Inhibitors, Agonists, Screening Libraries

Data Sheet

**Product Name:** Procyanidin B2
**Cat. No.:** HY-N0796
**CAS No.:** 29106-49-8
**Molecular Formula:** C_{30}H_{26}O_{12}
**Molecular Weight:** 578.52
**Target:** PPAR
**Pathway:** Cell Cycle/DNA Damage; NF–κB
**Solubility:** 10 mM in DMSO

**BIOLOGICAL ACTIVITY:**
Procyanidin B2 exerts a potent and beneficial role in reducing granulosa cell apoptosis and inducing autophagy process, and exerts a variety of potent protective pharmacological effects on diabetic complications.

In vitro: Procyanidin B2 treatment decreased FoxO1 protein level, improved granulosa cell viability, upregulated LC3–II protein level, and reduced granulosa cell apoptosis rate. Under a condition of oxidative stress, Procyanidin B2 reversed FoxO1 nuclear localization and increased its level in cytoplasm. In addition, FoxO1 knockdown inhibited the protective effects of Procyanidin B2 induced.[1]

In vivo: Treatment with Procyanidin B2 resulted in an improvement in body weight increase and serum levels of triglyceride, total cholesterol, advanced glycation end products, and urinary albumin excretion in comparison with the vehicle–treated diabetic mice (P < .05), although these levels were still higher than those in the control group. Treatment with Procyanidin B2 significantly reduced the extent of glomerular basement membranes thickening, mesangial expansion, and glomerular area as well. Mimecan protein expressions in diabetes mellitus were decreased approximately by 28% when compared with those in the control group (P < .05), and restored remarkably after Procyanidin B2 treatment (P < .05). The expression of nuclear factor–κB (NF–κB) p65 in nuclear extracts, markedly higher in the diabetic mice than in the controls, was significantly suppressed by Procyanidin B2.[2] Procyanidin B2 were dissolved in distilled water.[2]

**PROTOCOL (Extracted from published papers and Only for reference)**
Animal administration: Mice[2] The forced swimming test was performed following the procedures as described previously. Briefly, 60 min after i.g. drug administration, the mice were individually forced to swim in an open cylindrical container (diameter 14 cm, height 20 cm, containing 12 cm of water maintained at 24 °C). The duration of immobility in the last 4 min of total 6 min test was recorded. Mice were considered immobile when they ceased struggling and remained floating motionless in the water, making only those movements necessary to keep their head above water. To investigate the possible involvement of 5–HT1A receptors in the behavioral effects of YL–0919, mice were treated with WAY–100635 (0.1, 0.3 mg/kg, s.c.) in combination with YL–0919 (2.5 mg/kg, i.g.), and the TST or FST was carried out 60 min later. The dose of WAY–100635 used was based on our previous study.

**References:**
Caution: Product has not been fully validated for medical applications. For research use only.
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