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The yeast plasmid: A hitchhiker on chromosomes

The native 2-micron plasmid of yeast is remarkable for its nearly chromosome-like stability. This selfish DNA element is optimized for its maintenance at an average copy number of 40-60 molecules per cell nucleus. A plasmid coded partitioning system, comprised of two partitioning proteins and a cis-acting partitioning locus is responsible ensuring the equal or nearly equal segregation of replicated plasmid copies into mother and daughter nuclei. Cumulative results from a variety of genetic, cell biological and biochemical experiments suggest that the partitioning proteins promote the physical association of plasmid molecules to yeast chromosomes. This chromosome tethering is reminiscent of a similar strategy used by the episomes of mammalian gamma herpes and papilloma viruses for propagation in infected cells during long-term latency. Our analyses using fluorescence-tagged single-copy derivatives of the 2-micron plasmid suggest that plasmid sisters formed by replication tether to sister chromatids in a symmetric fashion, thus elevating the plasmid to nearly chromosome status in 1:1 segregation. We are currently mapping potential plasmid-localizing sites on chromosomes using genome-wide approaches

Speaker Biography

Jayaram's research is focused on the *Saccharomyces cerevisiae* plasmid 2-micron circle—a small, high-copy extrachromosomal selfish DNA element with chromosome-like stability. Plasmid persistence is accomplished by a deceptively simple partitioning system consisting of two plasmid-coded proteins and a cis-acting partitioning locus. The partitioning system promotes the tethering of plasmid sisters formed by replication to sister chromatids, and 1:1 plasmid segregation by a hitchhiking mechanism. Copy number maintenance utilizes DNA amplification promoted by the plasmid-coded Flp site-specific recombinase. Amplification is initiated by a replication-coupled DNA inversion reaction. Plasmid gene expression circuitry is fine-tuned for prompt amplification response when needed, without the risk of runaway increase in copy number. Our research interests span mechanisms of (a)DNA rearrangements mediated by Flp and other site-specific recombinases, (b)chromosome-coupled plasmid segregation, and (c)*in vivo* regulation of Flp levels/activity to prevent inappropriate plasmid amplification. In summary, we wish to unveil the interplay of plasmid- and host-encoded mechanisms that promote their nearly conflict-free coexistence over evolutionary times.

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