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Influence of a Constant Magnetic Field on the Mechanism of Adrenaline Oxidation

Vyacheslav N. Kazin, Evgenii A. Guzov, Valentina A. Moshareva and Evgenii M. Pliss *

Institute of Fundamental and Applied Chemistry, P.G. Demidov Yaroslavl State University, Sovetskaya St. 14, Yaroslavl 150003, Russia; kaz@bio.uniyar.ac.ru (V.N.K.); evgeniy.guzov@yandex.ru (E.A.G.); valya-valya-m@mail.ru (V.A.M.)
* Correspondence: pliss@uniyar.ac.ru

Abstract: In order to establish the role of the magnetic effect in the key stages of the autoxidation and initiated oxidation radical-chain reactions, the experimental data and kinetic analysis of the influence of a magnetic field on the oxidative transformations of adrenaline are presented in this work. In the case of autoxidation, the process is being controlled by the rate of adrenaline consumption in the gross process of quinoid oxidation. The analysis of the obtained results is estimative and is based on the assumption of the leading role of superoxide radical during the autoxidation. Superoxide radical concentration increases with the increase in the applied magnetic field strength, which leads to the decrease in the rate of initiation of the quinoid process. In the case of initiated oxidation, the results obtained are based on the known radical-chain mechanism, and they were interpreted using the theory of radical pairs. The observed magnetic effect is explained by the influence of a constant magnetic field on the mechanism of chain termination of radical-chain oxidation and/or initiation of the autoxidation process.

Keywords: magnetic field effect; radical pairs; adrenaline; superoxide dismutase; auto- and initiated oxidation kinetics

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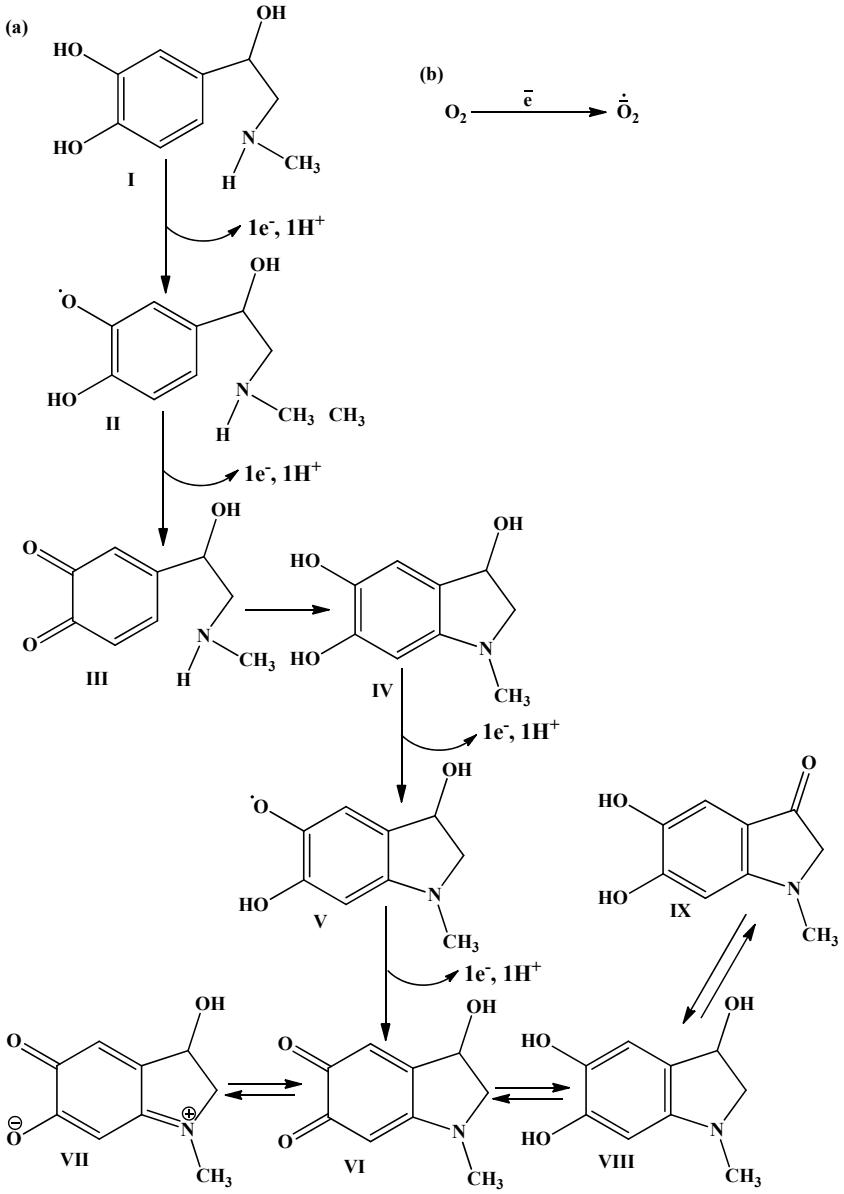
1. Introduction

