ABSTRACT: A mechanistic model based on synthesis and inactivation of enzymes, associated to the respiration pathway, was proposed to describe wound-induced respiration of fresh-cut produce. Carrots (Daucus carota L.) were cut into slices, sticks, a combination of both, and shreds to obtain different wounding intensities, defined by the ratio of new area created to tissue weight (A/W). Respiration rates obtained at 10 °C showed a typical increase, a maximum peak, and a decrease until reaching steady state. A 2-phase exponential decay equation described the process, with the obtained key parameters relating to A/W. This model can be used to understand wound-induced respiration and the possible implications in produce quality changes and packaging design.

Keywords: respiration, wounding, modeling, fresh-cut, produce, carrots

Introduction

The U.S. fresh-cut produce market was estimated to be about $10 billion to $12 billion in sales in the year 2000 (IFPA/PMA 1999). The fresh-cut process implies the use of some degree of wounding activity such as slicing, chopping, dicing, peeling, trimming, or shredding, which will divide the produce into smaller segments and shorten the storage life (Saltveit 1997).

Wounding may induce a complex series of events designed to repair the damage caused in the tissue (Rolle and Chism 1987). An immediate response may include a wound signal formed in adjacent and distant tissues, which elicits a wide range of physiological and biochemical responses (Saltveit 1997). The most common responses to wounding may include an increase in respiration rate, ethylene production, quality changes, and synthesis and/or loss of phytochemicals (Yang and Pratt 1978; Kahl and Laties 1989; Kader 1992; Brecht 1995; Tapadia and others 1995; Salveit 2000). Among these responses, respiration rate is considered an important indicator of produce shelf life. For example, whole produce with higher respiration rates (Kader 1986) and wounded tissues with increased respiration rates (Rolle and Chism 1987) are associated to a shorter shelf life. Respiration also plays a major role in packaging design, where the targeted atmosphere is usually determined using non-wounded respiration rates. This targeted atmosphere might not be maintained if wounded tissue with increased respiration rates are used (Saltveit 1997).

Wound-induced respiration has been related to an enhanced synthesis of enzymes involved in the respiration pathway, such as ATP-dependent phosphofructokinase involved in carbohydrate breakdown leading to pyruvate (Hajirezaei and Stitt 1991) and to an enhanced aerobic mitochondrial respiration related to changes in mitochondrial structure and number (Asahi 1978). Wound-induced respiration may also be associated in part to α-oxidation of long-chain fatty acids from membrane deteriorative processes (Latties 1964; Latties 1978), wound-induced ethylene (Rolle and Chism 1987), and oxidative reactions leading to browning (Saltveit 1997). Wounding may also enhance the synthesis of different enzymes in plant tissue, like phenylalanine-ammonia-lyase activity (Saltveit 1997; Toivonen and DeEll 2002). In general, the induction of these enzymes is ephemeral and could be explained by the presence of an inactivation system, which may be used by the tissue to regulate PAL and other enzymes (Creasy and others 1986). Wound-induced respiration will depend on the type of tissue, temperature, controlled atmospheres (Watada and others 1996), and degree of cutting (Zhu and others 2001).

Several respiration-rate mathematical predictive models have been proposed for use in packaging design. For example, respiration rate has been related by a linear function (Hayakawa and others 1975), quadratic function (Yang and Chinnan 1988), and by an enzyme Michaelis-Menten kinetics approach (Lee and others 1991) to O2 and CO2. More recently, respiration rate has been related to degree of cutting using a second-order linear regression function (Zhu and others 2001). However, all these models are limited to steady-state conditions and do not consider the burst in respiration observed through time after wounding.

In this article, we propose a novel mechanistic approach for modeling wound-induced respiration rates through time, based on synthesis and inactivation of enzyme activities from the respiratory pathway. Carrot tissues with different degrees of wounding representing fresh-cut slices, sticks, a combination of both, and shreds are used to validate experimentally the proposed model. The proposed model could be used to better understand the process of wound-induced respiration and eventually be applied in packaging design and to understand quality changes in fresh-cut produce.

