

Regulation of glucose-6-phosphate dehydrogenase in plants (review article)

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ABSTRACT

The regulation of G6PDH has been studied in plants, eukaryotic algae and cyanobacteria, but the gene encoding G6PDH has been isolated from only a few of these sources. In plants, Glucose -6-phosphate dehydrogenase (G6PDH) is present in the cytosol and in plastids. High sugar levels in the cytosol results in higher enzyme levels and G6PDH activity. In contrast, plastidic G6PDH can be stimulated by low NADPH to NADP⁺ ratios and is restricted by reductive inactivation. Both isoforms are feedback inhibited by NADPH.

Glucose- 6-Phosphate Dehydrogenase – General Properties

Glucose-6-phosphate 1-dehydrogenase (G6PDH, EC 1.1.1.49) is an enzyme that catalyses the rate-limiting step of the oxidative pentose phosphate pathway OPPP. It is a critical component of the oxidative stage of the pentose phosphate pathway, a metabolic network known to produce NADPH and ribose-5-phosphate, two metabolites essential for DNA synthesis [1]. It is the main focus of regulation of this pathway in both plants and animals. Almost all forms of G6PDH studied thus far are highly specific for their substrates, G-6-P and NADP⁺ for this reason; many of the properties of G6PDH have been preserved across evolutionary lines [2]. All G6PDH's have a single subunit type with the native enzyme existing as a dimer, tetramer or hexamer. The subunit size is also highly conserved between prokaryotes and eukaryotes between 50 to 67 kDa. The majority of characterized G6PDH enzymes show hyperbolic kinetics with some cyanobacterial enzymes showing sigmoidal kinetics [3]. A number of bacterial forms of the enzyme are capable of using NAD⁺ in place of NADP⁺ [4]. All G6PDH's are inhibited by NADPH, which acts as a competitive inhibitor. Metabolic effectors of G6PDH include Mg²⁺ (increases activity), ATP, ADP, AMP, long-chain acyl-CoA's (in mammalian liver, adipose tissue and erythrocytes), steroids, CoA and acetyl-CoA (in some plant species and bacteria this acts as a negative effector) [5]. Enzyme activity is to be measured spectrophotometrically by monitoring NADPH production by G6PDH at 340 nm.

G6PDH in photosynthetic organisms

The regulation of G6PDH has been studied in plants, eukaryotic algae and cyanobacteria, but the gene encoding G6PDH has been isolated from only a few of these sources [6-9]. Plant G6PDH is an unstable enzyme and kinetic characterization has usually been performed on either crude extracts or partially purified protein preparations. All plant cells contain plastids, specialized organelles that perform various biochemical processes including photosynthesis, biosynthesis of starch and oil biogenesis.

G6PDH is the first and a key control enzyme in the Oxidative Pentose Phosphate Pathway (OPPP) where NADPH is concomitantly produced from

NADP⁺ when G6PDH converts glucose-6-phosphate to 6-phosphogluconate. The OPPP serves as an alternative to glycolysis for the metabolism of glucose.

At least two compartment-specific isoforms of G6PDH, a cytosolic and a plastidic form exist in green plant tissues. G6PDH has been purified to apparent homogeneity from bacteria, yeast and animal tissues. In most cases it exists as a homodimer or a homotetramer. In higher plants, at least two G6PDH enzymes exist in two different cellular compartments, the cytosol and plastids. Each of these proteins in plants is encoded by a separate corresponding nuclear gene and share higher protein homology to each other (65%) than to cyanobacterial homologues (about 55%) [10].

G6PDH in higher plants

There are two compartment-specific isozymes of G6PDH in higher plants: one in the cytosol, the second in the plastids. The cytosolic isozyme has been purified from pea leaves [7] and potato tubers [6]. The chloroplast protein has been purified from pea leaves [8]. Another plastidic protein was cloned, but not directly purified, from potato shoots [10]. Attempts to purify G6PDH from barley root plastids were unsuccessful, as the protein was highly unstable [11]. The two isozymes are immunologically distinct, but compartment-specific forms share similarity between plant tissues [7].

