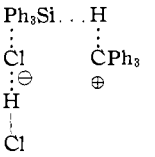
more positive in going to the transition state.[19] The reaction is markedly catalyzed by added hydrogen chloride, but the catalyzed reaction still seems to show first-order dependence on silane and halide (Fig. 3). Hydrogen chloride may possibly serve to promote the dissociation of the triphenylmethyl halide into the carbonium ion salt $Ph_3C^+ HCl_2^-$,[20] which would also function as an ion pair in the transition state



A similar four-center mechanism has been suggested by Russell for the reaction of silanes with alkyl halides in the presence of aluminum chloride.[21] The reaction of trityl halides with silanes may have some preparative value. Other methods are available for the conversion of silanes to silyl halides, but the conditions required are generally more drastic than those for the trityl halide reaction which may therefore be useful in halogenating sensitive silicon–hydrogen compounds. Also, it is often difficult to synthesize compounds containing both hydrogen and halogen bonded to silicon. Methods which have been used include the addition of a deficiency of a Grignard reagent to a halide such as $SiHCl_3$[22,23] or the reaction of a mixture of a dichlorosilane with a dihydride in the presence of aluminum chloride.[16] A troublesome separation step is necessary to obtain the desired product in either of these methods. The reaction of equimolar amounts of triphenylchloromethane and diphenylsilane in benzene led to a good yield of diphenylchlorosilane, Ph_2SiHCl, and it is possible that the reaction will be generally useful for the preparation of such mixed hydride–halides. For preparative purposes, the use of benzene as a solvent is especially convenient because nearly all of the triphenylmethane by-product precipitates from this solvent when the reaction mixture is concentrated.

Acknowledgments.—The authors are grateful to the Air Force Office of Scientific Research for a grant in support of this research; to the American Oil Foundation of the American Oil Co. for a grant to purchase the infrared spectrophotometer used in these studies, and to Prof. H. J. Dauben, Jr., and Mr. S. W. Tobey for helpful discussions.

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[CONTRIBUTION FROM THE DEPARTMENT OF CHEMISTRY, CASE INSTITUTE OF TECHNOLOGY, CLEVELAND 6, OHIO]

Retarded Autoxidation and the Chain-Stopping Action of Inhibitors

BY J. REID SHELTON AND DAVID N. VINCENT

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Oxidation of purified *cis*-1,4-polyisoprene, inhibited with secondary aromatic amines and a hindered phenol, was studied to determine if the kinetic isotope effects previously observed with deuterated oxidation inhibitors could be demonstrated at an earlier stage of the reaction. A linear plot for oxygen absorption *vs.* time demonstrated isotope effects in the initial stage of oxidation consistent with a rate-controlling hydrogen-abstraction process for the chain-stopping action. The decrease in apparent observed isotope effect with increasing inhibitor concentration and increasing oxidation temperature has been confirmed. This, and the observed change in inhibitor action from antioxidant to pro-oxidant with increasing inhibitor concentration, indicates that direct oxidation of inhibitor is a significant initiation process. The oxygen-absorption plots obtained in this study showed two discrete stages of retarded oxidation. The amount of oxygen absorbed at the well defined break between the initial stage and the faster second stage was independent of temperature and inhibitor concentration. It is suggested that this phenomenon is due to the onset of bimolecular hydroperoxide decomposition as a major initiation process. This hypothesis is supported by the observation that oxidation of samples preoxidized to the second stage, and then heated in a nitrogen atmosphere to decompose peroxides, again showed two constant-rate stages of retarded oxidation.

Introduction

The free-radical chain mechanism of autoxidation of organic materials is well established.[1,2] In the absence of oxidation inhibitors or added initiators, the only important initiation process involves peroxide decomposition, and the principal termination reaction involves the combination of two peroxy radicals. In the presence of materials which inhibit or retard the reaction of oxygen with organic substances, important changes occur in both the initiation and termination processes. Recent reviews on the subject of inhibition of oxidation are available.[3,4]

The rate of autoxidation of hydrocarbons in the absence of inhibitors is nearly independent of oxygen concentration,[2] but the rate of retarded oxidation in the presence of antioxidants increases in a regular fashion as the partial pressure of oxygen is increased.[5] This change reflects a shift in the initiation process from exclusively peroxide initiation to include reactions involving direct attack of oxygen upon the antioxidant, and possibly upon the hydrocarbon. The formation of free radicals as a result of direct oxidation of the inhibitor is also indicated by the observation that antioxidants often become pro-oxidants at concentrations greater than the optimum. Additional evidence for this reaction is provided in this paper and related studies.[6,7]

The important termination reaction in the mechanism of retarded autoxidation is the chain-stopping action of inhibitors. The widely used hindered phenol and secondary arylamine types probably function by donating hydrogen to a peroxy free radical. The resulting inhibitor free radical may combine with another $RO_2\cdot$ or it may participate in a chain-transfer process. Various kinetic studies[8,9] and product isolations[9-11] have given results that are in agreement with this mechanism.

