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Chromosomal Abnormality: A Study from Microorganisms

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Short Communication

Studies of chromosomal replication mechanisms started in tobacco mosaic virus and *Escherichia coli* [1]. Recently, the analysis of chromosomal replication mechanisms in the malaria parasite [2] and *Candida* [3] has begun uncovering many notions. Some microbes, including *Mycobacterium tuberculosis* [4] and *Listeria monocytogenes* [5], proliferate within the host cells and have unique chromosomal replication machinery (such as DNA) for chromosomal replication. On the other hand, protozoa such as the malaria parasite, and yeasts such as *Candida*, which can also grow in mammalian cells, have their own chromosomal replication machinery that is regulated by origin recognition complex (ORC) [6], which is even associated with virus replication. Growth rates and pathogenicity to humans differ among microorganisms, but the mechanisms by which proliferation and pathogenicity occur are not fully understood. Moreover, we do not know in detail the mechanisms by which pathogens can escape human immunity.

Recently, a lot of attention has been paid to biological defences, with a focus on signals like hormone and cytokine and analysis of the chromosomal replication mechanisms of viruses, budding yeasts, and *Drosophila* [1]. Early in *Drosophila* development, endoreplication and gene amplification occur in the four pairs of chromosomes [7]. These steps are maybe regulated by some signals like hormone [1]. Surprisingly, it has become clear that gene amplification occurs during the growth of the malaria parasite, too. Many researches therefore would like to uncover the relationship between the start of these mechanisms, the virulence of pathogenic microorganisms, and biological defences. Additionally, many new findings in this field could contribute to growth initiation and virulence in pathogenic microorganisms and in cancer.

References

1. Kohzaki H, Murakami Y, Transcription factors and DNA replication origin selection. Bio Essays. 2005; 27: 1107-1116.
2. Deshmukh AS, Srivstava S, Herrmann S, Gupta A, Mitr P, Gilberger TW, et al. The role of N-terminus of Plasmodium falciparum ORC1 in teromeric localization and var gene silencing. Nucleic Acids Res. 2012; 40; 5313-5331.
3. Koren A, Tsai HJ, Tirosh I, Burrack LS, Barkai N, Berman J. Epigenetically-inherited centromere and neocentromere DNA replicates earliest in S-phase. PLoS Genet. 2010; 6: e1001068.
4. Smith I. Mycobacterium tuberculosis pathogenesis and molecular determinants of virulence. Clin Microbiol Rev. 2003; 16: 463-496.
5. Kohzaki H, Asano M. Attempt of chromosome and genetic testing detection using ChIP assay. Front Biosci. 2016; 8: 298-302.
6. Den Bakker HC. Desjardins CA. Griggs AD. Peters JE. Zeng, Q. Young SK. Et al. Evolutionary dynamics of the accessory genome of Listeria monocytogenes. Plose One. 2013; 8: e67511.
7. Bell SP. Kobayashi R. Stillman B. Yeast origin recognition complex functions in transcription silencing and DNA replication. Science. 1993; 262: 1844-1849.

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