Memorial Sloan Kettering Cancer Center

John Callegari

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I study the molecular processes that allow cells to replicate areas of the genome that contain DNA damage. These processes, referred to as DNA damage tolerance mechanisms, are required for cell proliferation and generate many of the mutations that contribute to genetic variation and cancer development.

Our approach is to treat cells as a physicist might, introducing defined perturbations and accurately measuring system behavior using live-cell imaging and other quantitative methods. The goal of the research is to first understand the basic logic of the DNA damage response, and then develop predictive quantitative models of the molecular processes that govern the dynamics of DNA replication, DNA damage tolerance, and cell cycle progression.

Publications

Callegari AJ, Kelly TJ, Coordination of DNA damage tolerance mechanisms with cell cycle progression in fission yeast. *Cell Cycle*. 2016 15(2):261-273

see also: News & Views, *Cell Cycle* 15(3):314-315

Callegari AJ, Does transcription-associated DNA damage limit lifespan? *DNA Repair*. 2016 May; (41):1-7

Callegari AJ, Pneumann A, Clark E, Kelly TJ, Postreplication gaps at UV lesions are signals for checkpoint activation. *PNAS*. 2010 May 4; 107(18)8219-8224.

Callegari AJ, Kelly TJ, Shedding light on the DNA damage checkpoint. *Cell Cycle*. 2007. Mar 15; 6(6):660-6.

Callegari AJ, Kelly TJ, UV-irradiation induces a postreplication DNA damage checkpoint. *PNAS*. 2006 Oct 24; 103(43)15877-15882.

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