Plants are able to modify their development patterns in response to some kinds of stress. In Arabidopsis, PCC1 has been identified as a positive regulator of floral transition in response to the strees generated by UV-C irradiation. The analysis of transgenic *pPCC1::GUS* plants show that *PCC1* is expressed during the very early development in stomata and the vasculature of cotiledons. In true leaves under formation GUS staining can be detected in their basal region, including vasculature, and it can also be detected on the complete surface of true completely formed leaves. Arabidopsis lines expressing RNAi contructions specific for *PCC1* (iPCC1) show reduced levels of *FT* and, consequently, a delayed flowering time. The mechanism by which PCC1 could regulate flowering transition seems to be related to light signal transmition. Concomitantly, iPCC1 plants show partial scotomorphogenic phenotypes under the different qualities of light assayed, in a GA accumulation and signaling independent way. The diferential transcriptome of iPCC1 plants *versus* wild type plants show a clear role of PCC1 in defense-related proceses. Accordingly, we have observed that iPCC1 plants are more susceptible to the hemi-biotrof oomycete *Phytophtora brassicae* and more resistant to the necrotrof fungus *Botrytis cinerea*. Moreover, iPCC1 lines showed an upregulation of ABA-responsive genes, and a hypersensitivity to this phytohormone for the different phenotypes analyzed. Finally, among the altered genes in iPCC1 lines we have observed an overrepresentation of genes implied in lipid metabolism and transport. *PCC1* lost of function entails a reduction of 70% in the levels of phosphatidylinositol, and, in a lesser extent, of some other class of polar lipids such as phosphatidylserine and phosphatidylcoline. Moreover, the analysis of fatty acid composition of each type of polar lipids shows a higher insaturation degree of their lateral chains, mostly in phosphatidylserine and phosphatidyinositol. PCC1 associates the the plasma membrane by its carboxi-terminal region, which is as well responsible of the homodimer formation. Although it is still unclear the mechanisms by which PCC1 regulates such a different molecular, as with pathogen response and flowering transition, we have observed that PCC1 interacts with CSN5 subunit of the signalosome complex (CSN), what suggest that PCC1 could act as a CSN regulator, and ultimately as a regulator of protein ubiquitination.