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Fig. 1

Figure 1: Fig. 1

Abstract

Full Text

PHYSICAL CHEMISTRY

Z. K. MAIZUS, I. P. SKIBIDA, Corresponding Member of the USSR Academy of Sciences N. M. EMANUEL'

MECHANISM OF THE CATALYTIC DECOMPOSITION OF HYDROPEROXIDES UNDER THE INFLUENCE OF COPPER STEARATE

The use of salts of metals of variable valence as catalysts for liquid-phase oxidation of hydrocarbons makes it possible to substantially increase the rate of the process (⁀1). However, alongside the initiating action of the catalyst, in some cases a second, inhibiting function of salts of metals of variable valence is observed, appearing at relatively high catalyst concentrations. Thus, in the oxidation of *n*-decane in the presence of salts of divalent copper (⁀2) and of tetralin in the presence of salts of trivalent cobalt (⁀3), a sharp increase in the induction period is observed when a certain critical salt concentration is reached.

The mechanism of catalyzed oxidation of hydrocarbons differs from the mechanism of oxidation in the absence of metals of variable valence. Introduction of a catalyst primarily affects the mechanism of radical formation. In uncatalyzed oxidation the principal source of radicals is thermal decomposition of the hydroperoxide; in catalyzed oxidation it is its interaction with the catalyst.

In the present work, in studying the mechanism of decomposition of *n*-decyl hydroperoxide in the presence of copper stearate, it was established that the role of the catalyst is not always limited to its participation in the process of radical formation. In contrast to certain other catalysts (for example, cobalt stearate), copper stearate also promotes the molecular decomposition of the hydroperoxide without formation of radicals. This effect is enhanced when an inhibitor is introduced into the system.

Fig. 1. Dependence of the rate of copper stearate decomposition in the presence of copper stearate ($C_0 = 8 \cdot 10^{-4}$ mol/l) on the concentration of *n*-decyl hydroperoxide. Temperature 80°.

The decomposition of *n*-decyl hydroperoxide in the presence of copper stearate was carried out in a nitrogen atmosphere in a glass reactor at a temperature of 80°. The rate of radical formation was determined from the rate of consumption

of the inhibitor N-phenyl-β-naphthylamine. Special experiments showed that the rate of inhibitor consumption does not depend on its concentration, i.e., the inhibitor interacts only with free radicals and does not react directly either with the hydroperoxide or with other products present in the system.

The dependence of the rate of radical formation W_p on the concentration of copper stearate was investigated. It was found that, when the concentration of copper stearate was varied from $2 \cdot 10^{-4}$ to $2 \cdot 10^{-3}$ mol/l, the rate of radical formation increased linearly with increasing [St_2Cu]. Consequently, radical formation proceeds by a first-order reaction with respect to St_2Cu. The dependence of W_p on the hydroperoxide concentration at a catalyst concentration of $8 \cdot 10^{-4}$ mol/l is shown in Fig. 1. At very low concentrations of ROOH the rate of radical formation increases with increasing hydroperoxide concentration; at ROOH concentrations $>>$ 0.015 mol/l the value of W_p ceases to depend on [ROOH].

This form of the dependence of W_p on [ROOH] indicates that the formation of radicals occurs upon decomposition of the hydroperoxide complex with copper stearate; under the condition [St_2Cu] $\ll$ [ROOH], all the copper in the system proves to be bound in the complex, and a further increase in [ROOH] does not affect the rate of radical formation.

The mechanism of decomposition of hydroperoxides into radicals in the presence of St_2Cu may be described by the following scheme:

$$St_2Cu + n\mathrm{ROOH} \underset{2}{\overset{1}{\rightleftarrows}} [St_2Cu \cdot n\mathrm{ROOH}] \xrightarrow{3} \dot{RO}_2 + CuSt + HSt + (n-1)\mathrm{ROOH},$$

$$StCu + \mathrm{ROOH} \xrightarrow{4} CuSt(OH) + \dot{RO}.$$

For the rate of inhibitor consumption, taking into account that one inhibitor molecule reacts with two free radicals, we obtain

$$W_p = -d[\mathrm{InH}]/dt = k_3[CuSt_2 \cdot n\mathrm{ROOH}] + k_4[CuSt][\mathrm{ROOH}].$$

