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Metabolic profile of wound-induced changes in primary carbon metabolism in sugarbeet root

Abbas M. Lafta, Karen Klotz Fugate *

USDA-ARS, Northern Crop Science Laboratory, 1605 Albrecht Blvd., N., Fargo, ND 58102-2765, USA

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ABSTRACT

Injury to plant products by harvest and postharvest operations induces respiration rate and increases the demand for respiratory substrates. Alterations in primary carbon metabolism are likely to support the elevated demand for respiratory substrates, although the nature of these alterations is unknown. To gain insight into the metabolic changes that occur to provide substrates for wound-induced increases in respiration, changes in the concentrations of compounds that are substrates, intermediates or cofactors in the respiratory pathway were determined in sugarbeet (*Beta vulgaris* L.) roots in the 4 days following injury. Both wounded and unwounded tissues of wounded roots were analyzed to provide information about localized and systemic changes that occur after wounding. In wounded tissue, respiration increased an average of 186%, fructose, glucose 6-phosphate, ADP and UDP concentrations increased, and fructose 1,6-bisphosphate, triose phosphate, citrate, isocitrate, succinate, ATP, UTP and NAD^+ concentrations decreased. In the non-wounded tissue of wounded roots, respiration rate increased an average of 21%, glucose 6-phosphate, fructose 6-phosphate, glucose 1-phosphate and ADP concentrations increased, and isocitrate, UTP, NAD^+, $NADP^+$, and NADPH concentrations declined. Changes in respiration rate and metabolite concentrations indicated that localized and systemic changes in primary carbon metabolism occurred in response to injury. In wounded tissue, metabolite concentration changes suggested that activities of the early glycolytic enzymes, fructokinase, phosphofructokinase, phosphoglucose isomerase, and phosphoglucomutase were limiting carbon flow through glycolysis. These restrictions in the respiratory pathway, however, were likely overcome by use of metabolic bypasses that allowed carbon compounds to enter the pathway at glycolytic and tricarboxylic acid (TCA) cycle downstream locations. In non-wounded tissue of wounded roots, metabolic concentration changes suggested that glycolysis and the TCA cycle were generally capable of supporting the small systemic elevation in respiration rate. Although the mechanism by which respiration is regulated in wounded sugarbeet roots is unknown, localized and systemic elevations in respiration were positively associated with one or more indicators of cellular redox status.

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1. Introduction

Plant products inevitably are wounded from harvest, transport and storage operations. Wounding triggers an array of responses including cell division and the biosynthesis of callose, suberin, lignin, phytoalexins, and structural and defense proteins to assist in sealing the wound site from the environment, repairing damaged tissue, minimizing dehydration, and defending against opportunistic pathogens (reviewed in de Bruxelles and Roberts, 2001; Léon et al., 2001). Wounding also induces respiration, presumably to provide energy for these anabolic processes (Lipetz, 1970). Increases in respiration due to mechanical injury have been documented in numerous harvested plant products and are typically large, with respiration rate commonly increasing several fold (Passam et al., 1976; Kays and Paull, 2004; Serrano et al., 2004).

The increase in respiration in response to wounding is likely to require alterations in plant primary carbon metabolism. Although amino acids, proteins, lipids and organic acids can serve as substrates for respiration, carbohydrates, especially sucrose and starch, are the most common substrates for the process (Siedow and Day, 2000). Respiration of carbohydrates requires activity of sucrose and/or starch degrading enzymes as well as operation of the glycolytic pathway and the tricarboxylic acid (TCA) cycle. Combined these pathways provide the reduced compounds needed to fuel the electron transport chain and catalyze ATP formation via oxidative phosphorylation. While the flow of carbon through primary carbon catabolic pathways must increase to provide for the increased use of respiratory substrates in wounded plant organs, it is unknown how plants alter their metabolism to achieve this.

* Corresponding author. Tel.: +1 701 239 1356; fax: +1 701 239 1349.
E-mail address: karen.fugate@ars.usda.gov (K.K. Fugate).

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Determining the mechanism(s) by which metabolism is altered to support the large increases in respiration caused by wounding is complicated by the complexity of the respiratory pathway (Fig. 1). Sucrose degradation can occur by the action of three different enzyme activities, starch degradation requires at least three enzyme activities, and glycolysis and the TCA cycle require at least 10 and 9