1.1. The Base Mechanisms

The influence of a magnetic field on the processes occurring in living systems is of unrelenting interest to chemists and biologists [1–7]. The magnetic dependence of biosystems manifests itself at the level of elementary chemical processes in which spin particles are generated or participate. Unpaired electrons in these particles are carriers of spin magnetism: they are the ones that interact with magnetic fields. In a living system, they are the primary target for such interaction [2,8–13]. Within the framework of this concept, the adrenaline autoxidation reaction may serve as the marker of a magnetic field influence. In biophysics, this process is considered as a model for revealing the antioxidant properties of various compounds that inhibit the accumulation of the reaction product. It is assumed that, in an alkaline medium, oxygen molecules can predominantly act as electron acceptors being transformed to superoxide anion-radials ($O_2^{\bullet-}$). Therefore, the process of chemical transformation of adrenaline is caused by the formation of $O_2^{\bullet-}$ (Scheme 1) [14–20].

Most of the information was obtained for significant conversion depths and refers to the hypothetical gross process of adrenaline → adrenochrome transformation [12,13,15–18]. The detailed mechanism of the quinoid process is still actively discussed in the literature, and the role of magnetic fields is considered only from the standpoint of their possible effect on oxidative stress [1,4,7,14,18–21].

The inhibitor of adrenaline autoxidation is superoxide dismutase (SOD), which inhibits the process by trapping $O_2^{\bullet-}$ particles [17,22,23].



Scheme 1. The quinoid mechanism of adrenaline autoxidation. (**a**) Transformation of adrenaline **I** into adrenochrome **VI**; **II**–**V** are intermediate reaction products; possible mutual transitions of the products varying from **VI** (adrenochrome) to **VII**, **VIII**, and **IX**; (**b**) reaction of superoxide anion-radial formation.

It can be assumed that, along with the quinoid transformation, adrenaline {*p*,*m*-$(HO)_2C_6H_3CH(OH)CH_2N(H)CH_3$}, as a catecholamine, also participates in various stages of the classical radical-chain oxidation by molecular oxygen [22–25], which proceeds rather conjugated and/or may be its integral part (Scheme 2).

Almost no direct quantitative study of the kinetics of radical-chain oxidation of adrenaline by molecular oxygen has been carried out; there are no data on the effect of a magnetic field on this process. Although, this process has been studied in detail for other organic compounds and interpreted within the framework of the theory of radical pairs [8,9,11,25–28].