Using the steady-state condition for the concentration of monovalent copper,

$$d[CuSt]/dt = k_3[CuSt_2 \cdot n\mathrm{ROOH}] - k_4[CuSt][\mathrm{ROOH}] = 0,$$

we obtain

$$W_p = 2k_3[CuSt_2 \cdot n\mathrm{ROOH}].$$

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Assuming that the rate of establishment of equilibrium is sufficiently high and that reaction 3 does not disturb the equilibrium, we write for the concentration of the complex

$$[\mathrm{CuSt_2} \cdot n\mathrm{ROOH}] = K[\mathrm{CuSt_2}]_{\mathrm{eq}} \cdot [\mathrm{ROOH}]_{\mathrm{eq}}^{n}, \tag{1}$$

where K is the equilibrium constant*, and $[\mathrm{CuSt_2}]_{\mathrm{eq}}$ and $[\mathrm{ROOH}]_{\mathrm{eq}}$ are the concentrations of $\mathrm{CuSt_2}$ and ROOH not bound in the complex. Expressing $[\mathrm{CuSt_2}]_{\mathrm{eq}}$ and $[\mathrm{ROOH}]_{\mathrm{eq}}$ through the total concentrations $[\mathrm{CuSt_2}]_{\Sigma}$ and $[\mathrm{ROOH}]_{\Sigma}$,

$$[\mathrm{CuSt_2}]_{\mathrm{eq}} = [\mathrm{CuSt_2}]_{\Sigma} - [\mathrm{CuSt_2} \cdot n\mathrm{ROOH}]$$

and

$$[\mathrm{ROOH}]_{\mathrm{eq}} = [\mathrm{ROOH}]_{\Sigma},$$

we obtain

$$[\mathrm{CuSt_2} \cdot n\mathrm{ROOH}] = K[\mathrm{ROOH}]_{\Sigma}^{n}[\mathrm{CuSt_2}]_{\Sigma} / \left(1 + K[\mathrm{ROOH}]_{\Sigma}^{n}\right).$$

Thus, the rate of radical formation is equal to

$$W_p = 2k_3 K[\mathrm{ROOH}]_{\Sigma}^{n}[\mathrm{CuSt_2}]_{\Sigma} / \left(1 + K[\mathrm{ROOH}]_{\Sigma}^{n}\right). \tag{2}$$

At $1 \ll K[\mathrm{ROOH}]_{\Sigma}^{n}$, the quantity

$$W_p = W_{\infty} = 2k_3[\mathrm{CuSt_2}]_{\Sigma}$$

does not depend on the concentration of ROOH. Hence, using the value of the maximum rate of inhibitor consumption (Fig. 1), one can determine the constant k_3 for the rate of decomposition of the complex into radicals. For 80°, this value proved to be 0.06 min^{-1}. The dependence of W_p on [ROOH] makes it possible to determine the number of ROOH molecules bound in the complex with $\mathrm{CuSt_2}$, and the equilibrium constant K. For this purpose we rewrite formula (2) in the form

$$\frac{1}{W_p} - \frac{1}{2k_3[\mathrm{CuSt_2}]_{\Sigma}} = \frac{1}{W_p} - \frac{1}{W_{\infty}} = \frac{1}{2k_3 K[\mathrm{CuSt_2}]_{\Sigma}[\mathrm{ROOH}]_{\Sigma}^{n}}.$$

After taking logarithms we obtain

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Figure 2

Figure 2: Figure 2

$$\lg\left\{\frac{1}{W_p}-\frac{1}{W_\infty}\right\}=\lg\frac{1}{2k_3K[\mathrm{CuSt_2}]_\Sigma}-n\lg[\mathrm{ROOH}]_\Sigma.$$

From the slope of the straight line in coordinates

$$\lg\left\{\frac{1}{W_p}-\frac{1}{W_\infty}\right\}-\lg[\mathrm{ROOH}]_\Sigma$$

the value of n can be determined, and from the intercept cut off on the ordinate axis—the product k_3K.

