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KINETICS OF PIGMENT OXIDATION IN BEEF FROM STEERS SUPPLEMENTED WITH VITAMIN E

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INTRODUCTION

Supplementation of vitamin E to finishing steers can improve color and lipid stability of beef (Arnold et al., 1993). However, the mechanism by which α-tocopherol delays pigment oxidation in meat is not clear.

In this study, we describe a kinetic analysis for the production of metmyoglobin (Met) in beef derived from control and vitamin E supplemented animals. The model was applied to two muscles of different color stability: Longissimus lumborum (LL) and Gluteus medius (GM).

MATERIALS AND METHODS

Meat samples were obtained from the LL and GM muscles of 72 Holstein steers; 18 animals per treatment received 64 (control), 295, 550 and 2173 IU/steer daily of vitamin E (α-tocopheryl acetate , Hoffmann-La Roche, Inc., Nutley, NJ, USA) fed for two dose durations: 42 and 126 days. Muscles were removed from the carcasses, vacuum packaged and aged at 4°C for 13 days. Samples were cut, wrapped with fresh meat PVC film and displayed at 4°C.

Analysis for vitamin E content in meat was performed in duplicate by the method of Arnold et al. (1993). Surface pigment concentration was determined by reflectance spectrophotometry (Krzywicki, 1979) for triplicate cores. Data were analyzed as a split-plot design. Pigment variations over time were fitted by a nonlinear regression model.

RESULTS AND DISCUSSION

Table 1 shows the α-tocopherol concentration for each treatment combination in LL and GM. For both muscles, α-tocopherol content increased ($P<0.0001$) with dose and dose duration. Accumulation of α-tocopherol in the GM was higher ($P<0.0001$) than in the LL. α-tocopherol content differed among major muscles according to the ranking: Psoas major (PM) > gluteus medius > semimembranosus (SM) > longissimus (Chan et al., 1995; Liu et al.., 1995). Comparison of lipid stability across muscles followed a similar pattern (Chan et al., 1995). However, α-tocopherol accumulation contrasts with the relative color stabilities since the order in decreasing color stability is LL > SM > GM > PM (Liu et al., 1995).

Met formation in LL and GM was delayed ($P< 0.01$) by increasing vitamin E dose and duration. In both muscles, regardless of the duration of supplementation, an increase in dietary intake from 295 to 550 IU/day did not affect ($P>0.32$) Met or Oxy levels, so these data were pooled..For the different vitamin E treatments, GM accumulated more ($P<0.001$) Met than LL. Deoxy concentration was constant (Deoxy = 0.06) during the display period.

Fig. 1 shows Met formation over time for the different dose levels and dose durations used in this study. For LL, progress of oxidation was characterized by two distinct stages: a) an initial induction period which, within dose duration, increased with α-tocopherol content and b) a subsequent period where Met accumulation proceeded at a much faster rate. The presence of the induction period in the GM (Fig 2) was visually noticeable only at the highest dietary intake (2173 IU/day).

In buffer solutions, Met production is a combination of different processes which include Oxy autoxidation producing Met and superoxide anion ($O_2^{\cdot -}$), followed by the oxidation of another Oxy molecule by H_2O_2 from $O_2^{\cdot -}$dismutation (Tajima and Shikama , 1987). Yin and Faustman (1993) concluded that radicals generated by lipid autoxidation promote Met formation. α-Tocopherol is the most important radical scavenger within membranes and lipoproteins. By neutralizing lipid peroxy radicals, α-tocopherol provides an indirect protection to Oxy (Yin et al., 1993).

