論文内容の要旨

論文題目: **Evolution of the Shine-Dalgarno Interaction**
(Shine-Dalgarno相互作用の進化)

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1. Abstract

1.1. Background

Basic translational functions such as peptide bond synthesis are highly conserved across all domains of life, but translation initiation systems differ considerably between prokaryotes and eukaryotes. In prokaryotes, the site recognition that initiates translation is often mediated by rRNA-mRNA base pairing, known as the Shine-Dalgarno (SD) interaction. This mechanism is never observed in the nuclear genetic systems of eukaryotes. SD interactions adhere to a distinct base pairing rule, such that a pyrimidine-rich, anti-SD sequence in the 3′ tail of a small subunit rRNA binds to a complementary, purine-rich, SD signal sequence in the 5′ untranslated region (UTR) of an mRNA. A core motif (i.e., the anti-SD motif), 3′CCUCC, is conserved among anti-SD sequences, suggesting an extreme evolutionary constraint and a crucial role for SD interaction.

The anti-SD motif is so far known to be universal in prokaryotes, suggesting the universality of SD interaction, although its usage varies considerably. SD interactions were reported in eukaryotic organelles of prokaryotic origin, mitochondria and plastids; the interaction seems widely used in plastids, while used only in rare bacteria-like mitochondria. Exponentially increased genome data now provide an unprecedented chance to obtain more detailed understanding of SD interactions in various taxonomic groups. Here I conducted a large-scale analysis of all available complete genome sequences of bacteria and plastids for SD interactions with emphasis on their alterations and losses.

1.2. Parallel Losses of Shine-Dalgarno Interactions in Bacteria.

Contradicting to this belief, investigating over 1,000 bacterial genome sequences allowed me to find 15 bacteria without the canonical anti-SD motif (referred to as lost anti-SD bacteria). These lost anti-SD bacteria emerged independently in α-Proteobacteria, β-Proteobacteria, γ-Proteobacteria, Falavobacteria, and Mycoplasma. Loss of canonical anti-SD motifs in the lost anti-SD bacteria were accompanied by that of their SD sequences, suggesting that SD interaction no longer operates in them. Many of the lost anti-SD genomes belonged to obligate host-associated bacteria with highly reduced genomes (i.e., primary endosymbionts and mycoplasmas). The evolutionary forces toward large-scale gene/function loss during a period of host association may have forced the bacteria to sacrifice important but non-essential regulatory functions such as SD interaction. A-rich motifs at the corresponding areas of the SD sequences emerged in all surveyed Flavobacteria (regardless of the conservation of SD interaction). An unknown

translation initiation mechanism mediated by this motif may have replaced SD interaction, promoting the loss of SD interaction. In Mycoplasma, only a subdivision that infects red blood cells showed this loss, suggesting an environmental change to a hemotrophic condition is a likely driver for the loss.

1.3. Parallel Losses and Alterations of Shine-Dalgarno Interactions in Plastids.

My research hereinabove reported the rare loss of SD interactions in several bacterial lineages, most of which were under obligate association with eukaryotic host cells. This raised the question of what happened to SD interactions during the evolution of a cyanobacterial endosymbiont into modern plastids (including chloroplasts). My analysis of available complete plastid genome sequences revealed that the majority of plastids retained SD interactions but with varying levels of usage. Parallel losses of SD interactions took place in plastids of Chlorophyta, Euglenophyta, and Chromerida/Apicomplexa lineages, presumably related to their extensive reductive evolution. Interestingly, I discovered that the canonical SD interaction (3′CCUCC/5′GGAGG (rRNA/mRNA)) was replaced by an altered SD interaction (3′CCCU/5′GGGA or 3′CUUCC/5′GAAGG) through coordinated changes in the sequences of the core rRNA motif and its paired mRNA signal. These changes in plastids of Chlorophyta and Euglenophyta proceeded through intermediate steps that allowed both the canonical and altered SD interactions. This coevolution between the rRNA motif and the mRNA signal demonstrates unexpected plasticity in the translation initiation machinery.

1.4. Significance

This study demonstrates evolutionary plasticity of SD interactions by discovering their parallel losses (in bacteria and plastids) and alterations (in plastids) especially under host-associated conditions. Furthermore, alterations in SD interactions were achieved by stepwise and coordinated changes in rRNA motif and its complementary mRNA signal. This represents, to the best of my knowledge, the first report of rRNA-mRNA coevolution. This coevolution caused unexpected plasticity in the translation initiation machinery, likely driving genome evolution by affecting all genes with mRNA signals.