The dependence of the rate of radical formation on the concentration of ROOH during decomposition of n-decyl hydroperoxide in the coordinates

$$\lg\left\{\frac{1}{W_p}-\frac{1}{W_\infty}\right\}-$$

$$-\lg[\mathrm{ROOH}]_\Sigma$$

is presented in Fig. 2. The value determined from the slope of the straight line is $n=2$, and

$$k_3K=4.8\cdot10^3\ \mathrm{L^2/mol^2\cdot min}.$$

Using the found

* K represents the product of the equilibrium constants for formation of complexes containing from one to n

higher than the value of k_3, we obtain for the equilibrium constant $K=4\cdot10^4$ $\mathrm{l^2/mole^2}$. Thus, it may be considered established that the $\mathrm{CuSt_2}$ complex with ROOH contains 2 molecules of hydroperoxide.

Measurement of the rate of inhibitor consumption makes it possible to study only one pathway of hydroperoxide decomposition—the formation of free radicals.

Fig. 2. Dependence of $\frac{1}{W_p}-\frac{1}{W_\infty}$ on [ROOH]. Copper stearate concentration $8\cdot10^{-4}$ mole/l. Temperature 80°.

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Figure 3

Figure 3: Figure 3

In parallel with this reaction, molecular decomposition of hydroperoxide can occur without formation of free radicals, as well as chain decomposition induced by radicals. An estimate of the ratio between the rates of these three pathways of ROOH decomposition can be made by comparing data on the kinetics of consumption of hydroperoxide and of inhibitor. In this case, the decrease in the rate of hydroperoxide consumption upon introduction of inhibitor will indicate the presence of a chain consumption pathway, while the difference between the rate of hydroperoxide consumption in the presence of inhibitor and the rate of inhibitor consumption will correspond to the rate of molecular consumption of hydroperoxide without formation of free radicals. When an inhibitor was introduced into the system n-decyl hydroperoxide–$CuSt_2$, the rate of inhibitor consumption proved to be much lower than the rate of hydroperoxide consumption. Thus, at an initial concentration ROOH = 0.047 mole/l and [InH] = $2.2 \cdot 10^{-4}$ mole/l, the rate of ROOH consumption is $1.2 \cdot 10^{-3}$ mole/l $\cdot$ min, while the rate of inhibitor consumption is $1.9 \cdot 10^{-4}$ mole/l $\cdot$ min.

The rate of hydroperoxide consumption increases linearly with increasing ROOH concentration (Fig. 3a). Introduction of inhibitor markedly accelerates decomposition of the hydroperoxide. The rate of decomposition increases linearly with increasing inhibitor concentration (Fig. 3b). Comparing this fact with the absence of a dependence of the rate of inhibitor consumption on its concentration (see above), we arrive at the conclusion that, in the presence of $CuSt_2$, the inhibitor accelerates molecular decomposition of the hydroperoxide, while the inhibitor itself is not consumed in this act. If the inhibitor accelerated radical decomposition of the hydroperoxide, then, with increasing concentration of the inhibitor, the rate of consumption of the inhibitor in reaction with free radicals would also have to increase.

Fig. 3. Dependence of the rate of consumption of n-decyl hydroperoxide in the presence of copper stearate ($8 \cdot 10^{-4}$ mole/l) on the concentration of hydroperoxide in the presence of the inhibitor N-phenyl-β-naphthylamine, $C_0 = 3 \cdot 10^{-3}$ mole/l (a), and on the concentration of the inhibitor N-phenyl-β-naphthylamine, hydroperoxide concentration $C_0 = 3.3 \cdot 10^{-2}$ mole/l (b). Temperature 80°.

The dependence of the rate of hydroperoxide consumption on the inhibitor concentration (Fig. 3b) indicates the absence of a chain pathway of hydroperoxide decomposition. Indeed, the intercept cut off by the straight line in the coordinates W–[InH] is exactly equal to the rate of hydroperoxide decomposition in the absence of inhibitor. In the case where the peroxide decomposed by a chain pathway, the intercept would have had to lie below the point corresponding to the rate of uninhibited hydroperoxide decomposition. The increase in the rate of molecular decomposition of hydroperoxide under the action of

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copper stearate may be one of the reasons for the inhibiting action of copper catalysts in oxidation reactions, observed at high concentrations of the copper salt. A decrease in the concentration of hydroperoxide due to its molecular decomposition lowers the rate of chain branching in the oxidation process.

Of considerable interest is also the acceleration of the molecular decomposition of hydroperoxide under the influence of N-phenyl-β-naphthylamine. It is possible that, in a number of cases, mixtures of copper salts and free-radical inhibitors may be used as effective stabilizers of the process.

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