Diseases mechanism identification of the effect of epigenetic changes on networks orchestrating the transcriptional changes regulatory pathways affected by DNA hypomethylation induced human lupus. However, the genes and the patterns of DNA methylation, contribute to the induced epigenetic changes, and in particular altered DNA hypomethylation.

DNA methyltransferase inhibitors such as the cytosine analog 5-aza-cytidine and 5-aza-2'-deoxycytidine can induce DNA hypomethylation.

Recent evidence indicates that environmentally-induced epigenetic changes, and in particular altered patterns of DNA methylation, contribute to the environment–host interaction in some forms of autoimmunity. T cell DNA hypomethylation has been implicated in the pathogenesis of idiopathic and drug-induced human lupus. However, the genes and the regulatory pathways affected by DNA hypomethylation are largely unknown.

Here we report an elucidation of the regulatory networks orchestrating the transcriptional changes observed by microarray experiments on methylation sensitive genes as a meaningful approach in identification of the effect of epigenetic changes on disease mechanisms.

**Materials and Methods**

**RNA analysis**

5-Aza (5-aza-deoxycytidine) treatment to gain new insights into molecular basis in autoimmune diseases.

**Summary**

- DNA hypomethylation perturbs the functions of several cellular processes.
- GO analysis of the 5-Aza responsive genes reveal that immune response genes are preferentially affected by demethylation in T cells.
- Some methylation sensitive immune genes appear to be co-regulated by the Transcription Factors common to the promoters of these genes.

**Conclusions**

Immune response genes are preferentially expressed and at higher levels in hypomethylated T cells. These data suggest that epigenetic changes associated with environmental factors may lead to stronger autoimmune T cell responses under conditions of repeated stimulation. Underlying regulatory networks enables us to gain new insights into molecular basis in autoimmune diseases.

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