Spotlight

By M.O.

The ups and downs of DNA methylation in gastric tumors

Yoshida *et al.,*
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DNA methylation has important effects on gene expression, retrotransposition and carcinogenesis. Methylation usually occurs at CpG dinucleotides, which are often found in repetitive elements in the genome, known as Alu, LINE1 and Satα loci. Methylation of these sites serves to silence the genome-damaging activities of these elements while their demethylation is linked to lower genome stability, enhanced gene recombination, chromosome translocation and carcinogenesis.

Infection with *Helicobacter pylori* (HP) is a major cause of gastric cancer. Interestingly, aberrant DNA hypermethylation is induced as a consequence of HP infection. This methylation occurs mainly at "CpG islands" located in promoter regions of genes, which are generally hypomethylated to promote gene expression. To clarify the status of global DNA methylation in gastric cancers, Yoshida and colleagues examined samples of gastric mucosa collected by endoscopic biopsy from HP-negative or HP-positive volunteers. These volunteers were either healthy or afflicted with gastric cancer; in this case, only noncancerous tissue was examined.

The authors showed that Alu and Satα loci are hypomethylated in HP-infected gastric mucosa of healthy volunteers, and that Alu, but not Satα, hypomethylation persisted in patients with cancer. In contrast, LINE1 elements were only hypomethylated in patients with cancer. The authors speculate that hypomethylation of Alu and Satα elements may represent early events of gastric carcinogenesis induced by HP infection whereas LINE1 hypomethylation may occur as a consequence of cellular transformation processes.