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Poster Submissions**Poster Title**

Analysis of Nuclear Architectural Changes Throughout Apoptotic vs. Non-Apoptotic Cell Death

Authors and their Affiliation

Zackary Hintze, Alla Yalonetskaya, Elizabeth Tanner, Margaret Barkett, Jeanne Peterson, Kimberly McCall: Boston University

Please describe the extent of your work in this research

I have completed all experiments in mid-oogenesis regarding the lamin breakdown presented in this work.

Abstract Submission

- [GSI-abstract-ZHintze.docx](#)

Would you like your abstract to be considered for an oral presentation (students and post docs only)?

No

During programmed cell death, the breakdown of the nucleus is a very important step. Drastic changes in the nuclear architecture occur during both apoptotic and non-apoptotic death as DNA becomes condensed and the nucleus is destroyed. However, little is known about the mechanistic differences underlying nuclear breakdown in different death modalities. Thus, we became interested in the scaffolding of the nucleus itself. The nuclear lamina, comprised of filamentous lamin proteins, plays an integral role in providing structure to the nucleus, and crippling laminopathies caused by defects in these critical proteins highlight their importance in proper functioning of the cell as a whole. To better understand the differences between apoptotic and non-apoptotic nuclear degradation, we focused on the changes in the lamina throughout both apoptotic and non-apoptotic death, using the *Drosophila* ovary as a model. *Drosophila* oogenesis has been categorized into 14 distinct stages. Each developing egg chamber consists of a germline syncytium of 15 nurse cells and one oocyte surrounded by an epithelial layer of somatic follicle cells. The 15 nurse cells undergo a developmental non-apoptotic death during the late stages of oogenesis. Additionally, we can induce apoptosis in mid-stage egg chambers using protein starvation. This allows us to perform an *in vivo* comparison of apoptotic and non-apoptotic nuclear breakdown within the same tissue. Using immunohistochemistry staining for B-type lamins, we have found that while lamins persist late into developmental death, they are degraded rapidly during apoptotic death. Double staining against the Dcp-1caspase shows that lamins begin to degrade immediately with the activation of caspases in wild-type flies. Overexpression of wild-type lamin protein still shows early degradation of the lamina with little effect on the progression of apoptosis. In contrast, overexpressing cleavage-resistant lamin results in persisting nuclear debris even into the final phases of apoptosis. Further work will investigate the dynamics of acidification and DNA fragmentation as well as additional aspects of the nuclear envelope's degradation.