**TP508 TFA**

Cat. No.: HY-P0316A  
Molecular Formula: C₉₉H₁₄₇N₂₈F₃O₃₈S  
Molecular Weight: 2426.46


Sequence Shortening: AGYPDEKRGDACEGDSGGPFV

Target: Thrombin; NO Synthase

Pathway: Metabolic Enzyme/Protease; Immunology/Inflammation

Storage:
- Powder: -80°C 2 years, -20°C 1 year
- In solvent: -80°C 6 months, -20°C 1 month

**BIOLOGICAL ACTIVITY**

**Description**
TP508 TFA is a 23-amino acid nonproteolytic thrombin peptide that represents a portion of the receptor-binding domain of thrombin molecule. TP508 TFA activates endothelial NO synthase (eNOS) and stimulates production of NO in human endothelial cells. TP508 TFA activates endothelial cells and stem cells to revascularize and regenerate tissues.[1][2].

**In Vitro**
TP508 (50 μg/mL; 24 hours; HCAEC) treatment reverses radiation-induced endothelial dysfunction (ED) and loss of NO signaling by attenuating the downregulation of eNOS expression. TP508 treatment is able to stimulate NO production in the irradiated cells.[1]

TP508 mitigates effects of nuclear radiation on human endothelial cells in culture restoring endothelial NO production, tube formation and accelerating repair of radiation-induced DNA double-strand breaks (DSB).[1]

TP508 acts as an antagonist for the effects of thrombin. TP508 peptide inhibits these thrombin-induced effects through a RGD and αvβ3-related mechanism.[3]

**Western Blot Analysis**[1]

<table>
<thead>
<tr>
<th>Cell Line:</th>
<th>Primary human coronary artery endothelial cells (HCAEC)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Concentration:</td>
<td>50 μg/mL</td>
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<tr>
<td>Incubation Time:</td>
<td>24 hours</td>
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<tr>
<td>Result:</td>
<td>Prevented the radiation-induced downregulation of eNOS.</td>
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</tbody>
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**In Vivo**
TP508 (10 mg/kg; intravenous injection; male CD-1 mice) treatment mitigates radiation-induced endothelial cell damage, also significantly increases survival of CD-1 mice when injected 24 h after 8.5 Gy exposure.[1]

**Animal Model:** Male CD-1 mice (12-15-week old) with γ irradiation[1]
Dosage: 10 mg/kg
Administration: Intravenous injection
Result: Mitigated radiation-induced endothelial cell damage, also significantly increased survival of CD-1 mice.

REFERENCES

