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SHORT COMMUNICATION

LETTER TO THE EDITOR

Searching for the Deep Root and Fundamental Mechanism of Biotic Aging

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HIGHLIGHT

Biotic aging is an ancient topic and has captured human interest for long time. However, our current understanding of biotic aging may be very limited because it is restricted to only the eukaryotic world of life. This letter urges researchers and readers to pay attention to the study on prokaryotic aging because it may help to reveal the deep root and fundamental mechanism of biotic aging.

KEY WORDS

Aging, Eukaryotic aging, Prokaryotic aging, Cell aging, Evolution

Dear Editor:

I am very delighted to see that aging once again became a focal point in scientific publication as reflected by the special issue of *Cell*, Reviews on Aging (Vol. 120. No. 4, Feb. 25, 2005). However, I am a little disappointed to see that current studies on biotic aging are still largely confined within the eukaryotic world. This situation is evident from the contents of the 11 reviews in this special issue of *Cell* because they all addressed aging in eukaryotic organisms, especially multicellular organisms. Aging in prokaryotic organisms was mentioned only in one review but no real insight was given (8).

The evolutionary tree of biotic aging as depicted right now goes deep only to unicellular eukaryotic yeast and back only to 1 billion years ago (6). However, with recent highlight on bacterial aging (5, 10), we may need to re-examine our perceptions on biotic aging. If aging also occurs in prokaryotic world, as predicted by theoretical

analyses (13, 15-17) and indicated by experimental observations (1, 16, 18, 19, 22), then our search for the root of biotic aging should go even deeper.

Facing such a challenge and opportunity, we may particularly need to re-evaluate the validity and suitability of some dominant theories of biotic aging that are based on or related to only the eukaryotic features. These include disposable soma model of aging (9), the mitochondria link for aging (2), and the telomere shortening hypothesis (3, 20). In my opinion, some of these theories more likely explain the consequences rather than identify the fundamental causes of biotic aging. We should also realize that some proposed aging mechanisms such as the endocrine regulation of aging (7), the sex and death connection (21), and the tumor suppression and longevity (4) may only address the superficial manifestations of aging and have an even limited applications because they are specific to more complex biotic features that exist only in the even higher forms of eukaryotic life.

The discovery of prokaryotic aging and, more importantly, the revised view of bacterial/cell life (13, 15-17) ends a long-standing dichotomy in biology and ushers in a unification of all life forms under some common fundamental principles (11). This unification of biology also eliminates a major road block against finding common and fundamental cause and mechanism for biotic aging across the divisions of phylogenetic domains or kingdoms. A hypothesis which links DNA aging with cell aging and combines genetics with epigenetics is proposed (12). This hypothesis may serve as a new theoretic framework for studying aging from molecular level to the cell/organismal level. This conceptual paradigm shift, along with a methodological paradigm shift in cell age-synchronization (14), should lead future study on biotic aging into a deep level and result in productive outcome.

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