BQ-788

Cat. No.: HY-15894A
CAS No.: 173326-37-9
Molecular Formula: C₃₄H₅₁N₅O₇
Molecular Weight: 641.8
Target: Endothelin Receptor
Pathway: GPCR/G Protein
Storage: Please store the product under the recommended conditions in the COA.

**BIOLOGICAL ACTIVITY**

**Description**
BQ-788 is a potent, selective ETB receptor antagonist with $IC_{50}$ of 1.2 nM for inhibition of ET-1 binding to human Girardi heart cells, poorly inhibiting the binding to ETA receptors in human neuroblastoma cell line SK-N-MC cells with $IC_{50}$ of 1300 nM.[1]

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<th>IC₅₀ &amp; Target</th>
<th>IC₅₀: 1.2 nM (ETB)</th>
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**In Vitro**
BQ-788 potently and competitively inhibits $^{125}I$-labeled ET-1 binding to ETB receptors in human Girardi heart cells (hGH) with an $IC_{50}$ of 1.2 nM, but only poorly inhibits the binding to ETA receptors in human neuro-blasteroma cell line SK-N-MC cells ($IC_{50}$, 1300 nM). BQ-788 shows no agonistic activity up to 10 $\mu$M and competitively inhibits the vasoconstriction induced by an ETB-selective agonist (pA2, 8.4). BQ-788 also inhibits several bioactivities of ET-1, such as bronchoconstriction, cell proliferation, and clearance of perfused ET-1.[1]

**In Vivo**
BQ-788 (3 mg/kg/h, i.v.) completely inhibits a pharmacological dose of ET-1- or sarafotoxin6c (0.5 nmol/kg, i.v.)-induced ETB receptor-mediated depressor, but not pressor responses in conscious rats. Furthermore, BQ-788 markedly increases the plasma concentration of ET-1, which is considered an index of potential ETB receptor blockade in vivo. In Dahl salt-sensitive hypertensive (DS) rats, BQ-788 (3 mg/kg/h, i.v.) increases blood pressure by about 20 mm Hg. It is reported that BQ-788 also inhibits ET-1-induced bronchoconstriction, tumor growth and lipopolysaccharide-induced organ failure.[1] BQ 788 (3 mg/kg) results in an eightfold leftward shift in the ET-1 dose-response curve, suggesting a significant involvement of ETB dilator receptors.[2] Mice are treated with 30 nmol BQ-788 by intraplantar, reduce mechanical hyperalgesia (47% and 42%), thermal hyperalgesia (68% and 76%), oedema (50% and 30%); myeloperoxidase activity (64% and 32%), and overt-pain like behaviours. Additionally, intraplantar treatment with clazosentan or BQ-788 decreases spinal (45% and 41%) and peripheral (47% and 47%) superoxide anion production as well as spinal (47% and 47%) and peripheral (33% and 54%) lipid peroxidation, respectively.[3]

**CUSTOMER VALIDATION**

REFERENCES

