The STAT3 and NF-κB B (NF-κB) pathways play a prominent role in IL-17 production and intestinal inflammation. We have shown that a novel drug named Vidofludimus (Vido, 4SC-101, SC12267) inhibited colonic IL-17 and murine colitis by establishing anti-inflammatory activity of this drug.

**METHODS**

**Splenocyte Cultures:** Splenocytes were isolated from mice and cultured in RPMI 1640 medium containing 10% FBS and a 1% L-glutamine. Then, 10,000 × 10^6 cells were plated in 6-well plates and cultured for 48 hours at 37°C. After 10 minutes, the culture media was collected and a minimum of 1.2 × 10^8 cells were utilized for these studies.

**Vidofludimus Attenuates Phospho-STAT3**

**IL-17 Secretion: Marine Splenocytes**

48 hour Data

<table>
<thead>
<tr>
<th>Data is from 2 studies</th>
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<tbody>
<tr>
<td>% Control</td>
</tr>
<tr>
<td>Vido-50 μg/kg</td>
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<tr>
<td>Vido-100 μg/kg</td>
</tr>
<tr>
<td>Vido-500 μg/kg</td>
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<tr>
<td>PBS Control</td>
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**Vidofludimus Inhibits Nuclear Binding of STAT3 in a Dose Dependent Fashion: Marine Splenocytes**

**IL-17 Secretion: Marine Splenocytes**

Synergistic activation by IL-23 + IL-1β

**Vidofludimus Inhibits Cytokine-Induced IL-17 Secretion: Marine Splenocytes**

**Vidofludimus Attenuates Phospho-AKT1**

**Expression: Marine Splenocytes**

**Vidofludimus Does Not Affect the Expression of IL-6 and STAT3**

**Staining: Marine TNBS Colitis**

**Vidofludimus Attenuates Phospho-p65**

**Staining: Marine TNBS Colitis**

**CONCLUSION**

The inhibition of STAT3 and NF-κB pathways by Vidofludimus reduces IL-17 production and may contribute to the established anti-colitis activity of this drug.