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Oxygen diffusion in meat tissues

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Abstract—Oxygen penetration in muscle tissue was mathematically modeled by means of a non-stationary statement of the problem. The parameters concerned: solubility, diffusion coefficient and oxygen consumption in the tissue, were evaluated at different temperatures. The results were compared with experimental data of penetration width and spectrophotometric measurements of oxymyoglobin relative concentration. The effect of oxygen partial pressure in the formation of metmyoglobin in muscle tissue was analyzed in terms of an equation obtained for solution systems.

INTRODUCTION

THE COLOR of meat is determined by the relative proportions of myoglobin derivatives. In the presence of oxygen the three pigments oxymyoglobin (MbO_2), metmyoglobin (MetMb) and myoglobin (Mb) are constantly being interconverted, constituting the dynamic color cycle in fresh meat [1].

The take up of oxygen by myoglobin converts the purple reduced pigment to the bright red oxygenated form, oxymyoglobin. This process produces the 'bloom' of fresh meats. The red complex, once formed, is stabilized by the formation of a highly resonant structure, and as long as the oxygen remains complexed to the heme, the pigment will undergo no further color changes. However, the oxygen which is continually associated and dissociated from the heme complex accelerates this process by certain conditions occurring at low oxygen pressures. When this occurs the reduced pigment is subjected to oxidation by oxygen or other oxidants. Brooks [2, 3] working with ox blood (hemoglobin) and George and Stratmann [4, 5] using pure horse heart myoglobin found that the rate of oxidation was very dependent on the partial pressure of oxygen in the system; the rate was maximal at low partial pressures. Similar results were reported by Ledward [6] working with beef muscle.

In meat, the situation is more complex. When a piece of meat is exposed to air (either directly or inside a plastic film package) oxygenation and oxidation reactions take place depending on oxygen penetration. At the surface, where oxygen tension is high, bright red oxymyoglobin is formed and its red color extends inwards until the point where the oxygen level falls almost to zero. At this point, brown metmyoglobin is formed and deeper inside the meat, myoglobin remains purple. As time passes the outer layer slowly oxidizes and the metmyoglobin layer extends towards the surface. In meat the oxidation is quasi-reversible because reducing endogeneous substances of the tissue constant re-reduce the metmyoglobin to the purple form [7].

Reflectance spectrophotometry has been used to measure the relative amounts of myoglobin derivatives at the surface of cut meat [8, 9]. As this technique is non-destructive it can be used to follow color changes occurring in intact beef samples being eminently suitable to study the conditions which cause these changes.

The penetration of oxygen in the tissue is governed by several factors: temperature, oxygen concentration at the surface, bacterial growth, oxygen uptake by the meat muscle and oxygen diffusion coefficient [2, 10–12]. This phenomenon was traditionally interpreted as a stationary problem [2].

One of the objectives of the present work is to model mathematically the oxygen penetration in the tissue by a non-stationary equation. Another proposed objective is to quantify intervening parameters in order to interpret experimental results corresponding to oxygenation rate and oxygen concentration profiles inside meats.

Referring to reactions of myoglobin autoxidation the purpose was to determine the effect of oxygen partial pressure on the kinetic formation of metmyoglobin because the existing models were obtained in solution systems and its applicability to muscles has not yet been verified.

ANALYSIS

Stationary state

The one-dimensional diffusion of oxygen in meat with simultaneous consumption (R) of oxygen by the tissue (zero order) in the stationary state is governed by

$$D\frac{d^2C}{dx^2} = R \tag{1}$$

where D is the effective diffusion coefficient of oxygen in the tissue with the following boundary conditions:

$$x = 0, \quad C = C_0 \tag{2}$$

NOMENCLATURE

C	concentration
C'	oxygen concentration corresponding to the maximum myoglobin oxidation rate
C_0	interphase concentration
C_i	concentration of component i
D	effective diffusion coefficient
$F(R_\infty)$	Kubelka–Munk function
$F_0(T_\infty)$	Kubelka–Munk function of the free pigment meat
ΔF	difference of Kubelka–Munk functions
H	Henry's constant
K_i	absorption coefficient of component i
K_e	equilibrium constant
p	partial pressure
p'	oxygen partial pressure corresponding to the maximum myoglobin oxidation rate
p_0	interphase partial pressure
R_0	asymptotic oxygen consumption
R_∞	reflectance of an infinite layer
S	scattering coefficient
S_i	scattering coefficient of component i
T	absolute temperature
t	time
x	Cartesian coordinate
x^i	relative pigment concentration
x'	molar fraction in the liquid phase.

