experimental values of the concentration $c(\tau)$, and the mean integral error can be calculated by the formula

$$\dot{E} = \int_0^{t_K} |M(t) - M^*(t)|\, dt. \quad (17)$$

Thus the expressions (12)–(17) and the experimental $f_K(r)$, $c(\tau)$, and $M^*(t)$ dependences form a non-linear system of equations which is not closed for three parameters [n, m, and K (or r_m)], and which must be determined by minimising the deviation (17) by one of the standard methods of multidimensional optimisation.

If the calculations by the proposed algorithm are valid the function $U_0(x)$ calculated by formula (8) should be close to the experimentally observed size distribution of the seeding crystals at the initial instant. This test can be simplified by comparing at $t = 0$ the features (9) of the distribution function with a third-order zero (which define respectively the number, the average size, the surface, and the mass of the crystals in unit volume of the two-phase medium) with the measured characteristics of the seeding material.

The results of processing our experimental results[6] on the kinetics of the selective crystallisation of the *D* and *L* isomers of racemic glutamic acid (*DL*-GA) by the above algorithm are shown in Figs. 1 and 2. Crystallisation was carried out in the unscreened periodic regime, i.e. for $\tau < \tau_K \ll \theta$, which allowed us to put $\exp(-t) \cong \exp(t_K) \cong 1$ in all the expressions (6)–(9), and to choose $\tau_m = \tau_K$ as the scale value. In Fig. 2 we show the results of calculations of the function $I(t)$ by Eqn. (7) using the experimental data on the crystallisation kinetics of the different GA isomers, and also the dependence of the rate of nucleation on the supersaturation, obtained by combining the plots of this function with those shown in Fig. 1 of our previous paper.[6] The nucleation surge in one of the experiments (curve *2* of Fig. 2*a*) is explained by an oscillation in the supersaturation, as confirmed by the monotonic outline of the corresponding curve in Fig. 2*b*. The maxima in curves *3* and *5* appear to be due to secondary nucleation, not allowed for in our model, or to errors in the measurement of the supersaturation.

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Received 19th April 1989

Translated from *Zhurnal Fizicheskoi Khimii* **64** 1774–1782 (1990)

U.D.C. 577.152.1

Formation of aldehydes from alcohols under the influence of the peroxidic form of haemoglobin and of hydroxyl radicals generated by Cu^{2+} and Fe^{3+} ions in the presence of H_2O_2

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ABSTRACT. We have shown that in the presence of hydrogen peroxide Cu^{2+} and Fe^{3+} ions, and also met-haemoglobin (met-Hb), produce hydroxyl radicals which oxidise aliphatic alcohols to aldehydes. In the presence of H_2O_2 met-Hb forms the ferriperoxidic form, which also catalyses the oxidation of alcohols to aldehydes. The effectiveness of the oxidation of aliphatic alcohols by met-Hb decreases as the length of their hydrocarbon chain is increased, in inverse proportion to the affinity of the alcohol for the protein. Under the influence of oxygen free radicals the mercapto groups of the cystein residues of the met-Hb molecule are oxidised to cysteic acid residues, and the high-spin met-Hb is converted into low-spin haemichrome.

In the animal organism ethanol is mainly oxidised to acetaldehyde by yeast alcoholdehydrogenase.[1,2] Alcohols are also oxidised to aldehydes by catalase,[3,4] haemoglobin (Hb),[5] and by an electron-transfer complex in a microsomal system conjugated to the R-450 cytochrome.[6] The oxidation of alcohols to aldehydes takes place under the influence of hydroxyl radicals,[7] and is induced by hydrogen atoms and by free radicals of different types.[8] Sources of free radicals include ionising radiation,[8] iron and copper ions,[9,10] and ions of other transition metals in reactions of the Fenton type,[7,11] as well as Hb auto-oxidation processes[12] and the reactions between the products of lipid oxidation by peroxides and transition-metal ions.[11,13,17]

