

prior to the start of the autoxidation, the oxygen absorption is slowed down to a rate which is similar to the autoxidation rate of the inhibitor alone under the same conditions (Figs. 2 to 4). After an amount of oxygen has been absorbed which is equivalent to the inhibitor present, the reaction rate rapidly becomes identical with that of the developing agent alone. With hydroquinone, Fig. 2, the observations are similar but the curve does not show a sharp break. Both inhibitor and developing agent appear to be oxi-

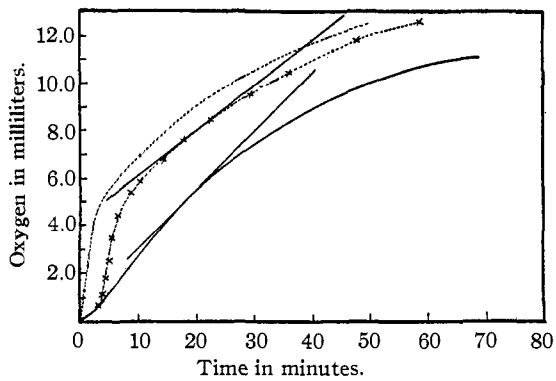


Fig. 5.—ψ-Cumohydroquinone in 20% dioxane, phosphate buffer, *p*H range 8.06–8.11: ψ-cumohydroquinone alone, ————; with ascorbic acid added before start, - - - -; with ascorbic acid added after start, -x-x-x-.

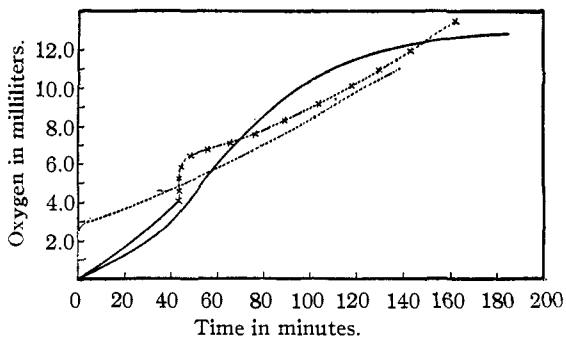


Fig. 6.—*p*-Phenylenediamine, carbonate buffer, *p*H range 9.00–8.75: *p*-phenylenediamine alone, ————; with ascorbic acid added before start, - - - -; with ascorbic acid added after start, -x-x-x-.

dized simultaneously during a transition period. The effect of dihydroxymaleic acid with the first three developing agents is similar to that of ascorbic acid, but the period of inhibition is much shorter and the amount of oxygen absorbed during this period is only about half that with ascorbic acid. Both of these effects are understood if we assume that before the dihydroxymaleic acid is exhausted, the developing agents are oxidized in sufficient amounts to raise the quinone concentrations beyond the saturation points of the quinone catalyses. It is in agreement with the suggestion that, in the later stages of the reactions, the solutions containing dihydroxymaleic acid and developing agents absorb oxygen faster than the developing agents alone, as shown by the crossing of the respective curves. The relative magnitude of the retardation is determined obviously by the rates of the various reactions involved. The lower reactivity of the dihydroxymaleic acid produces a milder effect than that observed with ascorbic acid. With hydroquinone, the weaker inhibitor produces practically no deviation from the blank.

When the inhibitor is added *after the start of the autoxidation*, the chances for a retardation are smaller than when it is added before the start. In the former type of experiments the inhibitor must overtake the formation of quinonoid oxidation products already in progress, in order to cause a retardation. Furthermore, the more of the quinone there is present, the better are the conditions for an acceleration of the type discussed above, or for an apparent increase of the reaction rate by the regeneration of the developing agent. Added after the start of the reaction, dihydroxymaleic acid causes no distinct deviation of the curves with *catechol*, *hydroquinone*, *p-aminophenol*, and *N-methyl-p-aminophenol*, while ascorbic acid, with the first three developing agents, produces a definite inhibition, though a much smaller one than that noted above. The curve is shown for catechol only. In the case of N-methyl-*p*-aminophenol, Fig. 4, an acceleration is observed when ascorbic acid is added after the start of the run.

The effects of both ascorbic acid and dihydroxymaleic acid upon *p-phenylenediamine* are very similar, and curves are shown for ascorbic acid only, Fig. 5. Both inhibitors are oxidized much more rapidly than the *p*-phenylenediamine. The oxygen-time curve for the developing agent alone exhibits an induction period. When the inhibitors are present before the start of the run, the induction period is intensified. When the inhibitors are added after the start of the runs, the autoxidations go through another induction period after the oxidation of the inhibitor is completed. The difference in rate in the later stages of the reactions is caused by the lowering of the *p*H through the oxidation products of the inhibitors.

