INHIBITION OF THE AUTOXIDATION OF ORGANIC SUBSTANCES IN THE LIQUID PHASE[1]

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I. INTRODUCTION

Several reviews have appeared in the past few years dealing with the autoxidation of organic substances in the liquid phase, i.e., oxidation by molecular oxygen without any accompanying flame (12, 44, 120, 196, 276). The normal curve for oxygen uptake during autoxidation consists of an initial period where very little oxidation occurs, known as the induction period. This is followed by a rapid increase in the rate due to autocatalysis by chain-branching intermediates that build up during the induction period. The rate soon reaches a maximum value and then slowly starts to decrease. Autoxidation can be inhibited, i.e., prevented or retarded, by the addition of certain compounds known as antioxidants, which lengthen the induction period or lower the maximum rate of oxygen uptake. Two reviews on the inhibition of autoxidation have appeared recently (94, 285), but they have concentrated on a single aspect of this subject. It is the purpose of the present review to summarize all the methods by which autoxidations in the liquid phase can be inhibited and to discuss the mechanism of the reactions involved. To this end the pertinent literature has been reviewed through 1960, but no attempt has been made to list the thousands of compounds that have been patented as antioxidants.

The autoxidations that are here considered involve a free-radical chain process described by the following reactions:

Initiation:

$$RH \xrightarrow{\text{activation}} R\cdot + (H\cdot) \quad (1)$$

Propagation:

$$R\cdot + O_2 \rightarrow RO_2\cdot \quad (2)$$

$$RO_2\cdot + RH \rightarrow ROOH + R\cdot \quad (3)$$

Decomposition of peroxide:

$$ROOH \rightarrow RO\cdot + \cdot OH \quad (4)$$

$$2ROOH \rightarrow RO\cdot + RO_2\cdot + H_2O \quad (5)$$

$$\left.\begin{matrix} RO\cdot \\ RO_2\cdot \end{matrix}\right\} + ROOH \rightarrow \text{various products} \quad (6)$$

$$ROOH \rightarrow \text{nonradical products} \quad (7)$$

Induced decomposition of peroxide:

$$X + ROOH \rightarrow \text{free radicals} \quad (8)$$

$$Y + ROOH \rightarrow ROH + YO \quad (9)$$

$$Z + ROOH \rightarrow \text{inactive products} + Z \quad (10)$$

$$M + ROOH \rightarrow \text{free radicals} \quad (11)$$

Self-termination:

$$RO_2\cdot + RO_2\cdot \rightarrow \text{inactive products} \quad (12)$$

Chain-breaking termination:

$$RO_2\cdot + IH \rightarrow RO_2H + I\cdot \quad (13)$$

RH represents the organic substrate, $RO_2\cdot$ is the corresponding peroxy radical, and ROOH is the hydroperoxide. X, Y, and Z are three different types of decomposers of organic peroxides. M is a metal, IH is a chain-breaking inhibitor or inhibitor of free radicals, and I· is its stable, comparatively unreactive radical.

There is still some discussion as to whether the oxidative steps in the above scheme involve a direct hydrogen-abstraction process or whether they involve the prior transfer of an electron to the oxidizing agent followed by the transfer of a proton (179, 277). However, since the overall result is the same in either case, the elementary reactions outlined above are not affected.

Oxidation of organic substrates is generally followed by measuring rates of uptake of oxygen gas, but

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other methods, such as measuring the rates of formation of hydroperoxide or acidic products, both by chemical and infrared techniques, have also been successfully applied. The point of oxidative attack is related to the structure of RH. The most easily removed hydrogen is one that is alpha to a double bond or to a conjugated system, followed by a tertiary, secondary, and primary hydrogen in that order. Secondary products formed from the initial molecular product ROOH have been identified for a large number of different types of organic substrate. The addition of small amounts of oxygenated products, labeled with ^{14}C, to the oxidizing substrate has proved to be a particularly useful method of identifying these secondary reactions (31, 109, 196). A similar technique has been applied in tracing and inhibiting the sources of the carbonaceous deposits formed in internal-combustion engines (279, 281, 343).

Owing to the extremely large number of reactions that can, and generally do, occur in both inhibited and uninhibited autoxidations, kinetic treatments by the usual steady-state methods have been avoided throughout this review. In order to obtain tractable equations that can be correlated with the rates and activation energies of elementary reactions it is generally necessary to make rather sweeping assumptions and simplifications (212). Although the absolute rates and activation energies of several of the elementary reactions that occur in the uninhibited autoxidation of a number of substrates have been obtained (12, 44, 45, 77, 91, 92, 162, 165, 196, 302, 304), there is comparatively little information about absolute, as opposed to relative, rates or activation energies of elementary inhibiting reactions (165, 266). The reader interested in a specific aspect of inhibition is advised to refer back to the original papers, probably half of which give kinetic treatments that are more or less suitable for the conditions employed. A few more general kinetic treatments of inhibition have also appeared (89, 94, 145, 165, 329), but these also tend to be oversimplified.

One other problem that is raised by the complexity of the overall reaction is the difficulty of devising suitable screening tests for antioxidants in the laboratory. In practical applications many organic substances may be required to withstand oxidation under mild conditions for periods of months or years. It is therefore necessary to screen antioxidants by an accelerated test, which generally involves raising the temperature and/or the oxygen pressure, or adding an oxidation catalyst. However, none of these methods can accelerate all the individual reactions to an equal extent; therefore many of the early laboratory tests showed very poor correlation with service performance. More recent tests are somewhat better designed, but the only final test is to try an antioxidant under actual service conditions. Another danger of accelerated tests is that oxidation at very high rates may be controlled by diffusion, in which case the nature of the surface film can affect the rate (309). However, in most studies of autoxidation diffusion has not been a critical factor.

Since rates of autoxidation can be affected by a change in the rate of any of the individual reaction steps, these steps are considered separately and in detail below, with particular reference to methods of reducing the overall rate of autoxidation.

II. INDIVIDUAL REACTIONS OCCURRING DURING AUTOXIDATION

A. Initiation

Reaction 1 is a general expression for the initiation process. This reaction is promoted by free radicals (e.g., reaction 3); therefore compounds which can form free radicals readily, such as hydroperoxides, benzoyl peroxide, α,α'-azobisisobutyronitrile, etc., promote autoxidation. Compounds of this type are known as initiators. In the absence of initiators the energy required for the production of the radical R· may come from heat, light, or ionizing radiation. The latter two generally produce unimolecular decomposition of the substrate, e.g.. in the photoöxidation of aldehydes (77, 162):

$$\mathrm{RCHO} \xrightarrow{h\nu} \mathrm{RCO\cdot} + \mathrm{H\cdot} \qquad (14)$$

The deleterious effects of light can usually be simply overcome by its exclusion from the substrate. When this is not feasible the addition of light absorbers can retard oxidation. An example of this is the addition of carbon black to certain polymers. Alternatively, inhibitors of free radicals may be added, but the particular type used is important, since some of them promote photoöxidation (103).

Thermal initiation may involve the reaction

$$\mathrm{RH} + \mathrm{O_2} \rightarrow \mathrm{R\cdot} + \mathrm{HO_2\cdot} \qquad (15)$$

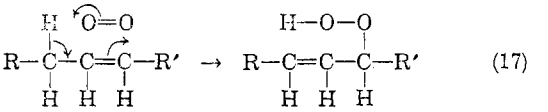
since this process requires considerably less energy (~30–45 kcal./mole (165)) than the direct thermal cracking process (70–100 kcal./mole). Initiation by reaction 15 is difficult to observe experimentally because of the higher endothermicity (~10 kcal./mole for a saturated hydrocarbon (165)) for this reaction than for reaction 4, which therefore becomes the predominant mode of initiation in the presence of even minute traces of hydroperoxide. There is also an increasing contribution to initiation from reaction 5 as the concentration of hydroperoxide rises (15, 16, 46). Therefore, although the rate of thermal initiation can be measured directly (272), it is generally more easily obtained by the addition of inhibitors of free radicals, which suppress secondary reactions (77, 91, 92, 162, 195). However, the activation energies obtained in this way are generally (77, 92, 162), though not always (165), appreciably lower than the estimated endother-

micity of reaction 15. Therefore, calculated rates of thermal initiation are many powers of ten lower than the experimental values obtained by the addition of inhibitors (92). This has led Denisov (92) to suggest that, since termolecular collisions are practically as frequent as bimolecular in the liquid phase, initiation might be a termolecular reaction.

$$RH + O_2 + HR \rightarrow R\cdot + H_2O_2 + R\cdot \qquad (16)$$

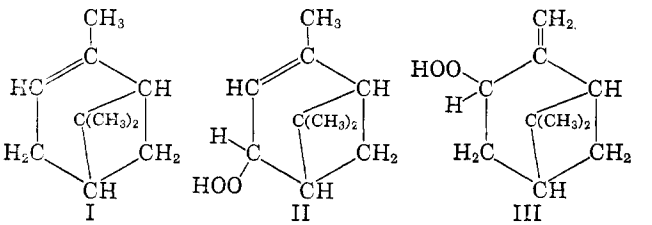
This reaction involves appreciably less energy than reaction 15 and therefore could become particularly important with compounds containing weak R—H bonds; e.g., when D(R—H) = 90 kcal./mole, ΔH_{15} = 43 and ΔH_{16} = 36 kcal./mole, and when D(R—H) = 80 kcal./mole, ΔH_{15} = 33 and ΔH_{16} = 16 kcal./mole (92). Although the steric factor will be very small, the lower endothermicity has given calculated thermal initiation rates in good agreement with experimental values (92). On the other hand, the kinetics most frequently observed favor reaction 15. That is, since the rate of autoxidation of an organic substance (V) is proportional to [RH][$RO_2\cdot$], i.e., to [RH] times the square root of the rate of initiation, reaction 15 gives $V \sim [RH]^{3/2}[O_2]^{1/2}$ and reaction 16 gives $V \sim [RH]^2[O_2]^{1/2}$. With few exceptions (131, 228) only the former kinetics have been observed (77, 162, 231, 272). An even greater objection to reaction 16 is given by the directly measured rate of thermal initiation of indene (272). The kinetics agree with reaction 15, the rate constant of which can be estimated by assuming a steric factor of 10^{-3}, a collision number of 10^{11}, and an activation energy equal to the endothermicity of the reaction (92). The R—H bond strength of indene will be less than that of toluene and will be assumed to be 72 kcal./mole, although by analogy with cyclopentadiene (121) it might be as much as 10 kcal./mole lower. D(H—O_2) is 47 kcal./mole and therefore $E_{15} = \Delta H_{15} = 72 - 47 = 25$ kcal./mole. Hence, $k_{15} = 10^{-3} \times 10^{11} \exp(-25{,}000/RT)$; i.e., at 50°C. $k_{15} = 1.2 \times 10^{-9}$ liter $mole^{-1}$ $sec.^{-1}$, in good agreement with the experimental value of 3.6×10^{-11} $mm.^{-1}$ $hr.^{-1}$, i.e., 1.7×10^{-9} liter $mole^{-1}$ $sec.^{-1}$, if it is assumed that the oxygen concentration in solution is 10 per cent of that in the gas phase. It seems, therefore, that even when D(R—H) is small, initiation still occurs predominantly by reaction 15. This suggests that the higher rates of initiation obtained with compounds having stronger R—H bonds (77, 91, 92, 162, 195) may be due to decomposition of hydroperoxide rather than to reaction 15 or 16, although, if this were the case, these rates should not show their observed dependence on oxygen pressure. The whole question of thermal initiation obviously requires further study.

It has also been suggested (115, 182) that thermal initiation with unsaturated substrates occurs by the direct addition of oxygen to the molecule without the prior formation of a free radical, e.g.,



(17)

The oxygen may perhaps also add directly to the double bond to give cyclic peroxides (12, 16, 155, 298), although it appears more likely that these are produced from hydroperoxides by isomerization (178). However, the bulk of the evidence suggests that the thermal initiation of oxidation of nonpolymerizable olefins involves the formation of a diradical or two free radicals (272). On the other hand, photosensitized oxidations almost exclusively produce hydroperoxides in which the double bond has migrated. For example (280), α-pinene (I) gives the hydroperoxide II on autoxidation but III on photosensitized oxidation. The latter almost certainly arises from a photoactivation by reaction 17.



In mixtures of hydrocarbons, such as petroleum oils, which are initially free from hydroperoxides it may be advantageous to remove the less stable components, i.e., those with weak carbon–hydrogen bonds. However, if thermal initiation contributes only slightly to the overall rate of initiation the less stable components may actually reduce the rate of oxidation of the mixture (see Section II,E).

B. Propagation

Reaction 2 is extremely rapid for nearly all hydrocarbon radicals, and therefore only peroxy radicals are of importance in chain propagation and termination except at very low partial pressures of oxygen or with very reactive organic substrates (12, 44).

In the absence of major steric effects the rate of reaction 3 depends on the resonance stabilization of the alkyl radical being formed (12, 44), i.e., on the carbon–hydrogen bond strength and on the availability of electrons at the carbon–hydrogen bond being broken (161, 271, 325). In any given substrate the rate can only be reduced by providing an alternate fate for the peroxy radical which will lower its steady-state concentration. This can be achieved by the addition of a more reactive compound such as an inhibitor of free radicals. Reaction 3 is basically similar to abstraction of

hydrogen by other free radicals, such as alkoxy radicals,

$$RO\cdot + RH \rightarrow ROH + R\cdot \quad (18)$$

and therefore the relative ease of oxidation of different substrates can be correlated with the relative rates of reaction 18 (54, 55).