Many biochemical reactions are accompanied by the formation of active forms of oxygen.[7,15] Some pathological processes arising under conditions of stress can activate free-radical reactions and increase the concentration of active forms of oxygen.[7,16,17] Free radicals are also produced by the metabolism of xenobiotics.[18] One of the most important enzyme systems generating free radicals while metabolising xenobiotics is the NADP.H-dependent microsomal cytochrome R-450.[19] By using traps for hydroxyl radicals it was shown that the microsomal oxidation of aliphatic alcohols can be strongly inhibited. This suggests that the mechanism

of the microsomal oxidation of ethanol or butanol includes the reaction between the alcohols and hydroxyl radicals.[20]

We have studied the formation of free radicals in solutions containing Fe^{3+}, Cu^{2+}, and H_2O_2, and also met-Hb and H_2O_2, and the oxidation of aliphatic alcohols by these radicals.

Experimental

Oxyhaemoglobin (oxy-Hb) was prepared from fresh donor blood by the standard method.[21] and was then crystallised.[22,23] The resulting preparation of oxy-Hb (100 mg ml^{-1}) was transferred to a column (2 × 20 cm) packed with DEAE-TS K-650 gel to complete the purification from catalase. The elution was carried out with a linear gradient of phosphate buffer (pH 8) of initial concentration 0.05 M and final concentration 0.2 M. The catalase activity was determined from the rate of suppression of the H_2O_2 absorption at 240 nm.[24] The oxy-Hb was oxidised to met-Hb by a six-fold molar excess of $K_3[Fe(CN)_6]$, followed by gel filtration on G-25 Sephadex. The protein was eluted with 0.05 M phosphate buffer at pH 7.4. The concentration of oxy-Hb and met-Hb was determined spectrophotometrically from the absorption at 576 nm (ε = 14250 M^{-1} cm^{-1}) and 630 nm (ε = 3700 M^{-1} cm^{-1}) respectively.[25] The peroxidic form of Hb was prepared by adding an excess of H_2O_2 to an aqueous solution of met-Hb.[26] The aldehydes were identified as their 2,4-dinitro-phenylhydrazones.[27] Acetaldehyde and ethanol were determined gas-chromatographically in a "Tsvet-106" chromatograph: column temperature 100 °C, evaporator temperature 120 °C, column 3 m long and 3 mm in diameter packed with 10% poly(ethylene glycol)-10000 on AW-HMDS Chromaton of particle size 0.2–0.26 mm, carrier gas nitrogen (30 cm^3 min^{-1}), air (300 cm^3 min^{-1}), or hydrogen (30 cm^3 min^{-1}). The absorption spectra were recorded with a Specord M-40 spectrophotometer (East Germany). The kinetic measurements were made by the stopped flow method[28] with automatic photo-recording of the kinetic curve displayed on an oscilloscope screen. The concentration of met-Hb bound in complexes with the alcohols was determined spectrophotometrically from the decrease in the optical density at 630 nm. The equilibrium constants for the association of the alcohols with met-Hb were determined by the formula for a tetrameric met-Hb molecule

$$K_a = c_c/c_a c_m,$$

where c_a and c_m are the concentrations of the free compounds (alcohol and met-Hb respectively), and c_c is the concentration of the alcohol–met-Hb complex. The hydroxyl radicals were determined from their reaction with dimethylsulphoxide by monitoring the resulting formaldehyde.[29] The mercapto groups of met-Hb in the cysteic acid residues were oxidised by the action of ultrasound of frequency 880 kHz and intensity 1.0–2.0 W cm^{-2} or by adding hydrogen peroxide. Acid hydrolysis of the protein was carried out for 24 h at 105 °C in 6 N HCl before determining the concentration of cystein and its oxidised forms on a T-339 amino acid analyser (Czechoslovakia).[30]

Results

The oxidation of ethanol by Fe^{3+} ions in the presence of hydrogen peroxide is shown in Fig. 1. The reaction is strongly inhibited by hydroxyl radical "traps": formic acid, thiourea, cysteine, cystine (Fig. 1). The dependence of the yield of acetaldehyde on the concentration of ferric ions and hydrogen peroxide is shown in Fig. 2. Large excesses of H_2O_2 over Fe^{3+} suppress the formation of acetaldehyde.

