

Rowan University

Rowan Digital Works

School of Osteopathic Medicine Faculty
Scholarship

School of Osteopathic Medicine

12-15-2017


Translocation of Cyclin C During Oxidative Stress Is Regulated by Interactions with Multiple Trafficking Proteins

Daniel G J Smethurst
Rowan University

Katrina F Cooper
Rowan University School of Osteopathic Medicine

Randy Strich
Rowan University School of Osteopathic Medicine

Follow this and additional works at: https://rdw.rowan.edu/som_facpub

 Part of the [Biological Phenomena](#), [Cell Phenomena](#), and [Immunity Commons](#), [Cell Biology Commons](#), [Fungi Commons](#), [Genetic Processes Commons](#), [Microbiology Commons](#), and the [Molecular Biology Commons](#)

Let us know how access to this document benefits you - share your thoughts on our [feedback form](#).

Recommended Citation

Smethurst, Daniel G J; Cooper, Katrina F; and Strich, Randy, "Translocation of Cyclin C During Oxidative Stress Is Regulated by Interactions with Multiple Trafficking Proteins" (2017). *School of Osteopathic Medicine Faculty Scholarship*. 126.
https://rdw.rowan.edu/som_facpub/126

This Poster is brought to you for free and open access by the School of Osteopathic Medicine at Rowan Digital Works. It has been accepted for inclusion in School of Osteopathic Medicine Faculty Scholarship by an authorized administrator of Rowan Digital Works. For more information, please contact brush@rowan.edu.

P3264

Board Number: B552

Translocation of cyclin C during oxidative stress is regulated by interactions with multiple trafficking proteins.

D.G. Smethurst¹, K.F. Cooper¹, R. Strich¹;

¹ School of Osteopathic Medicine, Rowan University, Stratford, NJ

Eukaryotic cells take cues from their environment and interpret them to enact a response. External stresses can produce a decision between adjusting to behaviors which promote surviving the stress, or enacting a cell death program. The decision to undergo programmed cell death (PCD) is controlled by a complex interaction between nuclear and mitochondrial signals. The mitochondria are highly dynamic organelles that constantly undergo fission and fusion. However, a dramatic shift in mitochondrial morphology toward fission occurs early in the PCD process. We have identified the transcription factor cyclin C as the biochemical trigger for stress-induced mitochondrial hyper-fragmentation in yeast (Cooper et al., 2014 Dev. Cell) and mammalian (Wang et al., 2015, MCB) cells. In response to PCD stimuli such as oxidative stress, cyclin C is released from the nucleus and associates with the mitochondrial fission machinery. Loss of cyclin C prevents mitochondrial fission while its ectopic introduction into the cytoplasm induces complete fragmentation in the absence of stress. Many of the details of the control of cyclin C localization within the cell have not been elucidated.

To initially investigate the regulation of cyclin C translocation events in *S. cerevisiae*, a two hybrid screen was used. We have identified multiple conserved trafficking proteins which interact with cyclin C. These

Tuesday-345

POSTER PRESENTATIONS-TUESDAY

include Gea1/2 and multiple components of the TRAPPII complex, both of which act as guanosine exchange factors (GEFs) for small GTPase regulators of vesicle traffic (Arf1/2 and Ypt31/32 respectively). Arf1 and its homolog Arf2 also mediate mitochondrial dynamics and contact with the ER. Gea2 interaction with cyclin C was confirmed by co-immunoprecipitation, and fluorescence microscopy data also indicates that the two proteins can colocalise. The use of conditional mutants has revealed that upon loss of functional Gea1/2 or Arf1/2, cyclin C is translocated to the cytosol without stress. Following from this we propose a model in which the protein trafficking machinery acts to keep cyclin C in the nucleus during healthy cell maintenance.