Peroxy radicals can also abstract a hydrogen atom, particularly one in the β-position, by an intramolecular process (278) and can add to olefinic double bonds (187, 223, 224, 231, 272). As the oxidation proceeds, the peroxy radicals generally tend to react more with the oxygenated products than with the as yet unoxidized molecules, since the former frequently contain more reactive carbon–hydrogen bonds (109).

C. Decomposition of peroxides

The thermal decomposition of a hydroperoxide generally gives some free radicals. It therefore leads to chain branching and accounts for the autocatalysis observed in many oxidations. Although the rate of branching is proportional to the concentration of hydroperoxide (195) the overall decomposition is very complex, since at least three different reactions can occur simultaneously. Moreover, the solvent generally plays an important role in the decomposition of hydroperoxide (295) (see Section II,D), for which reason the rate of decomposition changes during an autoxidation as the substrate is progressively oxidized (316).

At low concentrations of hydroperoxide the initial rate of decomposition is generally first order in hydroperoxide, but at higher concentrations there are substantial deviations (114) and the apparent rate constant increases (26, 119), probably owing to the increasing importance of bimolecular and chain reactions (reactions 5 and 6). The dependence of the rate on concentration can often be expressed as the sum of the rates of a first-order and a higher-order process (119, 296, 312), the first-order term being generally taken to represent reaction 4 (see, however, Section II,D). The true rate constant of the first-order process can be obtained by extrapolating the measured rate constants to zero concentration of hydroperoxide.

The rate of decomposition of hydroperoxide is accelerated by initiators which provide an additional source of free radicals (99, 283) and is retarded to a reproducible minimum value by the addition of inhibitors of free radicals (165, 166, 302, 304), including polycyclic aromatic hydrocarbons (18). The residual minimum rate of reaction, i.e., the fully inhibited rate, is generally taken to represent reactions 4 and 5. The importance of reaction 5 relative to reaction 4 decreases with increasing temperature and decreasing concentration of hydroperoxide (304), a result which is consistent with the suggestion (15) that the former results from a hydroperoxide dimer.

$$R{-}O{-}\underset{\displaystyle H}{\overset{\displaystyle |}{O}}\cdots H{-}O{-}O{-}R \rightarrow RO\cdot + H_2O + RO_2\cdot \quad (19)$$

The rate of the gas-phase decomposition of many organic substances is also reduced to a certain minimum value by the addition of gaseous inhibitors. However, it has been shown recently (203, 339) that the fully inhibited reaction is not necessarily a molecular process, but may represent a residual chain reaction in which the inhibitor both starts and stops the reaction chains. In order that the fully inhibited rate be independent of the particular inhibitor two equilibrium conditions must be fulfilled, *viz.*:

$$ROOH + I\cdot \rightleftarrows RO_2\cdot + IH \quad (20)$$

$$IH \rightleftarrows I\cdot + \cdot H \quad (21)$$

The second equilibrium would seem to be very improbable under the low-temperature conditions of most liquid-phase decompositions of hydroperoxide. A completely ionic mechanism with ions instead of radicals in the equilibria and chain-carrying steps also seems improbable in organic solvents. Therefore, although the fully inhibited reaction gives rise to free radicals it must, itself, be a molecular process, a conclusion which is confirmed by the fact that oxygen has no effect on its rate (217).

In order to inhibit an autoxidation it is obviously desirable that the initial concentration of hydroperoxide be as small as possible, thus reducing the rates of reactions 4 and 5. Although the rate of the latter reaction can be reduced by the addition of compounds with strong electron-donor or electron-acceptor properties (15), the effect of such additives on reaction 4 is not known, since it is generally masked by an overall increase in the rate of decomposition of hydroperoxide due to the occurrence of an induced decomposition (12, 15, 310). For example, the rate of decomposition of *tert*-butyl hydroperoxide is much greater in alcohols, ketones, ethers, *o*-cresol, aniline, and unsaturated solvents than in aromatic solvents, cyclohexane, carbon tetrachloride, or chloroform (295). Therefore, with the possible exception of sulfoxides (see Section II,D), it is probably not possible to reduce the rate of reaction 4 by complexing the hydroperoxide with an additive, the reaction being, rather, accelerated and diverted to a new path. Whether this new reaction is beneficial or harmful will depend on whether or not it yields free radicals.

tert-Butyl hydroperoxide dissolved in inert solvents, such as chlorobenzene and carbon tetrachloride, gives a quantitative yield of *tert*-butyl alcohol and oxygen both by thermal decomposition above 100°C. (26) and by photochemical decomposition at room temperature (219). In hydrocarbon solvents the free radicals attack the solvent and the amount of oxygen evolved is decreased (219). The following chain reaction, with B

representing the *tert*-butyl group, was proposed to account for the products:

$$BOOH \rightarrow BO\cdot + \cdot OH \quad (22)$$

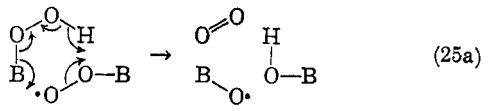
$$\left.\begin{matrix} BO\cdot \\ HO\cdot \end{matrix}\right\} + BOOH \rightarrow \left.\begin{matrix} BOH \\ H_2O \end{matrix}\right\} + BO_2\cdot \quad (23)$$

$$BO_2\cdot + BO_2\cdot \rightarrow 2BO\cdot + O_2 \quad (24)$$

An alternate reaction, which makes for a simpler chain, would be

$$BO_2\cdot + BOOH \rightarrow BO\cdot + BOH + O_2 \quad (25)$$

This reaction looks rather complicated, but a simple cyclic transition state is possible, the driving force for the reaction coming from the formation of molecular oxygen.



(25a)

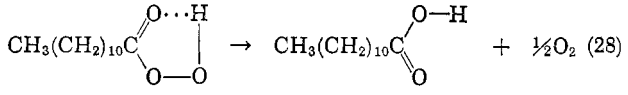
Thermal decomposition at lower temperatures gives the same products but may not involve an initial split into free radicals, since the rate is unaffected by the addition of an inhibitor of free radicals (240). The kinetics, in dodecane, were first order; therefore the following nonradical mechanism was proposed (240):

$$BOOH \rightarrow BOOH^* \quad (26)$$

$$BOOH^* + BOOH \rightarrow 2BOH + O_2 \quad (27)$$

The transition state of the second reaction would presumably be similar to that of reaction 25 above.

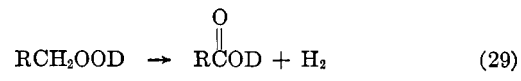
Certain other hydroperoxides can also apparently decompose without the production of free radicals. For example, perlauric acid in several solvents gives lauric acid and oxygen, by a first-order, nonradical, concerted decomposition, which is favored by the stereochemistry of the percarboxyl group (254):



(28)

On the other hand, perbenzoic acid decomposes to give free radicals in benzene solution, but not in alcoholic solution, presumably because the internally hydrogen-bonded, five-membered ring is not formed in the latter solvent (69a).

In contrast, the decomposition of pure primary hydroperoxides results in the evolution of hydrogen and formation of the corresponding acid and ester (341); the hydrogen comes from the peroxidic carbon atom.



(29)

(30)

The proposed reaction mechanism (105, 106) involves the initial formation of the aldehyde

$$RCH_2OOH \rightarrow RCHO + H_2O \quad (31)$$

followed by condensation of the aldehyde with further hydroperoxide to give an alkyl 1-hydroxyalkyl peroxide which decomposes via a cyclic transition state to give aldehyde, acid, and hydrogen (reaction 29) or ester and water (reaction 30). In spite of the fact that this reaction does not appear to involve free radicals (105, 106, 341), the addition of aldehydes to hydrocarbons generally catalyzes their oxidation, probably because any beneficial effects of the above reactions are more than outweighed by the free radicals produced from the direct oxidation of the aldehydes. Reaction 31 probably accounts for the inhibition of the oxidation of tetralin by strong dehydrating agents (163).

D. Induced decomposition of peroxides

The rate of decomposition of hydroperoxide can be accelerated by many different types of compounds. Inhibition results from reactions 9 and 10 and catalysis from reactions 8 and 11. The exact reactions involved with the many different types of decomposers of peroxides recorded in the literature (particularly in the patent literature (176)) is not always clear, but specific examples of all these reactions are known.

Reaction 8 involves induced decomposition by substances not containing a heavy metal, with the production of free radicals. It seems likely that alcohols (290), ketones and ethers (295), fatty acids (310), and olefins (321) fall into this class, e.g.:

$$ROOH + R'CH{=}CH_2 \rightarrow RO_2\cdot + R'\dot{C}HCH_3 \quad (32)$$

What is more surprising is that the rate of decomposition can be affected by different saturated hydrocarbon solvents (304) and even in these solvents the measured activation energy is generally (165, 302, 304), though not always (310), appreciably below the value obtained for comparable hydroperoxides in the gas phase (190). It has been suggested that the influence of saturated hydrocarbon solvents (SH) is due to a reaction similar to reaction 32 (304); i.e.,

$$ROOH + SH \rightarrow RO_2\cdot + S\cdot + H_2O \quad (33)$$

The evidence that this reaction serves as a major source of free radicals is rather conflicting. For example (195), by following the rate of consumption of α-naphthol, an inhibitor of free radicals that was added at different points during the autoxidation of *n*-decane, the rate constant for chain branching, i.e., for the production of radicals from the hydroperoxide products, was found to be 1.9×10^{-5} sec.$^{-1}$ at 130°C. Its temperature dependence was given by

$$k = 6 \times 10^8 \exp(-24{,}800/RT) \text{ sec.}^{-1}$$

This preëxponential factor is very low for a true unimolecular reaction, and the activation energy is much lower than the RO—OH bond strength (~ 35 kcal./mole). A rate constant at 130°C. of 3×10^{-5} sec.$^{-1}$ was later obtained by the same workers by following the rate of decomposition of the hydroperoxides, but the activation energy was not measured (217). The fairly close agreement between the two rate constants was taken to indicate that there was virtually no decomposition of hydroperoxide to nonradical products. When these same hydroperoxides are decomposed in chlorobenzene as a solvent (assumed to be inert) and in the presence of added hydrocarbon (R'H) the rate, measured by the consumption of α-naphthol, is given by k_1[ROOH] + k_2[R'H][ROOH] but does not contain a term involving [ROOH]^2 (218). The rate constant k_1 was 0.28×10^{-5} sec.$^{-1}$ at 130°C.; therefore this reaction is not nearly as important as the pseudo-unimolecular reaction at this temperature. k_2 increased as the R'—H bond strength decreased. In contrast to the foregoing (312), measurements at slightly higher temperatures of the rate of decomposition of decane hydroperoxides extrapolated to zero initial concentration (to eliminate interference from higher-order processes) gave

$$k = 10^{12} \exp(-31{,}700/RT) \text{ sec.}^{-1}$$

for the apparent unimolecular decomposition (i.e., $k = 0.7 \times 10^{-5}$ at 130°C.). Both the preëxponential factor and the activation energy suggest a true unimolecular process involving rupture of the RO—OH bond, and both are in good agreement with rate constants obtained in the same way by subsequent workers for the unimolecular decomposition of saturated hydroperoxides in saturated solvents (165, 302, 304). In conclusion it seems probable that both reactions can occur, and that the low-activation-energy, pseudo-unimolecular process will tend to predominate at lower temperatures and with substrates containing weak carbon–hydrogen bonds. The plots of activation energy should show some curvature, although this has not been reported. It also seems likely that even the high-temperature rate constants in saturated hydrocarbons contain a contribution from reaction 33 which may well account for the measured activation energies being in the range 30–32 kcal./mole, instead of the expected 35 kcal. or more.

Increasing the surface-to-volume ratio of the reaction vessel or the addition of powdered solids may increase (128), decrease (297), or have no effect (15) on the oxidation rate. An increase in rate implies that reaction 8 occurs on the surface, whereas a decrease implies that the surface promotes either reaction 10 or the destruction of radicals.

Reaction 9 involves a stoichiometric process such as that first suggested for certain sulfur (86, 87) and selenium compounds (88), in which the hydroperoxide is converted to the corresponding alcohol.

$$\text{ROOH} + \text{R'SR''} \rightarrow \text{ROH} + \text{R'S(O)R''} \quad (34)$$

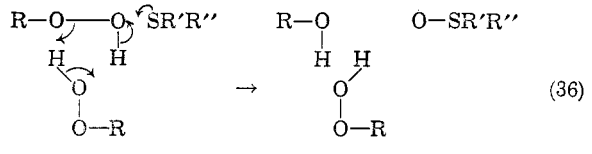
$$\text{ROOH} + \text{R'S(O)R''} \rightarrow \text{ROH} + \text{R'S(O)}_2\text{R''} \quad (35)$$

Monosulfides which contain at least one aliphatic or cycloaliphatic group attached to the sulfur atom are more effective antioxidants than mercaptans and disulfides, while diaryl sulfides and sulfones are inactive (87). The most active sulfides contain one *tert*-butyl group attached to the sulfur and a second group which should be isopropyl, *tert*-butyl, —CH(CH$_3$)CH=CHR, or —CH$_2$CH$_2$COR (12a). The structural requirements for active disulfides are not as stringent (12a).

Although many sulfides are fairly stable toward oxygen, both sulfoxides and sulfones are autoxidized rapidly; among their products are strong acids, which are probably sulfonic acids. These acids, which will almost always be produced when sulfides are used as antioxidants, are able to decompose further peroxide by a catalytic ionic rearrangement process (reaction 50) (157, 184).

