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

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\textsubscript{2} release with an IC\textsubscript{50} of 1–20 \textmu M. It also inhibits production of thromboxane B\textsubscript{2} (IC\textsubscript{50} = 10–50 \textmu M), transforming growth factor–\beta\textsubscript{1} (TGF–\beta\textsubscript{1}; IC\textsubscript{50} = 100–200 \textmu M), and interleukin-8 (IC\textsubscript{50} = 100 μM). Tranilast has no effect on tumor necrosis factor–\alpha production, nor does it inhibit the activity of either cyclooxygenase 1 or 2, or human type II\textsubscript{A} (14 kDa) or type IV (85 kDa) phospholipase A\textsubscript{2} at concentrations up to 1 mM. Tranilast inhibits fibroblast proliferation, and also attenuates the proinflammatory activity of human monocytes.

Tranilast-induced reduction in TGF–\beta 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–\beta 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–\beta (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.

In contrast, tranilast inhibits cyclin–dependent kinase activity through the induction of p21\textsuperscript{Waf1/Cip1} and, thus, arrests the proliferation of vascular smooth muscle cells \textit{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 enhanced expression of p21\textsuperscript{Waf1/Cip1} and decreased expression of proliferating cell nuclear antigen in the tranilast-treated graft coronary arteries.

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


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