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Title	MUC2: the Major Target Gene for the MSI in Colorectal Carcinoma
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Aim	The recent discovery of hypermethylation of the promoter of genes is a powerful epigenetic mechanism for the inactivation of some genes in colorectal and other cancers. Approximately, 95% of hereditary non-polyposis colorectal cancers (HNPCCs) and 15% of sporadic colorectal cancers (CRCs) are instable in their Mismatch repair system (MMR).
Materials & Methods	we analyzed by immunohistochemistry the immunostaining of MUC2 and MMR genes.
Result	Herein, we have shown that the MSI phenotype, resulting in the deficiency of a capital genes of MMR system such as MLH1, MSH2, MSH6 and PMS2. Moreover, all CRCs were found to be hypermethylated in the MUC2 promoter and this correlated with negative E-cadherin expression and especially microsatellite instability.
Conclusion	MSS cancers tend to be diploid or near diploid karyotype with similar mutations in the APC and K-Ras genes but reduced mutations in p53. Mutations found in MSI cancers but not MSS cancers include the TGF-β, IGFII, BAX and E2F4 genes and now MUC2. MUC2 appears to be a typical target gene of MSI phenotype in Tunisian patients with CRCs. Knowing that the Hypermethylation of the promoter may be reversed and gene function restored to a cell, identification of epigenetic alterations can be a powerful therapeutic approach throughout the inhibition of DNA-methylase enzyme.