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DEFENSE OF THE DOCTORAL

DISSERTATION

"Telomere Dysfunction-Induced Senescence in Placentas and Amniochorionic Membranes"

by

Mark Simpson

M.D./Ph.D. Program

B.S. 2013, Cornell University, Ithaca, NY
M. Eng. 2014, Cornell University, Ithaca, NY

Thesis Advisor:
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Tuesday, May 26th, 2020
1:00 P.M.
WebEx

Abstract

Cellular senescence is an irreversible proliferative arrest that is evoked by both cell intrinsic and extrinsic stresses. Recent studies have provided evidence that cellular senescence is involved in development of placentas (Pl) and amniochorionic membranes (ACm). Supporting this are data suggesting that senescent cells are abundant in human Pl and ACm at term. Similarly, deregulated activation of cellular senescence has been implicated in a number of gestational complications such as intrauterine growth restriction and premature birth. As the causes for cellular senescence in PL and ACm are currently unknown, therapies that would reduce burden of such complications are challenging to develop. We propose that a primary cause of cellular senescence in these tissues is telomere dysfunction, and cells in telomere-dysfunction induced senescence (TDIS) drive paracrine senescence through secreted factors. Our data show that: a) a gestational-age-associated increase of cells displaying hallmarks of TDIS in amniochorionic membranes and placenta, and b) TDIS cells can amplify senescence responses to neighboring cells via secretion of paracrine signaling molecules. Overall, our data reveal that telomere dysfunction may be a major contributor to cellular senescence in placental tissue.

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