During the induction period, Met production is inhibited by α-tocopherol. Following assumed oxidation of α-tocopherol, Oxy will react with the lipid prooxidant species increasing Met accumulation. The time at which α-tocopherol is consumed (t_0) marks the end of the induction period, its extension will be determined by the α-tocopherol concentration in the muscle. Assuming that in both stages, Met accumulation follows first order kinetics, the integrated solutions are:

$$Y = A_0 * \exp(-A_1 * t) + A_2 \quad \text{for } t < t_0 \qquad (1) \qquad Y = A'_0 * \exp(-A'_1 * (t-t_0)) + A'_2 \quad \text{for } t > t_0 \qquad (2)$$

With Y = Met; A_1 and A'_1 = slope; A_2 and A'_2 = asymptotic value of Y. When the initial α-tocopherol level was low enough or Met formation rate was sufficiently slow so that Met asymptotic value for each reaction stage was not reached then $A_2 = 0$ and $A'_2 = 0$ respectively and the following equations resulted:.

$$Y = A_0 * \exp(-A_1 * t) \qquad (3) \qquad Y = A'_0 * \exp(-A'_1 * (t-t_0)) \qquad (4)$$

Pigment concentrations vs time for both muscles were fitted by a segmented model consisting of Eq. (3) for the induction period and either Eqs (2) or (4) for $t > t_0$. Induction time (t_0) values for each treatment combination are shown in Table 1. When vitamin E was administered at the highest level, t_0 value corresponding to LL was not detectable within a display period of 12 days. For each muscle and dose duration, t_0 increased with the α-tocopherol content of the muscle. For both muscles, an increment in dose duration from 42 to 126 days enlarged t_0. Although GM accumulated more α-tocopherol than LL, t_0 values for this muscle were lower than for LL. This behavior can be explained by considering that GM has higher TBA values than LL (Chan et al.., 1995), therefore we can expect a greater production of oxidative lipid radicals. α-tocopherol requirements in the GM will be higher than in the LL. For equivalent levels of α-tocopherol in both muscles, GM will present lower t_0 values than LL.

We propose the following scheme to account for the α-tocopherol effect on beef pigments:

Oxy ---> Met + $O_2^{\cdot -}$ (1) $O_2^{\cdot -}$ ---> H_2O_2 (2) Oxy + H_2O_2 ---> Met (3) LOOH --->LOO' (4)
LOO' ---> X (5) α-TH + LOO' ---> α-T$^{\cdot}$ + LOOH (6) Oxy + X ---> Met (7)

With LOOH = lipid hydroperoxide; LOO' = hydroperoxide radical; X = lipid prooxidant; α-TH = α-tocopherol; α-T$^{\cdot}$ = α-tocopherol non-reactive radical.

During the induction period, α-tocopherol will react with LOO', thus steps (5) and (7) will not occur. When the antioxidant is depleted, Oxy will react with the lipid prooxidants producing a considerable increase in Met accumulation rate.

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TABLE 1 Relationship between α-tocopherol content in muscle and the extension of the induction period t_0

Vitamin E (IU/day)	LL				GM			
	42[a]		126[a]		42[a]		126[a]	
	α-toc[b]	t_0[c]	α-toc[b]	t_0[c]	α-toc[b]	t_0[c]	α-toc[b]	t_0[c]
64	0.45[d]	4.48	0.48[d]	4.48	0.65[d]	1.80	0.70[d]	1.86
295[b]	0.87[e]	5.91	1.46[f]	6.82	1.26[f]	3.69	2.08[j]	4.75
550[b]	1.37[f]	5.91	2.48[j]	6.82	2.14[j]	3.69	3.29[l]	4.75
2173	2.92[g]	7.12	5.57[i]	ND[n]	4.23[k]	5.60	7.18[m]	6.01

[a]dose duration (days); [b]μg/g; [c]induction time (days); [d,e,f,g,h,i,j,k,l,m]means lacking a common superscript letter differ ($P<0.05$); [n]length of the induction period not discernible