Greek symbols

α	coefficient of the exponential oxygen consumption
α_1, α_2	coefficients in equation (21)
μ	dynamic viscosity
σ	oxygen penetration distance
σ'	width of the oxymyoglobin layer.

Superscript

m	matrix.

Subscripts

L	refers to liquid phase
λ	corresponding to a determined wavelength.

$$x = \delta, \quad dC/dx = 0 \tag{3}$$

where C_0 is the dissolved oxygen concentration at the interface ($x = 0$) and δ the gas penetration distance. Equation (3) indicates that starting from δ there is no flux of gas towards the interior of the meat.

The solution of equations (1)–(3) leads to the following concentration profile:

$$C - C_0 = \frac{R}{2D}x^2 - \frac{R\delta x}{D}. \tag{4}$$

Accepting that in $x = \delta$ is $C/C_0 \ll 1$ the known expression of Warburg that permits the evaluation of oxygen penetration in muscle in the stationary state [2] was obtained

$$\delta = \sqrt{\left(\frac{2DC_0}{R}\right)} \tag{5}$$

Equation (5) leads to a constant value of δ; however, experimental observations show that this value of δ is not achieved instantaneously and that oxygen penetration increases with time [2, 11].

These findings do not agree with the pseudo-stationary formulation of the problem and conduced to a non-stationary statement of the system.

Non-stationary state

Assuming that oxygen consumption by the muscle decreases during post-mortem time [13] the diffusion of oxygen in the non-stationary state was represented by

$$\frac{\partial C}{\partial t} = D\frac{\partial^2 C}{\partial x^2} - R_0 e^{-\alpha t} \tag{6}$$

with the following boundary conditions:

$$C = C_0 \quad \text{in} \quad x = 0 \tag{7}$$

$$\frac{\partial C}{\partial x} = 0 \quad \text{in} \quad x = \delta \tag{8}$$

where $R(t) = R_0 e^{-\alpha t}$ is the oxygen consumption by the tissue, decreasing exponentially with time.

This sink is confined to the zone included between $x = 0$ and δ, because only the oxygen in contact with the tissue is consumed.

The solution of equation (6) with the corresponding initial and boundary conditions was obtained applying the Laplace transform that gives

$$C = C_0 - \frac{R_0}{\alpha}\left(\frac{\cos(\sqrt{(\alpha/D)}(\delta - x))}{\cos(\sqrt{(\alpha/D)}\delta)} - 1\right)e^{-\alpha t}$$

$$- \sum_{n=0}^{\infty} \frac{(-1)^n}{(2n+1)} e^{-D(2n+1)^2\pi^2 t/4\delta^2} \frac{\cos(2n+1)\pi(\delta - x)}{2\delta}$$

$$\times \left[\frac{16\delta^2 R_0}{\pi(4\delta^2\alpha - (2n+1)^2\pi^2 D)} - \frac{4C_0}{\pi}\right]. \tag{9}$$

At large contact times and assuming a very low concentration of oxygen in $x = \delta$ equation (9) is reduced to

$$\delta = \sqrt{\left(\frac{D}{\alpha}\right)} \arccos\left(\frac{1}{\frac{C_0\alpha}{R_0 e^{-\alpha t}} + 1}\right) \quad (10)$$

which developed in series leads to

$$\delta = \sqrt{\left(\frac{2DC_0}{R_0 e^{-\alpha t}}\right)}. \quad (11)$$

MATERIALS AND METHODS

In order to check the mathematical formulation of the problem oxygen penetration and oxymyoglobin concentration measurements were performed on samples of tissues obtained from gluteus medium bovine muscles removed from steers within 24 h post-mortem at 4°C.

Oxygen penetration

The penetration of oxygen into meat was determined by measuring the depth of the oxymyoglobin layer [2, 14]. A slice of tissue taken from the anerobic depth of the muscle and measuring about 5 × 4 cm was placed between two parallel glass plates. Diffusion of oxygen was permitted by only one of the borders, the others were sealed.

An S. R. Zeiss stereomicroscope equipped with an MC 63 camera was used to measure the extension of the bright layer.