(i) $I\ (+O_2, + RH) \rightarrow R^{\bullet}$

(1) $R^{\bullet} + O_2 \rightarrow RO_2^{\bullet}$

(2) $RO_2^{\bullet} + RH \rightarrow ROOH + R^{\bullet}$

(3) $ROOH \rightarrow RO^{\bullet} + {}^{\bullet}OH$

(4) $R^{\bullet} + R^{\bullet} \rightarrow RR$

(5) $R^{\bullet} + RO_2^{\bullet} \rightarrow ROOR$

(6) $RO_2^{\bullet} + RO_2^{\bullet} \rightarrow ROOR + O_2$

(7) $RO_2^{\bullet} + QH_2 \rightarrow ROOH + QH^{\bullet}$

$QH^{\bullet} \leftrightarrow Q^{\bullet-} + H^+$

(8) $RO_2^{\bullet} + Q^{\bullet-} \xrightarrow{+H^+} Q + ROOH$

(9) $Q^{\bullet-} + Q^{\bullet-} + 2H^+ \rightarrow QH_2 + Q$

(10) $Q^{\bullet-} + O_2 \rightarrow Q + O_2^{\bullet-}$

(2′) $O_2^{\bullet-} + RH \xrightarrow{+H^+,\ +O_2} H_2O_2 + RO_2^{\bullet}$

(6′) $O_2^{\bullet-} + O_2^{\bullet-} \xrightarrow{+2H^+,\ SOD} H_2O_2 + O_2$

(11) $Q \leftrightarrow Q'H_2$

(7′) $RO_2^{\bullet} + Q'H_2 \rightarrow ROOH + Q'H^{\bullet}$

Scheme 2. Mechanism of radical-chain oxidation of adrenaline. RH = $-CH_2N-$; QH_2 = *p,m*-$(HO)_2C_6H_3-$.

1.2. Purpose of the Work and Research Methodology

The purpose of this work was to establish the role of the magnetic effect in the key stages of adrenaline oxidative transformations both in the autoxidation process and in the initiated radical-chain reaction.

Based on the above literature data [2,9,11,17,18,25–28], it can be assumed that the magnetic field influence on the processes of auto- and initiated oxidation is expected to be realized for different key reactions and/or their probable superposition. In accordance with this assumption, the experimental approach to the studied transformations of adrenaline and analysis of the magnetic field effect was also formed.

2. Experimental

The kinetics of adrenaline autoxidation were studied using the rate of consumption (W) at T = 310 ± 0.1 K in carbonate buffer (pH = 10.40–10.70) using the method of electron spectroscopy on a Perkin Elmer Lambda 35 spectrophotometer (USA) similarly to [29].

Adrenaline (Adr) was used in the form of hydrochloride, the solutions of which were prepared immediately before the experiment. The experiments were carried out with gas (oxygen or air) purged at atmospheric pressure, SOD concentration was 30–90 U/mL, constant magnetic field induction B = 75–125 mT. The magnetic effect (MFE) was determined as the ratio of adrenaline consumption rates in the applied magnetic field W(H) and in the Earth's natural magnetic field W(0): MFE = W(H)/W(0). Experiment details and installation design are described in our previous works [12,13,29].

In the absence of an applied magnetic field, the kinetics of oxygen consumption during the Adr oxidation initiated by 2,2′-azobis(2-methylpropionamide) dihydrochloride (AAPH) in phosphate and carbonate buffer was studied at T = 310 ± 0.1 K using the computerized

biological oxygen monitor Yellow Springs Instruments Co. Model 5300A at atmospheric pressure; [SOD] = 0–200 U/mL.

In an applied magnetic field, the kinetics of oxygen consumption at constant pressure (1 atm) was studied at $T = 310 \pm 0.1$ K using a highly sensitive capillary microvolumemeter. The rate of radical initiation (W_i) was determined by the method of inhibitors in an anaerobic atmosphere (Argon). Nitroxyl radical 4-hydroxy-2,2,6,6-tetramethylpiperidine-1-oxyl (>NO•) was used as an inhibitor. Changes in [>NO•] were monitored by ESR method (spectrometer CMS 8400). The magnetic field effect (MFE) was determined as the ratio of the rates of initiated oxidation $W_0(H)$ in the applied magnetic field $B = 0.3$–0.6 T and in Earth's natural magnetic field $W_0(0)$: MFE = $W_0(H)/W_0(0)$. Experiment details and installation design are described in our previous works [11,25–28].

The rates of changes in adrenaline (W) or oxygen (W_0) concentrations were calculated from the primary experimental data using the Kinetics-2012 optimization program [30].

3. Results and Discussion

3.1. Autooxidation of Adrenaline

As pH increases, the increase in autoxidation rate is observed (Table 1). This trend takes place up to considerable conversion depths.

Table 1. Comparison of the rates of adrenaline consumption (W) at different pH. Carbonate buffer, $[Adr]_0 = 68$ μM, air atmosphere, 310 K.

pH	10.40	10.60	10.70
$W \cdot 10^3$, M·s^{-1}	3.8	6.5	10.2

The addition of SOD at a constant pH value leads to a decrease in adrenaline consumption rate, which decreases with gain in the enzyme activity (Figure 1, curves 1 and 3; Table 2).

