

## Poster C-20

### Maximum likelihood inference of ancestral gene content for the bacterial septosome



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**Short Abstract:** We apply maximum likelihood methods for inferring the ancestral gene content of a specific protein complex, the bacterial septosome, and correlate shifts in septosome composition to phenotype. We now are estimating the variance of rates of gain and loss of genes through simulations and validating putative horizontal transfer

#### Long Abstract:

In most bacteria, cell division occurs through binary fission and is driven by the formation of a highly organized structure, the septosome, a protein complex that determines the location of the cell division plane, drives the synthesis of cell wall at the forming septum, and might even provide a constrictive force for the invagination process. The main component of the septosome is the FtsZ protein, a tubulin homolog, which forms polymers that organize into a ring structure around mid cell and recruits several other proteins, both directly and indirectly, thus forming a network of protein interactions that must be carefully tuned, both in time and space, in order to allow the correct and timely generation of two identical daughter cells [1]. For such a highly organized multi-component system, gain or loss of a cell division gene is expected to be an important evolutionary event, with potentially high impact on a lineage's phenotype. Still, different lineages of bacteria that divide by binary fission exhibit different profiles of cell division genes, thus suggesting different mechanisms for regulating division.

As a first step to correlate phenotypes changes and gene content variation of the septosome complex, we inferred the septosome gene content for the ancestors of a set of completely sequenced bacteria. We retrieved the presence/absence pattern of 20 cell division gene families from 163 sequenced bacterial and archaeal genomes from the STRING database and combined this data with the recently published tree of life by Cicarelli et al. [2] as inputs to the BayesMultistates program [3]. Inference of ancestral gene content was performed using maximum likelihood and two variants of the independent characters model, with either equal or unconstrained rates of gene gain and loss. The best model for each gene was chosen using a likelihood ratio test (LRT).

For most genes, a high probability of being present in the genome of the last common ancestor of all bacteria is inferred, even when some important bacterial lineages are removed from the analysis. Our reconstructed last common ancestor of all bacteria would have 14 of the genes analyzed, including all site selection genes (Min operon and divIVA) and the most conserved components of the complex (ftsZ, ftsA, ftsK), as well as some less well characterized genes, whose products localize late to the dividing septum, such as ftsW, ftsI and amiC. The genes ftsQ, ftsB and ftsL, although coding for interacting components of a

sub-complex inside the septosome, were not acquired simultaneously in a single or a few lineages and then lost, but instead acquired independently and sequentially by different lineages. *ftsQ* is inferred to be present, with a high probability, in the last common ancestor of all bacteria, but *ftsB* is likely to have been independently gained by the ancestors of a few groups, providing an interesting example of horizontal gene transfer (HGT). *ftsL*, on the other hand, was independently gained at the root of two very divergent lineages, and might correspond either to another example of HGT or to two independent gene origins. The overall inferred picture suggests that the evolution of the septosome is driven primarily by gene loss events, with a limited, but perhaps relevant, impact of HGT.

We are now performing careful phylogenetic analyses of the septosome genes, as a means to further evaluate the hypothesis that most cell division genes families were not subjected HGT, with a focus on the possibility of xenologous gene displacement [4].

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[2] Ciccarelli FD, Doerks T, von Mering C, Creevey CJ, Snel B and Bork P. *Science*, 311:1283-1287 (2006).

[3] Pagel M. *Systematic Biology*, 48(3):612-622, (1999).

[4] Makarova KS, Ponomarev VA and Koonin EV. *Genome Biology* 2001, 2(9):research0033.1-0033.14.