In pea chloroplasts, G6PDH exists as a homotetramer of 56 kDa monomers, and has been purified to homogeneity [8]. It exhibits a pH optimum around 8.2 and has Km values of 2.4 μ M and 370 μ M for NADP⁺ and glucose-6-phosphate respectively.

The plastidic enzyme has been more difficult to purify from higher plants and most of the characterization has been performed on isolated plastid extracts [12]. The plastidic isoform from pea chloroplasts is similar to other higher plant G6PDH [8]. The native protein is a tetramer composed of four 56 kDa subunits, with a lower affinity for G-6-P and a greater affinity for NADP⁺ when compared to its cytosolic counterpart.

The regulation of G6PDH in higher plants occurs at the level of translation and transcription. Regulation by light/dark transitions is one way to study the metabolic changes and activity of chloroplast G6PDH [13]. The role of the ferredoxin/thioredoxin system has been examined in the light dependent inactivation of G6PDH. Isolated spinach chloroplasts follow a cycle of dark activation and light inactivation of the enzyme [12] as a way to regulate activity. G6PDH in spinach chloroplasts is inactivated by DTT (a sulfhydryl reagent that imitates the effects of thioredoxin). Light-dependent inactivation and DTT inactivation of G6PDH may not, be achieved through the same mechanism [12]. Root plastid G6PDH is also redox regulated in a similar fashion to spinach chloroplasts [11]. There has been limited study of the effects of metabolites on the chloroplast enzyme but the root plastid enzyme shows inhibition by ATP, UDP, citrate and acetyl-CoA [11].

Synthesis and turnover

Glucose-6-phosphate dehydrogenase is stimulated by its substrate Glucose-6-Phosphate. The usual ratio of NADPH/NADP⁺ in the cytosol of

tissues engaged in biosyntheses is about 100/1. Increased utilization of NADPH for fatty acid biosynthesis will dramatically increase the level of NADP⁺, thus stimulating G6PDH to produce more NADPH. G6PDH converts glucose-6-phosphate into 6-phosphoglucono- δ -lactone and is the rate-limiting enzyme of the pentose phosphate pathway.

Cellular Functions

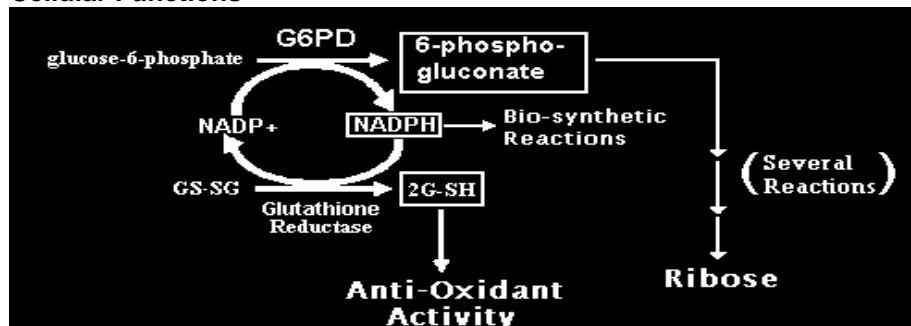


Fig.1: Cellular functions of Glucose-6-phosphate dehydrogenase

The G6PDH enzyme functions in catalyzing the oxidation of glucose-6-phosphate to 6-phosphogluconate, while concomitantly reducing nicotinamide adenine dinucleotide phosphate (NADP⁺ to NADPH); or, in terms of electron transfer, glucose-6-phosphate loses two electrons to become 6-phosphogluconate and NADP⁺ gains two electrons to become NADPH. This is the first step in the pentose phosphate pathway. In addition to producing the 5-carbon sugar ribose, G6PDH is also responsible for maintaining adequate levels of NADPH inside the cell (see Fig.1). NADPH is a required cofactor in many biosynthetic reactions. NADPH is also used to keep glutathione, a tri-peptide, in its reduced form.

