### Table I (Continued)

<table>
<thead>
<tr>
<th>MicroRNA</th>
<th>Up or down regulated</th>
<th>Gene targets top 10 predicted</th>
<th>Relation to T cell function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hsa-mir-106b</td>
<td>Down</td>
<td>ZNFX1, TNKS2, SERF1A, SERF1B, PKD2C7ORF43, VSX1, PLERHA3, ITGB8, E2F5</td>
<td>Mir-106b has been shown to modulate the TGF-signaling pathway (with TGF-involved in T regulatory cells differentiation and maturation).</td>
</tr>
<tr>
<td>Hsa-mir-9</td>
<td>Down</td>
<td>ONECUT2, TESK2, CSDA, RNFL11, COL15A1, POU2F1, PRDM6, BEND3, UBE3C, MUM1L1</td>
<td>Mir-9 enhances IL-2 production in activated human CD4+ T cells by repressing Blimp-1.</td>
</tr>
<tr>
<td>Hsa-mir-125a</td>
<td>Down</td>
<td>PCTP, TRIM71, TMEM87, ENPP1, SMC1, XIAA152, ENPEP2NSF5, HMACS1, ADAM9</td>
<td>MicroRNA-125a negatively regulates RANTES expression by targeting KLF13 in activated T cells.</td>
</tr>
<tr>
<td>Hsa-mir-15b</td>
<td>Down</td>
<td>CPEB2, C3NE1, FGF2, PIAG1, RSP53, STOX2, WEE1, ZBTB34, SLC20A2, MGA74A</td>
<td>Predominantly expressed in memory T cells (one of 7 mirs constituting 60% of the mirs expressed in CD8 cells).</td>
</tr>
</tbody>
</table>

### Discussion

This novel preliminary data generates the hypothesis that further investigation of miR profiles may help explain underlying differences in immune pathogenesis between MPO and PR3 positive disease. **Conclusion**—Further validation of these data in a larger population is required to establish the potential significance and function of these differences in miR expression. This may allow future use of microRNAs in AAV as biomarkers or to enable identification of novel therapeutic targets.

### Further readings


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### P177

**The interaction between C5a and sphingosine-1-phosphate in neutrophils for ANCA-mediated activation**

J. Hao, C. Wang, M. Chen, M.H. Zhao
Peking University First Hospital, Beijing, China

**Introduction**—C5a and the neutrophil C5a receptor play a central role in antineutrophil cytoplasmic antibody (ANCA)-mediated neutrophil recruitment and activation. The current study further investigated the interaction between C5a and S1P in neutrophils for ANCA-mediated activation.

**Methods**—The levels of S1P in plasma sequential samples from 29 patients with ANCA-associated vasculitis (AAV) in active stage and in remission were tested. The effect S1PR inhibitor was tested on respiratory burst and degranulation of C5a-primed neutrophils activated with ANCA.

**Results**—The level of S1P was significantly higher in active disease than that in remission (2034.2 ± 438.5 vs. 1489.3 ± 547.4, P < 0.001). S1P-primed neutrophils activated with MPO-ANCA-positive IgG and PR3-ANCA-positive IgG (314.0 ± 36.8 vs. 379.0 ± 20.6, P < 0.05; 314.0 ± 36.8 vs. 384.5 ± 11.4, P < 0.05). S1P-primed neutrophils induced by MPO or PR3-ANCA-positive IgG, the lactoferrin concentration in the supernatant increased from 448.0 ± 17.1 ng/ml in untreated cells to 1342.7 ± 29.5 ng/ml (P < 0.001) or 1338.3 ± 27.1 ng/ml (P < 0.001). The level of S1P increased from 18.0 ± 3.0 nmol/L in the non-primed neutrophils supernatant to 53.3 ± 4.2 nmol/L in C5a-primed neutrophils supernatant (P < 0.01), and decreased to 24.7 ± 3.2 nmol/L upon pre-incubation with C5aR inhibitor (P < 0.05). In C5a-primed neutrophils, subsequently activating with MPO or PR3-ANCA-positive IgG, the MFI value was 369.8 ± 18.8 (P < 0.01) or 377.3 ± 13.2 (P < 0.01), which decreased to 310.0 ± 12.5 (P < 0.01) or 323.8 ± 15.5 (P < 0.05) upon pre-incubation with S1PR inhibitor.

**Conclusion**—The level of S1P is significantly higher in active AAV patients than that in remission. S1P triggered by C5a-primed neutrophils acting as an autocrine or paracrine manner, which activate neutrophils again. Blocking S1PR may decrease C5a-induced activation of neutrophils by ANCA. The interaction between S1P and C5a may play an important role in neutrophils for ANCA-mediated activation.

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### P178

**Circulating level of high mobility group box 1 is associated with disease activity in antineutrophil cytoplasmic autoantibody-associated vasculitis**

C. Wang, M. Chen
Renal Division, Peking University First Hospital, Beijing, China

**Introduction**—High mobility group box 1 (HMGB1) is a kind of pro-inflammatory mediator and has been confirmed to be associated with inflammatory conditions and tissue damage. Previous studies have reported circulating HMGB1 levels were elevated in patients with active ANCA-associated vasculitis (AAV). The current study aimed to