Oxymyoglobin relative concentration

Relative pigment concentration was measured in a Varian Super Scan 3 spectrophotometer equipped with an integrating sphere reflectance attachment. Reflectance spectra were recorded between 400 and 800 nm using a barium sulfate coating as the reference standard. Meat samples of 3.2 × 2.3 cm in section and 2 cm thick were placed in special sample ports with muscle fibers parallel to the surfaces to be analyzed and were covered with a thin optical glass removing trapped air; the same glass was used to cover the reference standard. Absorbance readings of the glass slice were considered in further calculations. The thickness of the samples was selected to satisfy requirements for R_∞ measurements (R_∞ is the reflectance of a layer so thick that an increase in thickness does not change its value). To establish standard spectra, meat pigments were converted to the three forms of myoglobin. Reduced Mb samples were obtained from fresh cuts in deep zones of muscle and/or by spreading a thin layer of sodium hydrosulfite over the meat surface. For conversion to MetMb, potassium ferricyanide (1%) in an aqueous solution was sprayed over meat slices to a level of 0.1 ml per 100 cm^2 of beef surface three times at intervals of 1 h, at 4°C. Slices with myoglobin completely converted to MbO_2 were obtained by blooming in an oxygen atmosphere at 4°C during 2 h.

Diffuse reflectance refers to reflected radiant energy that has been partially absorbed and partially scattered by a surface with no defined angle of reflection. The most generally accepted theory concerning diffuse reflectance has been developed by Kubelka and Munk [15, 16] and it is valid for weakly absorbing substances in diffuse reflecting systems only. Most other theories are special cases or adaptations of the Kubelka–Munk theory. For the purpose of the Kubelka and Munk analysis, meat can be considered to be a light scattering matrix of cellular material, myofibrillar proteins, connective tissue and light absorbing pigments [17]. The intensity of reflected light and therefore its color and appearance is governed by the interrelationship of the light scattering components in the system, and the concentration and spectral absorption properties of the pigments. The red pigments are absorbers of light and the uncolored structure and myofibrillar proteins both scatter and absorb.

The Kubelka–Munk function, $F(R_\infty)$ in the case of meat tissue, can be expressed as

$$F(R_\infty) = \frac{(1-R_\infty)^2}{2R_\infty} = \frac{\sum K_{i_\lambda} C_i}{\sum S_{i_\lambda} C_i} \quad (12)$$

where K_i is the absorption coefficients of pigments (MbO_2, MetMb, Mb) and matrix; S_i the scattering coefficients; C_i the concentration of the components in meat tissue. Considering that scattering is attributed to the matrix of fibers ($S_\lambda^m = S_\lambda$), the following was obtained:

$$F(R_\infty) - F_0(R_\infty)\Big|_\lambda = \frac{K_\lambda^{oxy} C^{oxy} + K_\lambda^{myo} C^{myo} + K_\lambda^{met} C^{met}}{S_\lambda C^m} \quad (13)$$

where $F_0(R_\infty) = K^m/S$.

Reflectance spectra of beef shows a minimum at approximately 730 nm [18] where it is not dependent on pigment concentration and can be considered as the K/S value by free pigment meat.

Analysis of reflectance spectra of meat is concerned with changes in reflectance at specific wavelengths: 525 nm is isobestic for all three derivatives, 572 nm is isobestic for MbO_2 and reduced Mb, 473 is isobestic for MbO_2 and MetMb.

The applied equations for evaluating relative pigment concentration (x^i) were [19]

$$x^{met} = 1.58 - 1.05\frac{\Delta F_{572}}{\Delta F_{525}} \quad (14)$$

$$x^{myo} = 2.24 - 2.38\frac{\Delta F_{473}}{\Delta F_{525}} \quad (15)$$

$$x^{oxy} = 1.685x^{met} + 1.85\frac{\Delta F_{580}}{\Delta F_{525}} - 2.65 \quad (16)$$

where

$$\Delta F_\lambda = F(R_\infty)_\lambda - F(R_\infty)_{730}.$$

Table 1. Oxygen solubility in meat tissue (water content on wet basis = 0.74)

T (°C)	Henry's constant $10^{-4} \times H$ (atm)	Oxygen solubility $C_0 \times 10^6$ (m^3O_2 (kg tissue)$^{-1}$)
0	2.55	7.57
5	2.91	6.63
10	3.27	5.90
15	3.64	5.31

Table 2. Oxygen diffusion coefficient in muscle tissue

T (°C)	μ water $\times 10^3$ (kg m^{-1} s^{-1})	$D_{O_2\text{-muscle}} \times 10^9$ (m^2 s^{-1})
37	0.705	1.70†
10	1.307	0.839
5	1.520	0.709
0	1.787	0.590

† Lightfoot [20]

RESULTS AND DISCUSSION

Oxygen penetration was computed by fitting the following parameters: solubility diffusion coefficient and consumption of oxygen in the tissue in equations (9) and (11).

