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Commentary

Nucleotides imbalance as a Factor Distingishing Cell Expansion from Cell Proliferation

Sone Clia*

Department of Cell Science, University of Ibadan, Ibadan, Nigeria

DESCRIPTION

Over the course of the cell cycle, the majority of proliferating cells double each component of their mass; as a result, metabolic requirements change to support biosynthetic processes unique to various cell cycle phases. For RNA synthesis and to ensure correct and efficient DNA replication during the S phase, proliferating cells require sufficient quantities of each nucleotide species, which must be acquired. To produce biomass, ribosomal RNA and messenger RNA must be synthesized, which requires nucleotides. Since RNA makes up the great majority of the nucleic acids in cells, the creation of RNA both directly and indirectly contributes to biomass. Therefore, nucleotide acquisition is crucial for biomass synthesis, which enables cell growth, as well as cell cycle progression and division. This raises the question of how cells coordinate the many nucleotide requirements for promoting cell development and permitting genome replication, particularly during the S phase.

Ribonucleotide Reductase (RNR) catalysis the conversion of ribonucleoside diphosphates to deoxyribonucleoside diphosphates, which results in the generation of dNTP. It is possible that a substrate-level constraint for dNTPs can prevent DNA synthesis because RNR inhibition reduces DNA replication and causes replication stress signalling. Insufficient dNTP levels at the start of the S phase in budding yeast can cause replication stress signalling to be activated in unaffected cells, indicating that endogenous dNTP levels are within a range that can become limiting. Furthermore, RNR mutations that result in the depletion of particular dNTPs might hinder the advancement of the S phase in budding yeast, highlighting the significance of preserving the proper concentrations of individual dNTPs for DNA replication. By coordinating responses to stressful situations and nutrition availability, cells have developed conserved signalling networks that match growth with metabolic capacity. Development regulation pathways typically stop cell growth and reduce biosynthesis when nutrients become scarce in order to conserve resources. By controlling *de novo* nucleotide synthesis and coordinating RNA synthesis and the degradation, growth signalling contributes to nucleotide metabolism. For example, mTORC1 signalling encourages the generation of one-carbon substrates for purine synthesis while the mTORC1 substrate p70 S6 kinase phosphorylates and stimulates a crucial enzyme in pyrimidine synthesis. Purine levels govern the activity of mTORC1, and nucleotide availability can also be a significant input for growth regulation pathways. Nucleotide availability alone enables starvation survival in cells with impaired autophagy, underscoring the need of nucleotide homeostasis for cellular fitness under a variety of environmental circumstances. There is a wide spectrum of intracellular concentration variation and diverse roles for various nucleotide types in cellular metabolism. Physiological context affects the availability of extracellular nucleobases and nucleosides; however these species are frequently rare in the environment. The majority of cells must therefore rely on *de novo* synthesis to meet at least some of their nucleotide demands, even if many cells preferentially salvage available nucleobases and nucleosides. Purine and pyrimidine production both include numerous metabolic pathways that might be differently impacted by changes in the environment. Therefore, the relative quantities of different nucleotides in cells can be influenced by the availability of nutrients in the environment, including nucleotide precursors. The degree to which cells are aware of the relative availability of different nucleotide species and the mechanisms by which they maintain nucleotide homeostasis to adapt to changing demands during the cell cycle are yet unknown.

CONCLUSION

Nucleotide species imbalances prevent cell proliferation but are not detected by the traditional metabolic regulatory pathways. Instead, despite the nucleotide imbalance, cells proliferate and enter the S phase, which activates DNA replication stress signalling as a defence mechanism. Replication stress signalling also increases nucleotide availability during unaffected S phases, indicating that nucleotide balance detection and maintenance during typical proliferation may be aided by replication stress sensing.

Correspondence to: Sone Clia, Department of Cell Science, University of Ibadan, Ibadan, Nigeria, E-mail: sone.clia@inta.ng

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