(1) J. L. Bolland, *Quart. Rev.* (London), **3**, I (1949).
(2) L. Bateman, *ibid.*, **8**, 147 (1954).
(3) J. R. Shelton, *Rubber Rev. Rubber Chem. Technol.*, **30**, 1270 (1957); *Official Digest, Fed. Soc. Paint Technol.*, **34**, 590 (1962).
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$$InH + RO_2\cdot \longrightarrow In\cdot + ROOH$$
$$In\cdot + RO_2\cdot \longrightarrow \text{products}$$

Several researchers have sought to test this mechanism by looking for the predicted kinetic isotope effect when the active hydrogen of the inhibitor is replaced by deuterium. Hammond and co-workers[12,13] found no isotope effect on the rate of oxygen absorption of solutions of cumene or tetralin in chlorobenzene at 60–70° initiated by azobisisobutyronitrile (AIBN) and inhibited by several different N-deuterated aromatic amines. They concluded that some other mechanism should be considered. Pedersen[14] studied the oxidation of cracked gasoline stocks by the ASTM induction period method and found no significant change when the active hydrogens of N,N′-diphenyl-*p*-phenylenediamine were replaced by deuterium. A kinetic deuterium isotope effect has been demonstrated in this Laboratory[6,7] with both aromatic amine and phenolic type inhibitors by oxygen-absorption measurements on styrene–butadiene rubber polymer at 90°. Ingold[15] observed small isotope effects in the induction period for uninitiated oxidation of saturated white mineral oil at 160° which were originally reconciled with Hammond's mechanism. We have maintained that isotope effects as large as 1.8, obtained in our studies, are sufficient to show that the hydrogen-donation mechanism is involved.

The alternative mechanism proposed by Hammond is that the rate-determining step of the inhibition reaction is the equilibrium formation of a charge-transfer complex between peroxy radicals and the aromatic nucleus of the inhibitor with a subsequent rapid reaction of the complexed radical with a second peroxy radical

$$InH + RO_2\cdot \rightleftharpoons (InH, RO_2\cdot)$$
$$(InH, RO_2\cdot) + RO_2\cdot \longrightarrow \text{products}$$

This interpretation is supported by their observation[13] that the rate of oxygen absorption in the presence of certain amines is inversely proportional to the square root of the inhibitor concentration which "seems to demand some sort of reversible interaction between inhibitor and peroxy radicals." The possibility that the hydrogen-abstraction reaction might be reversible was also considered, but addition of hydroperoxide did not reduce the effectiveness of the inhibitor.[16]

Bickel and Kooyman[9] have reported a wide variety of kinetic laws exhibited by oxidation systems inhibited by phenols and amines. These included examples of the same kinetic variation which formed the basis for Hammond's mechanism. However, these authors showed that this inverse square root dependence can arise from a chain-transfer reaction in which the inhibitor radical, formed by hydrogen abstraction, reacts with the hydrocarbon substrate under oxidation conditions to re-form a reactive free radical. Bickel and Kooyman[17] have also shown that abstraction of hydrogen from 2,6-di-*tert*-butyl-4-methylphenol by diphenylpicrylhydrazyl radicals shows a significant deuterium isotope effect.

Harle and Thomas[18] used electron paramagnetic resonance to detect free radicals and to follow changes in the concentration of radicals in the oxidation of hydrocarbons in the presence of amine inhibitors. Their initial results were interpreted as being "closer to the prediction of Boozer and Hammond," but when Thomas[19] established the identity of the free radical, it was found to be a relatively stable diarylnitric oxide free radical which could be formed by a sequence of reactions including abstraction of hydrogen from the amine. No stabilized radicals were found in the systems inhibited with phenols. A recent paper by Thomas and Tolman[20] reconciles the variation in concentration of the diphenylnitric oxide, as observed in the oxidation of cumene in chlorobenzene in the presence of diphenylamine, with the Boozer and Hammond kinetics which ignore the possibility of a chain-transfer reaction. They also report that they looked for a deuterium isotope effect between the amine and $RO_2\cdot$ radicals, but did not find one.

In a recent review, Ingold[4] has questioned the validity of our interpretation of the isotope effects observed[6,7] on the basis that the effects were observed at a fairly late stage in the oxidation process. We had also been concerned about this limitation of the data, and the present study was undertaken to find a suitable system which would minimize the nonlinear character of the initial stages of oxidation. The work of Lorenz and Parks,[21] in which vulcanized rubber samples were extracted with solvents prior to oxidation, indicated that the initial rapid rate of oxidation was due to extractable nonrubber components.

A synthetic *cis*-1,4-polyisoprene was chosen for this study since it can be subjected to a relatively efficient purification procedure. The polymer is also more easily oxidized than the styrene–butadiene rubber previously used, thus allowing studies to be made at lower temperatures. We preferred a solid nonvolatile material to the use of low molecular weight liquid model compounds. Oxidation of the purified hydrocarbon gave the desired linear oxygen-absorption curve in the presence of inhibitors, and thus made possible this study of the deuterium isotope effect in the initial stages of oxidation.

Experimental Procedures

Purification of Substrate.—Purification of synthetic *cis*-1,4-polyisoprene, obtained from the Goodyear Tire & Rubber Co., was carried out in a glove-box under a nitrogen atmosphere. The polymer, cut into approximately 1-cm. cubes, was extracted for 48 hr. with acetone at reflux to remove the major portion of the phenolic inhibitor originally present. It was then dissolved in benzene, at reflux, to give a solution of approximately 5 to 7% polymer, which was filtered through four layers of nylon mesh and a layer of Pyrex wool to remove insoluble gel. An equal volume of a 5% solution of sulfuric acid in methanol was prepared and slowly added to the rapidly stirred polymer solution until the polymer just began to precipitate. After 2 hr., the remaining methanolic sulfuric acid was added plus three additional volumes of unacidified methanol, and the precipitated polymer washed by kneading in two portions of fresh methanol. The washed polymer was redissolved in benzene at room temperature, reprecipitated by slowly adding the polymer solution to three volumes of rapidly stirred acetone, and washed twice with fresh acetone. Three additional dissolutions in benzene and precipitations in acetone were made. Finally, the precipitated polymer was dried *in vacuo* for 96 hr. at room temperature and stored under nitrogen. It was expected that the acid treatment would remove and/or decompose catalyst residues and preformed peroxides, and the subsequent precipitations would remove any low molecular weight impurities.

