Increased Serum Levels of IL-28, IL-29 and Protective Effect of IL28B rs8099917 Polymorphism in Patients with Hashimoto's Thyroiditis

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Background
• Hashimoto's thyroiditis (HT) is thought to result from the decreased of T helper type 2 (Th2) responses, leading to progressive destruction of thyrocytes. IFN-α1, -α2, and -α3 (also known as IL-29, IL-28A, and IL-28B, respectively) is a recently described member of the IFN-α family and has been shown to decrease production of Th2 cytokines in vitro. However, the role and mechanism of IFN-α1 in Hashimoto's thyroiditis remain unknown.

Purpose
• The purpose of our study is to elucidate whether the IL-29 and IL-28B gene polymorphisms are susceptibility genes for the development of HT. Also we aimed to investigate the effects of IL-29 and IL-28 serum levels on pathogenesis of HT.

Material/Method
Using the polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP) method, the single-nucleotide polymorphisms (SNPs) of IL28B rs8099917 (IL28 G/T) and IL29 rs30461 (IL29 T/C) were studied in 99 patients with HT and 100 healthy controls.

Results
Considering the allelic distribution for IL28 G/T polymorphism a higher frequency of G allele was observed in the control group when compared to the HT group. So it was suggested that G allele may be a protective role for HT pathogenesis (OR= 0.388 95%-CI 0.217-0.693; p=0.001).

Also our findings demonstrate that there was statistically significant difference in serum IL-28 and IL-29 levels between case and control groups (p=0.001). The increased serum levels of IL-28 and IL-29 in patients with HT was determined.

Table 1. The primer sequences, annealing Tm, restriction enzymes for detecting each single nucleotide polymorphism (SNP)

<table>
<thead>
<tr>
<th>SNP</th>
<th>Reference SNP ID</th>
<th>Forward primer</th>
<th>Reverse primer</th>
<th>Annealing Tm</th>
<th>Restriction enzyme</th>
</tr>
</thead>
<tbody>
<tr>
<td>IL28 G/T</td>
<td>rs8099917</td>
<td>5’-CAT CCC TCA TCC CAC TTC TGG G-F</td>
<td>5’-TCG GGC CAC CAC AAT TCA TCA T-3 (R)</td>
<td>55 °C</td>
<td>BsrDI</td>
</tr>
<tr>
<td>IL29 T/C</td>
<td>rs30461</td>
<td>5’-TGA ACA TGG ACA GTI AGG CAC Y-F</td>
<td>5’-GAG CCA ATA GGA GCC CAG AC-3 (R)</td>
<td>54 °C</td>
<td>AvaII</td>
</tr>
</tbody>
</table>

Conclusion
In conclusion, IL28B gene polymorphism and serum IL-28 and IL-29 levels seemed to play a role in the pathogenesis of HT.