# CHRD 2024: Abstract Submission Form

**Presenter Name**
Carthy Caswell

**Presenter Status**
Undergraduate Students

**Role in the project**
Design
Perform Experiments
Analyze Data
Write Abstract

**Research Category**
Basic Science

**Title**
The Proteasome Inhibitor MG132 Restores Emg1 Protein Levels and Partially Rescues Growth Defects in a Yeast Model of Bowen-Conradi Syndrome

**Background**
Bowen-Conradi Syndrome is a rare genetic disorder which affects 1 in 355 live births in the Hutterites. It is characterized by severe developmental delays and a failure to thrive. BCS is due to a D86G variant in the EMG1 protein, a member of the SSU processome and a requirement for ribosome assembly. The BCS variant causes a decrease in Emg1 protein stability which likely targets it for degradation via the ubiquitin proteasome pathway.

**Objective**
This study examines whether the use of MG132, a proteasome inhibitor, could increase EMG1 protein levels and partially rescue the growth defect caused by the D86G variant. We also want to determine if the BCS EMG1 variant is ubiquitinated (ub) at a higher level compared to WT.

**Methods**
A yeast model containing the analogous BCS variant Emg1 protein was qualitatively analyzed for Emg1 abundance with and without treatment of MG132 to determine whether Emg1 abundance could be increased. Next, the mechanism by which Emg1 protein abundance increases in the presence of MG132 was analyzed via ubiquitin immunoprecipitation. Finally, a growth analysis was conducted to examine if the growth defects caused by the BCS Emg1 protein could be partially rescued via inhibition of the ub-proteasome pathway.

**Results**
With MG132, both WT and BCS variant Emg1 protein increased. We also determined that the mechanism that causes decreased abundance of Emg1 is in fact the ubiquitin proteasome pathway. Finally, growth analysis shows that the BCS Emg1 variant grown in the presence of MG132 grew at a higher rate than without the treatment.

**Conclusion**
The BCS variant Emg1 protein is targeted for degradation via the ubiquitin proteasome pathway. By using MG132, the BCS variant Emg1 protein levels are increased and the the growth defects are partially rescued. Examining the abundance of pre and mature rRNA is the next step in verifying that the growth defects can be rescued.

**Do you have a table/figure to upload?**
No

## Authors


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