When ascorbic acid is added to 2,4-diaminophenol (amidol), Fig. 3, either before or after the start of the autoxidation, an acceleration occurs. Dihydroxymaleic acid is not suited to these experiments, because it decomposes with evolution of carbon dioxide at a low *p*H.

With *ψ-cumohydroquinone*, ascorbic acid, when added before the start or during the course of the run, causes a strong acceleration of the oxygen absorption. The maximum velocity is, as should be expected, somewhat higher in the latter case.

After the ascorbic acid is exhausted, the absorption rate is for some time smaller than at corresponding points of the blank test, as is shown by the tangents marked at the curves. Dihydroxymaleic acid shows no effect upon the autoxidation of ψ-cumohydroquinone.

The mechanism of the acceleration observed with ψ-cumohydroquinone, 2,4-diaminophenol, and N-methyl-4-aminophenol was explained above. The question remains to be answered why the acceleration occurs particularly with these compounds. In all three cases, the stability of the quinone is relatively high, either by the nature of the quinone (ψ-cumoquinone), or by the *p*H conditions of the reactions. Furthermore, the reaction of the added reducing agent with the quinone is slower than in the other experiments. This is suggested for ψ-cumoquinone and ascorbic acid by the rate of decoloration mentioned above, and it follows for the other two cases from the *p*H dependence of the reactivity of the reducing agents (Table I) and the low *p*H of the reaction mixtures. Most likely, both factors, the small loss of quinone in reactions other than reduction by the added reducing agent, and the appropriate rate of the latter reaction, combine to create the conditions under which enough semiquinone is formed to cause an acceleration of the oxygen absorption.

The experiments with the developing agents are summarized in Table II.

TABLE II

EFFECT OF REDUCING COMPOUNDS ON THE AUTOXIDATION OF DEVELOPING AGENTS

Developing agent	Dihydroxymaleic acid added before start of autoxidation	Dihydroxymaleic acid added after start of autoxidation	Ascorbic acid added before start of autoxidation	Ascorbic acid added after start of autoxidation
Hydroquinone	0	0	– –	–
Catechol	–	0	– –	–
ψ-Cumohydroquinone	0	0	+	+
4-Aminophenol	–	0	– –	–
4-N-Methylaminophenol (Metol)	–	0	– –	+
2,4-Diaminophenol (Amidol)			+	+
p-Phenylenediamine	–	–	–	–

– indicates a retardation, – – a very strong inhibition, + an acceleration, 0 no noticeable effect.

The observations agree with the assumption that a quinone catalysis via the reactions (3) and (2) takes place in the autoxidation of all the developing agents tested and that the retardations are caused by the reduction of the catalysts. This suggestion is supported further by an experiment in which stannous tartrate inhibited the autoxidation of hydroquinone at *p*H 8.4, and by another one in which hydrazine has a similar effect at *p*H 8.0. Most revealing are the experiments with catechol, *p*-aminophenol, N-methyl-*p*-amino-phenol and hydroquinone, where the autoxidation of the developing agent almost stops when ascorbic acid is added before the start of the run. The inhibitor, most likely, does react not only with the quinone, but also with the semiquinones (5b). An acceleration results if the formation of the semiquinone according to (5a) overcompensates the elimination of the semiquinone according to (5b) or via the quinone (3) and (5). The stability of the quinones and the relative rates of the various reactions involved determine the result. The marked differences depending upon whether the reducing agents are added before or after the start of the autoxidations, are readily understood with the suggested mechanisms. They would be less easily explained on the assumption that the inhibitors interfere with reaction chains. It may be noted that, in the present research, retardations caused by the elimination of a quinonoid catalyst (5a) appear to occur simultaneously with retardations in which an active intermediate (Moureu and Dufraisse)[7] is disposed of (5b).

Photographically, the results of the present investigation are interesting in two ways. They afford a consistent explanation of the action of the various stabilizers which are used to diminish the aerial oxidation of developers,[4] including stannous tartrate.[14] Moreover, the high activity of the semiquinones of developing agents, which is demonstrated by the observed retardations and accelerations, should have some bearing on the mechanism of the development process itself. The present results make it likely that the formation of semiquinones in reactions of the developing agents with their quinonoid oxidation products, *i. e.*, quinone catalyses, play a role in photographic development proper. The investigation, in photographic development, of systems similar to those studied in the present paper should therefore be of interest.