Regulation

The enzyme, glucose-6-phosphate dehydrogenase (G6PDH, EC1.1.1.49), where it plays the key role in regulating carbon flow through the pentose phosphate pathway. Specifically, the enzyme catalyzes the first reaction in the pathway leading to the production of pentose phosphates and reducing power in the form of NADPH for reductive biosynthesis and maintenance of the redox state of the cell.

Regulation of G6PDH enzymes in the cytosol and in chloroplasts are governed by distinct mechanisms. Figure 2 shows a hypothetical model based on the elucidated regulation principles. High sugar levels in the cytosol trigger elevated transcription of the cytosolic G6PDH gene via sugar mediated signaling to the nucleus. Increased mRNA expression results in higher enzyme levels and G6PDH activity. C5 sugar phosphates formed in the cytosol can be used for nucleotide synthesis or are transported into plastids to replenish continuous withdrawals of Ribose-5-phosphate R5P and

Erythrose-4-phosphate E4P (by plastid-localized nucleotide synthesis and the shikimate pathway, respectively), which is especially important in metabolic sink situations (i.e. in darkness and in heterotrophic tissues). In contrast, G6PDH activity in plastids can be stimulated by low NADPH to NADP⁺ ratios (most likely via dephosphorylation of the existing enzyme pool) and is restricted in prolonged dark periods (probably via phosphorylation). This ensures quick adaptation of the OPPP to short-term NADPH shortage in the stroma and could help to poise the important but labile balance of stromal reduction charge in the night, when alternative mechanisms like the malate valve are inactive [14, 15].

In green tissues of plants during the light period, photosynthesis generates reduced nicotinamide adenine dinucleotide phosphate (NADPH), which is used in many reactions, for example, carbon fixation, fatty acid synthesis, and nitrogen assimilation. The oxidative pentose phosphate pathway (OPPP) is one of the major sources of NADPH in non-photosynthetic tissues as well as in photosynthetic tissues under darkness [16, 17].

The two dehydrogenases in the OPPP, glucose- 6-phosphate dehydrogenase (G6PDH, EC 1.1.1.49) and 6-phosphogluconate dehydrogenase (6PGDH, EC 1.1.1.44), use NADP⁺ as a cofactor and generate two moles of NADPH during the conversion of one mole glucose-6-phosphate (G6P) to ribulose-5-phosphate. The reaction catalyzed by G6PDH is considered the regulatory step in the OPPP because of its tight regulation by the redox balance, that is, NADPH/ NADP⁺ [18-19].

In plants, substantial evidence shows G6PDH to be involved in nitrogen assimilation [20-22], responses to stresses including oxidative stress [15, 23], pathogenesis [24, 25], salt stress [26], and metal toxicity [27].

Two subcellular localizations have been confirmed for plant G6PDHs, cytosolic and plastidic [28] and both isoforms are feedback inhibited by NADPH [29]. In chloroplasts, where photosynthesis generates NADPH, plastidic G6PDH isoforms are inactivated by thioredoxin reduction that involves two cysteine residues conserved only among plastidic isoforms [6, 7, and 13]. Thus for plastidic isoforms, two mechanistic principles for G6PDH regulation are known, feedback inhibition and reductive inactivation, providing the basis for an inverse relationship between photosynthesis and G6PDH activity. To date, only one study reports peroxisomal G6PDH (protein and activity) in pea leaves (*Pisum sativum*), but neither nucleotide nor amino acid sequences are available [30]. The authors speculated that G6PDH as well as 6PGDH, the second enzyme in OPPP, are involved in the regeneration of NADPH required for the ascorbate-glutathione cycle occurring in the leaf peroxisome. A G6PDH isoform not subjected to these regulatory mechanisms can be postulated and such an isoform could potentially play a role in providing NADPH in non-photosynthetic tissues or in green tissues independently of photosynthesis to support sudden increases in the demand for NADPH, for example, under oxidative stress [31]. In their phylogenetic study, Wendt *et al.* (1999) showed that plastidic isoforms are

separated into two groups, P1 and P2, which can be distinguished by specific antibodies [32].

