Interrelation of the chemical structure and inhibiting activity of sterically hindered phenols in methyl oleate oxidation in homogeneous and microheterogeneous systems

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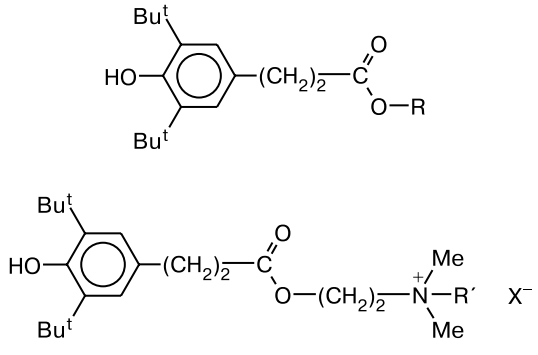
Specific features of the inhibiting activity of sterically hindered phenolic antioxidants (AOs) 3,5-Bu^t_2-4-$OHC_6H_2(CH_2)_2C(O)O(CH_2)_2N^+Me_2R \cdot X^-$ (R = H, Me, C_8H_{17}, $C_{10}H_{21}$, $C_{12}H_{25}$, $C_{16}H_{33}$; X = Br, I), being phenosan derivatives containing the ethanolamine residue substituted at the N atom by an alkyl substituent, were studied. The action of these AOs was studied in the initiated oxidation of homogeneous solutions of methyl oleate in chlorobenzene and an aqueous-emulsion medium in the presence of the surfactant sodium dodecyl sulfate. Phenolic AOs act in two directions: they react with peroxy radicals with a rate constant of $0.98 \cdot 10^4$ L mol^{-1} s^{-1} and decompose hydroperoxides to form molecular products. The effect of hindered phenols as AOs depends substantially on their chemical structure and oxidation conditions. In lipid solutions, they efficiently hinder the oxidation of methyl oleate, outperforming the action of α-tocopherol, dibunol, phenosan K, and its methyl ester taken in comparative concentrations. The inhibiting activity of the AOs decreases substantially with the chain elongation of the R substituent. For oxidation in an aqueous-emulsion medium, the inhibition effect of the AOs under study weakens compared to oxidation in a homogeneous solution, which is accompanied by the disappearance of differences in efficiency of different AOs.

Key words: antioxidants, α-tocopherol, peroxide oxidation, antioxidation activity, antiradical activity, methyl oleate, sterically hindered phenols, micelles.

One of the promising directions for the development of new highly efficient antioxidants (AOs) is the synthesis of "hybrid" molecules, whose structure combines several characteristic groups affecting (independently or synergetically) the oxidation of substrates in lipid or aqueous phases. Previously,[1] we have synthesized from phenosan-1, *viz.*, 3-(3,5-di-*tert*-butyl-4-hydroxyphenyl)propionic acid, a series of sterically hindered AOs containing the ethanolamine residue substituted at the quaternized N atom by one or several alkyl substituents. These inhibitors are known as ICPANs (Table 1).



Note. For decoding R, R´, and X, see Table 1.

It is also shown that AOs have no local and general toxic effects, do not affect the embriogenesis and development of posterity, exhibit antiacetylcholinesterase activity,[2] and control the plant cell growth.[3]

Analysis of the chemical structure suggests that some compounds (phenosan K and methyl ester of phenosan-1) are lipophilic in nature and form homogeneous solutions with lipids, while other AOs with a polar fragment in the structure (quaternized N atom) can spontaneously form micelles in which the hydroxy group of phenol can be hindered inside the microreactor due to the orientation of polar and nonpolar groups. Therefore, this work is aimed at studying the specific features of the inhibition of lipid

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Table 1. Kinetic characteristics of the antioxidants (AOs)

Antioxidant	R (R´)	X	$k \cdot 10^4$ /L mol^{-1} s^{-1}	τ* /min	A**
Phenosan K	K	—	2.20	920	35.4
Methyl ester of phenosan	Me	—	2.30	1050	40.4
ICPAN-9	H	Succinate	0.79	1025	39.4
ICPAN-10	Me	I	0.59	1125	43.3
ICPAN-10-C-8	C_8H_{17}	Br	1.06	350	13.5
ICPAN-10-C-10	$C_{10}H_{21}$	Br	0.98	500	19.2
ICPAN-10-C-12	$C_{12}H_{25}$	Br	0.97	425	16.3
ICPAN-10-C-16	$C_{16}H_{33}$	Br	0.94	1075	41.5