Estimation of parameters

Solubility of oxygen in the tissue. The solubility of oxygen in water can be represented by Henry's law $p = Hx'$ where p is the partial pressure of solute in the gas phase (atm) and x' is the molar fraction of solute in the liquid phase (moles of solute/moles of solution).

The variation of Henry's constant with temperature for the oxygen–water system is observed in Table 1. Considering that in air $pO_2 = 0.21$ atm and the average water content on the wet basis of muscle tissue is 0.74 g water/g tissue, values of solubility (C_0) of oxygen in meat tissue were calculated (Table 1).

Oxygen diffusion coefficient

Values of the oxygen diffusion coefficient in muscle tissue (D_L) at different temperatures (Table 2) were calculated using the Wilke–Chang equation

$$\frac{D_L \mu}{T} = \text{constant} \qquad (17)$$

and considering that $D = 1.7 \times 10^{-9}$ m^2 s^{-1} at 37°C [20]. In equation (17), μ is the liquid viscosity and T the absolute temperature.

Oxygen uptake in post-rigor bovine muscle

Oxygen uptake is the result from tissue respiration heme pigment oxygenation, dissolution into tissue fluids of low oxygen tension, lipid oxidation and/or bacterial demands [21].

Post-mortem respiratory oxygen consumption is produced because the muscle contains active enzyme systems and residual stores of respiratory substrates and intermediates. Active respiratory enzymes in post-mortem muscle have been demonstrated by several investigators [22–24] and substantial quantities of respiratory intermediates and substrates have been observed after various post-mortem storage times [24–26]. Significant respiratory oxygen consumption by post-rigor bovine muscle has been observed previously by Urbin and Wilson [27] and Bendall and Taylor [13]. De Vore and Solberg [21] demonstrated that post-rigor oxygen uptake as a result of lipid oxidation is negligible.

Table 3. Consumption of oxygen by Pseudomonas fluorescens

T (°C)	m^3O_2 s^{-1} cell $\times 10^{20}$
30	2.694
20	0.675
10	0.166
5	0.083
0	0.042

Bendall and Taylor [13] reported that the oxygen consumption rate in post-rigor muscle declines exponentially during storage time. According to these results oxygen consumption in semitendinosus muscle was modeled by the following equation:

$$R = R_0 e^{-\alpha t} \qquad (18)$$

where $R_0 = 1.1 \times 10^{-9}$ m^3O_2 (kg tissue s)$^{-1}$ and $\alpha = 0.964 \times 10^{-6}$ s^{-1}.

Oxygen uptake by contaminating bacterial population was also found insignificant [21]. According to Greig and Hoogerheide [28] one cell of Pseudomonas fluorescens requires 2.69×10^{-20} m^3 s^{-1} of oxygen at 30°C.

Table 3 shows the effect of temperature on oxygen bacterial consumption. At 5°C a population of 10^7 CFU cm^{-2} (CFU = colony forming units) consumes only 8.33×10^{-11} m^3 O_2 s^{-1} m^{-2}.

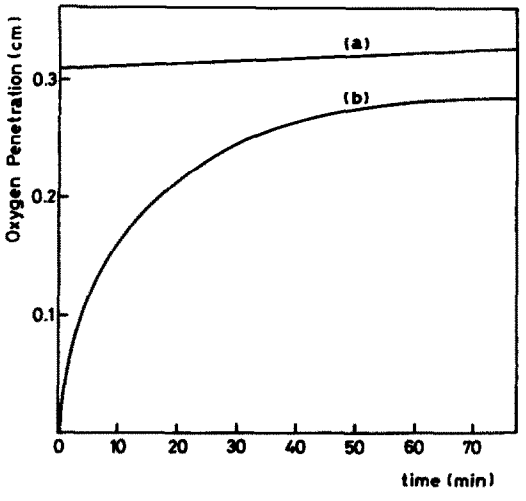

FIG. 1. Oxygen penetration curves at 0°C predicted by (a) pseudo-stationary model (equation (11)), (b) non-stationary model (equation (9)).

