

The molecular architecture of cell cycle arrest

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Abstract

The cellular decision governing the transition between proliferative and arrested states is crucial to the development and function of every tissue. While the molecular mechanisms that regulate the proliferative cell cycle are well established, we know comparatively little about what happens to cells as they diverge into cell cycle arrest. We performed hyperplexed imaging of 47 cell cycle effectors to obtain a map of the molecular architecture that governs cell cycle exit and progression into reversible ("quiescent") and irreversible ("senescent") arrest states. Using this map, we found multiple points of divergence from the proliferative cell cycle; identified stress-specific states of arrest; and resolved the molecular mechanisms governing these fate decisions, which we validated by single-cell, time-lapse imaging. Notably, we found that cells can exit into senescence from either G1 or G2; however, both subpopulations converge onto a single senescent state with a G1-like molecular signature. Cells can escape from this "irreversible" arrest state through the upregulation of G1 cyclins. This map provides a more comprehensive understanding of the overall organization of cell proliferation and arrest.

Keywords cell cycle; proliferation; quiescence; senescence; single-cell

Subject Category Cell Cycle

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Introduction

The decision of when and where to trigger cell division is fundamental to nearly all aspects of development and physiology. At the level of the individual cell, the molecular basis of the proliferation/arrest decision is embedded within a highly interconnected and dynamic network of cell cycle regulators. Progression through the proliferative phases of the cell cycle (G1/S/G2/M) is governed by a series of biochemical reactions that are coordinated in time and

space to ensure the successful replication of DNA and its division into two daughter cells. In addition to these four proliferative phases, cells may also "exit" the proliferative cell cycle into a state of cell cycle arrest, often referred to as G0. While arrested, cells still perform many essential cellular functions including metabolism, secretion, transcription, and translation. However, as long as they remain in the G0 state, arrested cells neither synthesize DNA nor undergo cell division. This five-state model has become the canonical cell cycle model found in most textbooks (Morgan, 2007) and the current literature (Spencer et al, 2013; Overton et al, 2014; Manesal & Cheeseman, 2020) and has shaped our thinking about the cell cycle for over 70 years (Howard & Pelc, 1953; Cameron & Greulich, 1963; Smith & Martin, 1973).

While the mechanisms that govern progression through the proliferative cell cycle have been studied extensively, we know comparatively little about what happens to cells after they exit the proliferative cell cycle. We know that cells may exit the cell cycle in response to various biochemical (e.g., DNA damage and oxidative stress) or environmental insults (e.g., lack of mitogens and high local cell density) triggered by different molecular mechanisms (Sages & Laporte, 2019; Manesal & Cheeseman, 2020). After exiting the cell cycle, cells may progress into deeper states of reversible ("quiescent") cell cycle arrest (Owen et al, 1989; Kwon et al, 2017; Wang et al, 2017), and in some cases can transition into an irreversible ("senescent") state of arrest (Marthandan et al, 2014; Sousa-Victor et al, 2014; Fujimaki et al, 2019; Fujimaki & Yao, 2020). Clearly, cell cycle arrest is far from a single, static molecular state (Collier et al, 2006; Klosinska et al, 2011; Sun & Basma, 2017), yet a systematic characterization of when and how cells arrest remains lacking.

In this study, we used a combination of hyperplexed, single-cell imaging and manifold learning to map the molecular architecture of cell cycle arrest. Previously, we used this approach to map the structure of the proliferative cell cycle in unperturbed, nontransformed retinal pigment epithelial (RPE) cells (Stallaert et al, 2022). Building upon this work, here, we exposed asynchronous RPE cells to three distinct stressors—hypomutagenic, replication, and oxidative—known to induce cell cycle arrest. For each stress, we identify the

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Inou pioneering cell biophysicist and winner of the 2003 International Prize for Biology The articles cover the discovery and elucidate the behavior in living cells of the dynamic molecular filaments which organize the cell and play a central role in cell division Other articles report on the development of microscopes especially those using polarized light and digital image enhancement which make possible studies of the ever changing molecular architecture directly in living cells This book also contains many high quality photo micrographs as well as an appended DVD with an extensive collection of video movies of active living cells After training in Tokyo and at Princeton University Dr Inou has held teaching positions at the University of Washington Tokyo Metropolitan University University of Rochester Dartmouth Medical School and University of Pennsylvania He is a member of the U S National Academy of Sciences and currently holds the title of Distinguished Scientist at the Marine Biological Laboratory in Woods Hole Massachusetts Visions of Cell Biology Karl S. Matlin, Jane Maienschein, Manfred D. Laubichler, 2018-01-19 Although modern cell biology is often considered to have arisen following World War II in tandem with certain technological and methodological advances in particular the electron microscope and cell fractionation its origins actually date to the 1830s and the development of cytology the scientific study of cells By 1924 with the publication of Edmund Vincent Cowdry's General Cytology the discipline had stretched beyond the bounds of purely microscopic observation to include the chemical physical and genetic analysis of cells Inspired by Cowdry's classic watershed

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