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Opinion Article

Comprehensive Study on Reactive Oxygen Species at Frigid Temperatures

John Burg*

Department of Systems Biology, University of Massachusetts Chan Medical School, Worcester, USA

DESCRIPTION

The ability of life to advance arbitrarily slowly could indicate key difficulties for living systems in maintaining thermal disequilibrium. They demonstrate that Reactive Oxygen Species (ROS) and a global gene expression speed quantitatively dictate the pace of life for budding yeast at subfreezing temperatures and impose temperature dependent speed constraints the shortest and longest conceivable cell doubling times. By lengthening the time that cells spend in the G1 phase, which comes before the S-G2-M phase, an increase in the concentration of ROS in the cells shortens the time it takes for cells to double. The rate at which genes are expressed limits how quickly ROS are reduced in cells and establishes the quickest doubling time. Cells require ROS concentrations below their threshold in order to reproduce.

Cells that have an enough amount of ROS stay in the G1 phase, grow uncontrollably large, and then burst. Therefore, yeast's replicative life cannot proceed arbitrarily slowly at a given temperature, and cells with the lowest ROS-levels duplicate the fastest. The thermal slowing of the lifetimes of other species may be constrained by underlying obstacles. How innumerable biological events collectively control the rate of life is a crucial question. There is the well-known, ill-defined idea that life moves towards death at a certain rate. But even for one cell, precisely describing and quantifying this rate, as well as figuring out how each intracellular event influences it, presents a significant difficulty. Physics-wise, solving this conceptual conundrum will improve their comprehension of how living cells maintain themselves outside of thermal equilibrium.

Understanding creatures that can't control their internal temperatures, such bacteria, plants, and cold-blooded animals which typically live in chilly environments is similarly important. Researchers have identified particular genes, stress reactions, and epigenetic pathways that support a cell's ability to survive under chilly conditions. They do not, however, currently understand how a complex web of interrelated processes interact to control and maybe restrain a cell's ability to advance in its life at extremely low temperatures.

Research began with the discovery that yeast cells cooperate with one another to survive in subfreezing conditions. They secrete and accumulate glutathione, an antioxidant, to neutralize potentially harmful ROS, which are a major cause of death for yeast in subfreezing conditions. By continually observing individual cells for weeks to months and using single-cell-level analysis, they were able to identify how the quantity of intracellular ROS affects yeast's capacity to multiply, thrive, and survive at extremely low temperatures. They discovered that the cause of all these ROS-induced effects is the same: ROS prolong the eukaryotic cell cycle's G1 (growth) phase, causing cells to grow continually while preventing the entry into the S-G2-M (replicative) phase.

As a result, yeast's life can be slowed down to any amount. But when the temperature gets closer to freezing, they discovered that while such ultra-slow self-replication is still theoretically feasible, it becomes incredibly rare. These findings collectively point to quantitative boundaries for the dynamics of self-replication at extremely low temperatures.

One can assert that a cell cannot divide at an arbitrary rapid rate for any temperature without knowing any specifics. However, it is not immediately clear that a cell cannot delay the end of the cell cycle indefinitely. Here, they identified the high- and low-speed limitations at each extremely cold temperature and explained how the protein synthesis rate and ROS interact to produce these limits.

Correspondence to: John Burg, Department of Systems Biology, University of Massachusetts Chan Medical School, Worcester, USA, E-mail: johnburg@mas.edu

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