The presence of a sulfur-containing compound in a system subject to autoxidation is a not unmixed blessing, since it may promote the development of resinous compounds and sludges (86, 118, 246, 335). Some of the disadvantages of sulfides can be overcome by the use of selenides (88), which result in fewer deleterious end-products and which are more effective decomposers of peroxide, dicetyl selenide being, for example, more than ten times as active as dicetyl sulfide. The selenides are converted to selenoxides by reaction with hydroperoxides, but the selenoxides appear to decompose to give about 50 per cent of the original selenide back again instead of being converted to the hexavalent state. This regeneration of the original decomposers of peroxide probably accounts for their powerful inhibiting action.

The decomposition of peroxides by sulfides is more complex than is suggested by reaction 34. The reaction of saturated sulfides with cyclohexenyl or *tert*-butyl hydroperoxide is bimolecular in hydroperoxide in hydrocarbon solvents and unimolecular in alcoholic solvents (14). The two mechanisms are depicted as follows:



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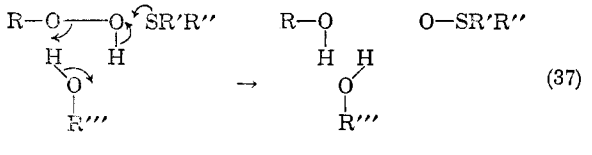


(37)

and provide a very satisfactory path for the clean abstraction of an oxygen atom from the middle of the formally linear hydroperoxide. Although this simple picture applies under a variety of conditions to the secondary hydroperoxide (14, 144), several alternate reactions can occur with *tert*-butyl hydroperoxide (BOOH). For example, the reaction may involve a two-stage process with a hydroperoxide–solvent complex as the oxidant (14). A chain reaction which appears to come into play in the presence of oxygen can also occur with saturated sulfides.

$$BOO\cdot + R'R''S \rightarrow BO\cdot + R'R''SO \quad (38)$$

$$BO\cdot + BOOH \rightarrow BOH + BOO\cdot \quad (39)$$

The chain tends to be suppressed in unsaturated solvents or by unsaturated sulfides (144). However, the sulfoxides produced from unsaturated sulfides in reaction 34, or by their direct autoxidation (13), may react further with hydroperoxide and sulfide to give a disulfide, water, and an unidentified peroxide (144). The disulfide is itself an inhibitor of free radicals (144) and may perhaps also be the source of the strong acids responsible for the catalytic decomposition of hydroperoxides.

Both mono- and disulfides owe a large part of their antioxidant activity toward olefins to their immediate oxidation products, i.e., sulfoxides and thiosulfinates (8, 12a), since these products show an immediate effect whereas, with the parent compounds, a small amount of oxygen must be absorbed before there is appreciable activity. Sulfoxides form complexes with hydroperoxides and can inhibit a partially oxidized substrate, but their inhibiting activity is destroyed by the simultaneous addition of an acidic substance such as stearic acid (12a). The activity of sulfur compounds cannot be wholly accounted for by their peroxide-decomposing action and although they suppress peroxide-initiated autoxidation, they do not suppress oxidations initiated by azobisisobutyronitrile (12a). It would appear, therefore, that the sulfoxide–hydroperoxide complex retards decomposition of the hydroperoxide to free radicals. However, active sulfoxides decompose very readily, e.g.,

$$2BS(O)B \rightarrow 2(CH_3)_2C{=}CH_2 + BS(O)SB + H_2O \quad (40)$$

and it is therefore more practical to use the parent sulfides, since they provide a "reservoir" for the supply of the active ingredient.

Kennerly and Patterson (180) have tested a variety of sulfur-containing compounds as decomposers of peroxides for preoxidized mineral oil and for cumene hydroperoxide in mineral oil. The reaction showed first-order dependence on peroxide, which is not surprising for the preoxidized oil, where products of the decomposition of peroxides, such as alcohols, can function as hydrogen donors (i.e., reaction 37), but it is rather surprising for cumene hydroperoxide in view of the results quoted above. However, it is quite possible that a change of mechanism has occurred, since the first-order kinetics were observed at 150°C. where the concentration of hydroperoxide dimer would be small, whereas bimolecular kinetics were observed at 50°C. (14). The rates of decomposition of cumene hydroperoxide at 150°C. in (liter) (mole)$^{-1}$ (min.)$^{-1}$ obtained with the following diphenyl sulfides were, for the unsubstituted sulfide, 0.6; for the 4-hydroxy sulfide, 6.0; for the 4,4′-dihydroxy sulfide, 60; and for the 4,4′-dimethoxy sulfide, 0.00. High yields of phenol were obtained, which suggested the occurrence of an ionic rearrangement process of the type catalyzed by sulfonic acids (reaction 50, see below). It was concluded that the sulfides were only precursors of the active decomposers, but since no obvious process exists for the conversion of the phenol sulfides to sulfonic acids which is compatible with the dependence of activity on structure given above, it was concluded that a mercaptyl radical or a phenoxy sulfide radical is the active species.

$$C_6H_5C(CH_3)_2OOH + \cdot SR \rightarrow C_6H_5OC(CH_3)_2O^+H + {}^-SR \rightarrow C_6H_5O\cdot + CH_3COCH_3 + HSR \quad (41)$$

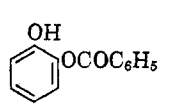
$$C_6H_5O\cdot + HSR \rightarrow C_6H_5OH + \cdot SR \quad (42)$$

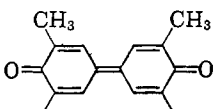
However, in view of recent work on the decomposition of peroxides by substituted phenols (95, 322), this mechanism seems open to serious question. The differing activities of the diphenyl sulfides may have been due to the presence or absence of the phenolic group, a point which could be cleared up by a comparison of 4,4′-thiobis(2,6-di-*tert*-butylphenol) and 4,4′-thiobis-(2,6-dimethylphenol) as decomposers of peroxides. The rates with these two sulfides should be similar if reaction 34 or an electron-transfer process occurs, but should be very different if the phenolic group is involved, since the highly hindered hydroxyl group in the former compound will be very unreactive (166, 322). The high yields of phenol may perhaps have been produced by the acid-catalyzed reaction of cumyl alcohol, the expected initial product, with unreacted hydroperoxide (186). In conclusion, sulfur-containing antioxidants react with hydroperoxides by a molecular process at low temperatures but perhaps by an ionic process at higher temperatures. The antioxidants are themselves oxidized to acids, which promote a concurrent catalytic decomposition.

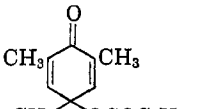
Although the reactions of phenols and amines with hydroperoxides have received little attention, their reactions with benzoyl peroxide have been studied very thoroughly in recent years. The conclusions derived from these studies are probably generally applicable to their reactions with hydroperoxides.

The decomposition of benzoyl peroxide in inert solvents occurs primarily by unimolecular fission of the O—O bond; the benzoyloxy radicals produced may

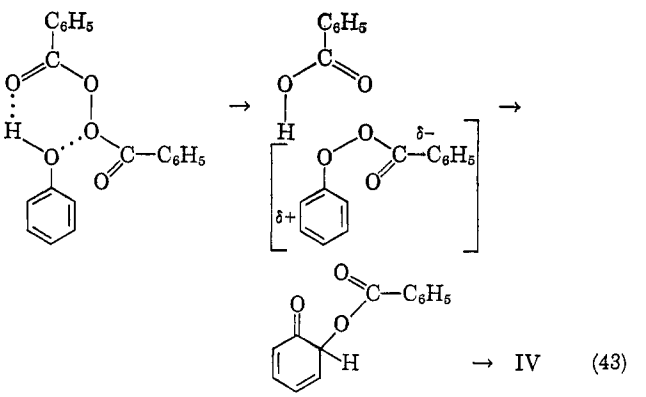
react with the solvent and initiate a chain decomposition of the peroxide. Since this peroxide disappears very rapidly from solution in liquid phenol, it was at first assumed that a chain reaction occurred (10, 306), although it had also been suggested that a fast nonradical reaction between peroxide and phenol might be involved (320). The products of the reaction have been identified in many cases (78). Phenols with free ortho positions gave chiefly catechol monobenzoates (IV), 2,6-dimethylphenol gave 3,3,3′,3′-tetramethyldiphenoquinone (V), and 2,4,6-trimethylphenol gave 4-benzoyloxy-2,4,6-trimethylcyclohexa-2,5-dienone (VI).


IV


V

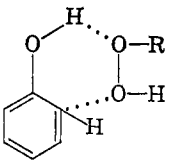

VI

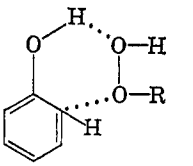
In every case most of the benzoyl peroxide residues not attached to the aromatic nuclei are recovered as benzoic acid. Analogous products have also been obtained with acetyl peroxide (332). The products were interpreted (78) as resulting from radical coupling and disproportionation, possibly while the radicals are still in the same solvent cage (17, 19). More recently, however, a simple bimolecular "four-center" mechanism, which does not involve free radicals, has been proposed (322) and confirmed by means of ^{18}O tracer experiments (95):


(43)

The reaction shows neither acid nor base catalysis, but the rate varies significantly with the solvent, being slow in strongly hydrogen-bonding media. It is accelerated by electron-supplying groups on the phenol, although there is no simple Hammett $\rho\sigma$ relation (140), and is retarded by bulky ortho substituents (322). Measurements with deuterated phenols give $k_H/k_D = 1.32 \pm 0.03$ in several systems (322).

The molecular reaction of phenols with hydroperoxides has received very little attention. When hydroperoxides have been studied the active species has generally been the peroxy radical (33, 51, 62), the only exception being a brief note on the rate of reaction of *tert*-butyl hydroperoxide with *p*-methoxyphenol (322). However, there is no reason to doubt that a mechanism similar to reaction 43 is generally operative. Phenols with free ortho positions might give either catechol (VII) or a catechol monoether (VIII).


VII


VIII

In either case reaction with more hydroperoxide would occur, since the activity of the product will be enhanced by the addition of an electron-donating OH or OR group to the original phenol. In this way each phenol molecule can decompose two or more molecules of hydroperoxide.

The products and kinetics of the reaction of benzoyl peroxide with primary, secondary, and tertiary amines have been studied extensively (319). The benzoyl peroxide undergoes an initial nucleophilic attack by the amine to give an ion-pair, the subsequent reactions of which are fairly complicated and may give rise to free radicals. With a secondary aliphatic amine the ions transfer a proton to give benzoic acid and a stable *N*,*N*-dialkyl-*O*-benzoylhydroxylamine (95), but with a secondary arylamine or alkarylamine the latter compound rearranges further to give a hydroxybenzanilide (95). The same sort of reactions are probably involved with primary amines, which give a complex mixture of products (319). Neither primary nor secondary arylamines show any deuterium isotope effect, and only a very small percentage of radicals are formed from the ion-pairs (234). On the other hand, an appreciable fraction of the ion-pairs produced by tertiary amines decompose to give free radicals, although quaternary imines (323) or enamines (58) may be formed concurrently.

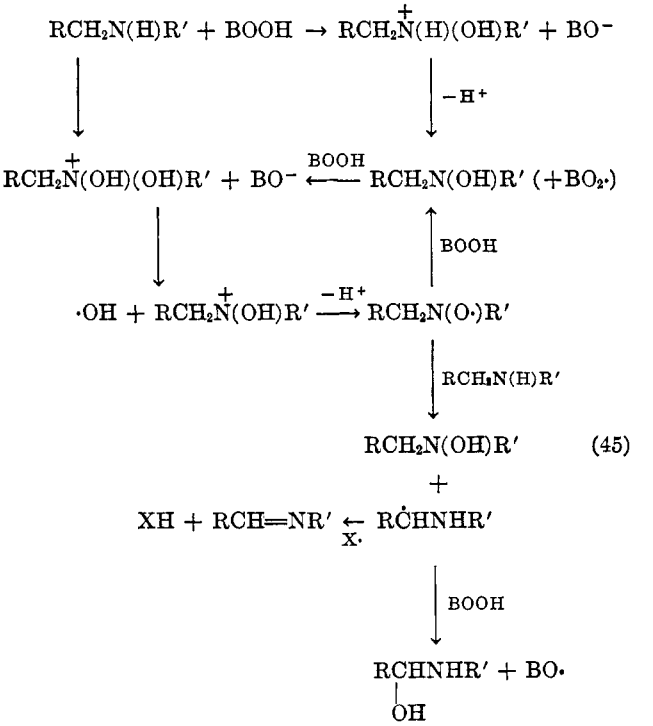
By analogy, the amine–hydroperoxide reaction probably also involves an initial polar reaction, the products of which rearrange to give either stable products (reaction 9) or free radicals (reaction 8). Thus, *N*,*N*-dimethylaniline is sometimes a weak catalyst (35, 169) and sometimes a weak inhibitor of hydrocarbon autoxidations (142); this suggests that this amine can give either free radicals or stable products on reaction with hydroperoxides, just as it can on reaction with benzoyl peroxide (323). Aromatic primary and secondary amines that are inhibitors of free radicals, on the other hand, appear to decompose hydroperoxides without the production of free radicals (167) (see Section IV). The evidence as to whether or not the majority of alkylamines produce free radicals is conflicting. Polyethylene

and polypropylene polyamines must give radicals on reaction with a tertiary hydroperoxide (cumene hydroperoxide), since this reaction initiates the emulsion polymerization of styrene (333), but they may not give radicals with secondary hydroperoxides (see Section IV). A representative selection of primary, secondary, and tertiary amines do not, however, initiate the polymerization of styrene on reaction with tertiary hydroperoxides (333). The hydroperoxides are instead converted in high yield to the corresponding carbinols, the rate of reaction following the order tertiary amines > secondary amines > primary amines (64, 85). Virtually all the oxygen from the hydroperoxide appears as water. No amine oxides were detected, a result which is surprising in view of the usual method of preparing amine oxides, *viz.*:

$$R_3N + H_2O_2 \rightarrow [R_3N{-}OH]^+[OH]^- \rightarrow R_3NO + H_2O \quad (44)$$

However, their absence from the products may be due to further reaction with hydroperoxides, since at least one amine oxide appears to be a more active decomposer of peroxides than its parent amine. Thus, the relative inhibiting efficiencies toward the autoxidation of a thermally cracked gasoline of *N,N'*-diphenyl derivatives of *p*-phenylenediamine, *p*-quinonediimine, and *p*-quinonediimine *N,N'*-dioxide are 1.0:0.79:0.91, respectively (256). The first compound probably acts mainly as a chain-breaking inhibitor (51) and only to a lesser extent as a decomposer of peroxides, but the second two compounds probably act only in the latter capacity.