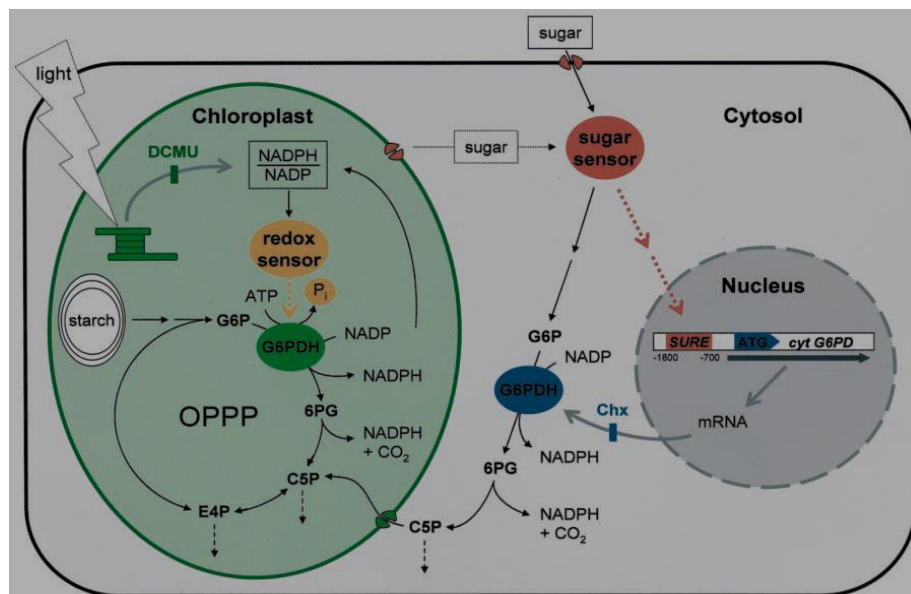


Figure 2. Model of G6PDH regulation in the cytosol and in chloroplasts of leaf tissue [15].

Distinct biochemical characteristics together with specific gene expression patterns were observed for a P1 and a P2 isoform in potato [1, 33]. Isoform P2 was less sensitive to both regulatory mechanisms by NADPH and the gene was ubiquitously expressed while the P1 gene was predominantly expressed in green tissues. The authors proposed that G6PDH isoforms belonging to the plastidic P1 group are tightly regulated by photosynthetic redox potential in green tissues, while those in the P2 group are less tightly regulated ubiquitous isoforms. A P2 isoform was also identified from barley roots with a low sensitivity to NADPH feedback inhibition, reinforcing this view [31]. Without a plant's genomic sequence, characterizing the full complement of isoforms for a given class of enzymes has not been possible in the past. In the post-genomic era, this limitation is no longer hampering genome-wide analysis.

Ferredoxin-thioredoxin regulation of enzymes

Photosynthesis fixes CO₂ into carbohydrates, a process that occurs in the light, while the majority of carbohydrate degradation occurs in the dark. In the light, enzymes of the Calvin cycle, or the Reductive Pentose Phosphate Pathway (RPPP) need to be activated and at the same time those of the OPPP need to be inactivated [34]. There are three possible mechanisms of

