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3rd Edition (Revised in January, 2014)

### **[ Product Information ]**

**Name:** Hirsutenone

**Catalog No.:** CFN98646

**Cas No.:** 41137-87-5

**Purity:** > 98%

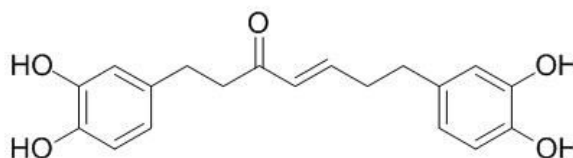
**M.F:** C<sub>19</sub>H<sub>20</sub>O<sub>5</sub>

**M.W:** 328.4

**Physical Description:** Oil

**Synonyms:**

(4E)-1,7-Bis(3,4-dihydroxyphenyl)-4-hepten-3-one



### **[ Intended Use ]**

1. Reference standards;
2. Pharmacological research;
3. Synthetic precursor compounds;
4. Intermediates & Fine Chemicals;
5. Others.

### **[ Source ]**

The leaves of *Alnus nepalensis*

## **[ Applications ]**

Treatment of MCF10A cells with 12-O-tetradecanoylphorbol-13-acetate (TPA) led to the expression of COX-2 and MMP-9. Hirsutenone at 12 microM inhibited the TPA-induced COX-2 expression at both the transcriptional and posttranscriptional levels. Hirsutenone also suppressed the synthesis of prostaglandin E(2), one of the major products of COX-2, and its catalytic activity. The upregulation of Metalloproteinases (MMP-9) by TPA was also significantly reduced by hirsutenone. Likewise, hirsutenone attenuated the invasiveness and motility of MCF10A cells stimulated with TPA. Hirsutenone blocked the TPA-induced DNA binding of nuclear factor kappa B (NF-kappaB) and translocation of p65, the functionally active NF-kappaB subunit, to the nucleus. The luciferase reporter gene assay revealed that hirsutenone abrogated the transcriptional activity of NF-kappaB. Treatment of MCF10A cells with N-alpha-Tosyl-L-phenylalanine chloromethyl ketone, a specific inhibitor of NF-kappaB, reduced the TPA-induced expression of COX-2 and MMP-9. In summary, hirsutenone inhibits the TPA-induced upregulation of COX-2 and MMP-9 in human breast epithelial cells, possibly by targeting NF-kappaB, which may contribute to its chemopreventive effects.

Hirsutenone, dexamethasone, ERK inhibitor or Bay 11-7085 (an inhibitor of NF-kappa B activation) reduced the lipopolysaccharide-induced production of cytokines IL-1 beta and IL-8, and the chemokine CCL17. Hirsutenone, ERK inhibitor or Bay 11-7085 also prevented the lipopolysaccharide-induced expression of Toll-like receptor 4, the phosphorylation of inhibitory kappa B-alpha, the activation of NF-kappa B and the expression of ERK. The results show that hirsutenone may reduce the lipopolysaccharide-stimulated production of inflammatory mediators in keratinocytes by suppressing the Toll-like receptor 4 expression-mediated NF-kappa B activation that is regulated by the ERK pathway. These findings suggest that hirsutenone may exert a preventive effect against microbial endotoxin lipopolysaccharide-induced inflammatory skin diseases through inhibition of ERK pathway-mediated NF-kappa B activation.

## **[ Solvent ]**

Chloroform, Dichloromethane, Diethyl ether, DMSO, Acetone, etc.

## **[ HPLC Method ]**

Mobile phase: Methanol : 0.1% Acetic acid H<sub>2</sub>O=65:35;

Flow rate: 1.0 ml/min;

The wave length of determination: 280 nm.

## **[ Storage ]**

2-8°C, Protected from air and light, refrigerate or freeze.

## **[ References ]**

1. *European Review for Medical and Pharmacological Sciences*, 2012, 16, 853-859.
2. *Phytomedicine*, 2013, 20(2), 124-132.
3. *FEBS Lett.*, 2006, 580(2), 385-392.
4. *INTERNATIONAL IMMUNOPHARMACOLOGY*, 2010, 10(4), 520.