The decomposition of *tert*-butyl hydroperoxide (BOOH) by trishydroxyethylamine (64), 4-methyl-2-pentylamine (85), or tri-*n*-propylamine (85) gives over 80 per cent yields of *tert*-butyl alcohol but no acetone. This might suggest that the *tert*-butoxy radical (BO·) is not formed, since it rapidly rearranges to acetone and a methyl radical. However, appreciable amounts of acetone are produced by *tert*-octylamine, a primary amine having no α-hydrogens, which reacts more slowly than the other amines (85). This suggests that BO· is produced in all cases, but that if the amine has an α-hydrogen atom the radical is rapidly converted to BOH. This view is supported by the fact that the decomposition induced by amines containing α-hydrogen atoms is inhibited by oxygen and by inhibitors of free radicals. Moreover, the identification of 2,6-di-*tert*-butyl-4-*tert*-butylperoxy-4-methylcyclohexadienone from the reaction inhibited by 2,6-di-*tert*-butyl-4-methylphenol proves that the *tert*-butyl peroxy radical is also present (85) (see reaction 67a). Radicals of the type R(R')NO· were also identified in the reaction by the electron paramagnetic resonance technique (85). The proposed reaction mechanism (85) involves both ionic and free-radical intermediates; e.g., with a secondary amine



The ketimine (RCH=NR') undergoes a variety of further reactions (85). Other ionic processes not involving the production of free radicals may also be occurring concurrently:

$$RCH_2N(H)R' + BOOH \rightarrow [RCH_2N(H)(OB)R']^+[OH]^- \quad (46)$$

$$[RCH_2N(H)(OB)R']^+[OH]^- \rightarrow H_2O + BOH + RCH{=}NR' \quad (47)$$

Possibly the hydroperoxide can also react with amines containing α-hydrogen atoms via a cyclic transition state to give ketimine, alcohol, and water, directly.

In view of the fact that some free radicals must be involved in the reaction it is surprising that alkylamines only rarely have a catalytic effect on an autoxidation (35). On the contrary, most examples recorded in the literature indicate that these amines are either inactive (107) or are weak inhibitors (4, 169). It seems probable that their inhibiting activity is due to the fact that peroxide-decomposition reactions which do not lead to the production of radicals (e.g., reaction 52) outweigh the catalytic effect of those decomposition reactions which do lead to free radicals. The ready transfer of the α-hydrogen atoms to free radicals may also contribute to their inhibiting power, but it can also lead to a chain-transfer process in which the new chain-carrying species promotes an increased rate of oxidation (35).

Further examples of reaction 9 come from the reactions of hydroperoxides with trisubstituted phosphines (159) and trialkyl phosphites (326) to give phosphine oxides and trialkyl phosphates, respectively, together with the corresponding alcohol.

$$ROOH + R'_3P \rightarrow ROH + R'_3PO \quad (48)$$

$$ROOH + (R'O)_3P \rightarrow ROH + (R'O)_3PO \quad (49)$$

Two alternative transition states have been proposed (96),

$$[R_3'POH]^+[OR]^- \quad \text{or} \quad R_3'P\cdots O\cdots O{-}R \;(H)$$

but a cyclic transition state of the same type as for the sulfide–hydroperoxide reaction could also account for the products. Trialkyl phosphites are also destroyed by a direct reaction with peroxy radicals to give the phosphate and an alkoxy radical (326). The same reaction probably also occurs with trisubstituted phosphines.

In conclusion, reaction 9 appears to be general for many compounds containing elements from Groups V and VI of the Periodic Table, but further work is needed to elucidate the detailed reaction mechanisms, which appear to change not only from one element to another but also to vary with temperature and with the type of compound, i.e., with the substituents attached to the reactive element. Although the reaction is not catalytic, the stoichiometry, with respect to hydroperoxide, is frequently greater than unity; moreover, the products may promote a concurrent catalytic decomposition of the hydroperoxide. Many of the compounds recorded as decomposers of peroxides or as inhibitors in the patent literature (176) contain two or more of these reactive elements. While the majority of these compounds undoubtedly function mainly as decomposers of peroxides, a great many are also active as inhibitors of corrosion and as detergents (279) (see below).

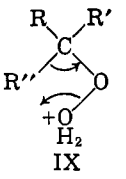
Reaction 10 provides the most attractive mechanism for peroxide decomposition for inhibition purposes. The best known example is an ionic rearrangement of the type catalyzed by strong acids in the Lewis sense (e.g., ferric chloride in benzene (184).

$$RR'R''COOH + A \rightarrow RR'CO + R''OH + A \quad (50)$$

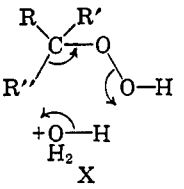
The migrating group (R'') will be aryl or allylic to the complete exclusion of alkyl groups or hydrogen. Among substituted aryls, the group of greatest electron-donating power migrates (307). This reaction was originally formulated as a chain reaction involving a rearrangement of the alkoxy cation (184), i.e.,

$$RR'R''CO^+ \rightarrow RR'C^+OR'' \xrightarrow{AOH^-} RR'CO + R''OH \quad (51)$$

However, the absence of exchange between 1-phenylethyl hydroperoxide and $H_2{}^{18}O$ in acid solution has shown that a stable alkoxy cation is not formed (11). The reaction may occur by way of a four-membered cyclic transition state (IX) in the absence of water or by a six-membered ring (X) in the presence of water (300).



IX



X

Although acids can reduce rates of oxidation, they frequently direct the oxidation toward the formation of undesirable products, such as resins (86), sludges (246, 335), corrosive compounds (343), and dark-colored products. For this reason lubricating oils and cracked distillate fuels are frequently treated with alkalis before use (57, 149, 246) and are kept alkaline throughout most of their service life by the addition of highly alkaline additives (244, 262, 264, 284). By maintaining a high alkalinity the rate of oxidation of substances such as lubricating oils (168), cumene (28, 138, 291), tetralin (138, 163), and aldehydes and ketones (108) is increased, but sludges and colored products are either not formed (168) or are selectively oxidized (116). Corrosive substances are neutralized by the alkali to give products with good detergency properties (116, 244, 264). The increased rate of oxidation of these substrates by bases is probably due to a catalytic decomposition of hydroperoxides to give free radicals. The reaction may involve the formation of a complex between the alkali salt of the hydroperoxide (ROOM) and a second molecule of hydroperoxide, this complex reacting with RH to give the conventional chain reaction (291). However, it has also been suggested that ROOM forms a complex with RH that rearranges to oxygenated products without the formation of free radicals (138).

The oxidation of paraffin wax (164), the oxidation of diethylene glycol (211), and the cobalt-catalyzed oxidation of 1-decene (150) are all inhibited by bases, which suggests that the catalytic decomposition of hydroperoxide does not result in free radicals in these cases. Secondary hydroperoxides apparently lose the hydrogen attached to the carbon atom rather than that attached to the oxygen atom to give mainly alcohols and ketones (27, 307). Primary hydroperoxides give alcohols and probably aldehydes (341), presumably by the same mechanism.

$$B \;H{-}C(R)(R'')-O-O-H \rightarrow BH^+ + RR''CO + OH^- \quad (52)$$

Tertiary hydroperoxides decompose via the peroxy anion to give oxygen and the corresponding alcohol (185, 307).

$$ROOH + OH^- \rightarrow RO_2^- + H_2O \quad (53)$$

$$RO_2^- + ROOH \rightarrow RO^- + ROH + O_2 \quad (54)$$

The transition state of reaction 54 is probably similar to that of reaction 25a.

Reaction 11 represents the induced decomposition of hydroperoxides by a heavy metal. Although this reaction is fairly well understood, the overall effect of metals on autoxidations appears to be quite complex. A number of heavy metal ions, particularly those which possess two or more valency states with a suitable oxidation reduction potential between them (e.g., iron, cobalt, copper, manganese, etc.) can react with hydroperoxides to produce free radicals (12, 65, 318). The metallic ion can act either as a reductant:

$$Fe^{++} + ROOH \rightarrow Fe^{3+} + RO\cdot + OH^- \qquad (55)$$

or as an oxidant:

$$Ce^{4+} + ROOH \rightarrow Ce^{3+} + RO_2\cdot + H^+ \qquad (56)$$

Certain metals (e.g., cobalt and manganese) can act as both oxidant and reductant and are therefore catalysts for the decomposition of peroxides. For these metals reaction 56 may be rate determining, since the metal is found to be chiefly in its more highly oxidized state (318). An induction period is often observed during catalysis by Mn^{++} and Co^{++}, which can be eliminated by the addition of hydroperoxide (65). The occurrence of an induction period is due to the formation of an intermediate catalyst–hydroperoxide complex, which subsequently decomposes to yield the metal ions in their catalytic, higher-valency state (65, 93, 123). Any ions remaining in their lower valency after the initial oxidation stage apparently act as inhibitors (93), probably because of a termination reaction with free radicals (65, 93). In contrast to these results ferric chloride is an inhibitor of the oxidation of cumene and tetralin in aromatic solvents (143). The extent of inhibition is increased by the initial addition of hydroperoxides, presumably because they are able to oxidize ferrous compounds to the ferric state. On the other hand, ferric chloride is an initiator in polar solvents (143). This variation with solvent could be due to several causes, such as a difference in the reactions of ferric chloride monomer and dimer (143), the formation of ferric ions in polar solvents, changes in the coördinating tendency of the solvents towards the ferric chloride (65, 314), or a shift in the equilibrium of the radical–metal ion complex (183).

It has been shown that complexes of metal ions and hydroperoxides are the active catalytic agents that react with more hydroperoxide (30, 199); this is quite reasonable in view of the strong catalytic effect of some metal chelates on the decomposition of hydroperoxides (71), the oxidation of olefins (65, 314), and the oxidation of alkylbenzenes (201). The anion associated with the metal can influence its catalytic activity either by affecting its redox potential or by "blocking" the formation of a catalyst–hydroperoxide complex, and the effect of a given anion will depend on both the metal and the anion (65). Coördination of the solvent with the metal can also affect catalytic activity (65, 314).

In general heavy metals function as powerful prooxidants of organic autoxidations (12, 318). Catalysis by metallic surfaces is proportional to their surface area (65) and therefore, except for colloidally dispersed catalysts, appreciable activity occurs only in so far as the metal goes into solution (48, 305). The rate of dissolution of the metal can be reduced by the addition of "corrosion inhibitors" or "metal passivators," which form a strong chemisorbed film on the metallic surface (279, 308, 343). In addition, the oxidation products from noncorrosive substrates can also lacquer metal surfaces (221). The addition of corrosion inhibitors to lubricating oils becomes particularly important when the oil also contains "detergents" or "dispersants," which have been added to prevent the settling out of solid deposits, since the metal surfaces are then kept clean and vulnerable to attack (176). Incidentally, the term "detergent" is a misnomer, since most commercial detergents act mainly to prevent sludge and resin formation rather than to peptize these insoluble products (268). The formation of protective films on metal surfaces can also help to reduce the mechanical wearing and scuffing of surfaces subjected to high loads (313).

The dissolution of metals by organic acids in hydrocarbon media is dependent on the presence of oxygen, peroxy radicals, or peroxides (86, 261). Therefore the most effective types of corrosion inhibitors are sulfur- and phosphorus-containing compounds (such as thiophosphates and condensation products of phosphorus pentasulfide and unsaturated hydrocarbons (132, 279, 308, 343)), which can not only form a protective film but can also decompose the peroxides in the substrate. Some inhibitors of free radicals such as *N,N'*-diphenyl-*p*-phenylenediamine are also very effective for a similar reason (293).