Deuteration of Inhibitors.—N-Phenyl-2-naphthylamine, 2,6-di-*tert*-butyl-4-methylphenol, and N,N′-diphenyl-*p*-phenylenediamine were deuterated by exchange with deuterium oxide in a manner similar to that described by Shelton, McDonel, and Crano,[7,22] using cyclohexane, carbon tetrachloride, and chloro-

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(15) K. U. Ingold and I. E. Puddington, *ibid.*, **51**, 1322 (1959).
(16) G. S. Hammond and U. S. Nandi, *J. Am. Chem. Soc.*, **83**, 1217 (1961).
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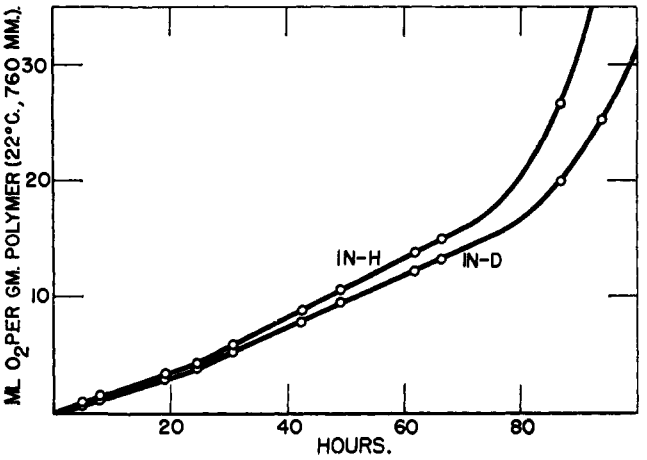

Fig. 1.—Oxidation of polyisoprene at 90°, 760 mm. oxygen pressure; inhibitor, N-phenyl-2-naphthylamine, 8.82 × 10^{-5} mole per gram.

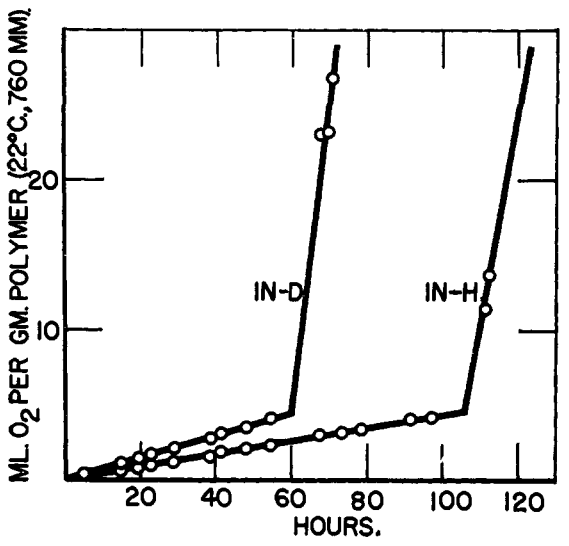

Fig. 2.—Oxidation of polyisoprene at 75°, 760 mm. oxygen pressure; inhibitor, N-phenyl-2-naphthylamine, 4.41 × 10^{-5} mole per gram.

benzene–ligroin, respectively, as solvents for the inhibitors. The extent of deuteration of the inhibitors, after separation from the water layer and evaporation of solvent, was determined by measuring the infrared absorbance of the remaining N–H (or O–H) in a solution of known molar concentration and relating to the appropriate calibration plot of absorbance *vs.* concentration of inhibitor. It was approximated that this method gives results within 5% of the true deuterium concentration. The extent of deuteration, as determined by infrared absorption, was 70% for N-phenyl-2-naphthylamine, 94% for 2,6-di-*tert*-butyl-4-methylphenol, and 95% for N,N'-diphenyl-*p*-phenylenediamine.

Samples of undeuterated inhibitors for use in the oxidation studies were dissolved in the appropriate solvent and treated with water in the same manner as in the deuteration procedure.

Preparation of Oxidation Samples.—The desired amounts of polymer, inhibitor, and nitrogen-purged, azeotropically dried benzene (sufficient to give an approximately 10% polymer solution) were mixed in a capped bottle on a roller until the polymer dissolved. Polymer films obtained by evaporation of the solvent under a nitrogen atmosphere were pressed (under 3000 p.s.i. at room temperature) onto a tared aluminum screen which had been washed with benzene and acetone. The samples were vacuum dried at room temperature to remove the last traces of solvent and weighed. The samples contained from 1.5 to 2.0 g. of polymer. The sample thickness could not be accurately measured, owing to the woven nature of the supporting screen, but it was estimated that the polymer film thickness was less than 0.025 in.

Oxygen-Absorption Studies.—The volumetric oxygen-absorption technique, described in previous publications from this Laboratory,[23-25] was used to measure the rate of oxygen uptake

(23) J. R. Shelton and H. Winn, *Ind. Eng. Chem.*, **32**, 71 (1946).

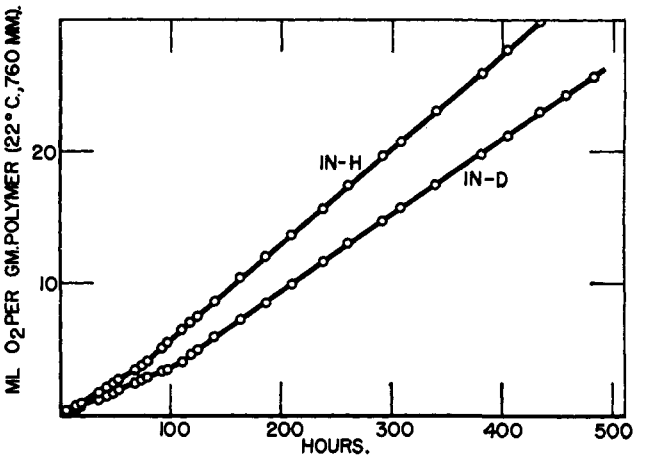

Fig. 3.—Oxidation of polyisoprene at 75°, 760 mm. oxygen pressure; inhibitor, N-phenyl-2-naphthylamine, 13.2 × 10^{-5} mole per gram.