Materials and Methods

Carrots and wounding intensity

Whole-topped carrots (Daucus carota L.) cultivar Caropak from California were obtained from a local market (Albertsons, College Station, Tex., U.S.A.) and stored at 10 °C before use. Carrots were cut into slices, sticks, and a combination of both cuts and shreds to obtain different wounding intensities (A/W), defined as the ratio of the new surface area created over the tissue weight. To obtain carrots of similar initial weights of about 75 g, carrot samples were cut at both ends to obtain cylinders of 13.5-cm length and 2.5-cm dia.
Modeling wound-induced respiration...

All control samples had a wounding intensity corresponding to the new area created to form the carrot cylinder shape. When making slices, samples were cut cross section to the carrot cylindrical axis to obtain 2, 4, 8, 16, or 32 sections. Sticks were made by cutting along the carrot cylindrical axis to obtain 2, 4, 8, or 16 sections from each cylinder. For the combination of slices and sticks from each carrot cylinders, cutting was done in 2 steps. The 1st step consisted in obtaining 8 slice sections as described above, followed either by 2, 4, 8, or 16 stick sections obtained as described previously. The combination of 8 slices with 2, 4, 8, and 16 sticks gave pie-form sections. Finally, carrot shreds were obtained using a Magic Chef® food processor (model JK-357, Maytag Cooperation, Newton, Iowa, U.S.A.) using each carrot cylinder, and the new area created was estimated from 40 pieces of shredded carrots of about 5.1 g, which gave an area of about 119.7 cm².

Carrot samples showed a vascular xylem tissue with about a 0.5-cm dia in a cross section cut. For each type of cut studied, we evaluated the contribution of different type of cells to the wounding response by defining the ratio of vascular xylem tissue area to the total new surface area created (Axylem/A).

Respiration-rate measurement

Wound-induced respiration studies had 2 parts. First, we studied the respiration rate for each type of cut under different wounding intensities. Each type of cut was obtained using a different carrot batch (or purchase date). Sliced carrots and sticks were obtained from carrots purchased on April 20 and May 1, and a combination of cuts and shreds was obtained from carrots purchased on May 15, 2001. For the 2nd part of the study, we determined the effect of the contribution of different type of wounded cells to respiration rate. One batch of carrots (purchased July 30, 2001) was used for all 3 types of cuts (slices, sticks, and combination cuts), maintaining a constant A/W with different Axylem/A values.

Carrot samples were placed in glass jars connected to an air-flow through system supplied by an air GAST pump (model DOA-P104-U.S.A.). Exposure of carrot tissues to <1% CO₂ for short periods of time: was allowed to accumulate CO₂ to concentrations <1% for about 1 h in the closed system, and gas samples were withdrawn using syringes and analyzed using a Horiba CO₂ infrared gas analyzer (model PIR-2000, Horiba Instruments Inc., Irvine, Calif., U.S.A.). Exposure of carrot tissues to <1% CO₂ for short periods of time will not cause adverse physiological effects. Carrot controls and each type of cut were studied using 4 replicates.

Wound-induced respiration model

The proposed model of wound-induced respiration rate is based on the concept that wounding induces in the plant cells the synthesis of enzymes related to the respiratory pathway (Hajirezaei and Stitt 1991; Nanos and others 1994), and at the same time the induction of a degrading or inactivation system of the enzymes being formed (Kahl 1978).

The wound-induced respiration-rate model ($rCO₂^{w}$) will be formed by 3 components: an inherent non-wound respiration-rate baseline ($rCO₂^{i}$), an increased respiration rate caused by the synthesis of new enzymes ($rCO₂^{s}$), and a component equivalent to the respiration rate that stopped being synthesized due to the inactivation system formed ($rCO₂^{d}$). Thus,

$$rCO₂^{w} = rCO₂^{i} + rCO₂^{s} - rCO₂^{d}$$

**New enzyme synthesis.** Assuming that the new enzyme is synthesized according to 1st-order kinetics:

$$E = E_f (1 - e^{-ks t})$$

where $E$ is the new synthesized enzyme at any time, $E_f$ is the maximum amount possible of new enzyme synthesized, $k_s$ is the enzyme synthesis kinetics constant (d⁻¹), and $t$ is time (d). If we assume that the amount of $E$ is proportional to respiration rate, then