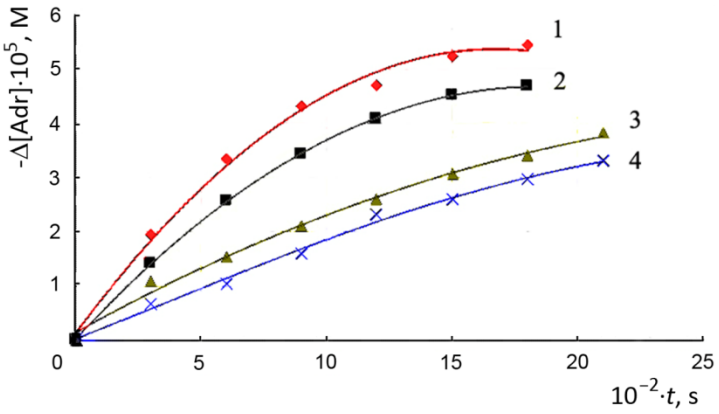


Figure 1. Kinetics of adrenaline consumption in carbonate buffer. pH = 10.60, $[Adr]_0 = 68$ μM, air atmosphere, 310 K; $B = 0$ (1); $B = 90$ μT (2); $B = 0$, [SOD] = 60 U/mL (3); $B = 90$ μT, [SOD] = 60 U/mL (4).

Table 2. Effect of SOD activity on the rate of adrenaline consumption. Carbonate buffer, pH = 10.60, $[Adr]_0 = 68$ μM, air atmosphere, 310 K.

$[SOD]_0$, U/mL	0	30	60	90
$W \cdot 10^3$, M·s^{-1}	6.5	5.0	2.8	1.5

A similar effect was also described in [19]: the same method was used to determine the activity of superoxide dismutase by its ability to inhibit the reaction of adrenaline autooxidation in an alkaline medium.

The results obtained are associated with the formation of reactive oxygen species (ROS): superoxide ($O_2^{\bullet -}$), hydroperoxyl ($HO_2^{\bullet}$), hydroxyl ($HO^{\bullet}$) radicals, and hydrogen peroxide (HOOH) [18,22,23]. Carbonate/bicarbonate buffer systems can also act as prooxidants: these biologically active components are involved in redox reactions, so the formation of radical anions $CO_2^{\bullet -}$ and $CO_3^{\bullet -}$ is possible [14,15,18].

The magnetic field effect leads to a decrease in the rate of adrenaline consumption at a constant pH value, and with an increase in *B*, the MFE decreases (Figure 1, curves 1 and 2, Table 3).

Table 3. Influence of the magnetic field strength (*B*) on the rate of adrenaline consumption (*W*) and the values of the magnetic field effect. Carbonate buffer, pH = 10.60, $[Adr]_0$ = 68 μM, air atmosphere, 310 K.

***B*, μT**	**0**	**75**	**90**	**125**
$W \cdot 10^3$, $M \cdot s^{-1}$	6.5	4.9	4.7	3.2
MFE ± 0.02	1	0.77	0.72	0.53

The addition of SOD in an applied magnetic field leads to an even greater drop in MFE values (Figure 1, curves 3 and 4; Table 4).

Table 4. Dependence of adrenaline consumption rate (*W*) and magnetic field effect (MFE) values on SOD activity in the absence of a magnetic field and in its presence. Carbonate buffer, pH = 10.60, $[Adr]_0$ = 68 μM, air atmosphere, 310 K.

[SOD], U/mL	**0**	**30**	**60**	**90**
		$W \cdot 10^3$, $M \cdot s^{-1}$		
B = 0	6.5	4.9	2.8	1.5
B = 90 μT	4.7	3.9	2.3	1.2
MFE ± 0.1	0.72	0.61	0.33	0.18

The presence of dissolved oxygen determines the following sequence of transformations [14,18,22,23]:

$$O_2 \xrightarrow{+e^-} O_2^{\bullet -} \xrightarrow{+e^- (+2H^+)} H_2O_2 \xrightarrow{+e^-} HO^{\bullet}(+HO^-) \Big) \xrightarrow{+e^- (+2H^+)} 2H_2O$$

An important role is also played by the equilibrium $HO_2^{\bullet} \leftrightarrow H^+ + O_2^{\bullet -}$, which, at low pH values (physiological conditions), is strongly shifted towards the formation of $HO_2^{\bullet}$, while at high pH values (alkaline media) the share of $O_2^{\bullet -}$ increases [22,23].

