Knowledge Systematization for Cellular Senescence Processes by Homeostasis Imbalance Process Ontology

Yuki Yamagata ^{1, 2}, Shuichi Onami ^{1, 2}, and Hiroshi Masuya^{2, 3}

¹ RIKEN Center for Biosystems Dynamics Research, 2-2-3 Minatojima-minamimachi, Chuo-ku, Kobe 650-0047, Japan, ² RIKEN Information R&D and Strategy Headquarters, 2-2-3 Minatojima-minamimachi, Chuo-ku, Kobe 650-0047, Japan, ³ RIKEN BioResource Research Center, Kouyadai 3-1-1 Tsukuba, Ibaraki 305-0074 Japan

Abstract

In the science of aging, controlling cellular senescence attracts increasing attention. This study focuses on cellular senescence and systematizes knowledge by developing the homeostasis imbalance process ontology (HoIP). We uniformly represent a series of processes related to cellular senescence as an imbalance between stress and its response. Moreover, we show how cellular senescence brings benefits and deleterious effects in the embryonic, acute, and chronic course.

Keywords

cellular senescence, homeostasis imbalance process ontology, knowledge systematization

1. Introduction

Cellular senescence is a crucial factor in aging. This abstract introduces an organization of knowledge concerning the processes of cellular senescence. We have developed a homeostasis imbalance ontology process (https://bioportal.bioontology.org/ontologies/HO IP) [1]. We report the representation of the course of cellular senescence in HoIP.

2. Methods and Results

We manually reviewed textbooks and articles using Protégé 5.5.0. This work captured the process of cellular senescence as the outcome of homeostasis imbalance between stress (e.g., DNA damage) and stress response. Referring to upper ontology BFO and OBO-Foundry ontologies in biomedicine, we created a new definition of the course of cellular senescence in HoIP.

HoIP describes each course in terms of causal processes. For example, in the chronic cellular senescence course, we confirmed that sustained senescence-associated secretory phenotype secretion could cause chronic inflammation related to oncogenesis and type 2 diabetes. In contrast, transient SASP might cause DNA repair, tissue repair, and tissue remodeling in the embryonic and acute courses.

Proceedings ICBO 2022, September 25-28, 2022, Ann Arbor.

ORCID: 000-0002-9673-1283 (A. 1)

© 2020 Copyright for this paper by its authors. Use permitted under Creative Commons License Attribution 4.0 International (CC BY 4.0). CEUR Workshop Proceedings (CEUR-WS.org)

EMAIL: yuki.yamagata@riken.jp (A. 1)

3. Discussion

This study shows that cellular senescence benefits the embryonic and acute course via the homeostatic imbalance. Especially in embryos, the homeostatic imbalance might lead to dynamic equilibrium, homeorhesis i.e., [2]. framework also highlights the fact that chronic cellular senescence has deleterious tissue effects. HoIP will aid in understanding the fundamental mechanisms of cellular senescence.

4. Acknowledgements

This work was supported by JSPS KAKENHI Grant Number JP22K17959 and the RIKEN Open Life Science Platform Project.

5. References

- [1] Yamagata et al. Homeostasis Imbalance Process Ontology: a Study on COVID-19 Infectious Processes, VDOS-2021, 2021.
- [2] Waddington, C. H. (2014). The strategy of the genes. Routledge.