Although reactions 55 and 56 are probably chiefly responsible for the catalysis of oxidation by metallic ions, there may also be a direct initiation by metallic ions in some cases; e.g. (20, 65, 110),

$$RH + M^{(n+1)+} \rightarrow M^{n+} + R\cdot + H^+ \qquad (57)$$

or (43, 314)

$$O_2 + M^{n+} \rightleftarrows [M^{(n+1)+}O_2^-] \xrightarrow{RH} \text{radicals} \qquad (58)$$

particularly at temperatures below 100°C. (201). It has, in fact, been suggested that the initiation of the autoxidation of even purified natural fats may be due to the catalysis of reaction 58 by trace metals (314). In general, the catalytic effect of metal salts reaches a constant value at quite low concentrations of catalyst. This may be due either to chain termination by the catalyst (65, 93, 129, 130) or to the occurrence of a steady-state concentration of hydroperoxide, which

is achieved when reactions 3 and 11 proceed at the same rate (340). Under these conditions no chain branching occurs, since every decomposition of ROOH leads to the formation of only one new ROOH molecule. Once oxidation has started the metal can sometimes be completely precipitated from solution without affecting the subsequent rate of oxidation (194).

Copper stearate inhibits the ferric stearate-catalyzed oxidation of tetralin and the cobalt stearate-catalyzed oxidation of normal paraffins (129), the rate falling to about the values obtained for the straight copper stearate-catalyzed reactions. Although chain termination by the catalyst by reactions such as

$$RO_2\cdot + Cu^{++} \rightarrow Cu^{+} + R^{+} + O_2 \quad (59)$$

$$RO\cdot + Cu^{+} \rightarrow Cu^{++} + RO^{-} \quad (60)$$

$$RO_2\cdot + Cu^{+} \rightarrow Cu^{++} + RO_2^{-} \quad (61)$$

could be responsible (21, 185, 187), recent work suggests that the free radicals are not completely free in the presence of copper salts (90, 183, 188, 205). The formation of a radical–copper ion complex would probably decrease the reactivity of the radical. Inhibition of iron- and cobalt-catalyzed oxidations would therefore be due to a more stable radical–metal ion complex with copper than with the other two metals.

The formation of radical–metal ion complexes, as well as the possibilities of termination by reactions 59, 60, and 61, probably accounts for the few cases where the oxidation of organic substances has been reported to be inhibited by heavy metals. Thus the rate of oxidation of toluene is reduced by copper naphthenate (248), and of *p*-xylene by uranium, vanadium, and cupric naphthenates (1, 247), while the manganese salt has been reported both to catalyze (247) and to inhibit (1) the oxidation of the latter compound. Metal ion–radical complexes are probably particularly important with aromatic substrates, since the free radicals are already largely complexed with aromatic molecules (275, 324), and it has been suggested that these complexes are further stabilized by metal ions (183). Another termination reaction that might also be important with alkyl aromatic substrates is

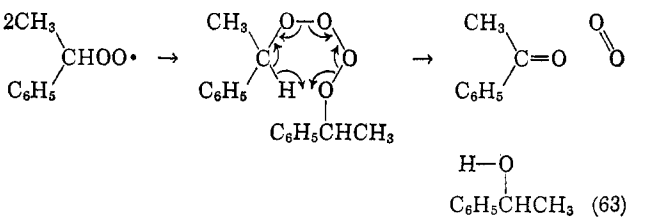
$$RO\cdot + M^{++} \rightarrow RO^{+} + M^{+} \quad (62)$$

followed by rearrangement of the alkoxy cation to a phenolic inhibitor (reaction 51). Other examples of inhibition by metal ions include the effect of cobalt stearate on *n*-heptaldehyde (129) and of copper salts on purified gasoline (255) and on the fatty acids from soybean oil (189). Cupric stearate acts on *n*-decane (192) and copper naphthenate on *p*-xylene (247) as a catalyst at low concentrations and as an inhibitor at high concentrations. This change from catalysis to inhibition suggests that at high concentrations destruction of free radicals by reactions 59 to 62 becomes important enough to reduce their steady-state concentration and thus reduce their rate of attack on the substrate.

The generally deleterious effects of metals on organic oxidations can be most readily overcome both by deactivating dissolved metals by the addition of chelating (complex-forming) agents such as ethylenediamine tetraacetic acid or *N,N'*-disalicylidene-1,2-propanediamine (65, 255, 330, 333), and, when metal surfaces are present, by the addition of metal passivators as described above. The antioxidant activity of these compounds naturally only appears in the presence of metals. Useful chelating agents are effective by virtue of their steric effect in preventing the formation of metal ion–hydroperoxide complexes and also by modifying the redox potential of the ion so as to suppress reactions 55 or 56 (65). The change in the latter property can actually favor an increase in the catalytic power of the metal (65), even though the chelating agent might act as an inhibitor in the absence of the metal (177). Furthermore, the metal chelate may itself be subject to direct oxidation, even though the chelating agent is unreactive in the absence of the metal (229). One interesting feature exhibited by lubricating oils, as opposed to pure hydrocarbons, is that they can generally "tolerate" a certain critical amount of metallic catalyst without appreciable effect on their oxidation rate (83), but the nature of the deactivators they contain has not been established.

E. Self-termination

Primary and secondary peroxy radicals terminate reaction chains by way of a cyclic transition state (273):



(63)

Tertiary peroxy radicals terminate reaction chains less readily, since they lack a hydrogen atom on the α-carbon. It is generally assumed (26, 42, 84, 219, 308a) that these radicals undergo a nonterminating interaction to produce alkoxy radicals

$$2R_3CO_2\cdot \rightarrow 2R_3CO\cdot + O_2 \quad (64)$$

which may dimerize, possibly while still in the same "solvent cage," disproportionate with the transfer of an alkyl group (3), decompose, or abstract a hydrogen from the substrate. In the last two cases termination of the chains has not occurred. The autoxidation of cumene by a mixture of normal oxygen and $^{18}O^{18}O$ has shown that the oxygen evolved in this reaction arises from both cumyl peroxy radicals, i.e., it contains $^{16}O^{18}O$ (308a). It seems unlikely, therefore, that the peroxy

radicals disproportionate with loss of oxygen (52), i.e.,

$$2C_6H_5C(CH_3)_2OO\cdot \rightarrow C_6H_5C(CH_3)_2OOH + C_6H_5C(CH_3)=CH_2 + O_2 \quad (65)$$

and a suggested cyclic transition state for this reaction (300) (similar to that for reaction 63 but involving cleavage of a C—O bond instead of an O—O bond) cannot be correct, since all the evolved oxygen would arise from a single peroxy radical, i.e., it would be $^{16}O^{16}O$ and $^{18}O^{18}O$. It has also been suggested that tertiary peroxy radicals can react with hydroxyl radicals to terminate chains (219, 230).

$$R_3COO\cdot + \cdot OH \rightarrow R_3COH + O_2 \quad (66)$$

The absolute rate of self-termination of a number of peroxy radicals has been determined by photochemical methods, using the rotating sector technique (12, 77, 162).

Because secondary (and primary) peroxy radicals terminate reaction chains more rapidly than tertiary it is possible to reduce the rate of oxidation of a compound which gives the latter radicals by the addition of a compound which gives the former, even though the added compound may be the more reactive in the pure state (2, 271). For example, the rate of oxidation of cumene is reduced by the addition of small amounts of the more reactive hydrocarbon, tetralin (271). This addition causes a relatively high proportion of the peroxy radicals produced in the propagation steps to be secondary radicals. These radicals terminate chains more rapidly than the cumyl peroxy radicals and thereby diminish the steady-state concentration of radicals and the rate of oxidation. Mixtures of pure compounds can, of course, also give oxidation rates higher than the sum of the individual rates, e.g., benzaldehyde and decanal (162), methyl linoleate and dimethylbutadiene (181).

F. Chain-breaking inhibition

The chain-breaking step by which inhibitors of free radicals reduce the rate of oxidation of organic compounds has generally been considered to involve a hydrogen-abstraction reaction:

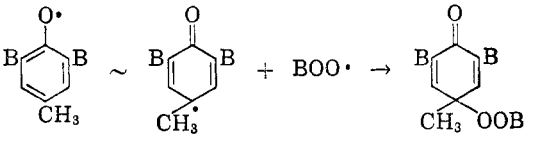
$$RO_2\cdot + IH \rightarrow ROOH + I\cdot \quad (13)$$

This reaction was first proposed for phenolic inhibitors (47) but was soon extended to cover substituted anilines.

The free radical I· is generally stabilized by resonance and may, therefore, be insufficiently reactive to start a new oxidation chain (47), particularly when the phenolic or amino group is surrounded by bulky substituents (34, 35). It will be destroyed by reaction with another free radical. Analysis of the stable products of interaction between peroxy radical and inhibitor by a number of workers under a variety of experimental conditions has shown that the overall reaction can follow several paths. The product analyses have been most successful with phenolic inhibitors; only one amine inhibitor has been successfully examined (51). The following radical–radical reactions have been identified:

$$I\cdot + RO_2\cdot \rightarrow ROOI \quad (33, 51, 62) \quad (67)$$

For example (33, 62),



(67a)

where B represents the *tert*-butyl group.

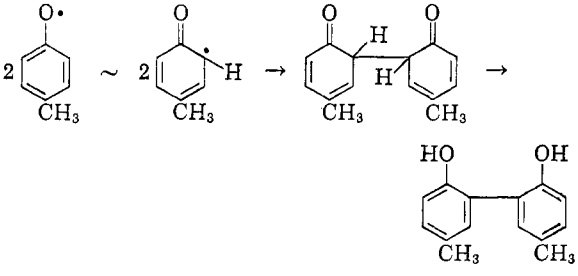
$$I\cdot + RO_2\cdot \rightarrow ROOH + I' \quad (47, 51, 104) \quad (68)$$

For example (51),

$$p\text{-}C_6H_5NHC_6H_4\dot{N}C_6H_5 + CN(CH_3)_2COO\cdot \rightarrow p\text{-}C_6H_5N{=}C_6H_4{=}NC_6H_5 + CN(CH_3)_2COOH \quad (68a)$$

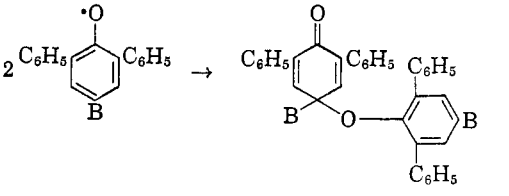
$$I\cdot + I\cdot \rightarrow I_2 \quad (72, 74, 154, 208, 209, 238, 241, 329,336; \textit{cf.} \text{ also } 233) \quad (69)$$

For example (238),



(69a)

Similar reaction products have been identified in the reactions of alkylphenols with hydroxyl radicals (79). Although a dimerization product of this type will undergo further reactions with peroxy radicals, other dimerizations can lead to inactive products: e.g. (336),



(69b)

$$I\cdot + I\cdot \rightarrow IH + I' \quad (47, 75) \quad (70)$$

For example (47),

$$2p\text{-}HOC_6H_4O\cdot \rightarrow p\text{-}HOC_6H_4OH + p\text{-}O{=}C_6H_4{=}O \quad (70a)$$

2,6-Dialkyl-4-methylphenols under certain oxidative conditions form dimers of the corresponding hydroxybenzyl radicals. It was at first suggested that the hydroxybenzyl radicals were produced in the initial step, i.e., reaction 13 (33, 72, 154, 238), but it has since been shown that these radicals are secondary products arising from isomerization of the initially formed phenoxy radicals (74). There is now, in fact, abundant evidence from electron paramagnetic resonance measure-

ments that the phenoxy radical is the initial oxidation product of alkylphenols (7, 22, 29, 113, 242). These radicals may be quite stable or may undergo rapid secondary reactions. On the other hand, the first moderately stable radicals that can be detected by electron paramagnetic resonance during autoxidations inhibited by primary and secondary aromatic amines are derivatives of nitric oxide (303). These radicals (which are also present during the induced decomposition of hydroperoxides by alkylamines (85)) are apparently formed by reaction of a very reactive nitrogen radical with a second peroxy radical:

$$(C_6H_5)_2NH + RO_2\cdot \rightarrow (C_6H_5)_2N\cdot + ROOH \quad (71)$$

$$(C_6H_5)_2N\cdot + RO_2\cdot \rightarrow (C_6H_5)_2NO\cdot + RO\cdot \quad (72)$$

The diphenyl nitric oxide radical is itself an inhibitor capable of terminating half as many chains as diphenylamine. No stable radical products were detected by this technique during inhibition by tertiary amines (303).

For many inhibitors, particularly those in which the active center is not protected by bulky substituents, the radical I· can initiate a new chain by reaction with the substrate (34, 35, 329):

$$I\cdot + RH \rightarrow IH + R\cdot \quad (73)$$

or

$$IO_2\cdot + RH \rightarrow IOOH + R\cdot \quad (74)$$

The radical I· can also react with hydroperoxides (226), add to double bonds (148), and add oxygen (75, 76, 241).

$$I\cdot + O_2 \rightarrow IO_2\cdot \quad (75)$$

$$IO_2\cdot + I\cdot \rightarrow IOOI \quad (76)$$

The rates of uninhibited oxidations are independent of oxygen pressure, except at very low pressures (12). In contrast, the rates of inhibited oxidations, or their induction periods, depend on oxygen pressure (165, 180, 285), so it is probable that most inhibitors react directly with oxygen (180, 256).

$$IH + O_2 \rightarrow I\cdot + HO_2\cdot \quad (77)$$

Under some conditions the hydroperoxide of the inhibitor is produced (32, 70, 134), but under others, extensive rearrangements may occur (342). Oxidation will generally reduce inhibitor efficiency, although in certain cases the products may be more efficient than the original compound, leading to an increase in overall efficiency as the inhibitor is oxidized (169). In some substrates, an otherwise good inhibitor may promote the formation of sediments (246, 335). The reactions of phenols and aromatic amines with hydroperoxides have been discussed previously.

