# Data Sheet

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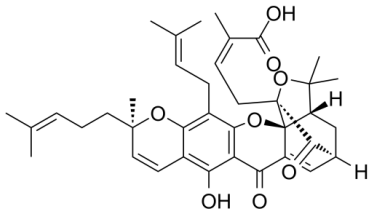
| **Product Name:** | Gambogic Acid |
| **Cat. No.:** | CS-1456 |
| **CAS No.:** | 2752-65-0 |
| **Molecular Formula:** | C38H44O8 |
| **Molecular Weight:** | 628.75 |
| **Target:** | Autophagy; Bcl-2 Family |
| **Pathway:** | Apoptosis; Autophagy |
| **Solubility:** | H2O : < 0.1 mg/mL (insoluble); DMSO : ≥ 100 mg/mL (159.05 mM) |



## BIOLOGICAL ACTIVITY:

Gambogic Acid (Beta-Guttiferrin) is derived from the gamboges resin of the tree Garcinia hanburyi. Gambogic Acid (Beta-Guttiferrin) inhibits **Bcl-$X_L$**, **Bcl-2**, **Bcl-W**, **Bcl-B**, **Bfl-1** and **Mcl-1** with **$IC_{50}$**s of 1.47 μM, 1.21 μM, 2.02 μM, 0.66 μM, 1.06 μM and 0.79 μM. IC50 & Target: IC50: 1.47 μM (Bcl-$X_L$), 1.21 μM (Bcl-2), 2.02 μM (Bcl-W), 0.66 μM (Bcl-B), 1.06 μM (Bfl-1) and 0.79 μM (Mcl-1)[1] **In Vitro:** Gambogic Acid (Beta-Guttiferrin) is a medicinal compound derived from the gamboges resin of the tree, Garcinia hanburyi. Gambogic Acid has documented cytotoxic activity against tumor cell lines in culture, with concentrations required for killing 50% of cells ($LD_{50}$ of ~1 μM). The activity of Gambogic Acid against the 6 human anti-apoptotic Bcl-2-family proteins is contrasted, using FPAs. Gambogic Acid displaces to various extents FITC-BH3 peptide binding to all 6 proteins, with apparent $IC_{50}$ 1.47 μM for Bcl-$X_L$, 1.21 μM for Bcl-2, 2.02 μM for Bcl-W, 0.66 μM for Bcl-B, 1.06 μM for Bfl-1, and 0.79 μM for Mcl-1[1]. The growth inhibitory effects of Gambogic Acid or Cisplatin (CDDP) on A549, NCI-H460, and NCI-H1299 cells are assessed by the MTT assay after 48 h exposure. A concentration-dependent inhibition of cell growth is observed with Gambogic Acid and CDDP, with $IC_{50}$s of 3.56±0.36 and 21.88±3.21 μM for A549 cells, 4.05±0.51 and 25.76±4.03 μM for NCI-H460 cells, and 1.12±0.31 μM and 25.21±4.38 μM for NCI-H1299 cells[2]. **In Vivo:** To further investigate whether Gambogic Acid (Beta-Guttiferrin) synergises CDDP against tumour growth in vivo, A549 tumors are implanted in SCID mice. When mice are treated with CDDP combined with Gambogic Acid, the tumor inhibition rate is 69.3%, whereas those of mice treated with CDDP and GA alone are 57.2% and 29.0%, respectively[2].

## PROTOCOL (Extracted from published papers and Only for reference)

**Cell Assay:** [2]The in vitro cell viability effects of Gambogic Acid, CDDP alone, or combined treatments are determined by MTT assay. The cells ($2\times10^4$ cells per mL) are seeded into 96-well culture plates. After overnight incubation, **A549 cells** are treated with **Gambogic Acid (0.44, 0.88, 1.75, 3.5, 7, 10.5 and 14 μM)**; **NCI-H460 cells** are treated with **Gambogic Acid (0.5, 1, 2, 4, 8, 12 and 16 μM)**; **NCI-H1299 cells** are treated with **Gambogic Acid (0.125, 0.25, 0.5, 1, 2 and 4 μM)**. For the combined treatment in NSCLC cells, three sequences are tested: (a) Gambogic Acid followed by CDDP cells are exposed to Gambogic Acid for 48 h, and then after washout of Gambogic Acid, cells are treated with CDDP for an additional 48 h; (b) CDDP followed by Gambogic Acid cells are exposed to CDDP for 48 h, and then after washout of CDDP, cells are treated with Gambogic Acid for an additional 48 h; and (c) concurrent treatment cells are exposed to both Gambogic Acid and ADM for 48 h. The nature of the drug interaction is analysed by using the combination index (CI)[2].

**Animal Administration:** [2]Mice[2]

To determine the in vivo antitumour activity of Gambogic Acid combined with CDDP, viable A549 cells ($5\times10^6$/100 μL PBS per mouse) are subcutaneously injected into the right flank of **7- to 8-week-old male SCID mice**. When the average tumor volume reach 100 $mm^3$, the mice are randomly divided into four treatment groups, including control (saline only, n=5), **Gambogic Acid (3.0 mg/kg per 2 days, intravenously; n=6)**, CDDP (4 mg/kg per week, intravenously; n=6), and sequential combination (CDDP treatment one day before Gambogic Acid treatment, n=6). CDDP (4 mg/kg, weekly) is generally administered at doses less than the maximum-tolerated

dose in an attempt to allow any additive effects of combination treatment with platinum-based agents and Gambogic Acid to be more easily detected. Tumor size is measured once every 2 days with a calipre. Body weight is recorded once every 2 days. After 14 days, the mice are killed and the tumors are excised and stored at -80 °C until further analysis.

## References:


[1]. Zhai D, et al. Gambogic acid is an antagonist of antiapoptotic Bcl-2 family proteins. Mol Cancer Ther. 2008 Jun;7(6):1639-46.

[2]. Wang LH, et al. Gambogic acid synergistically potentiates cisplatin-induced apoptosis in non-small-cell lung cancer through suppressing NF-κB and MAPK/HO-1 signalling. Br J Cancer. 2014 Jan 21;110(2):341-52.


## CAIndexNames:

2-Butenoic acid,2-methyl-4-[(1R,3aS,5S,11R,14aS)-3a,4,5,7-tetrahydro-8-hydroxy-3,3,11-trimethyl-13-(3-methyl-2-buten-1-yl)-11-(4-methyl-3-penten-1-yl)-7,15-dioxo-1,5-methano-1H,3H,11H-furo[3,4-g]pyrano[3,2-b]xanthen-1-yl]-,(2Z)-

## SMILES:

O=C(O)/C(C)=C\C[C@@]1(C2=O)OC(C)(C)[C@@](C[C@@]2([H])C=C34)([H])[C@]31OC5=C(C(O)=C(C=C[C@](CC/C=C(C)/C)(C)O6)C6=C5C/C=C(C)\C)C4=O

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