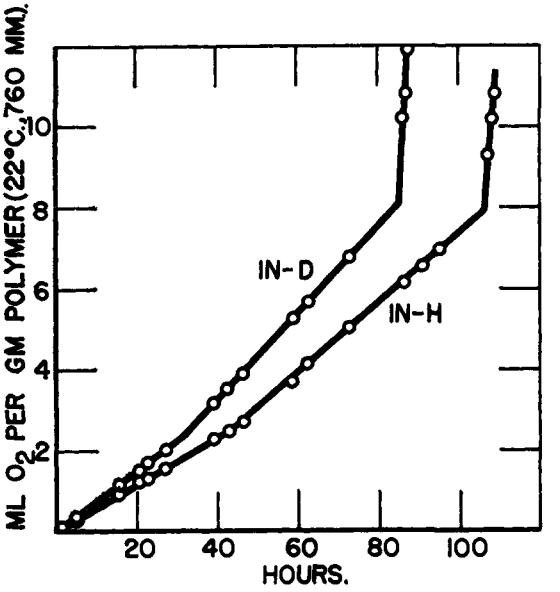

Fig. 4.—Oxidation of polyisoprene at 90°, 760 mm. oxygen pressure; inhibitor, 2,6-di-*tert*-butyl-4-methylphenol, 4.41 × 10^{-5} mole per gram.

by the polymer. All oxygen volumes were measured at 760 mm. with temperature corrections to 22°. The data reported represent averages of values obtained with two or more samples.

Several preliminary determinations of the oxidation rates were made in order to check the methods used in the preparation of samples. Samples of unpurified polymer prepared by the standard method, as well as similar samples containing twice and one-half the usual weight of polymer, all gave essentially the same oxidation rate at 90°, indicating that sample weight and corresponding variations in sample thickness were not controlling factors.

Results and Discussion

Samples of polymer purified by the method described and oxidized at 90° in the absence of added inhibitors showed an extremely rapid rate of oxygen absorption corresponding to approximately 18 ml. of O_2 per gram of polymer per hour. The unpurified polymer absorbed 0.066 ml. of O_2 per gram per hour.

The oxygen-absorption rate plots obtained with inhibited samples were reproducible, and showed the presence of three definite stages, as illustrated by Fig. 1 and 4. There is an initial linear absorption, followed by a more rapid linear absorption, and, finally, by the rapidly in-

(24) G. W. Blum, J. R. Shelton, and H. Winn, *ibid.*, **43**, 464 (1951).

(25) J. R. Shelton and E. T. McDonel, *J. Appl. Polymer Sci.*, **1**, 336 (1959).

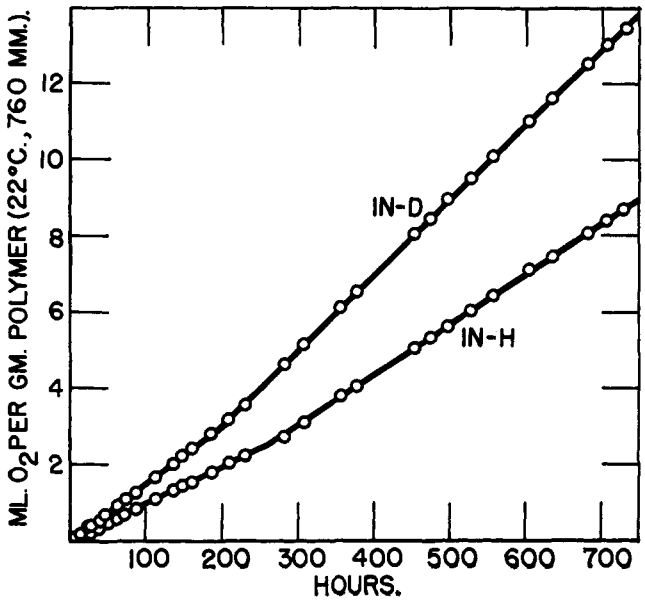

Fig. 5.—Oxidation of polyisoprene at 75°, 760 mm. oxygen pressure; inhibitor, 2,6-di-*tert*-butyl-4-methylphenol, 8.82 × 10^{-5} mole per gram.

creasing autocatalytic stage. The kinetic isotope effects were calculated from the slopes (oxygen-absorption rates) of the initial linear stages and also from the second linear stage when the data were available. The good agreement in the values from the initial stage and second linear stage of oxidation support the original premise of the earlier papers from this Laboratory[6,7] that the presence of a constant-rate stage, even rather late in the oxidation, indicates that the oxidation rate is still controlled by the inhibitor, and that kinetic isotope effects related to the inhibitor can also be determined in this stage. In addition, the kinetic isotope effect can be determined by the ratio of the induction periods, defined as the time to the onset of the autocatalytic stage. In the cases where these data are available, the isotope effects calculated in this manner are in agreement with those obtained from the rate data.

The Kinetic Deuterium Isotope Effect.—A definite and relatively large kinetic isotope effect has been observed in these systems. The decrease in the kinetic isotope effect with increasing inhibitor concentration and with increasing oxidation temperature that was previously reported[6,7] has been verified. In contrast to these previous reports where only qualitative data could be obtained at lower inhibitor concentrations, owing to the absence of a well defined constant-rate stage, the present study quantitatively demonstrates the regular change in magnitude of the isotope effect which ultimately results in a reversal of the direction with both temperature and inhibitor concentration.

