

***FLO/LFY* homologue in *Physcomitrella patens* is necessary for sporophyte formation**

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FLORICAULA/LEAFY (*FLO/LFY*) homologous genes have been isolated from angiosperms, gymnosperms, ferns, fern allies, mosses and liverworts. *Arabidopsis thaliana LFY* positively regulates the expression of some MADS-box genes that decide floral organ identity. This function is conserved in gymnosperm *FLO/LFY* homologues, while the fern, *Ceratopteris richardii FLO/LFY* homologues are not likely to induce MADS-box genes, because the expression patterns of its *FLO/LFY* homologues and MADS-box gene homologues are different. Further approach to the function of its *FLO/LFY* homologues is difficult to make because there is no reliable transformation system. To assess the original function of *FLO/LFY* homologues, *Physcomitrella patens* was used to characterize the genes using its feasible gene targeting technique.

We isolated two *FLO/LFY* homologous genes, *PpLFY1* and *PpLFY2*, from *Physcomitrella patens*. These genes had about 90% identical nucleotide sequences and showed very similar expression patterns. Both *PpLFY1*-GUS and *PpLFY2*-GUS proteins were detected strongly at shoot apices, in young leaves, axillary buds, and archegonia. In the case of expression in archegonium, GUS expression was observed in an entire archegonium at an early stage of development and gradually limited to the venter as it developed.

We obtained six lines of *PpLFY1/PpLFY2* double mutant by retransforming *PpLFY2* single mutant; the *pplfy2* normally developed until sporophytes were formed and had no particular phenotypic difference to the wild type. Double mutants also had no morphological difference from the wild type in protonemata and gametophores and formed normal antheridia and archegonia. However, they never formed sporophytes. Some mature archegonia of the double mutants had a brown plug in the canal, suggesting that fertilization had occurred. Because we could not observe further developed archegonium whose venter enlarged more, we infer that the double mutants arrest development during embryogenesis. Results of further observations including phenotype of *PpLFY1* single mutant will also be reported.