

Nam-Chon Paek

Department of Plant Science, Seoul National University, Seoul, Korea

Most plants regulate flowering time in response to seasonal changes in daylength, whose information is perceived by photoreceptors and transmitted to the circadian clock components controlling flowering-time genes. However, the molecular mechanisms underlying this process remain largely unknown. Here we show that COP1 mediates daylength signaling from CRY2 to ELF3 for the regulation of flowering time. Photoperiod-insensitive early flowering of *cop1* mutants is entirely due to circadian defect. COP1 interacts with and polyubiquitinates ELF3. COP1 E3 activity towards ELF3 decreases under the presence of C-termini of CRY2 (GUS-CCT2) in a concentration-dependent manner. Thus we propose that ELF3 polyubiquitination by COP1 are essential for ELF3 function to maintain the circadian rhythm for photoperiodic flowering.