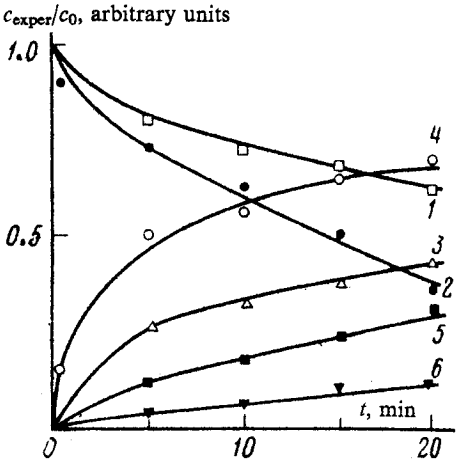

Figure 1. Consumption of ethanol (*1*, *2*) and production of acetaldehyde (*3*–*6*) in aqueous solutions containing $FeCl_3$ and $CuSO_4$ upon adding H_2O_2 and the hydroxyl radical traps cystine (*5*) and cysteine (*6*): *1*, *3*, *5*, *6*, $[FeCl_3] = 5 \times 10^{-3}$ M, 1.7×10^{-2} M ethanol, $[H_2O_2] = 0.3$ M; *2*, *4*, $[CuSO_4] = 1.37 \times 10^{-2}$ M, 1.13×10^{-2} M ethanol, $[H_2O_2] = 0.1$ M; *5*, 4×10^{-4} M cystine; *6*, 1×10^{-2} M cysteine; pH 4.0.

Copper ions also generate hydroxyl radicals in the presence of H_2O_2, and thus allow the ethanol oxidation to proceed like the Fe^{3+} ions. On the other hand Fe^{2+} ions in the presence of H_2O_2 were completely inactive towards the oxidation of aliphatic alcohols. The oxidation of ethanol to acetaldehyde was observed also in the presence of met-Hb and H_2O_2.

After the addition of H_2O_2 met-Hb gives a peroxidic form which slowly reverts to met-Hb as the excess of hydrogen peroxide is decomposed (Fig. 3). Adding alcohols to the peroxidic form of Hb in the absence of an excess of H_2O_2 does not produce significant amounts of aldehydes. This suggests that the oxidation of ethanol to acetaldehyde is controlled by the $OH^{\cdot}$ radicals generated in the oxidation of met-Hb by hydrogen peroxide to the peroxidic form. The formation of hydroxyl radicals was established from their reaction with dimethyl sulphoxide, leading to the formation of an equivalent amount of formaldehyde.[29]

Different forms of Hb differ in their ability to produce hydroxyl radicals when incubated with hydrogen peroxide. The incubation of oxy-Hb and Hb with H_2O_2 produces several times more $OH^{\cdot}$ radicals than the incubation of met-Hb. On the other hand met-Hb in the presence of hydrogen peroxide is the most effective catalyst for the oxidation of alcohols to aldehydes. In the case of the free Fe^{3+} and Cu^{2+} ions the yield of aldehydes does not increase with the length of the hydrocarbon chain.

In the presence of aliphatic alcohols the transformation of the peroxidic form of Hb into met-Hb with simultaneous oxidation of the alcohol to an aldehyde is accelerated (Fig. 4). Alcohols also protect the functional groups in the side-chain amino acids of the met-Hb residues from poisoning by high concentrations of H_2O_2. The effectiveness of the oxidation of alcohols by met-Hb in the presence of hydrogen peroxide decreases with increasing length of the hydrocarbon chain of the aliphatic alcohol, though the affinity of the alcohol towards the met-Hb increases in the same sequence.

