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Correction of transposon induced malignancy using recombinant stem cells

Sanjay R Nair

Transposable element (TE) activation is a source of new mutations in cancer cells. Recent work has identified a newly proposed mechanism involving extensive recruitment of TE-derived promoters to drive the expression of oncogenes and subsequently promote oncogenesis. As a result, dedifferentiation of cancer cells is documented along with TE expression. As an ideal remedy for this, recombinant stem cells can be introduced. The enzyme, transposase, acts like DNA scissors, cutting through the double-stranded DNA to remove a transposon from one genomic site. So, using non-homologous recombinant techniques, we can design genetically modified stem cells that express the transposase enzyme, which, once introduced into the site of malignant cells, cleaves off the TE. Genes encoding transposases are the most prevalent genes in nature. For example, transposase Tn5 is a member of the RNase superfamily of proteins, which includes retroviral integrases. They can be obtained from Shewanella and Escherichia bacteria. With the introduction of the gene encoding transposase into the DNA of induced pluripotent stem cells (IPSCs), non-homologous recombination may be possible, and it forms a heteroduplex and results in gene conversion. Further, this recombinant stem cell, once introduced into the malignant cells, translates the enzyme and might remove TE. Further research in this aspect could bring revolutionary treatment procedures.

Keywords: Oncogenesis, Dedifferentiation, Transposable element, Non-homologous recombination, Heteroduplex

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