$$E_f = a(rCO₂^{smax})$$

$$E_f = b(rCO₂^{dmax})$$

where $a$ is a proportional constant, $rCO₂^{smax}$ is the respiration rate caused by the new enzyme synthesis (mL CO₂/kg h), and $rCO₂^{dmax}$ is the maximum amount of respiration rate caused by the presence of $E_f$. Substituting Eq. 3 and 4 into Eq. 2 gives us an expression for respiration rate due to enzyme synthesis:

$$rCO₂^{s} = rCO₂^{smax} (1 - e^{-ks t})$$

**Inactivation system.** Assuming that the inactivation system is synthesized according to 1st-order kinetics we obtain:

$$IS = IS_f (1 - e^{-kd t})$$

where $IS$ is the synthesized inactivation system at any time, $IS_f$ is the maximum amount possible of the synthesized inactivation system, and $k_d$ is the inactivation system synthesis kinetics constant (d⁻¹). If we assume that the amount of $IS$ is proportional to the amount of CO₂ that stopped being produced because of the synthesis of the inactivation system, then

$$IS_f = b(rCO₂^{dmax})$$

where $b$ is a proportional constant, $rCO₂^{dmax}$ is the respiration rate that stopped being synthesized due to the inactivation system formed (mL CO₂/kg h), and $rCO₂^{dmax}$ is the maximum respiration rate possible that stopped being synthesized in the presence of $IS_f$. Substituting Eq. 7 and 8 into Eq. 6 gives an expression for the respiration rate that stopped being synthesized due to the inactivation system:

$$rCO₂^{d} = rCO₂^{dmax} (1 - e^{-kd t})$$

Substituting Eq. 5 and 9 into Eq. 1 gives a general expression for wound-induced respiration rate as a function of time:

$$rCO₂^{w} = rCO₂^{i} + rCO₂^{smax} (1 - e^{-ks t}) - rCO₂^{dmax} (1 - e^{-kd t})$$

In the case when the wound-induced respiration rate returns back to the initial value of respiration, the terms $rCO₂^{smax}$ ≡ $rCO₂^{dmax}$, thus:

$$rCO₂^{w} = rCO₂^{i} + rCO₂^{smax} (1 - e^{-kd t} - e^{-ks t})$$

Equations 10 and 11 are a 2-phase exponential decay equation. The key parameters of the equation would be related to factors such as A/W, temperature, and oxygen tension, among others.
Modeling wound-induced respiration...

The statistical analysis was done using the SAS Statistical Analysis System for Windows v8.1 (SAS Inst. Inc, Cary, N.C., U.S.A.). The means were compared with Tukey’s Studentized Range test at α = 0.05. Model fitting was done using a GraphPad Prism software version 3.02 (GraphPad Software, Inc, San Diego, Calif., U.S.A.).

Results and Discussion

Wound-induced respiration and use of the proposed model

The initial non-wound respiration-rate values were similar for all control samples (about 19 mL CO₂/kg h). However, when wounded, the respiration rate of carrot tissue showed a typical increase, a maximum peak, and a decrease through time until reaching steady-state respiration values similar to that of carrot tissue before wounding (Figure 1). Respiration rate showed an about 2.1-fold to 2.3-fold increase using stick, slice, and combination cuts. The peak in respiration rate was reached after 1 d for slices, 2 d for sticks, and 3 d for combination of both cuts. Wound-induced respiration rate for each type of cut increased with A/W. The range of A/W values obtained depended on the type of cut (Table 1) and showed the following decreasing order, combination of both cuts > sticks > slices.

The proposed wound-induced respiration model for fresh-cut produce is formed by a 2-phase exponential decay equation (Eq. 10). The model has 2 first-order kinetics equations. It describes an increase in respiration due to enzyme synthesis and a simultaneous decrease due to the action of an inactivation system that degrades or inactivates the enzyme being formed. Fitting the equation to the experimental data shows a good visual fitting (Figure 1) with \( r^2 \) values ranging from 0.708 to 0.942 (Table 1). This would suggest that the wound-induced respiration process might be described using this mechanistic model approach.