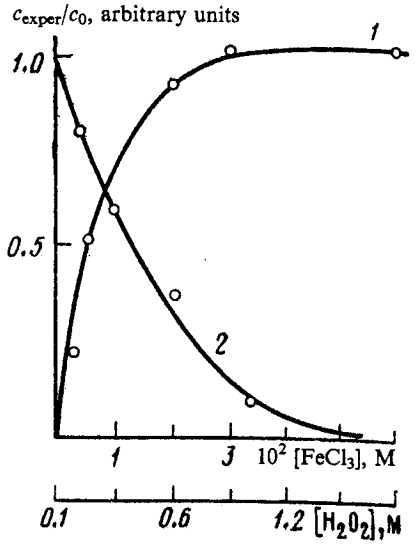

Figure 2. Dependence of the yield of acetaldehyde in aqueous solutions of ethanol (1.7×10^{-2} M) on the concentration of $FeCl_3$ (*1*) and of H_2O_2 (*2*); $[H_2O_2]$ = 0.3 M (*1*), $[FeCl_3] = 5 \times 10^{-3}$ M (*2*); pH 4.0

The increase in the interaction between the alcohols and the met-Hb can be easily detected spectrophotometrically (Fig. 5). The presence of isobestic points in the spectrum of met-Hb with different alcohol contents suggests an equilibrium in the solution between the two forms: met-Hb and its low-spin complex with alcohols (haemichrome). The long-wave shift of the absorption bands of the metal porphyrin in met-Hb indicates bonding of the alcohols in the hydrophobic pocket of the protein, and probably also an interaction of their hydroxyl groups with an iron ion. The equilibrium constants for the bonding of the alcohols with met-Hb, calculated for a tetrameric protein molecule, are 0.16, 0.3, and 0.6 M^{-1} for methanol, ethanol, and propanol respectively. The Hb ferro-forms (oxy-Hb and desoxy-Hb) accelerates the conversion of the peroxidic form into met-Hb:

$$Hb(Fe^{4+}{=}O)+HbO_2+2H^+ \rightarrow 2Hb(Fe^{3+})+O_2+H_2O. \quad (1)$$

The hydroxyl radicals formed by the interaction of H_2O_2 and met-Hb and the hydrogen peroxide oxidise the functional groups of the protein molecule. Under these conditions some of the met-Hb is converted into haemichrome, which does not absorb at 630 nm. Similar changes in the absorption spectrum of met-Hb are observed after the application of ultrasound to a solution of the compound.

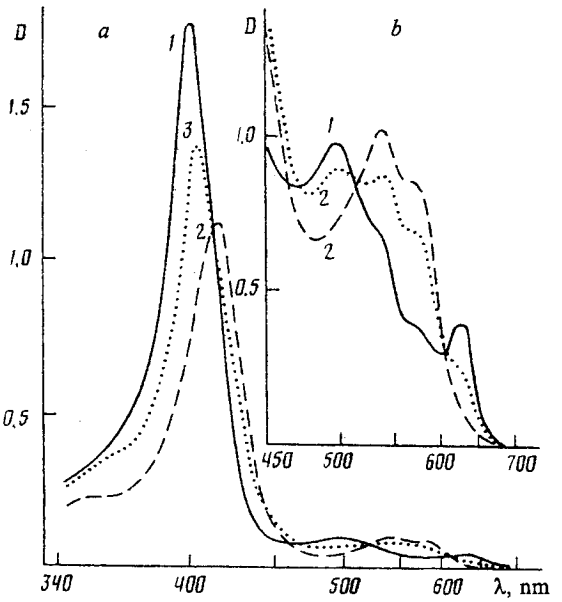

Figure 3. Absorption spectra of different forms of Hb for an absorbing layer 0.1 (*a*) and 1.0 cm thick (*b*): *1*, met-Hb; *2*, met-Hb + H_2O_2; *3*, ditto after 30 min; [met-Hb] = 1.05×10^{-4} M, $[H_2O_2]$ = 0.3 M; pH 7.0.

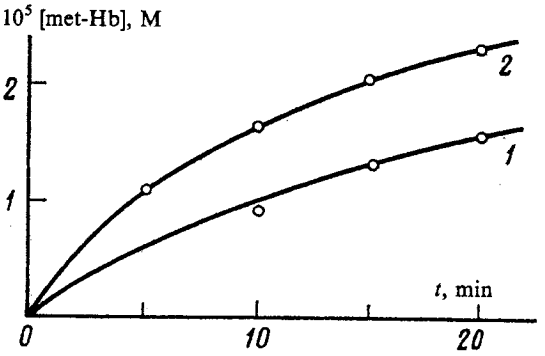

Figure 4. Kinetics of the reduction of met-Hb from the ferri-peroxidic form of Hb without alcohol (*1*) and in the presence of 1.25 M alcohol (*2*): [met-Hb] = 2.96×10^{-5} M, $[H_2O_2] = 3.53 \times 10^{-2}$ M; pH 6.8