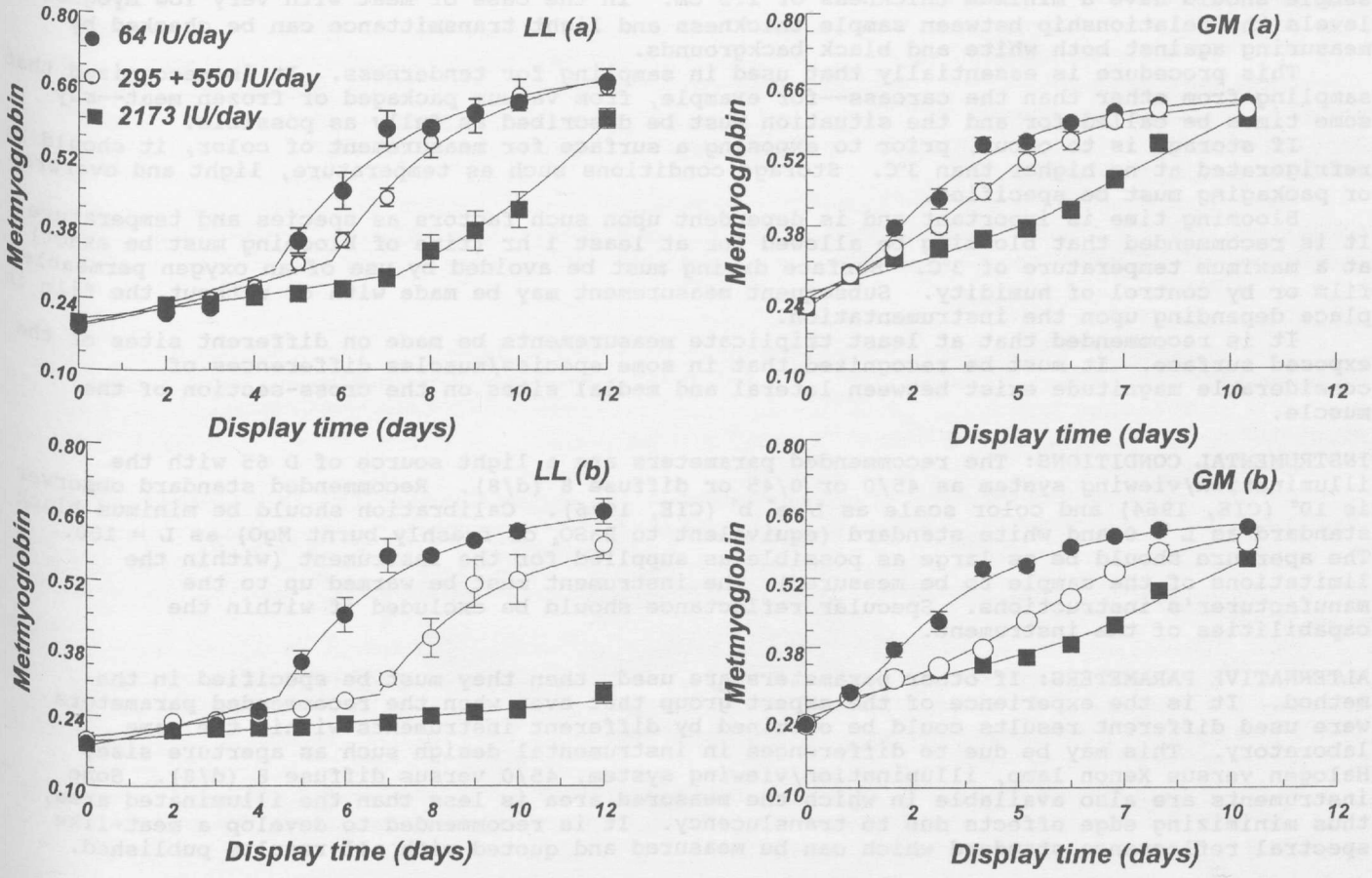

Fig.1 Observed and predicted metmyoglobin proportions. Dosage time = 42 days (a); 126 days (b)