A wide set of intermediate products appears [18–20], in which many reactions are "sensitive" to pH value. For example, it is assumed that autooxidation products participate in catalytic transformations with peroxy radicals formation [31].

In accordance with [11,25–28], the influence of a magnetic field increases the disproportionation rate constant of superoxide radicals, lowers their concentration, which leads to a drop in *W*. By intercepting $O_2^{\bullet -}$, the superoxide dismutase catalyzes the conversion of highly reactive superoxide into relatively less active hydrogen peroxide and molecular oxygen, which also reduces *W*. The net effect of these two factors is realized as a drop in MFE value.

As it has been already noted in the Section 1, reactions of molecular oxygen with the phenolic fragment of adrenaline and with semiquinone radical-ion play an important role, but these reactions are not taken into account in the quinoid mechanism [18–20].

Based on considerations described above, a series of experiments was carried out on the initiated oxidation of adrenaline with molecular oxygen in phosphate and carbonate buffers at low conversion depths (≤1%) when the effect of oxidation products can be neglected.

3.2. Radical-Chain Oxidation of Adrenaline

In the absence of an applied magnetic field, the kinetics of oxygen consumption are linear, and with increasing pH, the initial rate of O_2 consumption (W_0) increases (Figure 2).

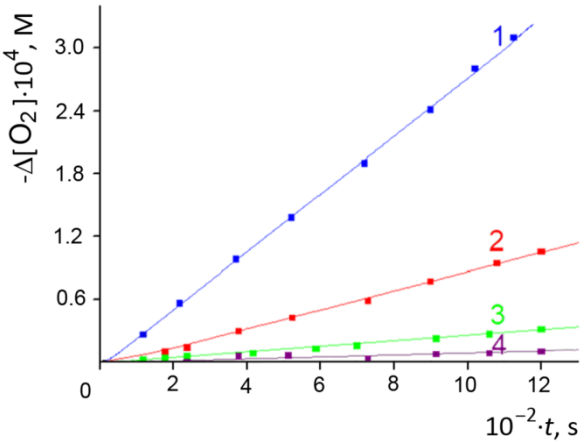


Figure 2. Kinetics of oxygen consumption during the oxidation of adrenaline in phosphate buffer. [AAPH] = $4 \cdot 10^{-3}$ M; $[Adr]_0 = 1 \cdot 10^{-3}$ M; pH = 9.0 (1), 8.2 (2), 7.4 (3), 6.0 (4); 310 K.

Note that at constant values of W_i and pH, W_0 linearly depends on $[Adr]_0$ (as can be seen from Figure 2). With an increase in conversion depth, W_0 increases, and in some cases reaches its maximum value followed by the relative decreasing or reaching the limit (Figure 3).

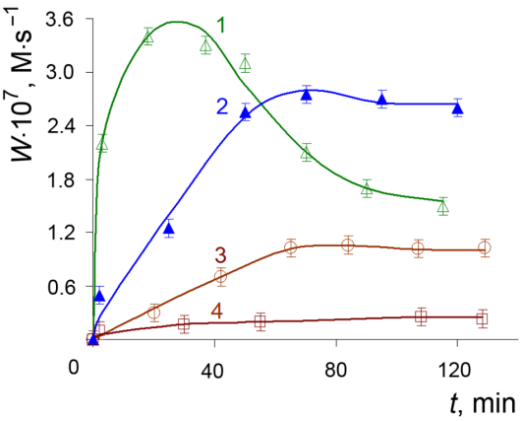


Figure 3. Dependence of oxygen consumption rate on time during the oxidation of adrenaline in phosphate buffer. [AAPH] = $4 \cdot 10^{-3}$ M; $[Adr]_0 = 1 \cdot 10^{-3}$ M, pH = 9.0 (1), 8.2 (2), 7.4 (3), 6.0 (4); 310 K.