The amino acid analysis results indicate oxidation of the mercapto groups of met-Hb to cysteic acid residues (Table 1). The change in the conformation of the haemichrome molecule induced by the oxidation of the mercapto groups (in contrast to the high-spin met-Hb) suppresses the reduction of haemichrome (by $NaBH_4$ or $Na_2S_2O_4$)

into the stable ferro-form. Of course, the irreversible haemichrome can be rapidly reduced to the ferro-form, but since the protein globule cannot create effectively an appropriate hydrophobic (haem) environment we observe a very fast reverse oxidation of the iron ion to the ferri-form by atmospheric oxygen.

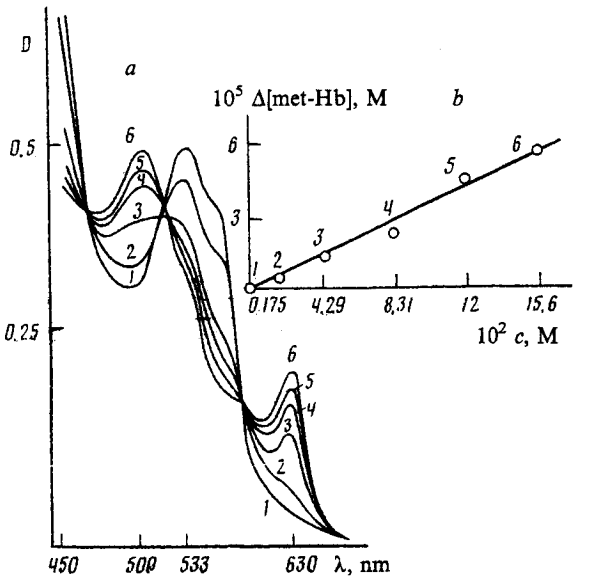

Figure 5. Formation of haemichrome from met-Hb as a function of the concentration of propanol in solution: *a*, spectral measurements; *b*, loss of met-Hb, determined by the absorption of 630 nm; pH 6.8; *c* is the concentration of propanol.

Discussion

In mixtures containing Fe^{3+} and H_2O_2 hydroxyl radicals are formed in the following chain of reactions:

$$Fe^{3+}+H_2O_2 \rightarrow Fe^{2+}+2H^++O_2^-, \quad (2)$$

$$Fe^{3+}+O_2^- \rightarrow Fe^{2+}+O_2, \quad (3)$$

$$Fe^{2+}+H_2O_2 \rightarrow Fe^{3+}+OH^{\cdot}+OH^-, \quad (4)$$

$$OH^{\cdot}+H_2O_2 \rightarrow H_2O+H^++O_2^-. \quad (5)$$

The resulting $OH^{\cdot}$ radical reacts with ethanol (rate constant 1.88×10^9 M^{-1} s^{-1} [7,8]) or with other aliphatic alcohols with formation of an alcohol radical which is oxidised by Fe^{3+} ions to an aldehyde:

$$CH_3CH_2OH+OH^{\cdot} \rightarrow CH_3\dot{C}HOH+H_2O, \quad (6)$$

$$Fe^{3+}+CH_3\dot{C}HOH \rightarrow Fe^{2+}+CH_3COH+H^+. \quad (7)$$

Therefore the concentration of the resulting aldehyde is proportional to that of the Fe^{3+} ions.

The inhibiting effect on the alcohol oxidation reaction of large excesses of hydrogen peroxide over Fe^{3+} is probably associated with the reaction of the $OH^{\cdot}$ radical with the H_2O_2 molecule [reaction (5)], which limits the formation of the alcohol radicals [reaction (6)].

As was stated above Fe^{2+} ions in the presence of H_2O_2 are much weaker oxidising agents for alcohols than Fe^{3+} ions. Hydrogen peroxide oxidised the bivalent iron ions, giving Fe^{3+} and $OH^{\cdot}$ radicals. The rate constant of this reaction is relatively low (76 M^{-1} s^{-1}).

