Lewis University
STEM Undergraduate Research Experience (S.U.R.E.) 2019
Faculty Mentor – Project Application

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Research Project Title: Evaluation of Transcript Levels in the Presence of Ectopically Expressed Wild-Type or Mutated Cyclin D3.

Research has focused on identifying target molecules that, when disrupted, selectively compromise the viability of cancer cells. One putative target is cyclin D3, which is mutated in a variety of cancers. Cyclin D3, along with its binding partner CDK4/6, is known to play an important role in controlling cell proliferation; yet, more recent work has implicated cyclin D3 in regulating gene expression, and it seems to help cells know when to use only one of the two alleles for a gene. Thus, the role of cyclin D3 in cancer may not be limited to abnormal cell division. The mechanism by which cyclin D3 changes the genetic profile of the cell remains unknown, but may hinge upon dimerization of two cyclin D3 protein molecules. To understand the dual functionality of this protein and how it is altered by the mutations observed in various cancers, site-directed mutagenesis has been used to generate mutant clones of the human *Ccnd3* gene. Expression of these gene constructs in an immature B cell lymphoma cell line creates a model system in which the altered cyclin D3 protein can be expressed and, due to changes in protein folding and/or protein-protein interactions, potentially impact cellular phenotypes. In particular, this project will evaluate changes in the transcriptional profile of a cell upon ectopic expression of normal (wild-type) cyclin D3 versus mutated forms of the protein. Ultimately, these data will help explain the currently underappreciated functionality of cyclin D3 and how it may contribute to the cancerous state.