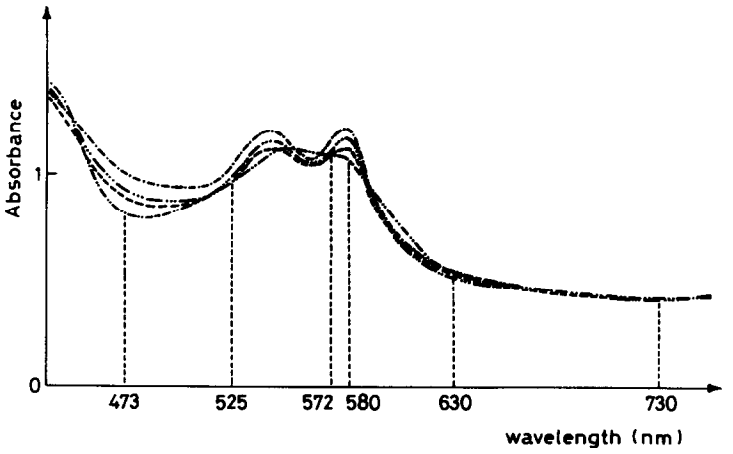

FIG. 2. Diffuse reflectance spectra during oxygenation of beef tissue: —··—, $t = 0$; ———, $t = 5$ min; —···—, $t = 12$ min; — · — ·, $t = 19$ min.

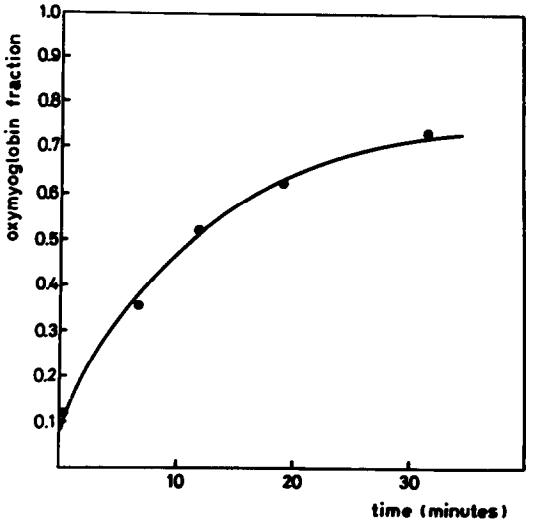

FIG. 3. Increase of the oxymyoglobin relative concentration during oxygenation in air. The sample was previously packaged in EVA/SARAN/EVA for 4 days.

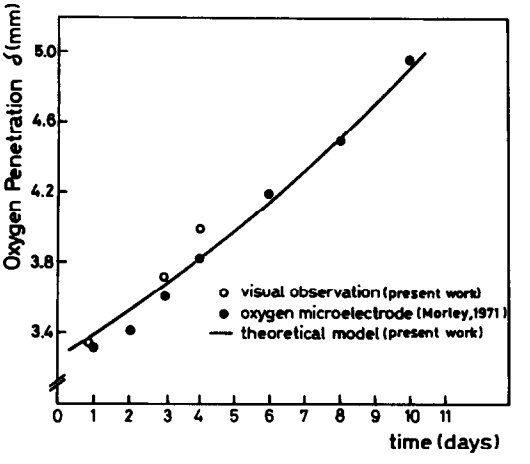

FIG. 4. Comparison between oxygen penetration values predicted by equation (9) valid for long contact times and experimental observations.

Penetration oxygen curves vs time (Fig. 1) predicted by non-stationary and pseudo-stationary models (equations (9) and (11), respectively) at contact times shorter than 1 h were obtained by feeding in the described parameters. The increase of δ predicted by the non-stationary equation is progressive. The fact is experimentally supported by oxymyoglobin percentage curves as a function of time obtained in muscle having all the pigment in the reduced form (myoglobin).

Figure 2 presents diffuse reflectance spectra for muscle tissue as a function of time during myoglobin oxygenation. From these curves oxymyoglobin percentage changes on the meat surface at short contact times, calculated using equation (14), were plotted in Fig. 3.

As curve (b) of Fig. 1 and the curve of Fig. 3 are similar it can be observed that the oxygenation is a phenomenon controlled by the oxygen diffusion in the muscle.

By comparing the solutions of equations (9) and (11) it was observed that at times longer than 6 h both solutions are coincident. That is starting from this time the pseudo-stationary formulation (equation (11)) is valid to interpret the oxygen penetration in the post-mortem muscle. Using this equation δ values were calculated for an exposure period of 1–10 days.