Table 1. Amino acid analysis results for the oxidation of the cystine and cysteine residues in Hb to cysteic acid.

L	t_{us}, min	c_1	c_2	c_1	c_2
	without ethanol			with ethanol	
H_2O_2	–	70	30	90	10
$H_2O_2+CuSO_4$	–	64.6	35.4	97.8	2.2
–	10	93.5	5	98.5	1.5
–	20	86.5	13.5	99	1
–	30	84	15.9	99	1
HbO_2	5	94.7	4.5	98.5	1.5
HbO_2	15	92.7	4.7	98.4	1.6
HbO_2	45	91.2	86	98.4	1.6

Notes. L are the compounds added to the solution of cystine; c_1 and c_2 are the concentrations of cystine and cysteic acid respectively (%). Initial concentrations: cystine 2×10^{-4} M, ethanol 1.29 M, H_2O_2 1.1×10^{-2} M, $CuSO_4$ 2×10^{-3} M, HbO_2 5×10^{-4} M. The HbO_2 is converted into met-Hb by exposure to ultrasound (us) for 15 min.

The hydroxyl radicals formed by the decomposition of H_2O_2 can rapidly oxidise the bivalent iron ions:

$$Fe^{2+}+OH^{\cdot} \rightarrow Fe^{3+} \rightarrow OH^-. \quad (8)$$

The rate constant of this reaction is high (3×10^8 M^{-1} s^{-1}), and comparable to the rate constant of the reaction between the $OH^{\cdot}$ radicals and ethanol (1.9×10^9 M^{-1} s^{-1}). This results in the interception of the $OH^{\cdot}$ radicals by the Fe^{2+} ions, especially at high concentrations of Fe^{2+} ions.

In the case of oxy-Hb other protein groups (in addition to the iron cation) can act as "traps" for $OH^{\cdot}$ radicals, and this strongly inhibits the oxidation of alcohols to aldehydes. We have previously shown that met-Hb accelerates the oxidation of free radicals from aliphatic alcohols to the corresponding aldehydes

$$\text{met-Hb}+R-\dot{C}H-OH \rightarrow Hb(II)+R-COH+H^+ \quad (9)$$

most effectively when the affinity of the alcohol for the met-Hb is highest.[30] In our case, conversely, the rate of oxidation of alcohols by met-Hb and H_2O_2 to aldehydes decreased with increasing length of the aliphatic chain. We can therefore assume that in the case of met-Hb other reactions induced by $OH^{\cdot}$ radicals (in addition to the oxidation of alcohols) are being observed. As we know, the haem cation and the His-92 and Cys-93 residues in Hb form a single conjugated system. Therefore the oxidation of met-Hb can involve

both the SH group and the iron(III) cation:

$$\text{met-HbS}^- + H_2O_2 \rightarrow \text{met-HbS}^{\cdot} + OH^{\cdot} + OH^- \rightarrow \text{Hb(IV)S}^- + OH^{\cdot} + OH^- \quad (10)$$

$$\text{Hb(III)S}^- + H_2O_2 \rightarrow \text{Hb(IV)S}^- + OH^{\cdot} + OH^-. \quad (11)$$

The generation of $OH^{\cdot}$ radicals assists the oxidation of the alcohols. These cases have been discussed above.

Met-haemoglobin and met-myoglobin are oxidised by hydrogen peroxide to the peroxidic Hb(IV) form also by a two-electron mechanism, which produces an oxidised group in the protein:[26,31,32]

$$\text{met-Hb} + H_2O_2 \rightarrow \text{Hb}^+\text{(IV)=O} + H_2O, \quad (12)$$

where Hb^+ is the protein with a charge on the protein itself or on a porphyrin ring. In this case two centres on the protein are being oxidised simultaneously: Fe^{3+} and the SH group. The presence of the mercapto radical in the protein, resulting from the reaction of met-Hb with hydrogen peroxide, was detected in an ESR spectrometer by the stopped flow method.[33]

