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Double Rolling Circle Replication (DRCR): A mode of amplification of oncogene as well as drug-resistant genes and replication of HSV and chloroplast DNA

Takashi Horiuchi, Masashi Tanaka, Minoru Kimura and Taka-Aki Watanabe
Tokai University, Japan

It is well established that eukaryote nuclear chromosomes are duplicated from multiple origins of replication. It remains a mystery, however, how genomes of some viruses, such as HSV (Herpes simplex virus) and *Baculoviridae* or chloroplasts are replicated. We found recently that: (1) Double Rolling Circle Replication (DRCR), originally found responsible for replication of yeast 2-micron plasmid DNA, can lead to amplification of oncogenes as well as drug resistance genes and (2) DRCR is highly recombinogenic. In addition, we will present our model, based on these findings, that DRCR is involved in DNA replication of HSV-1, chloroplasts and some mitochondria. The model could explain how DRCR contributes to replication-recombination coupling of HSV and how it promotes amplicon shortening during gene amplification.

Biography

Takashi Horiuchi has his research interest is genome dynamics, especially the physiological role in DNA replication fork blocking events in *E. coli* and *S. cerevisiae*, successful conversion from the circular genome of *E. coli* to linear, molecular mechanism of gene amplification of rDNA in yeast and oncogene (drug-resistant gene) in higher eukaryotes and molecular mechanism of DRCR (Double Rolling Circular Replication) in Herpes Simplex Virus (HSV) and Chloroplast DNA. He has received Kihara Prize in 2007 from Japan Society of Genetics for identification and characterization of DNA replication fork blocking event.

horiwood2002@yahoo.co.jp

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