Typical oxygen-absorption curves for purified *cis*-1,4-polyisoprene inhibited with N-phenyl-2-naphthylamine are shown in Fig. 1–3. In each case a linear plot of oxygen absorbed *vs.* time was obtained for the initial stage. Figures 1 and 3 exhibit two linear stages of retarded oxidation with a sharp break at approximately 4 ml. of O_2 absorbed per gram of polymer. In Fig. 2, the autocatalytic stage begins before this critical value is attained. The significance of this unusual observation of two successive stages of retarded oxidation will be discussed after consideration of the isotope effects observed.

Figure 2 shows an isotope effect in the expected direction if the arylamine inhibitor functions by giving up its reactive hydrogen to an $RO_2\cdot$ radical. Figure 3 demonstrates that the direction of the isotope effect can be reversed by increasing the concentration of inhibitor.

Table I includes data obtained at three temperatures and with three concentration levels of N-phenyl-2-naphthylamine. The rate data at 75° illustrate the frequently observed shift from antioxidant to pro-oxidant behavior with increasing amine concentration.[3,5] The minimum rate was observed at the intermediate concentration which corresponds to approximately two parts of inhibitor per hundred of polymer. The isotope effect (R_D/R_H) of 1.71 observed at the lower concentration is canceled out by the competing pro-oxidant effect at the optimum concentration, and reversed in direction at the higher concentration. The largest isotope effect detected in this study was 2.14 observed with the intermediate concentration at 60° and with 70% deuteration of the inhibitor. Increasing the temperature to 90° at the same concentration level canceled this effect and reversed the direction, as shown in Fig. 1, consistent with a higher activation energy associated with the competing pro-oxidant process.

TABLE I

OXYGEN ABSORPTION RATES FOR *cis*-1,4-POLYISOPRENE INHIBITED WITH N-PHENYL-2-NAPHTHYLAMINE

Temp., °C.	Inhib.	Inhib. concn., moles/ × 10^5	Rate, ml. O_2/g./hr. (22°, 760 mm.) Initial stage	Second stage	Isotope effect R_D/R_H Initial stage	Second stage
90	InH	8.82	0.174	0.253	0.86	0.89
	InD		.150	0.226		
75	InH	4.41	.0436	...		
	InD		.0744	...	1.71	...
75	InH	8.82	.0130	0.0172		
	InD		.0134	.0169	1.03	0.98
75	InH	13.2	.0513	.0709		
	InD		.0370	.0596	0.72	0.84
60	InH	8.82	.00215	...		
	InD		.00460	...	2.14	..
60	InH	13.2	.0147	0.0183		
	InD		.0126	0.0154	0.86	0.84

Similar effects were exhibited in oxidations inhibited with 2,6-di-*tert*-butyl-4-methylphenol. Figure 4 shows the three stages of oxidation, and Fig. 4 and 5 exhibit the sharp break between two linear stages of retarded oxidation with significant isotope effects in both cases. The largest isotope effect observed with this inhibitor, as reported in Table II, was 1.76 obtained at 60° with

TABLE II

OXYGEN ABSORPTION RATES FOR *cis*-1,4-POLYISOPRENE INHIBITED WITH 2,6-DI-*tert*-BUTYL-4-METHYLPHENOL

Temp., °C.	Inhib.	Inhib. concn., moles/g. × 10^5	Rate, ml. O_2/g./hr. (22°, 760 mm.) Initial stage	Second stage	Isotope effect R_D/R_H Initial stage	Second stage
90	InH	4.41	0.0584	0.0861	1.27	1.25
	InD		.0750	.1077		
90	InH	13.2	.0273	.0393	0.79	0.82
	InD		.0216	.0321		
75	InH	8.82	.00968	.0132	1.56	1.49
	InD		.0151	.0197		
75	InH	13.2	.00710	.00962	0.92	0.95
	InD		.00653	.00913		
60	InH	4.41	.00251	....	1.76	..
	InD		.00441	0.00549		
60	InH	13.2	.00124	....	1.16	..
	InD		.00144	....		

the lower concentration of the hindered phenol which was 94% deuterated. The largest reverse isotope effect, R_D/R_H = 0.79, was observed at the highest temperature, 90°, and highest inhibitor concentration. The

effect of concentration upon magnitude and direction of the isotope effect is evident at each temperature studied, and comparison of any one concentration at different temperatures shows that these effects are the same for the phenol as for the amine.

A third type of inhibitor included in this study was N,N′-diphenyl-*p*-phenylenediamine. Figure 6 shows the same sharp break between the initial stage and the second stage of retarded oxidation, and also illustrates the largest reverse isotope effect observed in this study, $R_D/R_H = 0.64$, obtained at the higher concentration of inhibitor. Although this inhibitor was studied in less detail than the other two, the data of Table III show a similar effect of concentration upon magnitude and direction of the isotope effect.

One may question whether the observed isotope effect arises from the hydrogen abstraction reaction or from a nonradical peroxide decomposition reaction. Although these inhibitors can, in many cases, act as peroxide decomposers, Hawkins[26] has stated that secondary aromatic amine and hindered phenol type inhibitors do not decrease the peroxide content when added to a partially oxidized cumene substrate. Furthermore, it has been demonstrated that similar hydrogen abstractions by radicals do lead to sizable kinetic deuterium isotope effects.[17,27] The isotope effect associated with the nonradical decomposition of peroxides by phenols[28] has been shown to be small (about 1.3). The considerably larger values we have obtained suggest that the observed isotope effects are mostly due to the hydrogen-abstraction process.