The results show that the peroxidic form of myoglobin is similar in structure to component (I) of peroxidase, but not identical to it.[31] The peroxidic form of Hb, which contains a mercapto radical, can probably also be obtained by a subsequent two-electron oxidation. Met-Hb can react with the $OH^{\cdot}$ radical to give met-Hb containing a mercapto group on the protein globule:

$$\text{Hb(III)SH} + OH^{\cdot} \rightarrow \text{Hb(III)S}^{\cdot} + H_2O. \quad (13)$$

Because of the relatively high effectiveness of its reaction with the $OH^{\cdot}$ radical the mercapto radical undergoes the reaction

$$\text{Hb(III)SH} + OH^{\cdot} \rightarrow \text{Hb(III)SOH}, \quad (14)$$

resulting in the oxidation of the mercapto group to a cysteic acid residue (Table 1). The peroxidic form of Hb, containing a group on the protein, can react with alcohols with formation of aldehydes:

$$\text{Hb}^+\text{(IV)=O} + \text{R}-\text{CH}_2\text{OH} \rightarrow \text{Hb(III)} + H_2O + \text{RCOH}. \quad (15)$$

The peroxidic form of Hb is reduced to met-Hb by its ferro-forms oxy-Hb and desoxy-Hb, but not carboxy-Hb, as well as hydrogen peroxide (Fig. 3).

By combining these results we can represent the reaction between met-Hb and H_2O_2 as follows:

$$\text{met-Hb} + H_2O_2 \rightarrow \text{Hb}^+\text{(IV)=O} + H_2O, \quad (16)$$

$$\text{Hb}^+\text{(IV)=O} \xrightarrow{e} \text{Hb(IV)=O} \xrightleftharpoons{H_2O_2} \text{Hb(III)} + H_2O_2 + O_2^-, \quad (17)$$

$$H_2O_2 + O_2^- \rightarrow OH^- + OH^{\cdot} + O_2. \quad (18)$$

As we know, the affinity of aliphatic alcohols for met-Hb increases with the length of the hydrogen chain,[5] and the bonding of the alcohols takes place either through the direct interaction of the hydroxyl with the Fe^{3+} of haem or in the vicinity of the haem, in the hydrophobic cavity of the protein. This is convincingly shown by the absorption spectra in Fig. 5. Alcohol bonded near an active centre will probably contribute more effectively to the reactions initiated by hydrogen peroxide than free alcohol (not bonded to the protein). However, kinetic measurements by the stopped flow method show that the pseudo-first order constants for the conversion of met-Hb into the peroxidic form under the influence of H_2O_2 decrease in the presence of aliphatic alcohols with hydrocarbon chains of increasing length (Fig. 6). This effect is due [see reactions (16)–(18)] to the higher rate of the transition of the peroxidic form of Hb into met-Hb, and therefore also of the oxidation of the alcohols. A good correlation is found between the logarithms of the rate constants and the Taft inductive constants of the alkyl substituents ($\rho^* = -0.63$, $r = 0.98$). Therefore we may conclude that although oxidation reactions of alcohols to aldehydes under the influence of hydroxyl radicals [reactions (6) and (7)] do take place in mixtures of met-Hb and hydrogen peroxide their contribution is marginal. In this case we would more probably observe an increase in the yield of aldehydes with the affinity of the alcohols (or, more exactly, of the $\text{R}-\dot{\text{C}}\text{H}-\text{OH}$ radicals) for the met-Hb [reaction (9)].

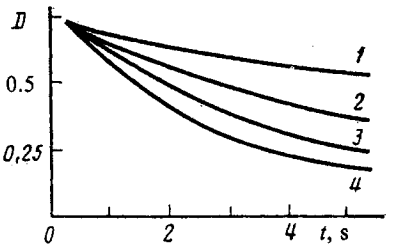

Figure 6. Kinetics of the loss of met-Hb (λ_{reg} = 406 nm) in the presence of H_2O_2 and aliphatic alcohols (*1*–*3*) and of H_2O_2 only (*4*); *1*, methanol; *2*, ethanol; *3*, propanol; *4*, 0.02 M Na phosphate buffer at pH 7.4. The concentration of alcohols is 5%, that of met-Hb and H_2O_2 1.20×10^{-5} M. The rate constants of the reactions of met-Hb with H_2O_2 and the alcohols (*1*–*3*) and with H_2O_2 only (*4*) are 0.065, 0.108, 0.158, and 0.256 s^{-1} respectively.