* Induction period at $C_{AO} = 1 \cdot 10^{-3}$ mol L^{-1}.
** $A = (\tau_{AO} - \tau_{MO})/\tau_{MO}$ (MO is methyl oleate).

oxidation by these compounds in homogeneous and micellar solutions, showing an interrelation of the efficiency and structure of the inhibitors, and establishing the mechanism of their action. The antioxidation effect of ICPANs was compared with the activity of the known reference AOs: dibunol and α-tocopherol (TP).

Experimental

α-Tocopherol (6-hydroxy-2,5,7,8-tetramethylphytylchromane) (Serva, Germany), dibunol (1-hydroxy-2,6-di-*tert*-butyl-4-methylbenzene) (Serva, Germany), ICPAN-9 ((*N*,*N*-dimethyl-2-ammonioethyl)-3-(3,5-di-*tert*-butyl-4-hydroxyphenyl)propionate succinate), ICPAN-10 ((*N*,*N*,*N*-trimethyl-2-ammonioethyl)-3-(3,5-di-*tert*-butyl-4-hydroxyphenyl)propionate iodide), ICPAN-10-C-8 ((*N*,*N*-dimethyl-*N*-octyl-2-ammonioethyl)-3-(3,5-di-*tert*-butyl-4-hydroxyphenyl)propionate bromide), ICPAN-10-C-10 ((*N*,*N*-dimethyl-*N*-decyl-2-ammonioethyl)-3-(3,5-di-*tert*-butyl-4-hydroxyphenyl)propionate bromide), ICPAN-10-C-12 ((*N*,*N*-dimethyl-*N*-dodecyl-2-ammonioethyl)-3-(3,5-di-*tert*-butyl-4-hydroxyphenyl)propionate bromide), and ICPAN-10-C-16 ((*N*,*N*-dimethyl-*N*-hexadecyl-2-ammonioethyl)-3-(3,5-di-*tert*-butyl-4-hydroxyphenyl)propionate bromide) were used. The ICPANs were synthesized by a known procedure[1] at the Institute of Biochemical Physics of the Russian Academy of Sciences. Phenosan K (potassium 3-(3,5-di-*tert*-butyl-4-hydroxyphenyl)propionate) and methyl ester of phenosan-1 (methyl 3-(3,5-di-*tert*-butyl-4-hydroxyphenyl) propionate) were synthesized at the N. N. Vorozhtsov Institute of Organic Chemistry (Siberian Branch of the Russian Academy of Sciences). All AOs were "chromatographic purity" grade.

The antiradical activity of the AOs was tested in the system of the initiated oxidation of ethylbenzene by the luminescence method.[4] The kinetics was studied by the method of oxygen absorption in the Warburg-type manometric setups using the oxidation of methyl oleate (MO) as a model substrate in the presence of an inert solvent (chlorobenzene) or in an aqueous-emulsion medium in the presence of sodium dodecyl sulfate at the 1 : 1 ratio of the aqueous to lipid phases. The process was initiated by the thermal decomposition of AIBN at 60 °C. Under experimental conditions, the initiation rate (W_i) was $4.2 \cdot 10^{-8}$ mol L^{-1} s^{-1}. The inhibitor effects were estimated by the A value, which was quantitatively determined from the equation

$$A = \tau_i/\tau_{st},$$

and from the ratio of the induction periods τ_i/τ_s, where τ_i and τ_s are the induction periods for substrate oxidation in the presence and absence of the studied AO, respectively; τ_{st} is the induction period of the inhibitor taken as standard. The kinetics of hydroperoxide accumulation was studied by iodometric back-titration for the autooxidation of methyl oleate at 60 °C in chlorobenzene.

Micelle formation was studied by the refractometry[5] and Rebinder[6] methods.