The variation of the isotope effect from a significant value in the predicted direction to a significant inverse value can be explained, as before,[7] by the occurrence of two competing reactions. Deuteration of inhibitor would slow down the hydrogen-abstraction chain-stopping reaction, and the observed rate of oxygen absorption would increase, leading to a kinetic isotope effect (R_D/R_H) greater than one. Deuteration of inhibitor would also slow down the direct oxidation of inhibitor, which leads to the formation of a rather active radical ($HO_2\cdot$), which would act as an initiator: $InH + O_2 \rightarrow In\cdot + HO_2$. Depending on the relative contribution of this initiation reaction, which is favored by higher temperatures and higher inhibitor concentrations, the kinetic isotope effect due to the hydrogen-abstraction chain-stopping reaction can be reduced or completely submerged to give a kinetic isotope effect of less than one.

TABLE III

OXYGEN ABSORPTION RATES FOR *cis*-1,4-POLYISOPRENE INHIBITED WITH N,N′-DIPHENYL-*p*-PHENYLENEDIAMINE

Temp., °C.	Inhib.	Inhib. concn., moles/g. × 10^5	Rate, ml O_2/g./hr. (22°, 760 mm.) Initial stage	Rate, ml O_2/g./hr. (22°, 760 mm.) Second stage	Isotope effect R_D/R_H Initial stage	Isotope effect R_D/R_H Second stage
75	InH	4.41	0.0216	....	1.18	..
	InD		.0255	....		
75	InH	13.2	.0592	0.0776	0.64	0.68
	InD		.0381	0.0526		

Recent communications by Ingold and Howard[29] report a study of the AIBN-initiated oxidation of cumene at 65° in the presence of 2,6-di-*tert*-butyl-4-methylphenol. They have found that the deuterated phenol is susceptible to a very rapid exchange with traces of moisture or hydroperoxide in the reaction mixture which probably accounts for the failure of many

(26) W. L. Hawkins, paper presented to the Akron Polymer Lecture Group, May 4, 1962.

(27) G. A. Russell, *J. Am. Chem. Soc.*, **79**, 3871 (1957).

(28) C. Walling and R. B. Hodgdon, *ibid.*, **80**, 228 (1958).

(29) K. U. Ingold and J. A. Howard, *Nature*, **195**, 280 (1962); *Can. J. Chem.*, **40**, 1851 (1962).

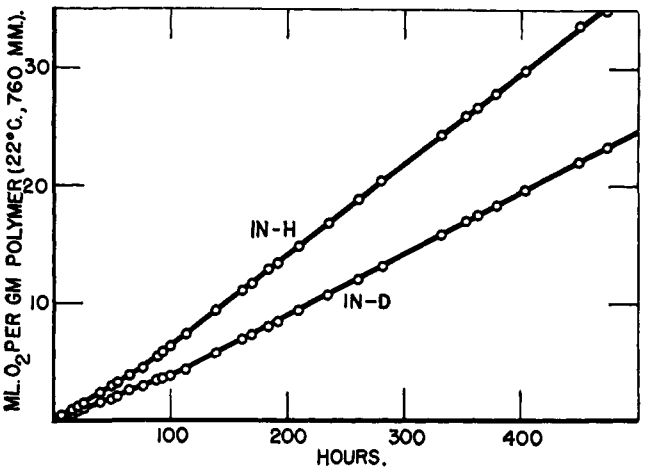

Fig. 6.—Oxidation of polyisoprene at 75°, 760 mm. oxygen pressure; inhibitor, N,N′-diphenyl-*p*-phenylenediamine, 13.2 × 10^{-5} mole per gram.

workers[11,14,20] to observe the expected isotope effect. This was previously suggested by Shelton, McDonel, and Crano[7] and rejected by Hammond and co-workers.[16] (This effect is apparently less important in a rigid medium such as SBR polymer or polyisoprene.) By adding deuterium oxide to the reaction mixture to keep the inhibitor in the deuterated state, Ingold and Howard[29] observed an isotope effect of 4.2. In similar experiments using styrene as a substrate, an isotope effect of 10.6 was observed. They conclude that the inhibition mechanism is a normal hydrogen-abstraction process exhibiting a kinetic isotope effect of about the expected magnitude for a transition state involving appreciable stretching of the O–H bond. These results give strong support to the position that we have maintained for some time,[3,6,7] that there is an isotope effect, and that it establishes the hydrogen-donation reaction as the rate-controlling step in the chain-stopping action of oxidation inhibitors.

The "Break" in the Oxygen-Absorption Rate Plot.—The plots of oxygen absorption *vs.* time in Fig. 1, 3, 4, 5, and 6 show the presence of two linear stages of retarded oxidation. The change in slope from the initial oxidation stage to the faster rate of oxygen absorption in the second stage produces a relatively sharp break in the plot. The position of this break with respect to the time axis is variable, apparently dependent upon the rate of the initial oxidation. The quantity of absorbed oxygen at the break is nearly independent of both the oxidation temperature and the inhibitor concentration and appears to be only a function of the particular inhibitor used. The observed oxygen-absorption levels at the break between the initial and second stage of retarded oxidation are reported in Table IV. This in-

TABLE IV

OXYGEN-ABSORPTION LEVELS AT THE BREAK POINT

Inhibitor	Inhib. concn., moles/g. × 10^5	Temp., °C.	Isotope effect R_D/R_H	Abs. at break, ml. O_2/g. (22°, 760 mm.) InH	Abs. at break, ml. O_2/g. (22°, 760 mm.) InD
N,N′-Diphenyl-*p*-phenylenediamine	13.2	75	0.64	4.7	4.3
N-Phenyl-2-naphthylamine	8.82	90	0.86	4.3	3.6
	8.82	75	1.03	3.7	3.7
	13.2		0.72	3.8	3.6
2,6-Di-*tert*-butyl-4-methylphenol	4.41	90	1.27	2.5	2.4
	13.2		0.79	2.7	2.9
	8.82	75	1.56	2.5	2.9
	13.2		0.92	2.4	2.5
	4.41	60	1.76	...	3.0

crease in oxidation rate for the second stage was observed in all oxidations except for those in which the autocatalytic stage began, or the oxidation was terminated, before the required level of oxygen absorption was reached. Some curvature at the break would be expected and could probably be demonstrated with additional points plotted on an expanded time scale.