Results were plotted together with the measurements made by Morley [11] employing an oxygen microelectrode with a detection level of 0.5% O_2 (3.8 mm Hg) and with the visual oxymyoglobin layer width measurements (Fig. 4) denoting satisfactory coincidence.

In the case of visual observations it was taken into account that oxymyoglobin layer measurements do not yield actual δ values. This is due to the fact that inside the muscle there is a concentration profile of O_2 and partial pressure decreases towards the interior of the muscle.

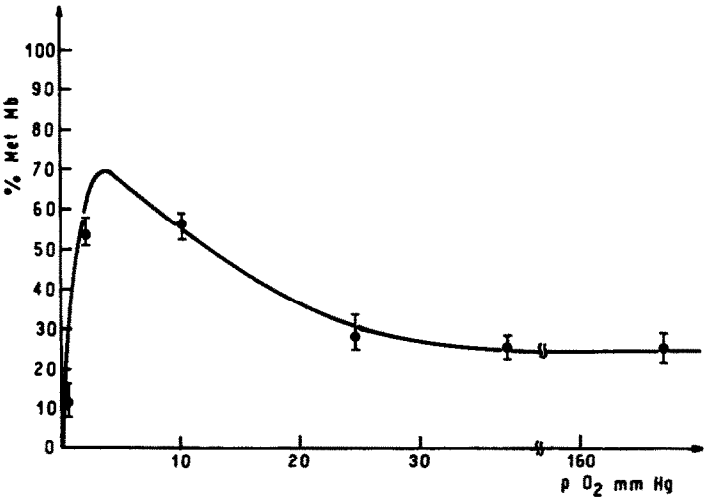

FIG. 5. Effect of oxygen partial pressure on myoglobin autoxidation rate. Experimental values in semitendinosus muscle at 0°C (Ledward [6]): ——, equation (21).

At low pO_2 the autoxidation is accelerated with formation of metmyoglobin [6]. This fact implies that when the stationary state is reached the width of the oxymyoglobin layer (δ') does not coincide exactly with the total penetration of oxygen (δ) in the muscle.

The relationship between δ' and δ can be determined by substituting equation (4) in equation (3) which leads to

$$\frac{\delta'}{\delta} = 1 - \left(\frac{C'}{C_0}\right)^{1/2} = 1 - \left(\frac{p'}{p_0}\right)^{1/2} \qquad (19)$$

where C' is the concentration of oxygen for which the maximum rate of autoxidation occurs and p' is the corresponding partial pressure.

Considering that at the interface the partial pressure of oxygen dissolved in the water content of the tissue (74%) p_0 is: $760 \times 0.21 \times 0.74 = 118.1$ mm Hg, and substituting in equations (19) for p' the values reported by Ledward [6]: 6 mm Hg at 0°C and 7.5 mm Hg at 7°C (semitendinosus muscle), the following ratios were obtained: $\delta'/\delta = 0.77$ at 0°C and 0.74 at 7°C. As can be observed the width of the oxymyoglobin outer strip (δ') is smaller than the total distance of oxygen penetration (δ); this fact was considered affecting visual observation by these correction factors.

Temperature effect on oxygen penetration

Several experimental reports evidenced that oxygen penetration increased with decreasing temperature [2, 11].

Comparing the stationary δ values (equation (9)) at 0 and 10°C, the following relationship was obtained:

$$\frac{\delta_0}{\delta_{10}} = \left(\frac{D_0}{D_{10}} \cdot \frac{C_{O,0}}{C_{O,10}} \cdot \frac{R_{10}}{R_0}\right)^{1/2}. \qquad (20)$$

Substituting the values of oxygen consumption, diffusion coefficient, and solubility: $R_0/R_{10} = 0.304$; $D_0/D_{10} = 0.703$; $C_{O,0}/C_{O,10} = 1.28$. Equation (20) leads to: $\delta_{0°C}/\delta_{10°C} = 1.72$ that verifies experimental findings.

Effect of oxygen partial pressure on myoglobin autoxidation

To interpret the effect of oxygen partial pressure on myoglobin autoxidation in meat tissue, the oxygenation of myoglobin as a previous step must be analyzed. This reaction is considered reversible and can be expressed as

$$Mb + O_2 \leftrightharpoons MbO_2$$

with equilibrium constant

$$K_e = (Mb)(pO_2)/(MbO_2).$$

The equilibrium is displaced to MbO_2 at oxygen pressures higher than 30 mm Hg resulting in K_e independent of pO_2; but at low pO_2 the relative equilibrium concentrations of MbO_2 and Mb depend on oxygen concentration in meat tissue.

