

AN ABSTRACT OF THE THESIS OF

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Abstract approved:

Christine Kelly

Fanconi's Anemia (FA) is an autosomal recessive chromosomal instability disorder. The symptoms include congenital abnormalities, defective hematopoiesis (formation of cells of the blood), and high risk of developing acute myeloid leukemia and solid tumors. Fanconi researchers have long been puzzled by how Fanconi Anemia (FA) cells, which are known to be more likely to perish under harsh conditions, somehow morph into cancer cells at an alarming rate since abnormalities usually cause cells to initiate apoptosis (programmed cell death). Some findings by Dr. Bagby's lab in murine ovarian tissue indicated that Fanconi cells were more sensitive to cytokines than wild-type cells of that tissue type. Therefore, preliminary studies exposing other cell types to cytokines were deemed to be useful. Studies comparing the proliferation of wild-type and Fanconi cells after TNF-alpha, IFN-gamma, and FAS ligand exposure were conducted. These results suggest that FAS has an effect on MEF Fanconi cells and that Fanconi cells are more likely to induce apoptosis when exposed to cytokines than wild-type cells.

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The Effect of Cytokines on
Wild-type and Fanconi Mouse Embryonic Fibroblasts

by

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I understand that my project will become part of the permanent collection of Oregon State University, University Honors College. My signature below authorizes release of my project to any reader upon request.

Katherine L. Wagner, Author

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