One of the reasons for the observed effects is the accumulation of hydroperoxides, which occurs faster with an increase in W_i and the initial concentration of substrates. This process leads to the formation of mixed micelles. The drop in oxidation rate after reaching $W_{0(max)}$ may be due to the substrate consumption. Detailed analysis of this situation was carried out in [32].

Table 5 shows the estimated dependence of the oxidation rate order (n_i) on the initiation rate (W_i) for various time intervals.

Table 5. Change in the order of initiated reaction of adrenaline oxidation (n_i) with respect to W_i over time. Phosphate buffer, $[Adr]_0 = 1 \cdot 10^{-3}$ M, pH = 7.4, air atmosphere, 310 K.

$W_i \cdot 10^9$, M s^{-1}	$W_{0(20)} \cdot 10^8$, M s^{-1}	$W_{0(70)} \cdot 10^8$, M s^{-1}	$W_{0(100)} \cdot 10^8$, M s^{-1}	$W_{0(150)} \cdot 10^8$, M s^{-1}
1.0	0.82	0.81	0.80	0.78
4.0	1.74	1.66	1.62	1.55
$n_i \pm 0.02$	0.54	0.52	0.51	0.50

The numbers in brackets indicate the time point, in minutes, for which the adrenaline oxidation rate was calculated.

It is noteworthy that during the entire time interval the oxidation rate is proportional to $\sqrt{W_i}$, i.e., the quadratic chain termination takes place [22] (we will return to the analysis of this result below).

Figure 4 illustrates the comparison of the influence of SOD and buffer type on radical-chain oxidation of adrenaline at a constant pH value.

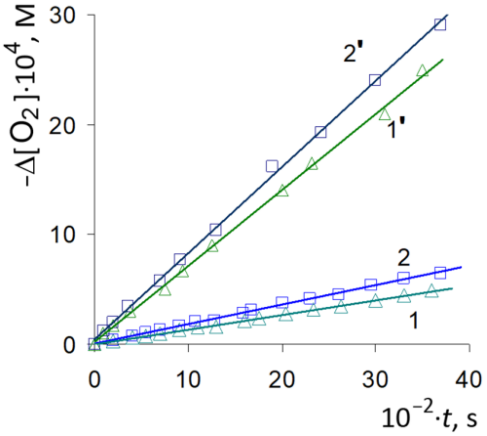


Figure 4. Kinetics of oxygen consumption during the oxidation of adrenaline. [AAPH] = $4 \cdot 10^{-3}$ M; $[Adr]_0 = 1 \cdot 10^{-3}$ M; H = 9.0; 310 K. Carbonate buffer: $[SOD]_0$ = 100 U/mL (1′ Δ), $[SOD]_0$ = 0 U/mL (2′ □); phosphate buffer: $[SOD]_0$ = 100 U/mL (1 Δ), $[SOD]_0$ = 0 U/mL (2 □).

The rate of the process in carbonate buffer is actually an order of magnitude higher than in phosphate buffer. It indicates a significant effect exerted by the radical anions $CO_2^{\bullet -}$ and $CO_3^{\bullet -}$ [15,17,18,27]. We emphasize that the effect of SOD is much weaker than in the case of measurement by the rate of adrenaline consumption (see Figure 1 and Table 2). This is due to the fundamental difference between the mechanisms of auto- and initiated oxidation.

Table 6 contains the obtained MFE values during the initiated oxidation of adrenaline.

Table 6. Values of the magnetic field effect (MFE) for the initiated oxidation of adrenaline. Phosphate buffer, pH = 7.4, [Adr] = 0.5 M, air atmosphere, $W_i = 1.2 \cdot 10^7$ M·s^{-1}, 310 K.

B, T	**0.3**	**0.4**	**0.5**	**0.6**
[SOD], U/mL		MFE ± 0.02		
0	1.11	1.31	1.52	1.72
100	1.14	1.35	1.56	1.74
200	1.17	1.38	1.58	1.77

As can be seen from Table 6, the influence of SOD and magnetic field strength on MFE values is noticeable only at $B \geq 0.3$ T. Most likely, the magnetic field has a dominant effect.