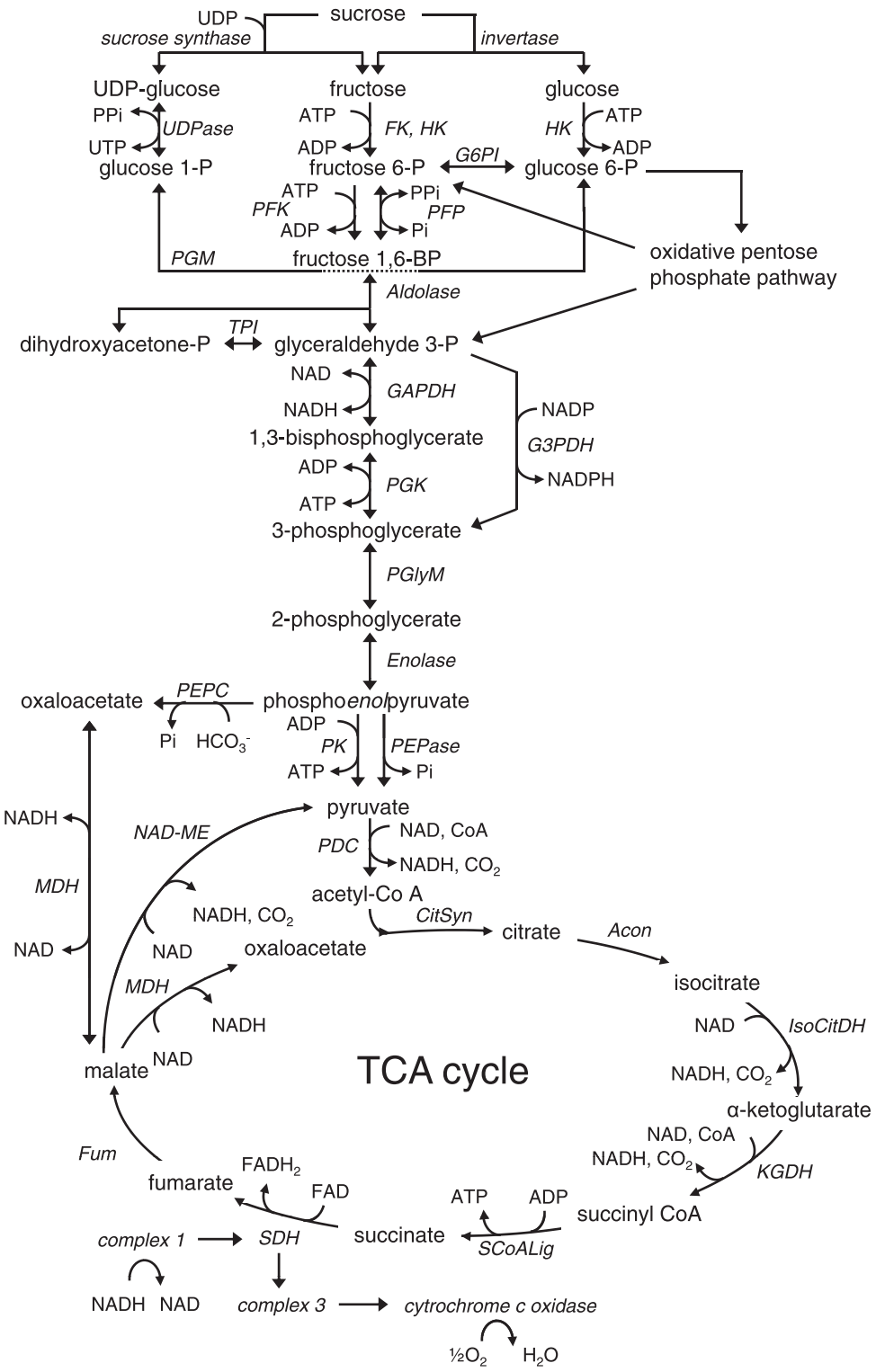

Fig. 1. Schematic of the enzymatic steps and metabolic intermediates in the respiration of sucrose. Enzyme abbreviations: Acon, aconitase; CitSyn, citrate synthase; FK, fructokinase; Fum, fumarase; G3PDH, NADP-dependent non-phosphorylating glyceraldehyde 3-phosphate dehydrogenase; G6PI, glucose 6-phosphate isomerase; GAPDH, glyceraldehyde 3-phosphate dehydrogenase; HK, hexokinase; IsoCitDH, isocitrate dehydrogenase; KGDH, α-ketoglutarate dehydrogenase; MDH, malate dehydrogenase; NAD-ME, NAD-malic enzyme; PDC, pyruvate dehydrogenase complex; PEPase, phospho*enol*pyruvate phosphatase; PEPC, phospho*enol*pyruvate carboxylase; PFK, phosphofructokinase; PFP, pyrophosphate-dependent phosphofructokinase; PGK, phosphoglycerate kinase; PGlyM, phosphoglyceromutase; PGM, phosphoglucomutase; PK, pyruvate kinase; SCoALig, succinyl coenzyme A ligase; SDH, succinate dehydrogenase; TPI, triose phosphate isomerase; UDPase, UDP-glucose pyrophosphorylase. Chemical compound abbreviations: CoA, coenzyme A; dihydroxyacetone-P, dihydroxyacetone phosphate; fructose 1,6-BP, fructose 1,6-bisphosphate; fructose 6-P, fructose 6-phosphate; glucose 1-P, glucose 1-phosphate; glucose 6-P, glucose 6-phosphate; glyceraldehyde 3-P, glyceraldehyde 3-phosphate; Pi, inorganic phosphate; PPi, inorganic pyrophosphate.