The Roles of BmMre11 and BmRad50 Protein in DSB Repair

Masateru Takahashi¹, Takahiro Kusakabe¹, Aki Hayashgi¹, Tsukasa Shirao¹, Kazuhiro Okano², Kazuei Mita³, Tooru Shimada⁴, Yutaka Kawaguchi¹, and Katsumi Koga¹

¹Laboratory of Silkworm Science, Kyushu University Graduate School of Bioresource and Bioenvironmental Sciences, 6-10-1 Hakozaki, Fukuoka 812-8581, Japan, ²Laboratory of Molecular Entomology and Baculovirology, RIKEN (The Institute of Physical and Chemical Research), Wako, Saitama, Japan, ³Genome Research Group, National Institute of Radiological Sciences Inage-ku, Chiba, Japan, and ⁴Department of Agricultural and Environmental Biology, Graduate School of Agricultural and Life Sciences, The University of Tokyo, Yayoi 1-1-1, Bunkyo-ku, 113-8657, Tokyo, Japan.

A DNA double-strand break (DSB) is highly cytotoxic DNA damage that disrupts the genomic integrity of a cell. Unrepaired or misrepaired DSBs can kill a cell or lead to chromosome aberrations; thus the prompt and efficient repair of DSBs is fundamental for genomic stability and cancer prevention. A number of fundamentally different DSB repair pathways are available in eukaryotic cell, two of which are known as major pathway. One is nonhomologous end-joining (NHEJ), another is homologous recombination (HR). MRE11 and RAD50 were reported to make a heterotrimer complex with NBS1, and play an important role in early process of HR and NHEJ. In this study, we have cloned the cDNAs encording BmMRE11 and BmRAD50 from silkworm testis, and their nucleotide sequences were determined. Moreover we have examined the effect of BmMRE11 and BmRAD50 knockdown on a silkworm cultured cell by RNA interference (RNAi). The DNA cell cycle analysis, quantified by flow cytometry, showed that the treatment of BmN4 cells with BmMRE11 and BmRAD50 dsRNA tended to arrest the cells in G₂/M phase. Now we are trying to examine the relationship between these genes and HR using the silkworm cultured cells.