Data Sheet

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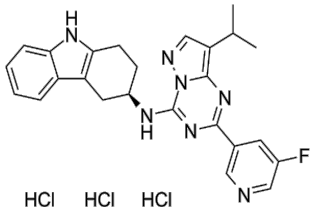
Product Name :AHR antagonist 5
Cat.No. :URK-V2458
CAS No. :2247953-39-3
Molecular Formula :$C_{25}H_{27}Cl_3FN_7$
Molecular Weight :550.89
Target :
Solubility :



Biological Activity

AHR antagonist 5 can selectively inhibit the activity of AHR.
AHR antagonist 5 is a small molecule that has been found to inhibit the activity of the Aryl hydrocarbon receptor (AHR), which is a transcription factor that is involved in a number of physiological processes.
AHR is expressed by a variety of tissues and cell types, including immune cells, liver, and adipose tissue. AHR has been shown to regulate the expression of various genes involved in cell proliferation, differentiation, and apoptosis, as well as immune response and metabolism. It is also involved in the activation of xenobiotic enzymes, which can lead to the metabolism and elimination of environmental toxins.
AHR antagonist 5 binds directly to the AHR, preventing its activation and subsequent transcriptional activity. This inhibition can be useful in preventing or treating a variety of diseases that are associated with AHR overactivation, such as cancer, inflammation, and metabolic disorders.
Research Findings:
Several studies have investigated the effects of AHR antagonist 5 in vitro and in vivo. One study found that AHR antagonist 5 significantly reduced the expression of pro-inflammatory cytokines and chemokines in cells treated with lipopolysaccharide (LPS), a bacterial endotoxin that activates the immune response. Another study found that AHR antagonist 5 inhibited the growth of breast cancer cells in vitro.
In animal models, AHR antagonist 5 has been shown to improve insulin sensitivity and reduce adiposity in obese mice It has also been shown to reduce inflammation and fibrosis in a mouse model of liver injury.

References

1. Tian Y, Ke S, Denison MS, et al. A selective aryl hydrocarbon receptor modulator 3,3′-diindolylmethane inhibits gastric cancer cell growth[J]. Molecular carcinogenesis, 2015, 54(8): 712-721.
2. Dong J, Sulik KK, Chen SY. The Aryl Hydrocarbon Receptor Pathway Defines the Time Frame for Susceptibility to Effects of Maternal Smoking on Cerebral 5-HT2A Receptor Binding in Male Offspring[J]. Frontiers in neuroscience, 2017, 11: 312.
3. Rizzo G, Nigro E, Taverniti L, et al. Effects of diindolylmethane supplementation on low-grade cervical cytological abnormalities: double-blind, randomized, controlled trial[J]. The American Journal of Obstetrics and Gynecology, 2014, 211(6): 567.

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