It is believed that the abrupt change in oxidation rate is probably due to a change in the initiation mechanism. Of the four possible initiation reactions listed here, it is suggested that only the first three may be operative, in varying degrees, during the initial stage of retarded oxidation.

Initiation reactions

$$RH + O_2 \longrightarrow R\cdot + HO_2\cdot$$
$$InH + O_2 \longrightarrow In\cdot + HO_2\cdot$$
$$ROOH \longrightarrow RO\cdot + HO\cdot$$
$$\downarrow\uparrow$$
$$ROOH \longrightarrow RO\cdot + RO_2\cdot + H_2O$$
$$\vdots$$
$$HOOR$$

The direct abstraction of hydrogen from the substrate by molecular oxygen has never been substantiated except for the case of rather active substrates.[30] Another possible reaction involving direct oxygen attack on the substrate is the addition of oxygen to a reactive double bond to form a biradical. Direct attack of oxygen on the inhibitor is indicated by the pro-oxidant effect observed at higher concentrations and by the reversal of the direction of the isotope effect as confirmed in this study. Since hydroperoxide will form at random in the solid substrate, the groups would be far apart at low concentration and only unimolecular homolytic cleavage would be expected initially. The constant rate of oxidation during a period in which peroxide concentration must increase suggests that peroxide decomposition contributes little to initiation in the initial stage.

It is proposed that as oxidation progresses the hydroperoxide concentration increases until a critical value is reached where the probability that the groups will be formed close enough together to associate through hydrogen bonding becomes significant. At this point the more energetically favored bimolecular decomposition reaction will also occur and bring about a more rapid rate of initiation. This would result in a steady-state hydroperoxide concentration since any excess above the concentration attained at the break in the oxygen-absorption plot would be rapidly decomposed.

For simple organic hydroperoxides in solution, a gradual change in kinetics has been shown[31-38] to begin at low concentrations of hydroperoxide (0.02–0.03 mole/l.). This change from first- to second-order kinetics as the hydroperoxide concentration increases corresponds to a change in the infrared O–H bond absorption from that of an unassociated O–H to that of a hydrogen-bonded species.[31]

Oxidation of a solid substrate, as in this study, yields hydroperoxides attached to a polymer chain. This restriction of mobility would prevent association at low concentrations and shift the concentration at which hydrogen-bonded association could occur to higher values and account for the more abrupt change. Oxygen is also consumed in the direct oxidation of the inhibitor and in the formation of cyclic peroxides in the oxidation of 1,5-dienes.[34] Consequently, the observed absorption of oxygen equivalent to a hydroperoxide concentration of approximately 0.10 mole/liter for the case of 2,6-di-*tert*-butyl-4-methylphenol-inhibited oxidations appears reasonable.

Although the break point occurs at a fairly constant oxygen-absorption level for each inhibitor, the average values for the oxygen-absorption levels at the break point (from Table IV) for the three inhibitors are quite different.

Inhibitor	Average O_2 absorption at break point, ml.
N,N'-Diphenyl-*p*-phenylenediamine	4.5
N-Phenyl-2-naphthylamine	3.8
2,6-Di-*tert*-butyl-4-methylphenol	2.6

Since the proposed hypothesis suggests that the break in the oxygen-absorption plots is due to the onset of bimolecular peroxide decomposition, it would be expected that this should occur at the same hydroperoxide concentration regardless of inhibitor. The observed order of oxygen-absorption levels probably reflects differences in the ability of the inhibitor to consume oxygen and to decompose hydroperoxide. The insensitivity of this value for a given inhibitor to changes in concentration indicates that increased consumption of oxygen at higher inhibitor concentration is paralleled by more rapid formation of hydroperoxide.

If the break is due to the onset of bimolecular hydroperoxide decomposition at some definite level of hydroperoxide concentration, a simple test of the hypothesis can be devised.[35] Heating preoxidized samples in an inert atmosphere should reduce the concentration below the critical value and make it possible to observe the break a second time on subsequent oxidation. The results of such a test with N-phenyl-2-naphthylamine and 2,6-di-*tert*-butyl-4-methylphenol are presented in Table V. Samples containing the amine were preoxidized past the break observed at 4.0 ml. of O_2 per gram to an absorption of 6 ml. and then heated in a nitrogen atmosphere. Subsequent oxidation showed the same type of break in the oxygen-absorption rate plots. Although the agreement between samples was only fair, the average for two samples heated 27 hr. at 150° shows the break between 3 and 4 ml., indicating that most of the hydroperoxide formed in the prior oxidation had been destroyed. Samples heated at 90° for 83.5 hr. in nitrogen show the break after the absorption of only 1 ml. of O_2 per gram, indicating that the rate of peroxide decomposition at concentrations below the critical level proceeds very slowly at 90°, which was also the temperature of the oxidation. Similar results are shown for samples containing the phenolic inhibitor which were preoxidized past the break at 2.5 ml. of O_2 per gram to an absorption of 3.5 ml. When oxidation was resumed at 90° after 28.5 hr. at 150° in nitrogen, the same samples showed the break at very close to 2.0 ml. of O_2 per gram. Samples heated at 90° show that the peroxide level was reduced only slightly below the critical level. Thus, the contribution of peroxide to initiation in the initial state must be very small at this temperature, and the major initiation reaction must involve direct oxidation of the inhibitor!

