A photorespiratory glyoxylate shunt in the cytosol supports photosynthesis and plant growth under high light conditions in Arabidopsis

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Photorespiration is a central aspect of plant metabolism that is tightly connected to photosynthesis, and functions in part to support photosynthetic performance, especially under stress conditions such as high light. However, our understanding of the mechanisms underlying the role and regulation of photorespiration in plant response to high light is limited. To identify modulators of photorespiration under high light, we isolated genetic suppressors of the photorespiratory mutant *hpr1*, which is defective in the peroxisomal hydroxypyruvate reductase 1. A suppressor that partially rescued *hpr1* under high light was mapped to *GLYR1*, which encodes the cytosolic glyoxylate reductase 1 enzyme that converts glyoxylate to glycolate. Independent *GLYR1* loss-of-function mutants also partially rescued *hpr1* and another photorespiratory mutant, *catalase 2*. Our genetic, transcriptomic and metabolic profiling analyses together suggested a novel connection between cytosolic glyoxylate and a non-canonical photorespiratory route mediated by the cytosolic HPR2 enzyme, which we named the photorespiratory glyoxylate shunt. This shunt is especially critical under high light intensities when a high rate of photorespiratory flux is required and in the absence of a properly functional major photorespiratory pathway. Our findings support the metabolic flexibility of photorespiration and may help future efforts to improve crop performance under stress conditions.

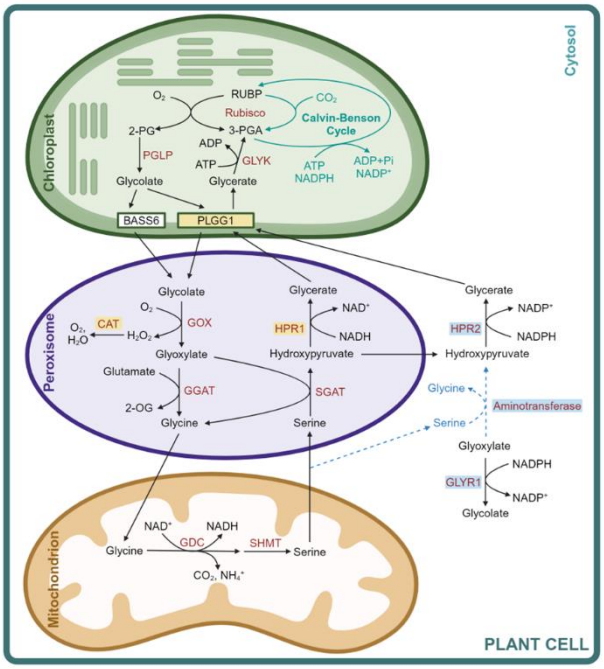

Figure. The established photorespiration pathway and the cytosolic glyoxylate shunt proposed in this study. Photorespiration involves a series of reactions in the chloroplast, peroxisome, mitochondrion, and cytosol. We propose that defective GLYR1 allows the accumulated free glyoxylate in the cytosol to react with serine, catalyzed by an unknown aminotransferase. The hydroxypyruvate produced can be further converted by HPR2 to glycerate, which re-enters the chloroplast.