light-dependent regulation of chloroplast enzymes: 1) effector-mediated, 2) ion-mediated and 3) protein-mediated. The effector-mediated mechanism leads to an allosteric activation or inactivation of enzymes, based on light-induced changes of relevant effector metabolites. For example Rubisco (RuBP- ribulose-1,5-bisphosphate) is activated in the light, by an increased level of NADPH and ATP. RuBP is further activated by intermediates of the Calvin cycle. Enzymes regulated by an ion-mediated mechanism change activity by altering pH or regulatory cations like Mg^{2+} . Some enzymes of the Calvin cycle have strict pH optima. In chloroplasts, favorable alkaline conditions occur only in the light. The third mechanism of protein-mediated regulation involves enzyme modification by another protein thioredoxin (Trx) that alters activity. Light regulation of enzymes using the ferredoxin-thioredoxin system is an example where the change in the redox balance occurs, increasing reduced reductant molecule levels upon illumination [35]. Thioredoxin regulates enzyme activity by changing the redox status, specifically of disulfide bridges between cysteine residues, of target proteins.

The ferredoxin-thioredoxin system consists of ferredoxin, thioredoxin and ferredoxin-thioredoxin reductase [35]. Ferredoxin is the terminal protein electron acceptor of the photosynthetic electron transport chain. Excitation of chlorophyll reaction centers namely photosystem one (PSI) by light, transfers electrons to ferredoxin and then to target acceptors like Fd-dependent enzymes and $NADP^+$ which are reduced. Electrons are passed to ferredoxin-thioredoxin reductase reducing a disulfide bond to its dithiol form. In a similar event, reduced ferredoxin-thioredoxin reductase then reduces thioredoxin which acts as the protein mediator of light regulation of a variety of enzymes, by changing their redox status. Thioredoxin (12 kDa) is a highly soluble protein that has been detected in plant, animal and bacterial systems; it regulates many cellular processes in photosynthetic and heterotrophic systems [35]. In photosynthetic systems, reduction by thioredoxin generally leads to activation of an enzyme. One notable, yet not surprising, exception is G6PDH, in the light; reduced thioredoxin inactivates this enzyme of the OPPP. This type of redox regulation by thioredoxin can be reversed. The target enzymes must be re-oxidized in the dark in order to inactivate the light-dependent pathways and to activate dark-dependent pathways. The mechanism of oxidation of these proteins in the dark is not well characterized and may occur through different mechanisms, including the oxidation of thioredoxin and subsequent oxidation of reduced enzymes [35]. Wenderoth et al identified the cysteine residues that were responsible for redox regulation in a higher plant chloroplast G6PDH by using site-directed mutagenesis [36]. Chloroplast G6PDH homologues all show the inactivation by redox modification of the ferredoxin-thioredoxin system [12, 35]. The redox chain pathway guarantees that specific target enzymes are activated and kept in the reduced state in the light. G6PDH activity is turned off and is only active in its oxidized state in the dark when photosynthetic electron flow ceases. The molecular basis for this control mechanism is disulfide-dithiol interchange of certain regulatory cysteine residues in the target enzymes. The reason for

such regulation prevents carbohydrate breakdown by the OPPP that would work against concurrent CO₂ fixation by the Calvin cycle [32].

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تنظيم إنزيم **Glucose-6-Phosphate Dehydrogenase** في النبات

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ان تنظيم إنزيم **Glucose-6-Phosphate Dehydrogenase** في النبات قد تمت دراسته في الخلية الحقيقية للطحلب والبكتيريا الزرقاء ولكن الجين المسئول عن نقل الرسالة لهذا الانزيم قد تم عزله من هذه المصادر القليلة ويوجد إنزيم **Glucose-6-Phosphate Dehydrogenase** في النبات في العصارة الخلوية والبلاستيدات وكلما ارتفع مستوى السكر في العصير الخلوي للخلية يرتفع مستوى إنزيم **Glucose-6-Phosphate Dehydrogenase** ونشاطه وعلي العكس من ذلك فإن الانزيم نفسه في البلاستيدات يمكن تحفيزه بالنسبة المنخفضة لـ $NADPH$ to $NADP^+$.

قام بتحكيم البحث

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