The onset of the autocatalytic stage, which corresponds to consumption of available inhibitor, occurred after the absorption of approximately 18 ml. of O_2 for the uninterrupted oxidation in Fig. 1. The similar samples employed for the interrupted oxidation, and

(30) C. A. Russell in "Peroxide Reaction Mechanisms," J. O. Edwards, Ed., Interscience Publishers, Inc., New York, N. Y., 1962, pp. 110–112.

(31) L. Bateman and H. Hughes, *J. Chem. Soc.*, 4594 (1952).

(32) L. Bateman, H. Hughes, and A. L. Morris, *Discussions Faraday Soc.*, **14**, 190 (1953).

(33) V. Stannett and R. B. Mesrobian, *ibid.*, **14**, 242 (1953).

(34) C. Walling, "Free Radicals in Solution," John Wiley and Sons, Inc., New York, N. Y., 1957, p. 442.

(35) E. M. Bevilacqua, private communication.

TABLE V

OXYGEN ABSORPTION OF PREOXIDIZED *cis*-1,4-POLYISOPRENE

Sample	Treatment	Rate at 90°, ml. O_2/g./hr. (22°, 760 mm.) Init. stage	Sec. stage	Abs. at break, ml. O_2/g. (22°, 760 mm.)
N-Phenyl-2-naphthylamine	Initial oxdn.	0.180	0.256	4.0
8.82×10^{-5} mole/g.	Oxdzd. then htd.	.200	.296	2.8
	26.9 hr. at 150° in N_2 atm.	.253	.426	4.4
	Oxdzd. then htd.	.266	.449	1.0
	83.5 hr. at 90° in N_2 atm.	.372	.540	1.1
2,6-Di-*tert*-butyl-4-methylphenol	Initial oxdn.	.0597	.0924	2.5
4.41×10^{-5} mole/g.	Oxdzd. then htd.	.0696	.0887	2.2
	28.5 hr. at 150° in N_2 atm.	.0802	.110	1.7
	Oxdzd. then htd.	.0760	.117	0.3
	86.9 hr. at 90° in N_2 atm.	...	.124	...

heated at 150° in nitrogen, absorbed a total of only 12 ml. of O_2 per gram before reaching the autocatalytic stage, indicating that considerable inhibitor was used up during the period of peroxide decomposition. This would account for the higher initial rates of oxidation and greater variations in the experimental data observed with the preoxidized and heat-treated samples.

Summary and Conclusions

The mechanism of chain-stopping action by amine and phenolic inhibitors in retarded autoxidation has been clarified by the demonstration of a significant kinetic deuterium isotope effect. The use of a purified *cis*-1,4-polyisoprene in this work permitted observation of the effect in the initial stage, thus confirming the validity of our prior conclusions based on isotope effects demonstrated in a later stage of retarded oxidation.[6,7] The decrease in magnitude and reversal of the direction of the deuterium isotope effect at higher temperatures and higher concentrations of inhibitor is attributed to competition resulting from an initiation reaction involving direct oxidation of the inhibitor which also shows an isotope effect.

These results show that the rate-determining step of the chain-stopping mechanism is the transfer of hydrogen from the inhibitor to a chain-propagation $RO_2\cdot$ radical. A reversible complexing of free radicals with the π-electrons of aromatic compounds present as inhibitor, solvent, or organic substrate is also possible as an additional, but not essential, feature.

Possible mechanism

$$RO_2\cdot + InH \rightleftharpoons (InH, RO_2\cdot)$$

$$\underset{\text{(or complex)}}{RO_2\cdot + InH} \xrightarrow[\text{controlling}]{\text{rate}} RO_2H + In\cdot$$

$$In\cdot + RO_2\cdot \xrightarrow{\text{(or complex)}} \text{inactive products}$$

It is also possible that, in some cases at least, the rate-controlling hydrogen-transfer reaction may occur more readily when a complex of $RO_2\cdot$ and inhibitor reacts with a second $RO_2\cdot$ radical. The last two steps in the suggested mechanism would then be replaced by

$$(InH, RO_2\cdot) + RO_2\cdot \xrightarrow[\text{controlling}]{\text{rate}} RO_2H + \text{other products}$$

The reaction sequence would then be the same as in Hammond's original postulate except for the important difference that the last reaction would be rate controlling, rather than fast as was originally proposed.

An unexpected result of this study was the discovery of two well defined linear stages of retarded oxidation prior to the onset of autocatalysis. It is proposed that this phenomenon results from a change in the initiation process and that the break between the initial stage and the faster second stage marks the point at which hydroperoxide concentration builds up to some critical level at which association by hydrogen bonding can occur, leading to the faster bimolecular decomposition of peroxide. Initiation by direct oxidation of inhibitor would occur in both stages, but at lower concentrations of hydroperoxide formed at random on polymer chains in a solid media only the slower unimolecular homolysis of the peroxides could occur. This hypothesis has been tested by preoxidizing samples past the break in the oxygen-absorption plot and then heating in an inert atmosphere to decompose part of the peroxide. Subsequent oxidation again demonstrated the characteristic change in rate, consistent with re-formation of sufficient hydroperoxide to attain the critical concentration. The sharp break observed in this study associated with two distinct constant-rate stages of retarded oxidation appears to be a consequence of our use of a purified solid hydrocarbon in an autoinitiated autoxidation in the presence of inhibitors.

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