Abstract Submission Guidelines

The following are instructions for completing and formatting your abstract submission.

Abstract Title - Capitalize only the first word of the title and proper nouns. Allowable Characters: Italics, Greek symbols/math operators, subscript and superscript. Character limit = 110

Authors - Separate the author's names with commas; do not include academic degrees or specialty certification.
Please **only** underline the presenting author's name. Allowable Characters: Italics, Greek symbols*/math operators, subscript and superscript. Character limit = 220

Affiliations - Enter the authors' professional affiliations at the time of the study. Affiliations should be listed in the order in which the authors' names are given in the byline. For authors affiliated with a university, the department, school or college, university, city, and state should be listed (in that order). If authors do not all have the same affiliation, the authors' last names should be given in parentheses to indicate affiliation. Allowable Characters: Italics, Greek symbols*/math operators, subscript and superscript. Character limit = 330

Abstract Content - Enter the content of your abstract. Do not include blank lines or paragraph breaks. Allowable Characters: Italics, Greek symbols*/math operators, subscript and superscript. Character limit = 1750

Research Grant - Enter information on any grants or the source of any third-party funding used to conduct this research (ask your mentor). Alternatively, enter "None" if there was no third-party funding for the research or "Unknown" if you do not know the source of research funding. Character limit = 200

Student Support - Enter information on the source of your student summer research stipend. Alternatively, enter "None" if you did not receive a stipend or "Unknown" if you do not know the source of your research stipend. Character limit = 95

Field of Research - Select your field of research from the drop-down box provided.

**Note: When entering your content, only use the symbols available from the Abstract Submissions page. Use of other symbols may be lost.* (I.e. α, β, γ, δ, ε, κ, λ, μ, ρ, χ, Δ, Θ, Σ)

Sample Abstract:

Please review the abstract below for an example of how your submission might look in the program booklet.

P21 Up regulation during *Campylobacter jejuni* CDT-induced epithelial H407 cell senescence

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Cytolethal distending toxin (CDT) is a genotoxin produced by *Campylobacter jejuni* (CjejCDT), a major cause of food- and water-borne zoonotic intestinal illness worldwide. CDT is a novel class of bacterial toxin with DNase activity that translocates to the nucleus of eukaryotic cells. There, it exerts genotoxic damage characterized by activation of an ATM-dependent DNA damage response (DDR) resulting in arrest of the cell cycle and apoptotic death. Previous studies in our laboratory indicate that eukaryotic cells intoxicated with sub-lethal concentrations

of CjejCDT undergo irreversible arrest of the cell cycle together with cytoplasmic accumulation of β-galactosidase and secretion of proinflammatory cytokines consistent with premature senescence, a protective mechanism against cancer progression. On the basis of these observations, we hypothesized that a key upstream activator of premature senescence, the cyclin dependent kinase inhibitor p21Cip1/Waf1 (p21) regulates the decision of intoxicated cell to undergo senescence rather than apoptosis. While H407 cells incubated with control medium or medium containing whole-cell lysate (WCL) of a *C. jejuni* mutant strain with a disrupted CDT gene (ΔCDT) continued to grow over 7 days, cells incubated with sublethal concentrations of CjejCDT obtained from the isogenic wild-type (WT) and ΔCDT strain complemented with a full length CDT operon showed complete growth arrest and a DDR characterized by ATM-dependent phosphorylation of histone H2AX (γ-H2AX). Sequential confocal microscopy and Western blot analysis of CjejCDT-intoxicated H407 cells correlated development of nuclear DNA lesions with up-regulation of p21 and loss of cytoskeletal integrity. These findings provide a molecular basis to explain the mechanism of CDT-induced premature eukaryotic cell senescence.

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