Results and Discussion

The rate constants of the reaction of the AOs under study with peroxy radicals *via* the commonly accepted scheme were estimated by the chemiluminescence method.

$$InH + RO_2^{\cdot} \xrightarrow{k} In^{\cdot} + ROOH$$

InH is inhibitor (AO)

The antiradical activity (k) of the ICPANs was compared with that for dibunol and TP. The k values for phenosan K and its methyl ester are shown to be close (see Table 1) and comparable with the k value for dibunol ($1.40 \cdot 10^4$ L mol^{-1} s^{-1}). The antiradical activity of ICPAN-9 and ICPAN-10 is 1.5—2-fold lower than that of the AOs listed above (see Table 1). A decrease in k for the ICPANs compared to the value for dibunol is caused by the influence of electron-withdrawing substituents that decrease the antiradical activity of the AOs.[7] The activities of ICPANs in the reaction with $RO_2^{\cdot}$ are considerably lower than that of TP ($k = 3.6 \cdot 10^6$ L mol^{-1} s^{-1}). The stoichiometric inhibition coefficient for the majority of the known AOs is equal to two. Thus, the data presented show that the effect of the AOs under study is caused by the antiradical activity toward peroxy radicals $RO_2^{\cdot}$ involved in the oxidation process; on the average, two free radicals decay on one inhibitor molecule.

The AO structure contains substituted amino groups or fragments of the quaternary ammonium base. These classes of compounds are known to be capable of decomposing hydroperoxides (ROOH).[8] The kinetically nonradical decomposition of ROOH can appear as a decrease in the initial (W^0) and maximum (W^{max}) oxidation rates of the substrate. Therefore, we studied the dependence of these parameters on the ICPAN structure (Fig. 1). The data in Fig. 1 show that the introduction of the ICPANs increases the induction periods compared to that in a reference experiment and also decreases the initial and maximum oxidation rates (Table 2).

It should be noted that ICPAN-9, ICPAN-10, and ICPAN-10-C-16 exert the most significant effect on W^0 and W^{max} (see Table 2). It is most probable that hydro-

Table 2. Kinetic parameters of methyl oleate (MO) oxidation in the presence of the antioxidants (AOs) taken in different concentrations (C_{AO}) ($W_i = 4.2 \cdot 10^{-8}$ mol L^{-1} s^{-1}, $T = 60$ °C)

Antioxidant	$C_{AO} \cdot 10^4$ /mol L^{-1}	τ_i /min	$W_{O_2}{}^0 \cdot 10^{-7}$	$W_{O_2}{}^{max} \cdot 10^{-7}$	$W_{O_2}{}^{max}$(MO)/ /$W_{O_2}{}^{max}$(AO)	f_{app}	
			mol L^{-1} s^{-1}			f_1	f_3
—	0	26	1.90	8.00	1.0	—	—
ICPAN-9	1	100	1.77	7.20	1.1	2.6	2.6
	2	200	1.24	4.93	1.6	2.6	2.6
	4	410	0.93	4.40	1.8	2.6	2.6
	6	600	0.74	3.45	2.3	2.6	2.6
	8	820	0.44	3.41	2.4	2.6	2.6
	10	1025	0.21	3.35	2.4	2.6	2.6
ICPAN-10	1	110	1.06	5.95	1.3	2.8	2.8
	2	210	0.74	4.52	1.8	2.8	2.8
	4	450	0.62	3.92	2.1	2.8	2.8
	6	670	0.54	3.31	2.4	2.8	2.8
	8	900	0.37	3.27	2.4	2.8	2.8
	10	1125	0.31	3.18	2.5	2.8	2.8
ICPAN-10-C-8	1	50	1.86	7.67	1.0	0.6	—
	2	60	1.49	7.08	1.1	0.6	—
	4	80	0.92	5.06	1.6	0.5	—
	6	100	0.73	4.15	1.9	—	0.6
	8	225	0.57	3.72	2.2	—	0.7
	10	350	0.29	3.47	2.3	—	0.9
ICPAN-10-C-10	1	60	1.24	5.58	1.4	1.3	—
	2	100	1.06	4.43	1.8	1.0	—
	4	190	0.93	4.13	2.0	1.0	—
	6	260	0.28	4.00	2.0	—	0.8
	8	400	0.21	3.72	2.2	—	1.0
	10	540	0.20	3.31	2.4	—	1.1
ICPAN-10-C-12	1	50	1.65	6.98	1.1	1.3	—
	2	90	1.49	4.65	1.7	1.3	—
	4	100	0.74	4.20	1.9	1.2	—
	6	180	0.72	4.10	2.0	—	1.1
	8	325	0.62	3.55	2.3	—	1.3
	10	440	0.60	3.35	2.4	—	1.4
ICPAN-10-C-16	1	75	1.06	4.22	1.9	1.9	—
	2	130	0.93	4.09	2.0	1.6	—
	4	200	0.90	3.65	2.2	—	—
	6	475	0.47	3.57	2.2	—	2.0
	8	750	0.37	3.44	2.3	—	2.4
	10	1075	0.35	3.19	2.5	—	2.7

