Transcriptome profiling of postharvest kiwifruit in response to exogenous nitric oxide

Rui Yang, Xiaocui Lin, Yuan Dou, Wei Zhang, Huaying Du, Chunpeng Wan, Jinyin Chen, Lili Zhang and Liqin Zhu

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Abstract

Nitric oxide (NO) is a signal molecule that can regulate fruit physiology. Several studies have indicated that NO can inhibit the ripening of kiwifruit, but the mechanism underlying this process is unresolved. This study used transcriptome analysis to identify the essential genes related to NO regulation during kiwifruit softening. NO gas fumigation (15 μl L^{-1}) significantly delayed kiwifruit softening. There were 736 differentially expressed genes (DEGs) between the NO treatment and the control. The expression levels of polygalacturonase (*PG*), pectate lyase (*PL*), β-galactosidase (*b-GAL*), pectinesterase (*PE*), and the beta-amylases-related genes decreased in response to the NO treatment, while those of four genes encoding cellulose synthase increased. The expression of genes related to ethylene biosynthesis and signal transduction also differed; the expression levels of 1-aminocyclopropane carboxylic acid oxidase (*ACO*), the ethylene receptors (*ERS1, ETR2*), and the ethylene-responsive transcription factors (*ERF016*, *ERF7*, *ERF010*, *ERF062*, *ERF110*, *ERF037*, *ERF008*, *ERF113*, *ERF12*, *ERF095*) were lower in the NO-treated kiwifruit. Expression of the calcium ion (Ca_2+) signal-related genes (*CNGC1*, *CPK1*, *CIPK2*, *CML31*, *CML48*, *ZIFL1*) significantly differed and may be involved in the regulation of the NO softening response. These findings add to our understanding of the molecular mechanisms of the NO-delayed softening response in kiwifruit.