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Corrigendum

Corrigendum to "Redox control of plant growth and development" [Plant Sci. 211 (2013) 77–91]

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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.

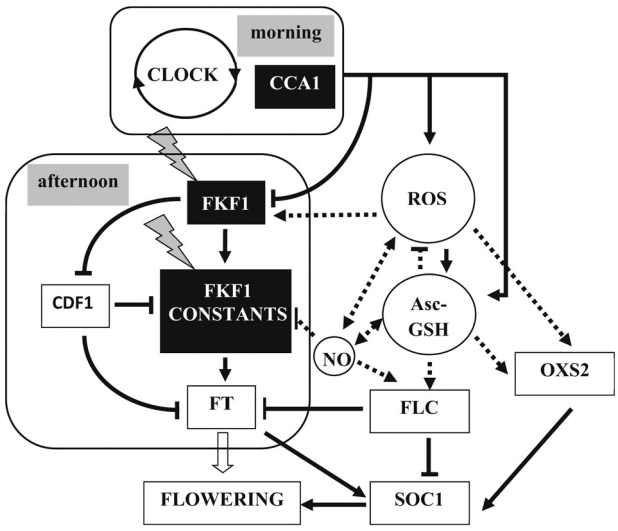

Fig. 7. Simplified model for the redox control of flowering. The left side of the figure shows the circadian clock-associated photoperiodic flowering pathway under long days. The *CIRCADIAN CLOCK ASSOCIATED1* (*CCA1*) gene is expressed in the morning and represses the transcription of the *FLAVIN-BINDING KELCH-REPEAT F-BOX1* (*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 STRESS2* (*OXS2*) transcription factor which regulates flowering due to its interaction with the *SUPPRESSOR OF OVEREXPRESSION OF CONSTANS1* (*SOC1*) gene. Solid lines indicate CCA1-, FKF1- and OXS2-dependent mechanisms. Dotted lines indicate Asc-, GSH- and NO-dependent pathways.

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