Note. τ_i is the induction period; $W_{O_2}{}^0$ and $W_{O_2}{}^{max}$ are the initial and maximum oxidation rates, respectively; f_1 and f_3 are the inhibition factors in the first and third regions of the concentration dependence, respectively.

peroxide decomposition is a specific feature of the mechanism of action of the AOs under study. To confirm this hypothesis, we studied the kinetics of hydroperoxide accumulation after the ICPANs were introduced into the partially oxidized substrate (Fig. 2). The data in Fig. 2 show that the hydroperoxide concentration decreases during the first hour and remains unchanged at a low level for the further 8 h, whereas peroxides continue to accumulate in a reference experiment (without AOs). The most efficient systems capable of decomposing hydroperoxides are ICPAN-9, ICPAN-10, and ICPAN-10-C-16 (Table 3). The results of two independent methods indicate that the AOs are involved in the decomposition of the primary oxidation products. Hydroperoxide decomposition is accompanied, most likely, by the formation of molecular adducts, because no secondary initiation of oxidation occurs.

Thus, the AOs under study can efficiently terminate the oxidation chains by the interaction with the peroxy radicals and present a possibility of the secondary initiation of the process due to hydroperoxide decomposition *via* the molecular route.

The effect of the ICPANs was studied in comparison with the known AOs, *viz.,* dibunol and TP. The kinetic

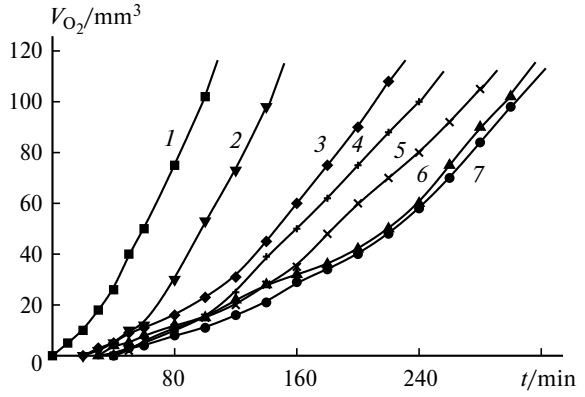

Fig. 1. Kinetic curves of oxygen absorption for the initiated oxidation of MO in the presence of different AOs: reference experiment (without AO) (*1*), ICPAN-10-C-8 (*2*), ICPAN-10-C-10 (*3*), ICPAN-10-C-12 (*4*), ICPAN-10-C-16 (*5*), ICPAN-9 (*6*), and ICPAN-10 (*7*) (MO : PhCl = 1 : 1, $C_{AO} = 2 \cdot 10^{-4}$ mol L^{-1}, $W_i = 4.2 \cdot 10^{-8}$ mol L^{-1} s^{-1}, $T = 60$ °C).

curves of oxygen absorption in the presence of comparable amounts of the ICPANs are presented in Fig. 1. It is seen that all compounds studied inhibit MO oxidation (see Table 1). Based on the character of the dependence of the induction periods on the AO concentration, we can divide the inhibitors into two groups. Phenosan K, methyl ester of phenosan-1, dibunol, ICPAN-9, and ICPAN-10 are characterized by linear dependences (Fig. 3, *a*). It should be noted that the inhibition effect of these ICPANs exceeds that of TP (almost twofold) and those of dibunol, phenosan K, and methyl ester of phenosan (by 30%) (see Fig. 3, *a*). The longest inhibition periods in the studied concentration range are observed for ICPAN-10.

