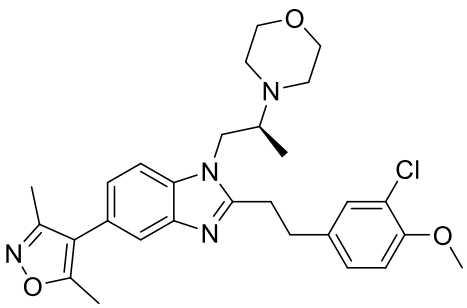
Product data sheet

MedKoo Cat#: 406442 Name: SGC-CBP30 CAS#: 1613695-14-9 Chemical Formula: $C_{28}H_{33}ClN_4O_3$ Exact Mass: 508.22412 Molecular Weight: 509.03962	
Product supplied as:	Powder
Purity (by HPLC):	≥ 98%
Shipping conditions	Ambient temperature
Storage conditions:	Powder: -20°C 3 years; 4°C 2 years.
	In solvent: -80°C 3 months; -20°C 2 weeks.

1. Product description:
SGC-CBP30 is a potent and selective inhibitor of CREBBP (CBP) and EP300; which are general transcriptional co-activators. CREBBP has also been associated with Amyotrophic Lateral Sclerosis (ALS) or Lou GehrigÂ's disease, a neurodegenerative disease with progressive degeneration of motor neurons in the brain and spinal cord, Alzheimer's disease and poly glutamine repeat diseases such as Spinal and Bulbar Muscular Atrophy and HuntingtonÂ's disease. (http://www.thesgc.org/chemical-probes/SGC-CBP30).

2. CoA, QC data, SDS, and handling instruction
SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under "QC And Documents" section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

3. Solubility data

Solvent	Max Conc. mg/mL	Max Conc. mM
DMSO	47.0	92.33
Ethanol	53.0	104.12

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	1.96 mL	9.82 mL	19.64 mL
5 mM	0.39 mL	1.96 mL	3.93 mL
10 mM	0.20 mL	0.98 mL	1.96 mL
50 mM	0.04 mL	0.20 mL	0.39 mL

5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator
Please refer the product web page under section of "Calculator"

6. Recommended literature which reported protocols for in vitro and in vivo study
In vitro study

1. Zhu YX, Shi CX, Bruins LA, Wang X, Riggs DL, Porter B, Ahmann JM, de Campos CB, Braggio E, Bergsagel PL, Stewart AK. Identification of lenalidomide resistance pathways in myeloma and targeted resensitization using cereblon replacement, inhibition of STAT3 or targeting of IRF4. Blood Cancer J. 2019 Feb 11;9(2):19. doi: 10.1038/s41408-019-0173-0. PMID: 30741931; PMCID: PMC6370766.
2. Sun J, Zhang W, Tan Z, Zheng C, Tang Y, Ke X, Zhang Y, Liu Y, Li P, Hu Q, Wang H, Mao P, Zheng Z. Zika virus promotes CCN1 expression via the CaMKIIα-CREB pathway in astrocytes. Virulence. 2020 Dec;11(1):113-131. doi: 10.1080/21505594.2020.1715189. PMID: 31957543; PMCID: PMC6984649.

In vivo study

1. Bi X, Jiang B, Zhou J, Fan X, Yan X, Liang J, Luo L, Yin Z. CBP Bromodomain Inhibition Rescues Mice From Lethal Sepsis Through Blocking HMGB1-Mediated Inflammatory Responses. Front Immunol. 2021 Feb 2;11:625542. doi: 10.3389/fimmu.2020.625542. PMID: 33603756; PMCID: PMC7884462.

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7. Bioactivity

Biological target:

SGC-CBP30 is a potent and highly selective CBP/p300 bromodomain (Kds of 21 nM and 32 nM for CBP and p300, respectively) inhibitor, displaying 40-fold selectivity over the first bromodomain of BRD4 [BRD4(1)] bound.

In vitro activity

In order to know whether SGC-CBP30 treatment has an effect on IL6 autocrine secretion in XG1LenRes cells, we measured IL6 in the culture media of XG1LenRes in the absence and presence of drug treatment. As shown in Fig,6c, SGC-CBP30 treatment reduced IL6 autocrine production from XG1LenRes in either the absence or presence of lenalidomide. Accordingly, STAT3 activation was inhibited by SGC-CBP30 treatment (Fig.6b). Finally, we demonstrated that SGC-CBP30 also increased the sensitivity to lenalidomide in three IMiD-sensitive HMCLs (supplementary Figure 9D–F). SGC-CBP30 treatment could change the chromatin structure in the IRF4 and MYC regulatory regions or regulate other genes in IMiDs-mediated activity, therefore inducing increased sensitivity to IMiD-induced signals (such as downregulation of IKZF1/IKZF3).

Reference: Blood Cancer J. 2019 Feb; 9(2): 19. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6370766/

In vivo activity

Lung injury in septic mice was characterized by alveolar septal thickening, extensive edema, massive infiltration of inflammatory cells, and alveolar congestion/collapse. In lungs from the SGC-CBP30-8 h therapy group, LPS- or CLP-induced histopathological damage and accumulation of inflammatory cells were attenuated (Figures 2A, B, first-line). Semi-quantitative assessment of lung histology revealed that administration of SGC-CBP30 at 8 h following LPS or CLP significantly decreased tissue injury in the lung, similar to the positive control DEX-0.5 h. In the SGC-CBP30-8 h treatment group, infiltrated inflammatory cells were significantly reduced and liver architecture was significantly improved (Figures 2A, B, third-line). As shown in Semi-quantitative statistics, administration of SGC-CBP30 at 8 h or DEX at 0.5 h after septic modeling markedly reduced liver injury scores. It is clear that the delayed SGC-CBP30 administration at 8 h significantly attenuated the histopathological deterioration and inflammatory cell infiltration in renal tissues. Together, these results demonstrate that selective CBP bromodomain inhibitors such as SGC-CBP30 may be effective not only to suppress inflammatory response in organs, but also to ameliorate organ injury caused by LPS-induced endotoxemia and CLP-induced sepsis.

Reference: Front Immunol. 2020; 11: 625542. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7884462/

Note: The information listed here was extracted from literature. MedKoo has not independently retested and confirmed the accuracy of these methods. Customer should use it just for a reference only.