Product Information

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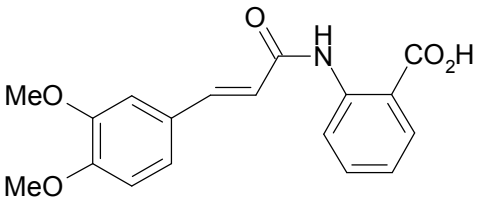
Tranilast

Catalog Number **T0318**
Storage Temperature 2–8 °C

CAS RN 53902-12-8
Synonyms: 3,4-DAA; SB-252218; Rizaben; N-(3,4-dimethoxycinnamoyl)anthranilic acid; 2–[[3–(3,4–Dimethoxyphenyl)–1-oxo–propenyl]amino] benzoic acid



Product Description
Molecular Formula: $C_{18}H_{17}NO_5$
Molecular Weight: 327.33

Tranilast was originally identified as an anti-allergy agent that inhibits mast cell degranulation. Tranilast also demonstrates potent immunomodulatory activities. It inhibits endotoxin–induced prostaglandin E_2 release with an IC_{50} of 1–20 μM. It also inhibits production of thromboxane B_2 (IC_{50} = 10–50 μM), transforming growth factor–β1 (TGF-β1; IC_{50} = 100–200 μM), and interleukin-8 (IC_{50} ~100 μM). Tranilast has no effect on tumor necrosis factor-α production, nor does it inhibit the activity of either cyclooxygenase 1 or 2, or human type IIA (14 kDa) or type IV (85 kDa) phospholipase A_2 at concentrations up to 1 mM. Tranilast inhibits fibroblast proliferation, and also attenuates the proinflammatory activity of human monocytes.[1]

Tranilast-induced reduction in TGF-β production appears to protect the kidney from obstruction-induced damage. Rats received either tranilast (150 mg/kg daily) or placebo for 14 days following the obstruction of one ureter. In placebo-treated controls, the obstructed kidney had a significantly higher TGF–β concentration than the unobstructed kidney (73.7 pg/mg versus 14.1 pg/mg) and significantly more fibrosis and tubular apoptosis. Tranilast treatment significantly decreased tissue TGF-β (15.9 pg/mg), fibrosis, and renal tubular apoptosis in the obstructed kidney. Tranilast also significantly increased the expression of proliferating cell nuclear antigen in both unobstructed and obstructed kidneys.[2]

In contrast, tranilast inhibits cyclin–dependent kinase activity through the induction of $p21^{Waf1/Cip1}$ and, thus, arrests the proliferation of vascular smooth muscle cells *in vitro*. In a study of mice receiving cardiac allografts, mice pretreated with tranilast showed reduced vascular occlusion (vasculopathy) but no effect on allograft rejection. Immunohistology revealed had enhanced expression of $p21^{Waf1/Cip1}$ and decreased expression of proliferating cell nuclear antigen in the tranilast-treated graft coronary arteries.[3]

Precautions and Disclaimer
This product is for R&D use only, not for drug, household, or other uses. Please consult the Material Safety Data Sheet for information regarding hazards and safe handling practices.

Preparation Instructions
Tranilast is soluble in DMSO (>10mg/ml). It is insoluble in water.

Storage/Stability
Store the product at 2–8 °C for up to twelve months. Store solutions at –20 °C for up to 3 months.

References

1. Capper, E.A., et al., Modulation of human monocyte activities by tranilast, SB 252218, a compound demonstrating efficacy in restenosis. J. Pharmacol. Exp. Ther., **295**, 1061-1069 (2000).
2. Miyajima, A., et al.,Tranilast ameliorates renal tubular damage in unilateral ureteral obstruction. J. Urol., **165**, 1714-1718 (2001).
3. Izawa, A., et al., Tranilast inhibits cardiac allograft vasculopathy in association with $p21^{Waf1/Cip1}$ expression on neointimal cells in murine cardiac transplantation model. Arterioscler. Thromb. Vasc. Biol., **21**, 1172-1178 (2001).

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