For the second group of inhibitors, the plots of the induction periods *vs.* concentration are not linear but S-shaped (Fig. 3, *b*). This group includes the AOs con-

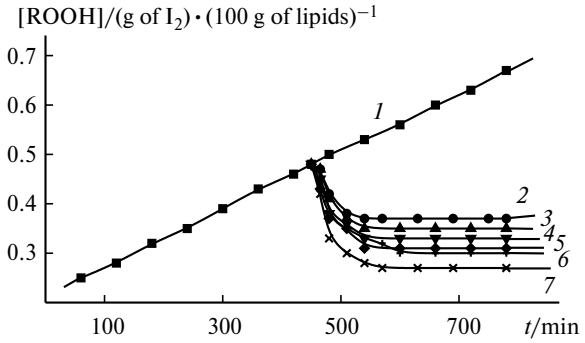

Fig. 2. Kinetic curves of hydroperoxide (ROOH) accumulation during MO autooxidation in the presence of equal concentrations of different AOs: reference experiment (without AO) (*1*), ICPAN-10-C-8 (*2*), ICPAN-10-C-10 (*3*), ICPAN-10-C-12 (*4*), ICPAN-9 (*5*), ICPAN-10-C-16 (*6*), and ICPAN-10 (*7*) ($T = 60$ °C).

Table 3. Kinetics of hydroperoxide decomposition during the autooxidation of methyl oleate in the presence of the antioxidants (AOs) (C_{AO} = const = $2 \cdot 10^{-4}$ mol L^{-1}, $T = 60$ °C)

Antioxidant	$W \cdot 10^{-4}$ /(g of I_2) (100 g of lipids)$^{-1}$ s^{-1}		Decomposition of ROOH* (%)
	accumulation	decomposition	
—**	5.90	—	—
ICPAN-10	—	5.6	54.7
ICPAN-9	—	4.8	48.4
ICPAN-10-C-8	—	4.2	41.0
ICPAN-10-C-10	—	4.5	44.1
ICPAN-10-C-12	—	4.8	46.8
ICPAN-10-C-16	—	5.1	50.1

* For 7 h.
** Reference experiment (without AO).

taining alkyl substituents with different numbers of C atoms at the N atom (see Table 1). The S-shaped curves for these AOs have three regions (see Fig. 3, *b*). In the first region, the dependence for all AOs is linear; however,

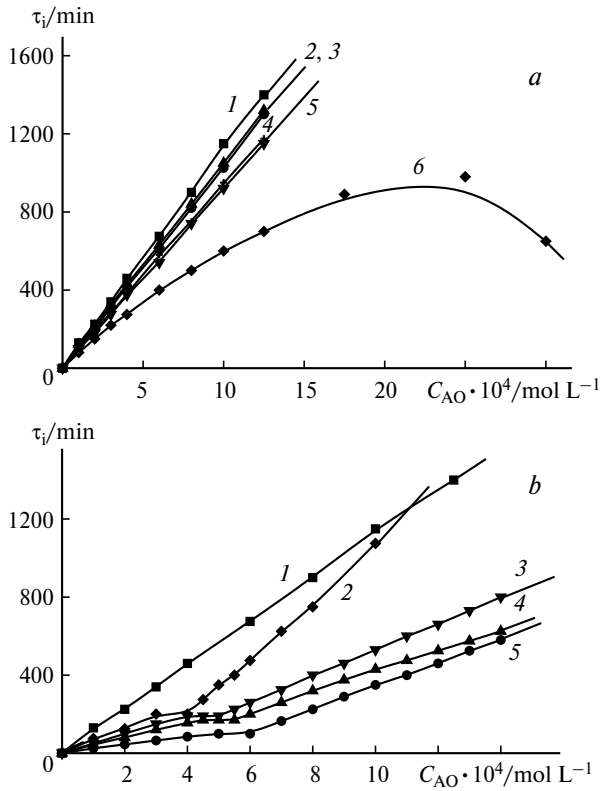

Fig. 3. Plots of the induction period (τ_i) *vs.* AO concentration (C_{AO}): (*a*) ICPAN-10 (*1*), methyl ester of phenosan (*2*), ICPAN-9 (*3*), phenosan K (*4*), dibunol (*5*), and TP (*6*); (*b*) ICPAN-10 (*1*), ICPAN-10-C-16 (*2*), ICPAN-10-C-12 (*3*), ICPAN-10-C-10 (*4*), and ICPAN-10-C-8 (*5*). For the oxidation conditions, see the caption for Fig. 1.

