

Cleveland State University

EngagedScholarship@CSU

Undergraduate Research Posters 2014

Undergraduate Research Posters

9-4-2014


Activation of DNA Damage Checkpoint Pathways During Skeletal Myoblast Differentiation and Apoptosis

Mofetoluwa Oluwasanmi
Cleveland State University

Greg Kliment
Cleveland State University

Crystal M. Weyman
Cleveland State University, c.weyman@csuohio.edu

Follow this and additional works at: https://engagedscholarship.csuohio.edu/u_poster_2014

 Part of the [Molecular Biology Commons](#)

[How does access to this work benefit you? Let us know!](#)

Recommended Citation

Oluwasanmi, Mofetoluwa; Kliment, Greg; and Weyman, Crystal M., "Activation of DNA Damage Checkpoint Pathways During Skeletal Myoblast Differentiation and Apoptosis" (2014). *Undergraduate Research Posters 2014*. 28.

https://engagedscholarship.csuohio.edu/u_poster_2014/28

This Article is brought to you for free and open access by the Undergraduate Research Posters at EngagedScholarship@CSU. It has been accepted for inclusion in Undergraduate Research Posters 2014 by an authorized administrator of EngagedScholarship@CSU. For more information, please contact library.es@csuohio.edu.



Activation of DNA damage checkpoint pathways during skeletal myoblast differentiation and apoptosis

College of Sciences and Health Professions

Student Researchers: Mofetoluwa Oluwasanmi* and Greg Kliment

Faculty Advisor: Crystal M. Weyman

Abstract

A subset of skeletal myoblasts undergo apoptosis rather than differentiation when cultured in differentiation media (DM: absence of growth factors). While the muscle regulatory transcription factor MyoD is known to control the process of differentiation, our lab has recently discovered that MyoD is also controlling the apoptotic process in response to culture in DM by direct up-regulation of the pro-apoptotic Bcl2 family member PUMA. We similarly discovered that MyoD plays a role in the increased expression of PUMA and apoptosis in response to the DNA damaging agent, etoposide. This led to the hypothesis that culture in DM may lead to stalled replication forks during DNA synthesis that are “recognized” as DNA damage. We are testing our hypothesis by determining if culture in DM results in the activation of pathways known to respond to DNA damage. We have determined that p38, p53, and c-abl are all up-regulated in response to culture in DM. Next, we will determine the significance of MyoD to the increased expression of these molecules.

**McNair Scholar*