The relative rates of reactions 3, 13, and 73 (or 74) have frequently been used to classify inhibitors into various groups. In general, an inhibitor is regarded as "strong" when reaction 13 is much faster than reaction 3; the strongest possible inhibitor is one which removes all the peroxy radicals before they can react with the substrate. An "effective" inhibitor is one for which reaction 73 or 74 is very slow, whereas with an ineffective inhibitor these reactions can become important chain-propagating steps. Inhibitors may therefore be classified into four groups according to whether they are strong or weak and effective or ineffective by measurements of the rates of the elementary reactions involved. Classifications of inhibitors by this method, particularly into strong and weak classes, have been made by a number of workers (34, 35, 47, 82, 89, 142), but it must be remembered that because of the variety of other reactions in which the inhibitor and its free radical can be involved these classifications apply only to the particular reaction conditions used in each case. Moreover, because of these alternate reactions, values of relative rates recorded in the literature may be erroneous, since they may reflect changes in the rates of reactions other than those that they are supposed to represent. Of more practical importance are direct qualitative comparisons of inhibitor efficiencies by measurements of induction periods for oxygen absorbtion, hydroperoxide build-up, etc. (37, 38, 39, 107, 200, 202, 213, 214, 232, 233, 239, 253, 256, 269, 328). Some general conclusions regarding the effect of structure on the efficiency of an inhibitor can be drawn from both kinds of tests. In general, the efficiency of a given inhibitor type is increased by an increase in the electron density at the reactive center. That is, the efficiency is increased by a decrease in the oxidation–reduction potential of the inhibitor or by a decrease in the I—H bond strength (47). However, too low an oxidation–reduction potential results in a decrease in efficiency, since the inhibitor then becomes susceptible to direct oxidation (47, 213). An increase in the degree of steric protection of the reactive center may either increase (256) or decrease (82) efficiency depending on the type of inhibitor.

In the absence of other reactions competing for the inhibitor, reaction 13 followed by reactions 67, 68, 69, or 70 suggests that each inhibitor molecule will react with two peroxy radicals. Estimates of the stoichiometry of inhibition have tended to confirm this conclusion (47, 50, 51). As a result of these reactions the inhibitor is itself oxidized, the first step being the formation of a free radical I· and the subsequent reactions of this radical giving rise to the observed products. However, this does not necessarily imply that the rate-controlling step of inhibition involves a hydrogen-abstraction reaction and there is, in fact, some quite compelling evidence from experiments with deuterated inhibitors that abstraction of hydrogen is not rate controlling.

Boozer and Hammond (49, 51, 142) have proposed an

alternative inhibition mechanism to account for their observation that *N*-methylaniline-*N*-*d* and diphenylamine-*N*-*d* have identical inhibiting actions on the oxidation of cumene and tetralin initiated by α,α'-azobisisobutyronitrile in chlorobenzene solution, compared with the corresponding undeuterated amines. The mechanism involves the reversible formation of a peroxy radical–inhibitor complex, followed by a very rapid reaction of the complex with a second peroxy radical.

$$RO_2\cdot + IH \rightarrow [RO_2\cdot \leftarrow IH] \quad (78)$$

$$RO_2\cdot + [RO_2\cdot \leftarrow IH] \rightarrow \text{inactive products} \quad (79)$$

Reaction 78 is written as a reversible process to account for the kinetics (*cf.* hydrogen abstraction from primary arylamines by the α,α-diphenyl-β-picrylhydrazyl radical (227)). It has since been shown (by the addition of hydroperoxide at the start of the reaction) that the kinetics are not the result of a reversible hydrogen abstraction, i.e., a reversible reaction 13 (143). This mechanism was extended to phenolic inhibitors and was also used to account for the inhibition of the oxidation of tetralin by *N*,*N*,*N'*,*N'*-tetramethyl-*p*-phenylenediamine. The formation of the Wurster cation with the latter inhibitor in the presence of water was assumed to be due to hydrolysis of the complex (49).

$$[p\text{-}(CH_3)_2NC_6H_4N(CH_3)_2 \rightarrow RO_2\cdot] + H_2O \rightleftarrows [p\text{-}(CH_3)_2NC_6H_4N(CH_3)_2]^+ + ROOH + OH^- \quad (80)$$

However, since the hydrogen atom on the α-carbon atom of amines is readily abstracted by the free radicals produced from alkyl peroxides (160, 315) and hydroperoxides (85, 183) in the liquid phase, or by oxidation in the gas phase (81), complex formation is probably not necessary to account for inhibition by this amine, i.e., inhibition might be due to the formation of the $p\text{-}(CH_3)_2NC_6H_4N(CH_3)CH_2\cdot$ radical, which could perhaps react with water to give the observed cation.

$$p\text{-}(CH_3)_2NC_6H_4N(CH_3)CH_2\cdot + H_2O \rightarrow p\text{-}(CH_3)_2NC_6H_4N(CH_3)_2^+ + OH^- \quad (81)$$

The absence of a deuterium isotope effect during inhibition by *N*,*N'*-diphenyl-*p*-phenylenediamine has led to the suggestion (256) that inhibition by aromatic amines (but not by phenols) is due to an electron-transfer reaction.

$$RO_2\cdot + IH \rightarrow RO_2^- + IH^+ \quad (82)$$

The Boozer and Hammond mechanism has received some support for the inhibitor *N*-phenyl-α-naphthylamine in octadecene, by electron paramagnetic resonance measurements of radical concentrations (147), although no free radicals were detected by this technique with phenolic inhibitors. The results obtained in this work could, however, also be interpreted in terms of reaction 13 and consumption of the inhibitor radical by some oxidation product which rises to a high concentration near the end of the inhibition period (147), e.g., hydroperoxide (226).

The activation energy for the reaction of diphenylethyl peroxy radicals with 2,6-di-*tert*-butyl-4-methylphenol has been reported to be close to zero (36) (see, however, reference 266), which led to the suggestion (36) that the absence of an isotope effect does not invalidate reaction 13 as the rate-determining step. Although this argument may be valid for this strong inhibitor, it cannot be applied to a weak inhibitor that owes its lower efficiency to an increase in the activation energy of reaction 13. Therefore, isotope effects are more likely to be observable with weak than with strong inhibitors. An examination of the effect of deuteration on the inhibition efficiencies toward a hydrocarbon oil of a number of sterically nonhindered very weak inhibitors, such as phenol, *o*-cresol, and diphenylamine, has shown the presence of a small isotope effect that decreases with increasing inhibitor efficiency (169). However, it was pointed out (169) that no firm conclusions about the mechanism could be derived because of the numerous reactions other than reaction 13 that might also give an isotope effect, in particular, reaction with hydroperoxide (322).

Many of these secondary reactions can be suppressed if the reactive center of the inhibitor is sterically protected; moreover, the chances of observing an isotope effect may be improved at the same time (6). For example, 4-substituted 2,6-di-*tert*-butylphenols react very slowly with benzoyl peroxide (322) and with hydroperoxides (166) and do not undergo chain-transfer reactions (34). For these reasons, a large number of 4-substituted 2,6-di-*tert*-butylphenols have been compared as inhibitors of the autoxidation of a white mineral oil at 160°C. (166). The relative induction periods (t) (which are related to the relative rates of the inhibition reaction) were correlated with the σ constants of the 4-substituent by means of the Hammett equation (140).

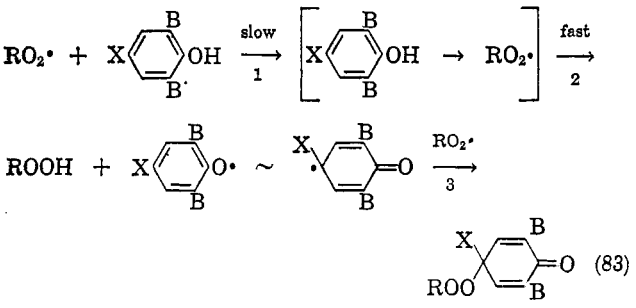
$$\log (t/t_0) = \rho\sigma = -0.71\sigma$$

The value of the reaction constant, ρ, for the inhibition reaction was calculated to be -1.06 from the observed kinetics. Bulky alkyl groups in the 4-position decreased inhibitor efficiencies. Their effect could be correlated by the Taft steric substituent constant (E_s) (301), which takes account of the physical size of the group.

$$\log (t/t_0) = \rho\sigma + \delta E_s = -0.71\sigma + 0.14E_s$$

However, it was concluded that, in spite of this relationship, the initial inhibition step could not be the addition of a peroxy radical at the 4-position to give a paraquinolide radical, since phenoxy radicals are known to be the initial products of the oxidation of alkylphenols.

The weakest inhibitors (i.e., those with electron-attracting 4-substituents) were deuterated at the phenolic hydrogen. No isotope effect could be detected within an accuracy of about 2–3 per cent. The following inhibition mechanism was suggested:



(83)

The reduction in inhibitor efficiency caused by bulky 4-substituents was attributed to a reduction in the rate of step 3 caused by steric effects; this is in agreement with the observation that the 2,4,6-tri-*tert*-butylphenoxy radical is an inefficient trap for benzoyloxy radicals (73). Step 1 may have been reversible, and it is also possible that the complex reacted directly with a second peroxy radical to give the observed products without a measurable isotope effect (*cf.* the phenylation of aromatic hydrocarbons (289)). In these phenols the O—H group lies in the plane of the benzene ring and is therefore protected from a direct abstraction reaction by the adjacent *tert*-butyl group (166). The peroxy radical probably approaches the inhibitor perpendicular to the plane of the ring and forms a complex with the π electron system (275, 324). If an electron-transfer reaction, of the type suggested for amines (256) and, on theoretical grounds, for phenols (126), were rate determining, or if a highly polar transition state for hydrogen abstraction were involved (274), the induction periods should be related to σ^+ (56, 274) rather than to σ constants. Unfortunately, σ and σ^+ constants are effectively the same for all except strongly electron-donating groups. The only compound studied that would have enabled this mechanistic distinction to be made was 2,6-di-*tert*-butyl-4-methoxyphenol. The induction period for this phenol followed the normal Hammett equation, but since it appeared to be slightly unstable under the conditions employed, an electron transfer or a highly polar transition state cannot be completely ruled out.

Completely different isotopic results, ranging from normal to inverse effects, have been reported for the oxidation of butadiene–styrene rubber inhibited by 2,6-di-*tert*-butyl-4-methylphenol (BMP) (286), *N*-phenyl-β-naphthylamine (PBN) (286, 287), and diphenylamine (DPA) (287). The following isotopic effects, measured by the rate of absorption of oxygen (k_D/k_H), were observed at 90°C. at the concentrations given (in parts of inhibitor per hundred of rubber): BMP 1 per cent, 1.0; 3 per cent, 1.3; PBN 3 per cent, 1.8; 5 per cent, 0.86; 7 per cent (saturated), 0.87; DPA 2 per cent, <1.0; 3 per cent, 0.78. At 80°C. 3 per cent of DPA showed no isotope effect. The negative isotope effects were readily explained as being due to initiation by direct attack of oxygen on the antioxidant (reaction 77). Deuteration will decrease the rate of this reaction and thereby conserve the antioxidant for inhibition. The negative isotope effect therefore becomes more important at high concentrations (i.e., proöxidant levels) of inhibitor. Moreover, reactions 67 and 73 are in competition for the radical I·. Since the latter reaction has the larger activation energy it becomes relatively more important, and therefore the negative isotope effect becomes more pronounced, as the temperature is raised.

It was concluded from the positive isotope effects that abstraction of hydrogen by peroxy radicals must be the rate-determining step of inhibition. This implies that deuterium exchange occurred in the work outlined above (49, 142, 166, 256), a result which seems unlikely in view of the substrates employed and has since been excluded in some of the work (49, 142) by carrying out the inhibition in the presence of an excess of heavy water (143). However, a closer examination of the large isotope effects obtained with 3 per cent BMP and PBN suggests that they are completely unrelated to reaction 13. In the first place, they correspond to a fairly late stage in the oxidation process (~10–20 ml. of oxygen per gram of rubber). During the initial stages k_D/k_H is only about 1.1 with both inhibitors, a value which is in not unreasonable agreement with previous work considering the limits of accuracy in all cases. In view of the complexity of oxidations after the initial stages and of the possibilities for isotope exchange between the inhibitor and the accumulating products, it is a very questionable procedure to report isotope effects on anything except initial rates or induction periods. Secondly, the high concentrations of inhibitor that must be employed in rubber because of the low rates of diffusion of the reactants increase the possibilities for side reactions involving an isotope effect. For example, *N*-phenyl-β-naphthylamine decomposes the hydroperoxides formed in certain rubbers (285)[2] and oils (166), probably without the formation of free radicals (167). If this reaction involves an isotope effect (which it may not, since the reaction with benzoyl peroxide does not involve one (234)), deuteration will

[2] Reports that *N*-phenyl-β-naphthylamine (PBN) does not react with the "stable rubber peroxides" formed in sodium–butadiene rubber (5, 202) do not invalidate this argument, since the extremely small amounts of peroxide formed in this system suggest that residual alkali is functioning as a decomposer of peroxide. Moreover, only a single concentration of PBN was examined, which can lead to completely erroneous conclusions (*cf.* Section III), and it is not clear from this work that the "stable peroxides" that were studied had actually been formed in the absence of PBN.

decrease its rate and leave more hydroperoxide to give free radicals by reactions 4 and 5, i.e., deuteration will lead to an increased oxidation rate. Similar arguments apply to 2,6-di-*tert*-butyl-4-methylphenol, for although this compound decomposes hydroperoxides very slowly at low concentrations, the rate can become appreciable at relatively high concentrations (166, 170). The reduced importance of this reaction at 1 per cent 2,6-di-*tert*-butyl-4-methylphenol can therefore account for the absence of an isotope effect at this concentration.