The purification (~100-fold) of the met-Hb solutions from catalase does not affect the rate of oxidation of the alcohols substantially. Therefore we may suggest that the contribution of catalase to the oxidation of alcohols under our experimental conditions is small and amounts (by our estimates) to a few per cent. This conclusion is confirmed by experiments with oxy-Hb: before the oxidation of oxy-Hb to met-Hb with hydrogen peroxide the formation of acetaldehyde is almost undetectable.

Our results lead to the conclusion that met-Hb decomposes hydrogen peroxide by the transfer of one electron from the iron cation or from the mercapto group of the protein molecule with formation of hydroxyl radicals. We also observe the formation of the peroxidic form of Hb, containing an oxidised functional group on the protein molecule. The peroxidic form of Hb is unstable and is rapidly converted into met-Hb under the influence of an excess of H_2O_2. The ferro-forms of Hb (oxy- and desoxy-Hb, but not carboxy-Hb) accelerate the transition of the peroxidic form into met-Hb. Alcohols also accelerate this conversion, and are thereby oxidised to aldehydes. The mercapto group of met-Hb is slowly oxidised by H_2O_2 to a cysteic acid residue.

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Received 27th April 1989

Translated from *Zhurnal Fizicheskoi Khimii* **64** 1783–1788 (1990)

U.D.C. 541.128.13.665.652.72

Metal–carbon fibre catalysts: structure of the carrier and dispersion of the active phase

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ABSTRACT. We have studied the structure of the carriers and the dispersion of metal–carbon fibre catalysts using cobalt, nickel, iron, platinum, and palladium as the active phase. The carbon fibres are shown to differ in specific surface and porosity. The dispersion of the active phase depends on the metal content of the catalyst and on the nature of the metal. The adsorption and desorption behaviour of carbon monoxide on the surface of the metal–carbon fibre system has been determined.

Carbon fibres (CF) are potential carriers for a series of catalysts. The use of carriers of this type promises improved technologies of catalytic processes, and it offers wider ranges of working temperatures and the possibility of varying the shape of the catalysts. As we have shown[1] Pt/CF catalysts are active in the synthesis of methane from CO and H_2, the Co/CF system[2] is active towards the preparation of C_1-C_4 hydrocarbons from CO and H_2. Very little is known about the physicochemical properties of the metal–carbon fibre system. The aim of the present work was to study the structure of the carrier surface (carbon fibre), the dispersion of the metallic phase of a series of catalysts, and the specific features of the adsorption of carbon monoxide on the surface of those systems.

Carbon fibres of various grades, prepared from cellulose hydrate, were used as carriers for the catalysts. They consisted of separate fibrils with non-uniform surfaces. In Fig. 1 we show micrographs of a CF-5 sample taken with a BS-101 instrument (Carl Zeiss, Jenna, East Germany). At the higher resolutions we note carbon islets, stripes, and projections. The break of the fibrils resembles a "cleavage". On the whole the micrograph suggests reproducible surface properties of the individual fibrils and uniformity of the CF as an assembly of fibrils.

An X-ray phase analysis of the carriers (carbon fibres) confirmed the absence of both metallic and non-metallic crystalline impurities. According to element analysis the CF samples consist of 99.99% carbon, mostly in an amorphous state.

The carbon fibres differ in their specific surface, which depends on the method of preparation and on the subsequent treatment of the CF. In Table 1 we use the following symbols: S is the specific surface, d is the average pore diameter, V is the total pore volume, c_{me} and c_{ma} are the numbers of mesopores and of macropores. Non-activated fibres, or fibres treated with a mixture of O_2 and O_3, have acidic properties (CF-1, CF-2), and the carbon fibres treated with superheated steam have basic properties (CF-3).[3]

The pore structure of the CF was studied with a mercury porosimeter, by the adsorption of benzene and of water, and by the