It is known that wounding triggers key enzymes from the respiration pathway, such as phosphofructokinase (Hajirezaei and Stitt 1991) and cytochrome oxidase (Nanos and others 1994), by increasing and decreasing their activity through time, allowing an increase and decrease in respiration rate. Enzyme activity changes, and the amount present are regulated by the rate of synthesis and degradation. In general, the increase in activity could be due to activation of previously inactive enzymes or to novo synthesis (Kahl 1978). The effect of wound-induced enzyme synthesis and degradation (Creasy and others 1986) is also observed in many other

### Table 1—Wound-induced respiration-rate model parameters (\( k_s \), \( k_d \), and \( r\text{CO}_2_{\text{max}} \)) according to wounding intensity (A/W) of carrot tissue

<table>
<thead>
<tr>
<th>Type of cut</th>
<th>Nr of sections</th>
<th>Nr of cuts</th>
<th>A/W (cm²/g)</th>
<th>( A_{\text{xylem}}/A )</th>
<th>( r\text{CO}<em>2</em>{\text{max}} )</th>
<th>( k_s )</th>
<th>( k_d )</th>
<th>( r^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control slices</td>
<td>0</td>
<td>0</td>
<td>0.131</td>
<td>4.00</td>
<td>5.078</td>
<td>1.294</td>
<td>0.190</td>
<td>0.866</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>1</td>
<td>0.262</td>
<td>4.00</td>
<td>10.50</td>
<td>1.423</td>
<td>0.407</td>
<td>0.743</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>3</td>
<td>0.524</td>
<td>4.00</td>
<td>8.783</td>
<td>1.478</td>
<td>0.298</td>
<td>0.775</td>
</tr>
<tr>
<td></td>
<td>8</td>
<td>7</td>
<td>1.047</td>
<td>4.00</td>
<td>14.11</td>
<td>1.630</td>
<td>0.317</td>
<td>0.889</td>
</tr>
<tr>
<td></td>
<td>16</td>
<td>15</td>
<td>2.094</td>
<td>4.00</td>
<td>19.04</td>
<td>1.740</td>
<td>0.220</td>
<td>0.882</td>
</tr>
<tr>
<td></td>
<td>32</td>
<td>31</td>
<td>4.189</td>
<td>4.00</td>
<td>31.96</td>
<td>2.255</td>
<td>0.281</td>
<td>0.892</td>
</tr>
<tr>
<td>Control sticks</td>
<td>0</td>
<td>0</td>
<td>0.131</td>
<td>4.00</td>
<td>19.00</td>
<td>0.594</td>
<td>0.514</td>
<td>0.802</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>1</td>
<td>1.031</td>
<td>17.97</td>
<td>19.89</td>
<td>19.89</td>
<td>13.00</td>
<td>1.000</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>2</td>
<td>1.931</td>
<td>18.91</td>
<td>13.00</td>
<td>13.00</td>
<td>21.30</td>
<td>1.361</td>
</tr>
<tr>
<td></td>
<td>8</td>
<td>4</td>
<td>3.731</td>
<td>19.44</td>
<td>21.30</td>
<td>21.30</td>
<td>63.70</td>
<td>1.501</td>
</tr>
<tr>
<td></td>
<td>16</td>
<td>8</td>
<td>7.33</td>
<td>19.71</td>
<td>63.70</td>
<td>63.70</td>
<td>50.40</td>
<td>1.002</td>
</tr>
<tr>
<td>Control pies</td>
<td>0</td>
<td>0</td>
<td>0.131</td>
<td>4.00</td>
<td>50.40</td>
<td>1.002</td>
<td>0.879</td>
<td>0.708</td>
</tr>
<tr>
<td></td>
<td>8 &amp; 2</td>
<td>7 &amp; 1</td>
<td>1.947</td>
<td>11.40</td>
<td>51.62</td>
<td>1.052</td>
<td>0.278</td>
<td>0.836</td>
</tr>
<tr>
<td></td>
<td>8 &amp; 4</td>
<td>7 &amp; 2</td>
<td>2.847</td>
<td>14.12</td>
<td>53.09</td>
<td>1.047</td>
<td>0.273</td>
<td>0.838</td>
</tr>
<tr>
<td></td>
<td>8 &amp; 8</td>
<td>7 &amp; 4</td>
<td>4.647</td>
<td>16.39</td>
<td>71.14</td>
<td>1.080</td>
<td>0.355</td>
<td>0.870</td>
</tr>
<tr>
<td></td>
<td>8 &amp; 16</td>
<td>7 &amp; 8</td>
<td>8.247</td>
<td>17.97</td>
<td>70.78</td>
<td>1.257</td>
<td>0.331</td>
<td>0.914</td>
</tr>
<tr>
<td>Shreds</td>
<td>-600</td>
<td>—</td>
<td>23.49</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