It can be concluded that the obtained data (in the first approximation) correspond to the kinetics of radical-chain oxidation with quadratic chain termination [22,23], i.e.,

$W_0 \sim [\text{Adr}]_0 \cdot \sqrt{W_i}$. Autooxidation of adrenaline (in the absence of radical initiator) proceeds at a high rate in an alkaline medium (pH > 9, see Tables 1–4, Figure 1). A similar trend is also observed for the initiated oxidation (Figures 2 and 3). The quadratic chain termination may be due to the oxidation of adrenaline under the action of AAPH and the disproportionation reactions of $HO_2^{\bullet}$, $QH^{\bullet}$, $Q^{\bullet}$, and $Q^{\bullet -}$ (see Scheme 2). In this case, the formal-kinetic oxidation mechanism can be approximately considered by analogy with the oxidation of other organic compounds [22,23].

Under experimental conditions, the chain termination step (reactions 4–6) is the only one spin-selective and, therefore, magneto-selective process [25–27]. The spin state of a radical pair can change due to spin evolution, for example, due to the Zeeman interaction of electron spins with an environment (with magnetic field, in this case [2,9,33,34]). In accordance with [2,9,11,26–28,33,34], magnetic field reduces the probability of recombination and cross-recombination of radical pairs. Therefore, the oxidation rate should increase with increase in field strength. It can be assumed that this provision also applies to ion-radical pairs: reactions (8), (9), and (6′). This means that the value of MFE = $W_{0(H)}/W_{0(0)}$ should increase with the growth of B, which is reflected in the data in Table 6. Of course, these considerations are only qualitative and require large-scale experimental confirmation with the determination of the rate constants of reactions (8), (9), and (6′). Obviously, such a study is far beyond the scope of this work.

4. Conclusions

We emphasize that the influence of the magnetic field on the processes of auto- and initiated oxidation is fundamentally different. In the first case, the process is being controlled by the rate of adrenaline consumption in the gross process of quinoid oxidation, the mechanism of which, as noted in Section 1, is still debatable. Therefore, the analysis of the obtained results is of an evaluative nature and is based on the assumption of the leading role of superoxide radical ($O_2^{\bullet -}$) in autoxidation. The concentration of $O_2^{\bullet -}$ increases with increasing the applied magnetic field strength, which leads to a decrease in quinoid process initiation rate and is reflected in MFE values. In the second case (initiated oxidation), the results obtained are based on the known radical-chain mechanism, and their evaluation was carried out within the framework of the theory of radical pairs. In this case, an increase in magnetic field strength leads to an increase in the overall process rate, which is interpreted as magnetic field effect.

We emphasize here the following circumstances:

- The quinoid process proceeds at high rates only in carbonate buffer at high pH values. Radical-chain oxidation proceeds at a measurable rate in both carbonate and phosphate buffers. However, the considered mechanism is valid for physiological values of pH = 7.4. Therefore, each process must be analyzed separately, although their superposition is not excluded;
- The effect of SOD on the rate of autoxidation (measured by the rate of adrenaline consumption) is significantly higher than on the rate of oxygen consumption;
- One should take into account the multiphase nature of the processes, the analysis of which still requires proving the eligibility of using liquid-phase kinetic models.

However, even in this phenomenological form, the presented material may be relevant for studying the effect of a stationary magnetic field on lipid peroxidation. These data can become the basis for interpreting new results under varying experimental conditions (phase state, magnetic field induction, pH, substrate concentration, etc.).

It is necessary to significantly expand the methodological arsenal of research by combining electrochemical (voltammetry, potentiometry) and spectral (electron spectroscopy) methods with the ones of oxygen concentration monitoring (microvolumemetry, oxygen monitor) and paramagnetic particle analysis (ESR) methods. The use of kinetic and quantum-chemical modeling is also obvious. Only such an approach is going to make it possible to aim for elucidation of the detailed mechanism of the process. We must not forget that in a real process, most of the reactions under consideration are catalyzed by enzymes.

At that, the mechanism of the effect of a stationary magnetic field on the modulation of ROS concentration in cells still remains unclear. Further research on the relationship between magnetic field induction and cellular reactive oxygen radical concentration will help understand the way magnetic fields affect cellular ROS levels and the development of oxidative stress.

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