In conclusion, the reaction of peroxy radicals with 4-substituted 2,6-di-*tert*-butyl phenols involves either no isotope effect or else a rather small one. It is difficult to see why this should be the case, since the apparently similar abstraction of hydrogen from cumene by peroxy radicals ($\rho = -0.43$ (271)) shows a large isotope effect (~5.5 at 60°C. (273)). Since the activation energy for the inhibition reaction is not zero (266), particularly for those phenols with electron-attracting para substituents which were deuterated (166), it must be concluded (141, 271, 273, 334) that stretching or twisting of the O—H bond proceeds to only a small extent in the complex in reaction 83 or in the transition state if that is all the complex represents. The question remains as to whether a scheme similar to reaction 83 also applies to nonhindered phenols and amines in which the $RO_2\cdot$ is not constrained to approach at right angles to the ring, i.e., in which the hydrogen is not protected from a direct abstraction process by bulky neighboring groups. The general failure to detect isotope effects with nonhindered inhibitors at low temperatures (49, 142, 256) suggests that the isotope effects observed at high temperatures (169) may have been partly or wholly due to a reaction with hydroperoxide. The importance of this reaction as a major cause of inhibition by these compounds has since been confirmed by experiments with mixed inhibitors (167) (*cf.* Section IV). A generally similar mechanism for all inhibitors is also favored by the nearly equal inhibiting efficiencies of 2,6-di-*tert*-butyl-4-methylphenol and 2,4,6-trimethylphenol (82, 167, 214, 232), although the degree of steric protection afforded to the OH group must be quite different in these compounds. A unified reaction scheme has been proposed (165):

$$RO_2\cdot + IH \rightleftarrows RO_2\cdot{,}IH \rightarrow ROOH + I\cdot \xrightarrow{RO_2\cdot} \text{final products} \quad (RO_2\cdot\text{ from } RO_2\cdot{,}IH \text{ to final products}) \tag{84}$$

It has been shown that changes in the relative rates of the elementary reaction steps can account for the different kinetics observed both with inhibitors of different efficiency and with the same inhibitor at different concentrations (165). The complex may represent either a transition state with little or no stretching of the I—H bond or a definite entity stabilized by partial or complete electron transfer, depending on the inhibitor and the reaction conditions.

Compounds other than phenols and amines can also inhibit autoxidations, but they are all very much less efficient than the better known inhibitors. Quinones appear to react with the radical R·, which may add either to the C=C (59) or the C=O (237, 317) double bond. The low inhibiting efficiency of quinones is due to the competition between this reaction and reaction 2. Olefins retard the oxidation of benzaldehyde (337), probably because the alkenyl peroxy radical is much less reactive than the acyl peroxy radical (16a).

Aliphatic alcohols (124, 267) and aromatic hydrocarbons, particularly polycyclic aromatics (125, 204, 311, 338, 344), are weak inhibitors for the oxidation of some hydrocarbons. The inhibition by aromatic hydrocarbons appears to be partly due to decomposition of their hydroperoxides to phenolic inhibitors (204, 282), which probably accounts for the frequently observed autoretardation of the rate of oxidation of aromatics, i.e., decreasing rate with increasing extent of oxidation (204), a phenomenon that is generally not observed with nonaromatic substrates. Aromatic hydrocarbons may also act as inhibitors by complexing with (275, 324) or adding to (23, 151, 207) the chain-carrying radicals so as to give less active radicals. In this case, electron-donating groups on the aromatic compound slow down radical addition while electron-withdrawing groups accelerate it (151). The effectiveness of aromatic hydrocarbons as inhibitors of the oxidation of lubricating oils passes through a maximum as their concentration is increased (125, 344).

III. ADDITION OF ANTIOXIDANTS DURING THE COURSE OF OXIDATION

In the absence of an initiator the rate at which an antioxidant is consumed in an autoxidizing system increases with time, since reaction 1 is complemented to an increasing extent by the decomposition into free radicals of the hydroperoxide products (146, 147). In the presence of a sufficient initial concentration of hydroperoxide the rate of consumption of inhibitor will, of course, be constant and equal to the rate of formation of free radicals from the hydroperoxide (5, 94, 195, 210, 245). Therefore, since the rate of consumption of antioxidant depends on the rate of initiation (89), both inhibitors of free radicals and decomposers of peroxides are most effective when they are added to a system that has undergone little or no prior oxidation, since the concentration of hydroperoxide (which is generally the chief source of free radicals) is then at a minimum. In practice, inhibitors of free radicals are generally added to substances that are initially free from hydroperoxides but must be stored without deterioration for comparatively long periods

of time (e.g., gasoline and edible fats). As the initial concentration of hydroperoxide rises the efficiency of these inhibitors decreases (180), and therefore decomposers of peroxides are generally added to substances such as automotive lubricating oils which often have to be added to an already partially oxidized substrate.

The technique of adding antioxidants at various points during an oxidation can yield valuable information about the reactions of both the antioxidant itself and the oxidation products of the substrate (94). For example (91), the addition of sufficient concentrations of α-naphthol, a strong inhibitor of free radicals, during the uncatalyzed oxidation of cyclohexane drastically reduces the steady-state concentration of free radicals and thereby prevents further oxidation until it has been consumed. Of the products, the concentration of hydroperoxide is decreased whereas that of cyclohexanol and of cyclohexanone is unaffected. The hydroperoxide is therefore probably formed by a free-radical reaction (i.e., reaction 3) and decomposed by a molecular process (reactions 4 and 5, and also probably by an induced decomposition, since α-naphthol is a strong decomposer of peroxides (166, 171)), while the alcohol and ketone are both formed and consumed by radical reactions. It was also shown (91) that with increasing extents of oxidation the rate of chain initiation increases (because of increased hydroperoxide) and the chain length decreases (because self-termination by reaction 12 is bimolecular). Moreover, the reactivity of the peroxy radicals decreases rather irregularly during oxidation because of the changing composition of the substrate. In a similar way the addition of α-naphthol to oxidizing n-decane has been used to measure the rate of formation of free radicals from n-decyl hydroperoxides (195, 217) (see Section II,D).

Strong decomposers of peroxides behave like strong inhibitors of free radicals in that they are able to prevent further oxidation of an already heavily oxidized substrate (170, 171). Weak inhibitors of free radicals, on the other hand, have little or no effect unless they are added before the start of oxidation (170, 171, 191, 267). Their behavior is probably due both to a low rate of reaction with peroxy radicals and, because of the decreased reactivity of the peroxy radicals present in the oxidized substrate, the inhibitor radical (I·) may have a reactivity toward the substrate comparable to the other chain-carrying species present in the later stages of oxidation.

The addition of antioxidants to a substrate at different points during its oxidation, combined with their peroxide-decomposing ability, has been used to classify antioxidants into three groups (170, 171, 173, 270). Group I antioxidants are effective only prior to the start of oxidation and do not react with hydroperoxides (see below). Group II antioxidants are effective whenever they are added and accelerate the decomposition of hydroperoxides. Group III antioxidants are effective only if added at the start or during the initial autocatalytic stages of oxidation and are rather ineffective decomposers of peroxides. It has also been reported (172), on the basis of one example from each group, that antioxidants of Groups I and III, but not of Group II, can inhibit the oxidation of white oil initiated by methyl radicals derived from the decomposition of acetyl peroxide, whereas antioxidants of Groups II and III, but not of Group I, inhibit the oxidation initiated by cumyl peroxy radicals derived from cumene hydroperoxide and cobalt naphthenate. It was, therefore, concluded that antioxidants of Groups I react only with R·, those of Group II with RO_2· and ROOH, and those of Group III with R· and RO_2·.

While there is no doubt that antioxidants can be classed in this fashion, the group that they occupy will depend on the substrate and also, probably, on the reaction conditions. Moreover, the basis of this classification and the conclusions derived from it appear to be rather doubtful for the following reasons. (*i*) In view of the high rate of reaction 2 it is questionable whether methyl radicals can have been a major source of initiation, as they would be rapidly converted to methyl peroxy radicals. (*ii*) No account was taken of the different rates of decomposition of acetyl peroxide and cumene hydroperoxide induced by the different antioxidants. (*iii*) The division according to peroxide-decomposing ability is based on conditions that do not correspond to the situation existing during the early stages of oxidation. That is, the rate of peroxide (cumene hydroperoxide) decomposition was measured with an antioxidant to peroxide concentration ratio of 1:50 (170, 171), whereas during the early stages of oxidation the antioxidant is in large excess over the hydroperoxide. These small concentrations of antioxidant can obviously have little effect on the rate of decomposition of peroxide unless they decompose it catalytically. Moreover, it has been shown (166, 167) that if this reaction is studied over a range of concentration of antioxidant (both phenols and aromatic amines) relative to hydroperoxide, the rate goes through a minimum. That is, the antioxidant at first suppresses the induced chain decomposition (reaction 6) and reduces the rate to a certain minimum value corresponding to the molecular decomposition, but as its concentration is increased the rate invariably increases again, i.e., an induced decomposition becomes important. Because of these two opposing factors, measurements at a single concentration of inhibitor are quite useless in evaluating the behavior of antioxidant–peroxide systems. (*iv*) It would be very remarkable indeed if the members of Group I (e.g., diphenylamine, p-hydroxydiphenylamine, N-phenyl-β-naphthylamine) reacted only with R· radicals while structurally similar com-

pounds belonged to Group II (e.g., *p*-phenylenediamine, *p*-aminophenol, α-naphthylamine) or Group III (e.g., *m*-phenylenediamine, *o*-aminophenol, *N*-phenyl-α-naphthylamine, β-naphthylamine, *N,N'*-diphenyl-*p*-phenylenediamine) (171). Moreover, many of the compounds that inhibit polymerizations in the absence of oxygen (and therefore must react with R· radicals), such as quinones and nitrobenzenes (100, 174, 317), are much less efficient inhibitors of autoxidations than are the members of Group I (51). It appears therefore that this classification of antioxidants mainly reflects changes in the rates of reactions 9 (or 10) and 13.

IV. SYNERGISM AND ANTAGONISM

By using two or more different types of antioxidants the resistance to oxidation of an organic substrate can frequently be improved to an extent greater than would be predicted on the basis of strict additivity. The two antioxidants are then said to show a "synergistic" effect toward one another and the component which is least active, or even inactive, by itself is called a synergist for the second component. The converse of synergism is "antagonism."

Probably the most generally effective synergistic mixtures of antioxidants are those in which one compound functions as a decomposer of peroxides and the other as an inhibitor of free radicals. The latter prevents the formation of long reaction chains, but some hydroperoxide is nevertheless formed by reaction 13. If this hydroperoxide then reacts with a decomposer of peroxides, rather than by decomposing into free radicals, the two antioxidants act together to complement one another. Moreover, the decomposer of peroxides may itself be subject to oxidation by peroxy radicals (8, 13, 14, 144, 326), and its efficiency will therefore be increased in the presence of an inhibitor of free radicals. The best known examples of this type of synergism involve the use of an inhibitor of free radicals and a decomposer of peroxides of the sulfur or phosphorus type, including phosphoric acid and organic phosphatides (24, 111, 139, 153, 158, 175, 180, 257, 259, 331). The synergistic effects generally observed between the natural sulfur compounds and the polynuclear aromatics present in lubricating oil (206) have been attributed to oxidation of the sulfur compounds to sulfonic acids (a peroxide-decomposition reaction), followed by the acid-catalyzed rearrangement of the aromatic hydroperoxides to phenolic inhibitors. The high efficiency of many antioxidants containing two or more functional groups (e.g., OH and NH, OH and S, etc.) is also undoubtedly connected with the occurrence of a different inhibiting reaction at each group.

A large variety of amines which do not themselves function as inhibitors of free radicals (i.e., primary, secondary, and tertiary alkylamines and tertiary alkarylamines) can also apparently fulfill the role of decomposer of peroxides with sterically hindered phenols (41, 63, 167, 260, 294), with nonhindered phenols (41, 63, 66, 122, 251, 294), and with aromatic amines (66, 97, 225, 251). The occurrence of this synergism between alkylamines and inhibitors of free radicals (66, 97, 167, 251, 294) provides further proof that, in autoxidations, alkyl amine–hydroperoxide reactions leading to the production of free radicals are comparatively unimportant (see Section II,D). For example, polyalkylene polyamines enhance the activity of phenolic antioxidants for the stabilization of edible fats and oils (68), whereas they rapidly decompose tertiary hydroperoxides to give free radicals (333). The difference in their behavior would seem to be due to the formation of a different type of hydroperoxide (i.e., nontertiary) in the fats and oils. That is, these amines (and other alkylamines) very probably decompose secondary hydroperoxides mainly by a nonradical process (reaction 52), whereas with tertiary hydroperoxides the production of free radicals predominates (reaction 45). However, alkylamines can readily transfer an α-hydrogen atom to a free radical and this process may also contribute to their synergism with inhibitors of free radicals (see below).