Figure 1—Wound-induced respiration rate \( r\text{CO}_2_{\text{w}} \) of carrots compared with time for different types of cuts. The symbols are the experimental points (\( n = 4 \)), and the lines are the fitted models (Eq. 11). ▲ = control; • = 2 sections; ○ = 4 sections; □ = 8 sections; and ◇ = 16 sections.
Wounding intensity effects on model key parameters

To understand the effects of A/W on respiration rate, we analyzed the relationship between the key parameters of the wound-induced respiration model and A/W. Results indicate that the enzyme synthesis kinetics constant, k_s, increased linearly with A/W for each type of cut (Figure 2). We expected these linear responses to overlap for each type of cut because we were using one type of product; however, there were 3 distinct linear responses.

The obtained k_s values showed the following decreasing order: slices > sticks > combination of both cuts. On the other hand, the inactivation system synthesis kinetics constant, k_d, was basically independent of A/W values and the type of cut (Figure 3). These k_d values fell in a single curve. In general, the k_d values were about 3 times higher than the k_s values, indicating that the rate of synthesis is faster than that of degradation in carrot tissue. For rCO_2smax or rCO_2dmax there was also a linear increase with A/W, and this relationship again depended on the type of cut (Figure 4). The obtained rCO_2smax values showed the following decreasing order, combination of both cuts > slices > sticks. All these linear responses can be described with equations that could be used to predict the model key parameters for any A/W.

An extreme case of A/W would be shredding (A/W about 23.5). The model fitted the wound-induced respiration for shredded carrots with an r^2 = 0.931 (Figure 5), giving rCO_2smax and k_d values higher and similar, respectively, to the other cuts. However, the k_s value was unexpectedly lower compared with the other cuts (Table 1), suggesting that extreme A/W decreased enzyme synthesis kinetics.

In general, the observed dependence of the model key parameters on the type of cut could be related to factors such as the use of different carrot batches in the experiments, the contribution of different types of wounded cells in each type of cut, or to both factors. For example, the use of a different carrot batch could be related to different tissue maturity levels, whereas for the case of different wounded cells, it is possible that the amount of vascular xylem tissue present in each type of cut could have played a major role in the response. In our study, slices and sticks had a contribution of vascular xylem tissue to total cut area (A_xylem/A) of about 4% and 20%, respectively, indicating that different types of cells were wounded for each type of cut. For the combination of both cuts, the A_xylem/A ranged from about 4% to 18% as A/W increased (Table 1).
To evaluate the contribution of different type of wounded cells (Axylem/A) in the respiration-rate response, we used only 1 batch of carrots and cut the tissue into slices, sticks, and combination of both cuts to obtain a similar A/W of about 2.0. Results indicated that there was not a significant difference ($P > 0.05$) among the experimental respiration-rate data obtained for each type of cut (Figure 6). Moreover, the model fits the data with a good visual fitting and a $r^2$ ranging from 0.91 to 0.98 (Table 2). Additionally, the obtained $k_s$, $k_d$, and $r_{CO_2,\text{max}}$ parameters from Eq. 11 were similar for the 3 types of cuts and were independent of Axylem/A, which ranged from about 4% to 19% (Table 2).

In general, these results would suggest that the batch factor influenced the model parameters more than the wounded cells type factor.

**Model potential and limitations**

The proposed model is intended to be used as a tool to understand the wound-induced respiration process and to help identify possible factors involved in the response. The model is considered mechanistic because it gives an interpretation of how respiration rises and declines through time. Wound-induced respiration is an immediate response and no lag phase is present, thus using 1st-order kinetics to describe enzyme or inactivation system synthesis seems suitable, however, other kinetic orders could be studied as well. The model may also be used in more practical terms for packaging design of fresh-cut produce and quality prediction by accounting for wound-induced respiration effects.

