2017

The hetZ Gene Regulates Heterocyst Formation in Anabaena sp. strain PCC 7120

Patrick Videau
Dakota State University

Vaille Swenson
Dakota State University

Michael Gaylor
Dakota State University, michael.gaylor@dsu.edu

S M. O'Hanlon
Dakota State University

L M. Cozy
Dakota State University

THE *HETZ* GENE REGULATES HETEROCYST FORMATION IN *ANABAENA* SP. STRAIN PCC 7120

P. Videau[1]*, V. A. Swenson[1], M. O. Gaylor[1], S. M. O'Hanlon[2], and L. M. Cozy[3]
[1]College of Arts and Sciences
Dakota State University
Madison, SD 57042
[2]School of Psychological Science
Oregon State University
Corvallis, OR 97331
[3]Department of Biology
Illinois Wesleyan University
Bloomington, IL 61701
*Corresponding author email: patrick.videau@dsu.edu

ABSTRACT

To form a complex multicellular organism, stem cells must differentiate into each cell/tissue type along proper spatiotemporal scales. The study of differentiation and organismal development has historically been conducted in prokaryotes due to their genetic and morphological simplicity. *Anabaena* sp. strain PCC 7120 is a multicellular filamentous cyanobacterium that differentiates a morphologically distinct secondary cell type, the heterocyst, in response to a lack of combined environmental nitrogen. Heterocysts are regularly spaced along filaments and fix atmospheric dinitrogen to maintain organismal viability in its absence. Previous work suggested that the *hetZ* gene is involved in heterocyst differentiation, but the insertional mutants created produced inconsistent phenotypes, so a specific role was not assigned. In this work, a clean *hetZ* mutant incapable of heterocyst differentiation was generated and the mutation was complemented with the reintroduction of *hetZ* alone. Overexpression of *hetZ* bypassed a mutation of *hetR*, the master regulator of heterocyst differentiation that controls biological pattern formation, but not a mutation of *hetP*, a regulator of commitment to a differentiated cell fate, which places *hetZ* roughly between these processes. A protein-protein interaction study showed that HetZ interacts with both HetR and itself. Assessment of transcriptional fusions between the *hetZ*, *hetR*, *hetP*, and *patS* (an inhibitor of HetR) promoter regions and GFP, and overexpression of HetR in a *hetZ* mutant resulted in the differentiation of heterocyst-like cells, together indicated that HetZ may act in concert with HetR as an early regulator of development. Taken together, these data describe a non-linear pathway of regulation leading to heterocyst development governed by both HetR and HetZ.