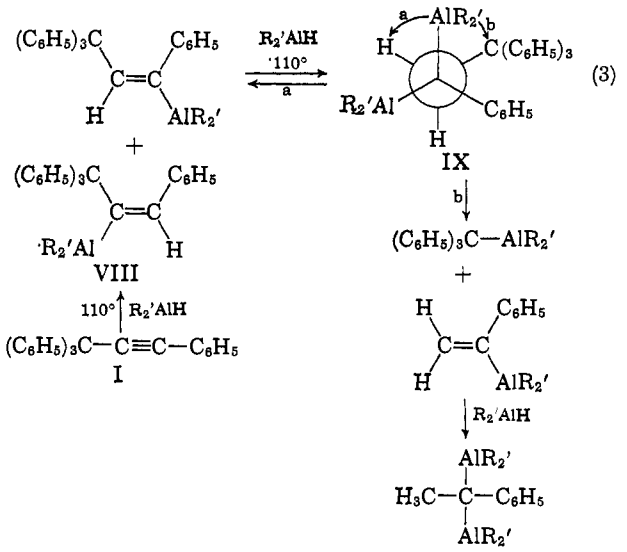

cis-adducts directly (*cf.* V), it can be stated that adducts VIII show no such isomerization tendency at 110°.

Further studies bearing upon the behavior of vinylaluminum compounds and the possible role of p_π–p_π effects are under consideration in this Laboratory.

Acknowledgment.—The authors wish to express their gratitude to the Petroleum Research Fund for the support of the foregoing research under Grant No. 723-A.

DEPARTMENT OF CHEMISTRY
UNIVERSITY OF MICHIGAN
ANN ARBOR, MICHIGAN

JOHN J. EISCH
WILLIAM C. KASKA

RECEIVED MAY 27, 1963

Influence of Cumene Hydroperoxide upon the Inhibited Oxidation of Cumene

Sir:

Considerable effort has been devoted to distinguishing between the conflicting oxidation inhibition mechanism of Bolland and ten Have,[1] which postulates simple hydrogen abstraction from aromatic amine and phenol inhibitors, and that of Boozer and Hammond,[2] which postulates rapid reversible complex formation between alkylperoxy radical and inhibitor. Recent electron spin resonance studies[3] indicating nil complex formation between cumylperoxy radicals and pyridine or triphenylamine have prompted us to reinvestigate alternative proposals which give kinetic results similar to the Boozer–Hammond proposal.

One such proposal by Bickel and Kooyman[4] suggests that data supporting the complex mechanism might be explained by a reversible reaction between intermediate inhibitor radical and hydroperoxide as shown in reactions 1 and 2. Hammond and Nandi[5] tested this possi-

$$RO_2\cdot + IH \longrightarrow RO_2H + I\cdot \qquad k_1 \qquad (1)$$

$$I\cdot + RO_2H \longrightarrow IH + RO_2\cdot \qquad k_2 \qquad (2)$$

bility by studying the influence of cumene hydroperoxide upon the oxidation rate of cumene at 70° when inhibited with several inhibitors including phenol. No effect was observed and this explanation was discarded. In contrast, we observe a significant accelerating effect of cumene hydroperoxide upon both phenol and diphenylamine inhibited oxidation rates of cumene initiated by azobisisobutyronitrile (AIBN) at 57.2°. Figure 1 presents initial phenol inhibited rates as a function of the concentration of added cumene hydroperoxide to the one-half power. On the other hand, inhibited oxidation rates with the highly hindered phenols, 2,6-*t*-butyl phenol and 2,6-*t*-butyl cresol, are uninfluenced by cumene hydroperoxide at the highest concentrations included in Fig. 1. In Fig. 2, inhibited rates are plotted against the inverse square root of phenol concentration for zero added hydroperoxide and for a hydroperoxide concentration of 0.012 *M*.

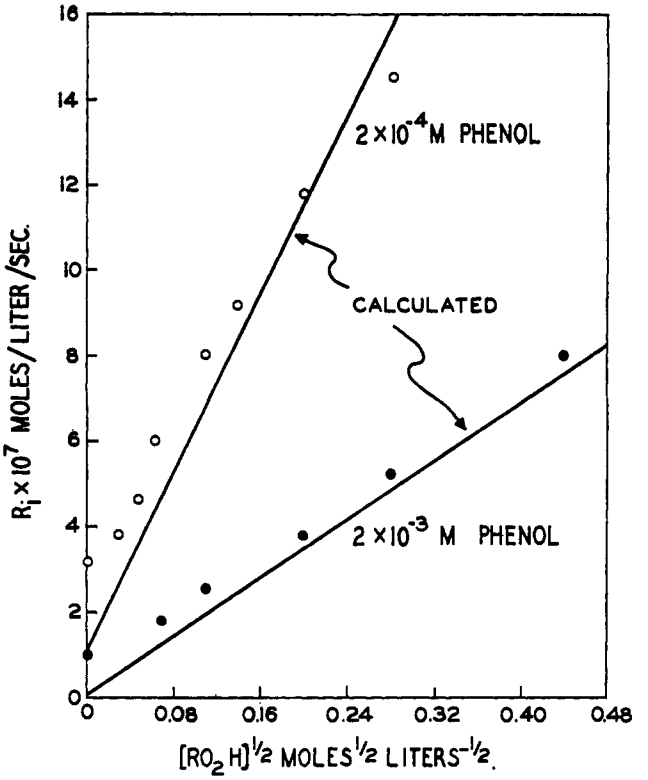

Fig. 1.—Phenol inhibited oxidation rates of cumene *vs.* square root of added cumene hydroperoxide: 5.68 *M* cumene; 8×10^{-3} *M* AIBN; 57°; 1 atm. of O_2; chlorobenzene diluent.

