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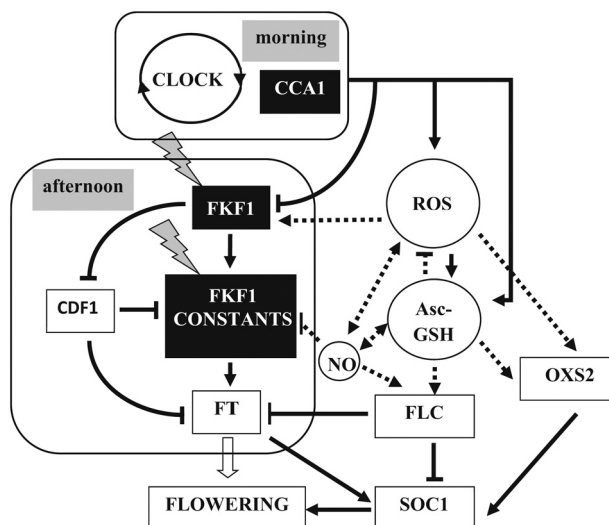
Plant Science

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## Corrigendum

Corrigendum to “Redox control of plant growth and development”  
[Plant Sci. 211 (2013) 77–91]Gábor Kocsy<sup>a,b,\*</sup>, Irma Tari<sup>c</sup>, Radomíra Vanková<sup>d</sup>, Bernd Zechmann<sup>e</sup>, Zsolt Gulyás<sup>a,b</sup>, Péter Poór<sup>c</sup>, Gábor Galiba<sup>a,b</sup><sup>a</sup> Agricultural Institute, Centre for Agricultural Research, Hungarian Academy of Sciences, Brunszvik u. 2., 2462 Martonvásár, Hungary<sup>b</sup> Research Institute of Chemical and Process Engineering, Faculty of Information Technology, University of Pannonia, Egyetem u. 10, 8200 Veszprém, Hungary<sup>c</sup> Department of Plant Biology, Faculty of Science and Informatics, University of Szeged, Középfasor 52, 6726 Szeged, Hungary<sup>d</sup> Institute of Experimental Botany, Academy of Sciences of the Czech Republic, Rozvojová 263, 16502 Prague, Czech Republic<sup>e</sup> Institute of Plant Sciences, Karl-Franzens-University of Graz, Schubertstr. 51, 8010 Graz, Austria

The authors regret that an incorrect version of Fig. 7 was published within their article. The text ‘GFK1’ was erroneously included within the lower, left hand box of the figure. The authors would like to apologise for any inconvenience caused; the correct Fig. 7 is published, in full, below.



**Fig. 7.** Simplified model for the redox control of flowering. The left side of the figure shows the circadian clock-associated photoperiodic flowering pathway under long days. The *CIRCADIAN CLOCK ASSOCIATED 1* (*CCA1*) gene is expressed in the morning and represses the transcription of the *FLAVIN-BINDING KELCH-REPEAT F-BOX 1* (*FKF1*) gene. In the afternoon, when the proportion of blue light increases, *FKF1* degrades *CYCLING DOF FACTOR 1* (*CDF1*) and stabilizes *CONSTANS*. Together with *CONSTANS*, *FKF1* induces the expression of *FLOWERING LOCUS T* (*FT*) gene. The induction of flowering depends on the day length. *CCA1* also affects the transcriptional regulation of ROS-responsive genes as shown on the right side of the figure. Changing ROS levels affect *FKF1* expression independently of the clock regulation. ROS thus influence flower induction. The Asc-GSH cycle may also affect flowering time, most likely through the induction of *FLOWERING LOCUS C* (*FLC*) expression. Nitric oxide (NO) induces *FLC* expression and suppresses *CONSTANS* expression. The right side of the figure also shows the redox control of the stress-induced early flowering. Its central component is the redox-responsive *OXIDATIVE STRESS 2* (*OXS2*) transcription factor which regulates flowering due to its interaction with the *SUPPRESSOR OF OVEREXPRESSION OF CONSTANS 1* (*SOC1*) gene. Solid lines indicate *CCA1*-, *FKF1*- and *OXS2*-dependent mechanisms. Dotted lines indicate Asc-, GSH- and NO-dependent pathways.

DOI of original article: <http://dx.doi.org/10.1016/j.plantsci.2013.07.004>.

\* Corresponding author at: Agricultural Institute, Centre for Agricultural Research, Hungarian Academy of Sciences, Brunszvik u. 2., 2462 Martonvásár, Hungary. Tel.: +36 22 569501; fax: +36 22 569576.

E-mail address: [kocsy.gabor@agrar.mta.hu](mailto:kocsy.gabor@agrar.mta.hu) (G. Kocsy).

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<http://dx.doi.org/10.1016/j.plantsci.2013.10.008>