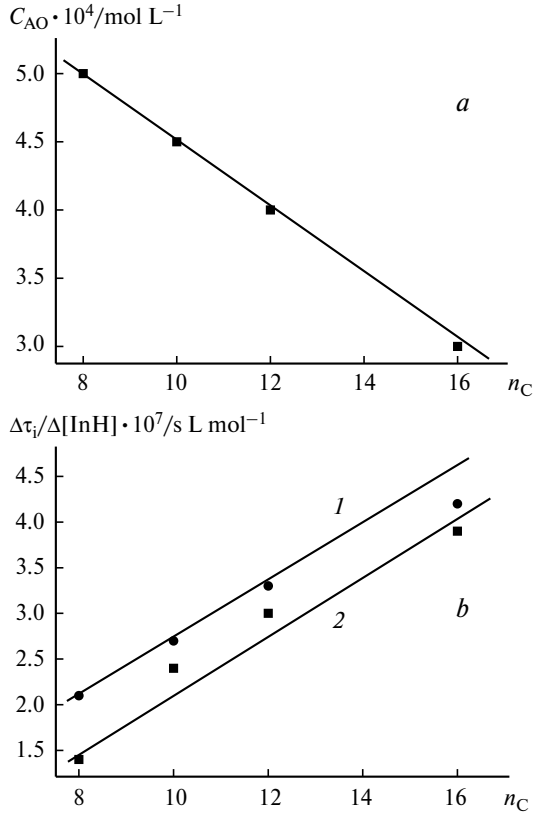

Fig. 4. "Threshold" micelle formation concentration (*a*) and $\Delta\tau_i/\Delta[InH]$ parameter (*b*) *vs.* chain lengths of the R´ substituent (n_C) in the third (*1*) and first (*2*) regions of the concentration dependence.

with an increase in the concentration, the second, rather narrow section appears in the curves. No change in the induction periods occurs within this section. The overall efficiency of inhibition, which is directly proportional to the AO concentration, again increases after some "threshold" concentration is exceeded.

These differences in the AOs effect are related to their structure. The "threshold" concentration corresponding to a break in the concentration curves decreases proportionally to the elongation of the R´ substituent chain (Fig. 4, *a*). This correlation indicates that macromolecular structures can be formed in solutions at certain AO concentrations, because, as known,[9] the critical micelle concentration decreases with the elongation of the hydrocarbon radical of a polar molecule.

The slopes of the concentration curves in the first and third regions differ substantially and are related to the chain length of the R´ substituent (Fig. 4, *b*). The slopes were quantitatively estimated from the $\Delta\tau_i/\Delta[InH]$ value, which is the ratio of the increment of the induction periods to the increment of the AO concentration. A direct interrelation between the $\Delta\tau_i/\Delta[InH]$ parameter and the chain length of the R´ radical is observed (see Fig. 4, *b*). From these data, taking into account that

$$\tau_i = f \cdot [InH]/W_i,$$

where f is the inhibition factor that shows the number of free radicals reacting with an inhibitor molecule, and the experimentally determined (by the inhibitor method) initiation rate value ($W_i = 4.2 \cdot 10^{-8}$ mol L^{-1} s^{-1}), we estimated the apparent f value (f_{app}). It has been shown above by the chemiluminescence method that for the AOs studied f is close or equal to two. The f_{app} values determined for the groups of the AOs, whose concentration plots have no breaks (see Fig. 3, *a*), remained numerically unchanged in the whole region of concentrations studied (see Table 2), while for the second group of inhibitors (ICPANs) the f values differed in the first and third regions of the curves.

The above-described regularities are related, most likely, to the formation of monolayered or bilayered (when the concentration increases) microheterogeneous structures. Evidently, they are formed easily due to the length of the hydrocarbon radical at the quaternized (ammonium) N atom. In the structure of monolayer micelles, the long-chain AO radicals are oriented, most likely, to the external surface, and the polar OH groups of phenol are directed to the internal surface. A hydrogen bond can appear between the adjacent OH groups, which decreases the possibility of their interaction with the peroxy radicals participating in the oxidation. When the bilayered structures are formed, the phenolic fragments of the molecules are localized on the external and internal surfaces of the bilayer, whereas the hydrophobic R´ radicals inside the structure are oriented toward each other. The localization of the OH groups of phenol on the external micellar surface allows them to interact with the peroxy radicals. This is kinetically expressed as anomalously high f_{app} values in the third region of the concentration curves.