Of considerable theoretical interest are combinations of what are generally regarded as strictly inhibitors of free radicals. The effects observed may be additive, antagonistic, or synergistic, depending on the inhibitors chosen and the substrate. As has already been mentioned, inhibitors of free radicals are generally able to decompose hydroperoxides (166, 167). This reaction has a very low rate with highly hindered phenols such as 2,6-di-*tert*-butyl-4-methylphenol. Therefore when synergism is observed with a highly hindered phenol as one component, the second component, which may be an aromatic amine (98, 167, 288) or another phenol (167, 197), probably functions as a decomposer of peroxides. Synergism has also been observed with mixtures of nonhindered phenols (67, 127, 135, 137, 167, 198), mixtures of nonhindered phenols and amine inhibitors (80, 122, 167, 169, 215, 252, 327), and mixtures of amine inhibitors (53, 167). In these cases also, one component probably functions as a decomposer of peroxides and the other as an inhibitor of free radicals.

Another interesting example of this type of synergism is the report that the resistance of mineral oils to oxidation is enhanced by mixtures of iron, nickel, or cobalt derivatives of alkyl dithiocarbamates, alkyl dithiophosphates, or alkyl xanthates and copper naphthenate or stearate (236). The first component must function as a decomposer of peroxides and the second is an example of copper functioning as an inhibitor of free radicals (*cf.* Section II,D) instead of as a prooxidant. The concentration of the copper compound

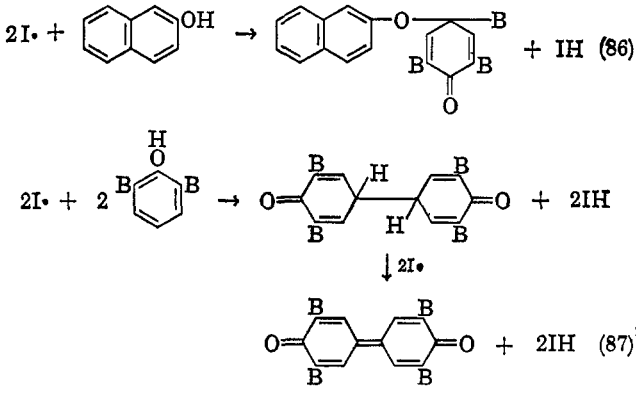
is, however, quite critical in this system; above a certain point it functions as a catalyst.

Mutual synergism of mixtures of peroxide decomposers has also been reported (133, 144, 180, 279). For example, alkyl triphenyl phosphites synthesized from mixtures of phenols are more effective antioxidants of mineral oil than any of the pure components (133). Similar effects might also be observed with mixtures of sulfides and selenides as decomposers of peroxides since, with catalysis by strong acids, selenoxides convert sulfides to sulfoxides and are themselves reduced to selenides (9), which are more potent decomposers of peroxides than are the sulfides (88).

Another mechanism that might give rise to synergism is the transfer of a hydrogen atom from the synergist to the inhibitor of free radicals after it has lost its active hydrogen to a peroxy radical, i.e.,

$$\mathrm{I}\cdot + \mathrm{SH} \rightarrow \mathrm{IH} + \mathrm{S}\cdot \qquad (85)$$

where SH represents the synergist. This mechanism is likely to be particularly favored with mixtures of inhibitors of free radicals, since even the less active component will give a resonance-stabilized free radical, S·. Reactions of this type have been observed between the 2,4,6-tri-*tert*-butylphenoxy radical (I·) and phenols, naphthols (243), other 4-substituted 2,6-di-*tert*-butyl phenols (208), and hydrazobenzene (75).



For equal concentrations of both components synergism due to these reactions cannot amount to a greater effect than that of twice the concentration of the stronger inhibitor. It has, however, been shown (167) that the synergistic effect on the induction period of a hydrocarbon oil at 160°C. of 2,6-di-*tert*-butyl-4-methylphenol (BMP) and certain less efficient nonhindered phenolic and amine inhibitors is greater than the effect observed with twice the concentration of BMP. For example, the induction periods in minutes obtained with the following inhibitors at 5×10^{-4} molar concentration by themselves, and with 5×10^{-4} molar BMP were, respectively: *p*-methoxyaniline, 30, 305; diphenylamine, 45, 325; *p*-methoxyphenol, 60, 345; *N*-phenyl-β-naphthylamine, 80, 335; and *N*-phenyl-α-naphthylamine, 125, 330; whereas BMP at 5×10^{-4} and at 10^{-3} molar concentrations gave induction periods of 170 and 275 min., respectively. It must, therefore, be concluded that even under these apparently favorable conditions hydrogen transfer cannot be solely responsible for synergism and these nonhindered inhibitors must owe an appreciable fraction of their inhibiting activity to their ability to decompose peroxides. However, the synergistic effects observed do not correlate very well with the relative rates of decomposition of peroxides by the different inhibitors (167). This lack of correlation is probably due to two main factors. Firstly, a nonhindered inhibitor that is a fairly weak decomposer of peroxides is also a fairly weak inhibitor of free radicals, since the rates of both reactions depend on the availability of electrons at the reactive center of the molecule (142, 166, 322). Such a compound may, therefore, show just as large synergistic effects with BMP as would be shown by a strong decomposer of peroxides, since a larger proportion of the latter will be destroyed by free radicals. Secondly, the radicals derived from the two inhibitors may undergo a rapid cross-combination reaction, i.e.,

$$\mathrm{I}\cdot + \mathrm{I}'\cdot \rightarrow \mathrm{II}' \qquad (88)$$

e.g., the second step in reaction 86. Provided the rate of this reaction is greater than that of the two self-recombination reactions, both inhibitor radicals are prevented from reacting with peroxy radicals (reactions 67 and 68) and therefore the inhibiting power of the combined inhibitors is decreased. This effect may even be large enough to overcome the factors promoting synergism, in which case the two antioxidants will be mutually antagonistic.

Many other combinations of additives have been reported to show synergistic effects. Although some of the combinations suggest that one component is an inhibitor of free radicals and the other a decomposer of peroxides, not all the results can be explained in this way. An interesting modification of the usual cause of synergism is frequently observed when the inhibitor of free radicals is also a strong decomposer of peroxides. In this case, its performance can frequently be improved by the addition of certain compounds that are completely ineffective by themselves. Since the additives alone are ineffective, it is improbable that they complex with hydroperoxide and prevent its decomposition into free radicals, particularly since such complexes generally accelerate the decomposition of peroxides (*cf.* Section II,D). These additives must, therefore, either preserve or regenerate the inhibitor. Preservation appears to involve a weak additive–inhibitor complex, which can no longer react with peroxides but can still react with free radicals. In this way the inhibitor is conserved for its more important role. This mechanism may also apply to some of the examples of synergism mentioned above. Regeneration of the inhibitor must generally

involve hydrogen transfer from the additive to the inhibitor radical (reaction 85).

In edible fats and oils that are free of natural inhibitors ascorbic and citric acids are generally completely ineffective inhibitors of autoxidation (136, 292). They sometimes show weak activity in impure substrates, but this is probably due to traces of natural inhibitors or to their metal-chelating ability (112, 127, 292). These acids show pronounced synergistic effects toward the stabilization of fats by phenolic inhibitors (25, 127, 198), particularly two naturally occurring and widely used compounds, nordihydroguaiaretic acid (NDGA) and the tocopherols (vitamin E) (40, 259). These natural phenolic inhibitors, or possibly their oxidation products, are strong decomposers of peroxides (258, 299), but in the presence of citric or ascorbic acid this reaction is suppressed (258). The synergistic effect of the acids is, therefore, probably due to the formation of a weak complex with the inhibitors which can react only with free radicals. This mechanism probably also accounts for the synergistic effect of the esters of these acids and phenolic inhibitors (193, 265). For example, tristearyl citrate is a synergist for the inhibition of the autoxidation of petroleum waxes by *tert*-butylhydroxyanisole and by lauryl gallate (101). But the complexity of synergistic effects is shown by the fact that, in the same substrate, tristearyl citrate antagonizes propyl gallate and triisopropyl citrate antagonizes both lauryl and propyl gallates, while citric acid is almost ineffective (101). Hydroxy acids and esters can also synergize aromatic amine inhibitors of free radicals, probably by the same mechanism. For example, ascorbic acid and ascorbyl palmitate are synergists for the stabilization of automotive gasolines by *N,N'*-di-*sec*-butyl-*p*-phenylenediamine (156).

Nordihydroguaiaretic acid and the tocopherols act only as inhibitors of the oxidation of fat at low concentrations; above a certain critical concentration they become proöxidants (259, 299), probably because of their direct oxidation by attack of molecular oxygen at their alpha carbon–hydrogen bonds rather than by reaction with hydroperoxides to give free radicals, as has also been suggested (258). Citric and ascorbic acids may be sufficiently strong acids to catalyze the rearrangement of the hydroperoxide formed from the inhibitor (reaction 50) to give polyhydroxyphenols of high inhibiting power. Since this reaction is catalytic it may explain why these acids show a greater synergistic effect at low rather than at high concentrations (259), where they may themselves be subject to direct oxidation. This rearrangement will undoubtedly be partly responsible for the synergism observed in these systems when phosphoric acid replaces the hydroxy acids (222, 259), although, in this case, some direct decomposition of fat hydroperoxides will occur concurrently (257). The suggestion (60) that the phosphoric acid merely absorbs excess energy from activated fat molecules seems very improbable.

Synergism due to donation of hydrogen by the synergist was originally proposed (61) to account for the inhibition of the oxidation of fat by mixtures of ascorbic acid and quinones, since both components are rather inactive by themselves. The ascorbic acid disappears very rapidly during the induction period and it was assumed that the acid, or its oxidation products, reduced the quinone to a semiquinone or hydroquinone (216), which then reacted with peroxy radicals. Ascorbic acid (and also citric acid) can probably act as a hydrogen donor, since it reduces slightly the rate of decomposition of fat hydroperoxide *in vacuo* (258), presumably by trapping free radicals and suppressing the self-induced chain decomposition (reaction 6). Donation of hydrogen was later extended to cover the synergistic action of ascorbic acid with tocopherols (135, 222). In this system the ascorbic acid is destroyed more slowly in the presence of tocopherol than in its absence (259), from which it has been concluded (259) that the acid does not function as a hydrogen reservoir. This conclusion is, however, quite unjustified since the tocopherol, by lowering the steady-state concentration of peroxy radicals, also reduces the rate of oxidation of the ascorbic acid by these radicals (i.e., acid and inhibitor exert a mutual sparing action on one another).

The hydrogen-donation reaction also seems to apply to several other mixtures. For example, α-amino acids (but not β-amino acids) are strong synergists for phenolic antioxidants (69, 152). The action of α-alanine on the hydroquinone-inhibited autoxidation of sunflower oil is reportedly due to its oxidative deamination to pyroracemic acid, which is able to reduce the oxidized hydroquinone (152). The synergistic effects of alkyl phosphonates on hindered phenols may also be partly due to this cause (190a).

Two additives which show synergistic effects in one substrate may be antagonistic toward each other in a different substrate (220, 251). However, if both components are individually active, many of the cases of antagonism reported in the literature are probably unrecognized examples of additivity or synergism since, in the absence of an initiator, a first-order relation between concentration of inhibitor and the degree of inhibition observed over a reasonable range of concentration is by no means as general as has frequently been suggested. As an example (167) 2,6-di-*tert*-butyl-4-methylphenol (BMP) and 2,4,6-tri-*tert*-butylphenol (TBP) at 5×10^{-4} molar concentrations gave induction periods of 170 and 110 min., respectively, for the autoxidation of a hydrocarbon oil at 160°C. The induction period for the combined antioxidants at the same concentrations was 230 min., appreciably below the value calculated on the assumption of simple additivity. However, an induction period of 110 min.

was also obtained with 2.5×10^{-4} molar BMP. Therefore, in the mixture the two inhibitors are equivalent to 7.5×10^{-4} molar BMP, a concentration which was found to give an induction period of 230 min., in agreement with the value obtained with the mixture, i.e., the effects of these two inhibitors are, in actual fact, additive. Genuine cases of antagonism have, however, been fairly frequently observed between inhibitors of free radicals of all chemical types (102, 117, 167, 169, 180, 250). This antagonism is probably generally due to the destruction of the radicals derived from the inhibitors by the rapid cross-combination reaction (reaction 88). As a consequence, these radicals are no longer available to destroy peroxy radicals. In some cases, particularly when fairly acidic phenols and fairly basic amines are involved (e.g., phenol and 2,4-dimethylaniline (169)), antagonism may also be partly, or wholly, due to the formation of a complex between the two inhibitors which is inactive toward peroxy radicals. The antagonistic effect of fatty acids on both phenolic and aromatic amine inhibitors (169, 249) may also be due to complex formation. Alternatively, it might be due to the formation of free radicals, either directly from the fatty acid (235) or by an induced decomposition of hydroperoxide (46, 65), although it is difficult to see why citric and ascorbic acid would not function in the same way. Antagonism may also, occasionally, be due to the reverse of reaction 85, i.e., transfer of hydrogen from the stronger inhibitor to the radical of the weaker inhibitor. This must, however, be a rather uncommon process, since the stronger inhibitor will tend to be consumed first.

In conclusion, synergism implies, of necessity, that the two component antioxidants play different roles during inhibition. Moreover, in a given mixture of antioxidants synergism may be due to more than one cause; e.g., a combination of an inhibitor of free radicals with a synergist that can decompose peroxides and can also donate hydrogen. Antagonism suggests that the two components interact directly with one another. The occurrence of true additivity over a range of concentration with two individually active components suggests that both of them are probably active in only a single inhibiting reaction.

V. References

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