The oxidation of myoglobin to metmyoglobin was considered by several authors [4, 19] as first order in unoxidized myoglobin (the sum of MbO_2 and Mb).

George and Stratmann [5], working with solutions of myoglobin, reported that the first-order constant showed a well-defined maximum value at 1–4 mm Hg partial pressure of oxygen and then decreased to a constant value above 30 mm. This rate constant was expressed by a complex function over the entire range of pO_2 according to

$$k = \alpha_1 \frac{K_e pO_2}{(K_e + pO_2)^2} + \alpha_2 \frac{(pO_2)^2}{(K_e + pO_2)^2} \qquad (21)$$

where the concentrations of MbO_2 and Mb were expressed in terms of the equilibrium constant of the oxygenation step.

Experimental data of Ledward [6] obtained work-

ing with beef muscle at 0°C, were fitted to equation (21) in order to verify the applicability of this expression to myosystems.

Values of α_1, α_2, and K_e were calculated using the Marquardt algorithm in a computer program for estimation of non-linear parameters.

The values obtained were $\alpha_1 = 2.6$; $\alpha_2 = 0.2$ and $K_e = 3$ mm Hg (K_e gives the maximum pO_2 for the autoxidation rate). Comparison between equation (21) and the experimental values of metmyoglobin equilibrium concentration at the surface of sterile muscle, reported by Ledward [6], are observed in Fig. 5 showing satisfactory coincidence.

CONCLUSIONS

The oxygen penetration in meat tissue at short contact times was represented by a non-stationary model of diffusion with simultaneous consumption of oxygen by the tissue (decreasing exponentially with time). At large contact times (>6 h) the pseudo-stationary and non-stationary models coincide with experimental results showing that the increase of the penetration distance (δ) is due to a decrease of the oxygen uptake by the tissue. Results evidence that oxygenation of myoglobin is controlled by oxygen diffusion in the tissue and that low temperatures increase δ values.

Effect of pO_2 on metmyoglobin rate constant was modeled by an equation that considered the influence of oxygen concentration in the reversible reaction of myoglobin oxygenation.

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DIFFUSION D'OXYGENE DANS LA VIANDE

Résumé—La pénétration d'oxygène dans les tissus musculaires est mathématiquement représentée au moyen d'un modèle instationnaire du problème. On évalue à différentes températures les paramètres concernés: solubilité, coefficient de diffusion et consommation d'oxygène dans le tissu. Les résultats sont comparés avec des données expérimentales de largeur de pénétration et des mesures spectrophotométriques de concentration relative d'oxymyoglobine. L'effet de la pression partielle d'oxygène dans la formation de metmyoglobine dans le tissu musculaire est analysé à l'aide d'une équation obtenue pour des systèmes de solutions.

SAUERSTOFFDIFFUSION IN FLEISCHGEWEBE

Zusammenfassung—Die Durchdringung von Muskelgewebe durch Sauerstoff wurde mit Hilfe einer nichtstationären Darstellung des Problems mathematisch modelliert. Die wichtigen Parameter (Löslichkeit, Diffusionskoeffizient und Sauerstoffverbrauch im Gewebe) wurden bei verschiedenen Temperaturen berechnet. Die Ergebnisse wurden mit experimentell ermittelten Werten der Durchdringungsbreite und spektrophotometrischen Messungen der Oxymyoglobin-Konzentration verglichen. Die Auswirkung des Partialdrucks des Sauerstoffs auf die Bildung von Metmyoglobin im Muskelgewebe wurde mit einer Gleichung für Lösungssysteme analysiert.

ДИФФУЗИЯ КИСЛОРОДА В МЫШЕЧНЫХ ТКАНЯХ

Аннотация—В нестационарной постановке дана математическая модель процесса проникновения кислорода в мышечные ткани. При различных температурах дана оценка таких параметров, как растворимость, коэффициент диффузии и расход кислорода в тканях. Проведено сравнение полученных результатов с экспериментальными данными по глубине броникновения и с результатами спектрофотометрических измерений относительной концентрации оксимиоглобина. Результаты использовались для анализа влияния парциального давления кислорода при образовании метмиоглобина в мышечных тканях.