

## **Dimethyl fumarate**

Catalog No: tcsc0909

**Available Sizes** 

#### **Size:** 5g

Specifications

#### CAS No:

624-49-7

#### Formula:

 $C_6H_8O_4$ 

#### Pathway:

NF-ĸB

#### **Target:**

Keap1-Nrf2

#### **Purity / Grade:**

>98%

#### Solubility:

DMSO : 9.6 mg/mL (66.61 mM; Need ultrasonic and warming)

## Alternative Names:

DMF

# **Observed Molecular Weight:** 144.13

### **Product Description**

Dimethyl fumarate is a nuclear factor (erythroid-derived)-like 2 (**Nrf2**) pathway activator and induces upregulation of antioxidant gene expression.

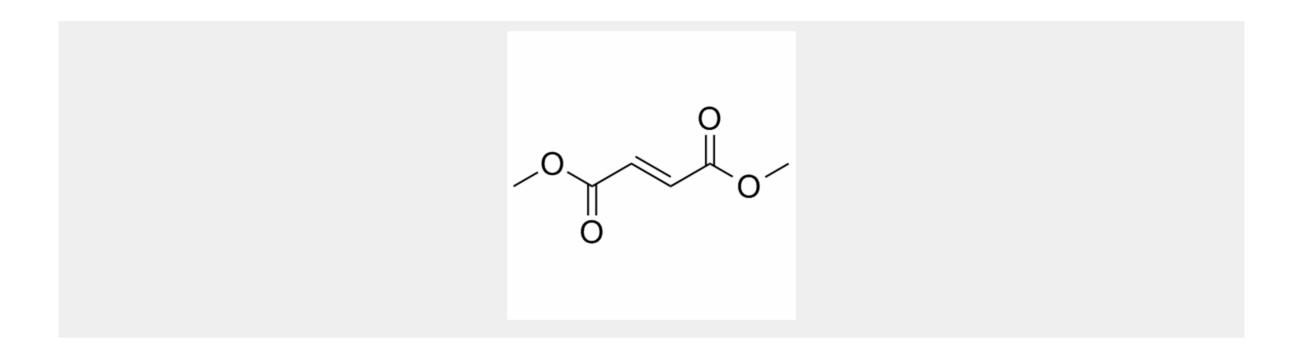
In Vitro: Dimethyl fumarate causes short-lived oxidative stress, which leads to increased levels and nuclear localization of the transcription factor nuclear factor erythroid 2-related factor 2 and a subsequent increase in glutathione synthesis and recycling in

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neuronal cells<sup>[1]</sup>. Dimethyl fumarate inhibits dendritic cell (DC) maturation by reducing inflammatory cytokine production (IL-12 and IL-6) and the expression of MHC class II, CD80, and CD86. Dimethyl fumarate impairs nuclear factor κB (NF-κB) signaling via reduced p65 nuclear translocalization and phosphorylation. Dimethyl fumarate inhibits maturation of DCs and subsequently Th1 and Th17 cell differentiation by suppression of both NF-κB and ERK1/2-MSK1 signaling<sup>[2]</sup>. Dimethyl fumarate inhibits TNF-alpha-induced nuclear entry of NF-kappaB in rat heart endothelial cells (RHEC)<sup>[3]</sup>. Dimethyl fumarate, an immune modulator and inducer of the antioxidant response, suppresses HIV replication and neurotoxin release. Dimethyl fumarate attenuates CCL2-induced monocyte chemotaxis, suggesting that Dimethyl fumarate could decrease recruitment of activated monocytes to the CNS in response to inflammatory mediators<sup>[4]</sup>.

*In Vivo:* Dimethyl fumarate inhibits nuclear entry of NF-kappaB in RHEC and reduces myocardial infarct size after ischemia and reperfusion in rats in vivo<sup>[3]</sup>. Dimethyl fumarate oral administration is shown to upregulate mRNA and protein levels of Nrf2 and Nrf2-regulated cytoprotective genes, attenuate 6-OHDA induced striatal oxidative stress and inflammation in C57BL/6 mice<sup>[5]</sup>.



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