In general, the model should be validated with different climatic and nonclimatic fresh-cut fruits and vegetables. Because these tissues are usually exposed to different temperature and altered gas compositions, there is need to understand how factors like temperature and oxygen tensions affect the model's parameters. For example, it is known that oxygen affects respiration rate following Michaelis-Menten kinetics and can be modeled by defining $V_m$ and $K_m$ parameters (Lee and others 1991). This set of Michaelis-Menten parameters most likely would be associated to a defined amount of E in the tissue, thus when wounding takes place, E increases and it is possible that $V_m$ may also increase. The result would be a new set of $V_m$ and $K_m$ parameters that would be associated to $r_{CO_2,\text{max}}$ through different oxygen levels. Similarly, the other 2 kinetic parameters, $k_s$ and $k_d$, would be dependent on oxygen tension as well.

There is also need to understand the effects of carbon dioxide and anaerobic conditions in wound-induced respiration rate and the parameters of the proposed model. For example, it is known that cytochrome oxidase may be stimulated or inhibited when the tissue is subjected to different concentration of CO$_2$ (Nanos and others 1994) or that phosphofructokinase (PFK) activity of shredded carrots increases under low O$_2$ tensions (Kato-Noguchi and Watada 1996).

One important factor, not considered in many studies, is the presence of moisture in the cut surface. This will impede gas diffusion, and with increasing respiration, this could possibly lead to anaerobiosis, causing deterioration of the tissue (Rolle and Chism 1987; Saltveit 1997). This factor may certainly affect the model parameters as well.

Finally, because it is known that the rate of quality deterioration depends on the amount and direction of cutting (Zhou and others 1992), the model proposed in this study may be used as a tool to understand quality changes. This can be done either by associating quality changes to the proposed wound-induced respiration

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**Table 2—Wound-induced respiration-rate model parameters ($k_s$, $k_d$, and $r_{CO_2,\text{max}}$) for different types of cuts and similar wounding intensity of carrot tissue (A/W about 2.0)**

<table>
<thead>
<tr>
<th>Type of cut</th>
<th>Nr of sections</th>
<th>Nr of cuts</th>
<th>A/W (cm$^2$/g)</th>
<th>Axylem/A</th>
<th>$r_{CO_2,\text{max}}$</th>
<th>$k_s$</th>
<th>$k_d$</th>
<th>$r^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Slices</td>
<td>16</td>
<td>15</td>
<td>2.09</td>
<td>4.00</td>
<td>29.95</td>
<td>3.03</td>
<td>0.27</td>
<td>0.979</td>
</tr>
<tr>
<td>Sticks</td>
<td>4</td>
<td>2</td>
<td>1.93</td>
<td>18.91</td>
<td>30.00</td>
<td>3.00</td>
<td>0.25</td>
<td>0.981</td>
</tr>
<tr>
<td>Slices and sticks</td>
<td>8 and 2</td>
<td>7 and 1</td>
<td>1.95</td>
<td>11.40</td>
<td>25.00</td>
<td>3.00</td>
<td>0.30</td>
<td>0.912</td>
</tr>
</tbody>
</table>

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**Figure 5—Maximum respiration rate ($r_{CO_2,\text{max}}$) compared with wounding intensity (A/W) for different types of cuts. $\bigcirc =$ carrot slices; $\square =$ carrot sticks; $\triangle =$ combination of slices and sticks.**

**Figure 6—Wound-induced respiration rate ($r_{CO_2}$) of carrots compared with time for different types of cuts and similar A/W of about 2.0. The symbols are the experimental points ($n=4$) and the lines are the fitted models (Eq. 11). $\bigcirc =$ 16 slices; $\square =$ 4 sticks; and $\triangle =$ 8 slices and 2 sticks.**
model or by defining a similar mechanistic model approach that could explain quality changes induced by wounding.

**Conclusions**

We propose a mechanistic model to describe wound-induced respiration of fresh-cut produce. It is based on the concept that wounding causes the synthesis of enzymes related to the respiration pathway and a system capable of degrading or inactivating the newly formed enzymes. The result is an increase in respiration rate, a maximum peak reached, and a final decrease of respiration rate through time until reaching a steady state. The wound-induced respiration rate and the key parameters of the model are dependent on A/W. This model can be used to better understand the wound-induced respiration process and be applied as a mean to identify factors that may affect the wound response. From a practical application, the proposed model has the potential to be used as a tool in packaging design as well as in understanding wound quality changes in fresh-cut produce.

**References**