To check the hypothesis about a possibility of AO structurization, we studied the change in the refraction index at different AO concentrations. At the "threshold" AO concentrations, the optical properties of the solutions change sharply, and a break caused by micelle formation appears in the concentration curves. The critical micelle concentration of the solutions was also estimated by the Rebinder method[6] and almost corresponded to the "threshold" concentrations. Therefore, data of independent methods confirm the assumption on the formation of microheterogeneous systems in lipid solutions.

Thus, the efficiency of inhibition is determined by the antiradical activity of the AOs under study and also by the formation of microheterogeneous systems involving these AOs.

The above-presented data stimulated us to study the kinetic features of lipid oxidation in an aqueous-emulsion

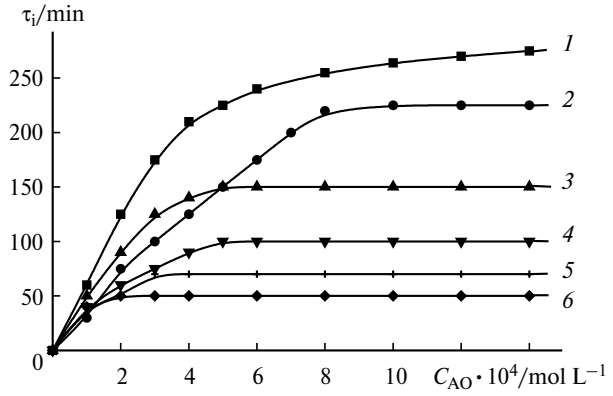

Fig. 5. Plots of the induction period (τ_i) *vs.* AO concentration (C_{AO}) for the initiated oxidation of MO in an aqueous-emulsion medium: ICPAN-10-C-10 (*1*), ICPAN-10-C-12 (*2*), ICPAN-10-C-8 (*3*), ICPAN-9 (*4*), ICPAN-10-C-16 (*5*), and ICPAN-10 (*6*). For the oxidation conditions, see the caption for Fig. 1.

medium in the presence of sodium dodecyl sulfate at the water to oil ratio equal to 1 : 1. The plots of the induction periods *vs.* AO concentration in an aqueous-emulsion medium are shown in Fig. 5. It is seen that these curves are of the same type regardless of the inhibitor structure: in a concentration range of $(2.0—4.5) \cdot 10^{-4}$ mol L^{-1} they reach a plateau, and the further increase in the AO amount does not elongate τ_i. The maximum τ_i values are shown to decrease with an increase in the number of C atoms in the chain of the R′ substituent. In essence, Fig. 5 reproduces the first region of the curves presented in Fig. 3, *b*. This correspondence is caused, probably, by the formation of microheterogeneous systems of the same type. It is likely that microreactors are formed, whose cores consist of active OH groups of phenols, and ionogenic ammonium groups linked with the alkyl R′ substituent are arranged outside a micelle. Under these conditions, sodium dodecyl sulfate provides the stabilization of the system due to the interaction of the hydrophobic fragments of inhibitor and surfactant molecules.[9]

Thus, the results obtained in this work show that the efficiency of the action of ICPANs as AOs depends on both their chemical structure and oxidation conditions. Compounds ICPAN-9 and ICPAN-10 in homogeneous and heterogeneous solutions outperform in activity the known synthetic and natural inhibitors of oxidation (dibunol, TP). Analogs with long-chain substituents at the quaternized N atom exhibit a relatively weak overall inhibiting activity due to the formation of microheterogeneous systems, and this activity is enhanced with the elongation of the R′ substituent.

It can be assumed that the sizes of the bilayered structures formed by the ICPANs with the R′ substituent length comparable with the chain length of highest aliphatic acids in the phospholipid structure (18—22 C atoms) correspond to the thickness of biological membranes. This fact would provide, most probably, their maximum retention in membranes and the stabilization of non-enzymatic oxidation processes on the internal cytoplasmatic and external surfaces of cellular membranes.

The relatively low toxicity and high efficiency of the ICPANs make it possible to consider them as antioxidants promising for the stabilization of the oxidation of homogenates and emulsions of edible and biologically active lipids of different origin.

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