Reactions 1 and 2 together with 3 give a moderately

$$RO_2\cdot + I\cdot \longrightarrow RO_2I \qquad k_3 \qquad (3)$$

good account of the experimental results as shown by the calculated curves in Fig. 1 and 2. These curves are due to the appropriate rate expression (4) where k_p and k_i are the known propagation and initiation rate constants, and using $k_1 = 4 \times 10^3$ l. mole^{-1} sec.$^{-1}$ and $k_2/k_3 = 5.7 \times 10^{-5}$. This expression calls for inverse first

$$R_i = \frac{k_p[RH]k_i[AIBN]}{4k_1[IH]}\left\{1 + \left(1 + \frac{8\,k_1k_2[RO_2H][IH]}{k_i[AIBN]k_3}\right)^{1/2}\right\} \qquad (4)$$

power dependence of the oxidation rate upon inhibitor concentration at zero hydroperoxide. If the mechanism is to apply, the observed inverse square root dependence must arise from hydroperoxide formed by reaction during the determination of the initial rate. By oxygen absorption measurement, the hydroperoxide generated during this period is approximately 10^{-3} *M* in all cases, and this value was used in (4) for the calculated curve. Treatment of starting cumene with activated silica reduced contaminating hydroperoxide to undetectable levels, less than 5×10^{-4} *M*. The concentration ranges covered in Fig. 1 and 2 are the maximum permissible except for the higher inhibitor concentration of Fig. 1. Limitations are imposed by the inhibited rate approaching the uninhibited rate (32

(1) J. L. Bolland and P. ten Have, *Trans. Faraday Soc.*, **43**, 201 (1947).
(2) C. E. Boozer and G. S. Hammond, *J. Am. Chem. Soc.*, **76**, 3861 (1954).
(3) J. R. Thomas, *ibid.*, **85**, 591 (1963).
(4) A. F. Bickel and E. C. Kooyman, *J. Chem. Soc.*, 2215 (1956).
(5) G. S. Hammond and U. S. Nandi, *J. Am. Chem. Soc.*, **83**, 1217 (1961).

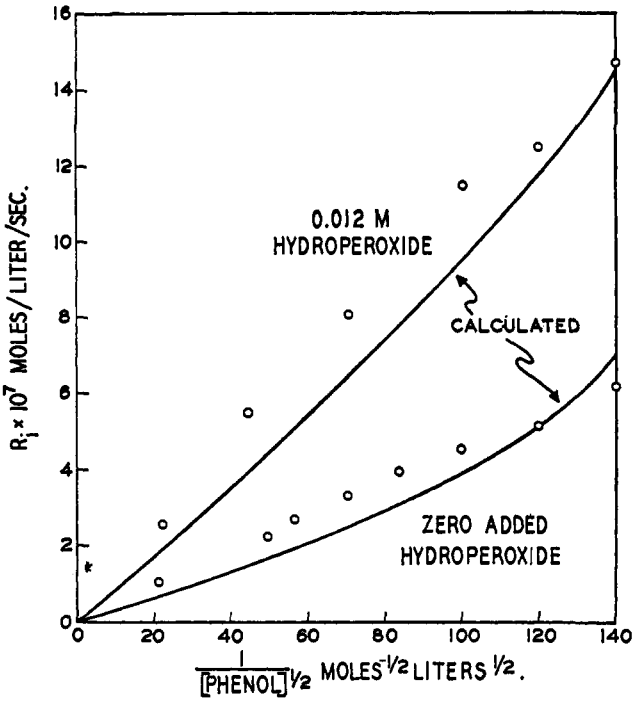

Fig. 2.—Oxidation rate of cumene *vs.* inverse square root of phenol concentration: 5.68 *M* cumene; 8×10^{-3} *M* AIBN; 57°; 1 atm. of O_2; chlorobenzene diluent.

$\times 10^{-7}$ mole l.$^{-1}$ sec.$^{-1}$), the chain length approaching unity or the inhibitor concentration becoming so low that reliable initial rates cannot be determined.

The following supplementary observations aid in excluding some alternative explanations of the hydroperoxide effect.