The mutual influence of oxidation-reduction systems on their reactivity, for which the present paper gives examples, appears also to be of physiological significance.[15]

(14) Désalme, *Bull. soc. franc. phot.*, [3] **8**, 192 (1921).
(15) Heard and Welch, *Biochem. J.*, **29**, 998 (1935).

Summary

The effect of ascorbic acid and dihydroxymaleic acid on the autoxidation of various organic developing agents is investigated. In two experiments, stannous ion and hydrazine are added to hydroquinone solutions.

A retardation is observed in most cases. This is explained on the assumption that the autoxidation of the developing agents is catalyzed by their quinonoid oxidation products, and that the added reducing agents eliminate these quinonoid products.

The quinones are assumed to catalyze the autoxidations by virtue of their reaction with the developing agents in which highly reactive semiquinones are formed. Most likely the latter are also reduced by the inhibitors.

In some cases, the reducing agents accelerate the autoxidation of the developing agents. The acceleration is caused by the formation of semiquinones in the reduction of the quinones. This interpretation is supported by experiments in which ψ-cumoquinone accelerates the autoxidation of ascorbic acid to an extent which cannot be explained by a reduction of the ψ-cumoquinone to the ψ-cumohydroquinone and autoxidation of the latter.

ROCHESTER, NEW YORK RECEIVED APRIL 12, 1943

[COMMUNICATION NO. 930 FROM THE EASTMAN KODAK LABORATORIES]

Investigation of Pyrazole Compounds. IV.[1] The Acylation of 3-Phenyl-5-pyrazolone and 3-Anilino-5-pyrazolone

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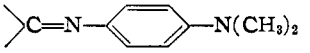
The acyl derivatives of 1-phenyl-3-amino-5-pyrazolone were described in the first paper of this series[2] where the constitution of the parent compound was established. The present paper deals with the acylation of 3-phenyl-5-pyrazolone,[3] and 3-anilino-5-pyrazolone.[1,4] The determination of the positions of the acyl groups was again assisted by color tests, in which the substance under investigation is heated with *p*-nitrosodimethylaniline,[1] or oxidized in the presence of *p*-aminodimethylaniline.[5]

A modification of the latter test, which uses the silver halide of a photographic film as the oxidizing agent, was most helpful. Originated by Fischer and Siegrist,[6] it has become the basis for several successful color photographic processes,[7] and is frequently used in color photographic research. The compound is dissolved in carbonate, an unsymmetrically dialkylated developing agent of the *p*-phenylenediamine class, *e. g.*, *p*-aminodimethylaniline, is added, and a strip of exposed photographic film is immersed in the solution. The *p*-phenylenediamine derivative reduces the silver halide of the emulsion and the compound under test, if it is of suitable constitution, couples with the oxidized developing agent to form a dye with a  group.[8] The film is washed, silver and excess silver halide are removed with a photographic reducer and thiosulfate, and a clear film is obtained. If a dye is formed, it gives information by its existence, color[6] and location, on the nature of the parent compound. The dye may be deposited throughout the emulsion layer of the film, at the emulsion surface only, or it may be found in the developer solution. If the molecular weight of the dye is low and the solubility in the alkaline developer high, the dye formed in the emulsion layer may bleed entirely or in part into the developing solution. On the other hand, coupling compounds which, because of their low rate of diffusion, do not penetrate into the gelatin, react only with that oxidized developing agent which diffuses out of the emulsion. These compounds form dyes on the surface of the film or as a precipitate in the liquid. In carrying out the film-strip test, a blank should be made to distinguish the dye formed as described above from colored oxidation and condensation products formed by the developing agent in the absence of couplers.

(1) Investigation of Pyrazole Compounds, III, THIS JOURNAL, **65**, 732 (1943).

(2) Weissberger and Porter, *ibid.*, **64**, 2133 (1942).

(3) Curtius, *J. prakt. Chem.*, **50**, 515 (1894).

(4) Worrall, THIS JOURNAL, **44**, 1551 (1922).

(5) Weissberger and Porter, *ibid.*, **65**, 52 (1943).

(6) Fischer and Siegrist, *Phot. Korr.*, **51**, 19 (1914).

(7) Davies, *Phot. J.*, **76**, 248 (1936); Forrest and Wing, *J. Soc. Mot. Pict. Eng.*, **29**, 248 (1937); Mees, *J. Frankl. Inst.*, **233**, 50 (1942).

(8) Mees, "The Theory of the Photographic Process," The Macmillan Co., New York, N. Y., 1942, p. 393.