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Opinion Article

The Impact of ROS on Yeast Growth at Low Temperatures

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DESCRIPTION

Reactive Oxygen Species (ROS) have a major impact on yeast development at subfreezing temperatures by controlling cell cycle dynamics. Increased ROS levels prolong the G1 phase, delaying the transition to the replicative S-G2-M phase, and slowing cell division. Higher ROS levels cause yeast cells to become excessively big and possibly burst, emphasizing the necessity of keeping ROS below a key threshold for optimum growth. This equilibrium between ROS and gene expression determines the fastest and slowest feasible cell doubling periods, exposing temperature restrictions on yeast replication and providing insights into how life survives under severe cold circumstances.

The ability of life to advance arbitrarily slowly may signal significant challenges for living systems in preserving thermal disequilibrium. They show that Reactive Oxygen Species (ROS) and a global gene expression speed quantitatively control the pace of life for budding yeast at subfreezing temperatures, imposing temperature-dependent speed restrictions on the lowest and longest possible cell doubling durations. An increase in the quantity of ROS in cells shortens the time it takes for cells to double by extending the time they spend in the G1 phase, which precedes the SG2-M phase. The rate at which genes are expressed limits how rapidly ROS are reduced in cells and determines the fastest doubling time. Cells require ROS concentrations below their threshold in order.

Cells with an enough level of ROS remain in the G1 phase, grow abnormally large, and then burst. As a result, yeast's replicative life cannot be arbitrarily slow at any given temperature, and cells with the lowest ROS levels replicate the fastest. Thermal slowing of other species' lives may be limited by underlying constraints. How numerous biological events jointly determine the rate of life is an important question. There is the well-known but ill-defined belief that life progresses toward death at a certain rate. Even for a single cell, precisely characterizing and quantifying this rate, as well as determining how each intracellular event effects it, is a considerable challenge. Solving this conceptual dilemma would increase their understanding of how living cells maintain themselves.

Understanding species that cannot alter their internal temperatures, such as bacteria, plants, and cold-blooded mammals, which often survive in frigid environments, is also critical. Researchers have found specific genes, stress reactions, and epigenetic pathways that aid a cell's ability to survive under cold circumstances. They do not yet understand how a complex network of interconnected processes interact to control and possibly constrain a cell's capacity to advance throughout its life at extremely low temperatures.

The revelation that yeast cells work together to survive in subfreezing temperatures sparked research. They release and accumulate glutathione, an antioxidant, to counteract potentially hazardous ROS, which are a primary cause of yeast death in subfreezing temperatures. They were able to determine how the amount of intracellular ROS impacts yeast's ability to proliferate, prosper, and survive at extremely low temperatures by continuously studying individual cells for weeks to months and employing single-cell analysis. They found that the cause of all these ROS-induced effects is the same: ROS prolongs the eukaryotic cell cycle's G1 (growth) phase, causing cells to develop continuously while delaying entry into the S-G2-M (replicative) phase.

As a result, yeast's life can be slowed to any degree. However, when the temperature approaches freezing, they discovered that, while such ultra-slow self-replication is theoretically possible, it becomes extremely rare. These findings collectively suggest quantitative limits for the dynamics of self-replication at exceptionally low temperatures. Without understanding the specifics, one can argue that a cell cannot divide at an arbitrary quick rate at any temperature. However, it is unclear if a cell can perpetually delay the conclusion of its cell cycle. The authors determined the high-and low-speed restrictions at each extremely cold temperature and explained how the protein synthesis rate and ROS combine to produce these limits.

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