1. Cumene hydroperoxide generated by oxidation before addition of inhibitor had exactly the same effect upon the inhibited rate as added hydroperoxide.
2. The inhibition periods with both phenol and diphenylamine are unchanged by hydroperoxide, showing that the observed effect is not due to chain initiation by hydroperoxide.
3. Direct chemical reaction between inhibitors and hydroperoxide is unimportant since the results are unchanged by aging their solutions prior to addition of initiator.

A number of alternative mechanisms have been examined with only one yielding calculated curves at all in agreement with experiment. This exception, which retains Boozer and Hammond's postulate, assumes that hydroperoxide forms a hydrogen bonded complex with inhibitor and that only free inhibitor reacts with $RO_2\cdot$ radicals. Agreement with experiment is obtained for an association constant of 300 l. mole^{-1}. However, infrared studies indicate an association constant between phenol and cumene hydroperoxide of only 12 l. mole^{-1} in carbon tetrachloride at 25°, making this explanation unlikely.

In addition to offering an explanation for the inverse square root inhibitor dependence so often observed with simple amines and phenols the hydroperoxide effect explains the lack of a deuterium kinetic isotope effect for diphenylamine even in the presence of excess deuterium oxide,[5] under which conditions Ingold[6] does find an isotope effect for 2,6-*t*-butyl cresol. It also explains why hindered phenols are more satisfactory inhibitors in autoxidation systems where chain initiation depends upon hydroperoxide.

By electron spin resonance techniques which permit the quantitative determination of cumylperoxy radical concentration in oxidizing cumene,[3] attempts were made to observe the steady-state concentration of the phenoxy radical in phenol inhibited systems. These experiments showed the phenoxy radical concentration to be 10^{-6} *M* or less even with phenol concentrations as high as 1.0 *M*. With $k_1 = 4 \times 10^3$ l. mole^{-1} sec.$^{-1}$ from above, $k_3 \geqq 4 \times 10^9$ l. mole^{-1} sec.$^{-1}$ and $k_2 \geqq 2.3 \times 10^5$ l. mole^{-1} sec.$^{-1}$. The values of these rate constants, particularly that of k_2, seem implausibly large and are the source of some reservation in wholeheartedly adopting this mechanism.

(6) K. U. Ingold, *Nature*, **195**, 280 (1962).

RICHMOND LABORATORY
CALIFORNIA RESEARCH CORPORATION
RICHMOND, CALIFORNIA
J. R. THOMAS

RECEIVED APRIL 20, 1963

The Configurational Stability of Primary Grignard Reagents[1]

Sir:

The n.m.r. spectrum of the methylene hydrogens of 3,3-dimethylbutylmagnesium chloride[2] in diethyl ether

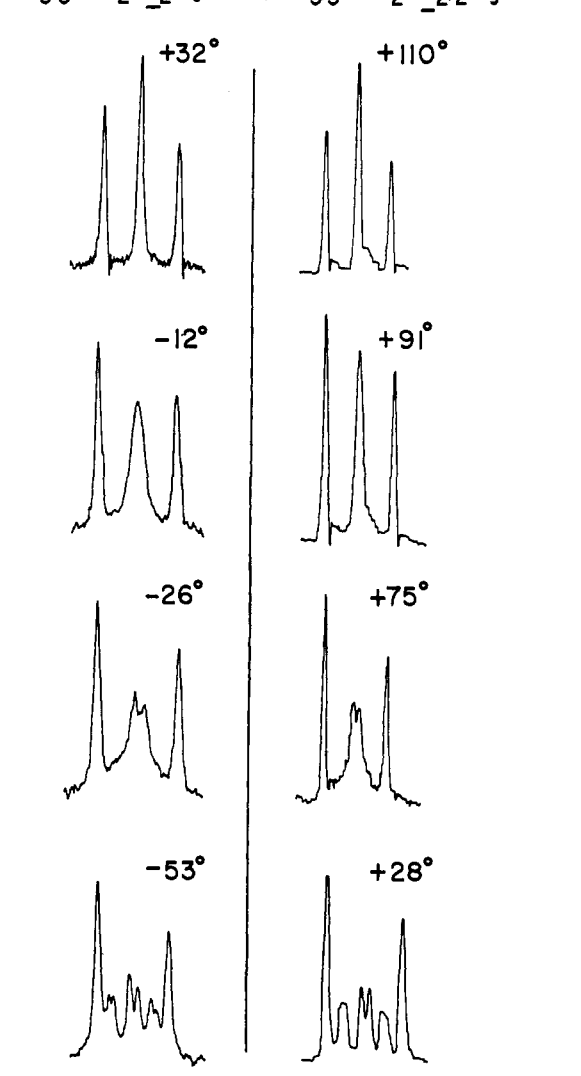

Fig. 1.—N.m.r. spectra of the $-C\underline{H_2}-Mg$ protons of 3,3-dimethylbutyl Grignard reagent and bis-(3,3-dimethylbutyl)-magnesium in diethyl ether solution as a function of temperature.

(1) Supported in part by the Office of Naval Research and The National Science Foundation.

(2) In this paper, the solvated organometallic compound prepared from 3,3-dimethylbutyl chloride and magnesium will be called 3,3-dimethylbutyl-