Table S1. Examples of Involvement of GSK-3 Isoforms in Cancer.

Cancer Type (alphabetical)	GSK-3 Isoform	Function	Type of Study	Reference
Bladder cancer	GSK-3β	Prognostic marker and therapeutic target. Inhibition of GSK-3 resulted in apoptosis. GSK-3 was serving as a tumor promoter. Aberrant nuclear accumulation of GSK-3β in 62% and 91% of noninvasive and invasive human urothelial carcinomas. GSK-3β nuclear staining was associated with poor prognosis.	Human tumor samples and in vitro	[1]
Brain cancer	GSK-3β	Brain-derived neutrophilic factor/TrkB induced phosphorylation of GSK-3β which resulted in its inactivation and contributed to chemotherapeutic drug resistance. GSK-3β was acting as a tumor suppressor.	In vitro	[2]
Brain cancer	GSK-3β	Inhibition of AKT mediated phosphorylation of GSK-3β by an AKT inhibitor reduced cell growth. GSK-3β was acting as a tumor suppressor.	In vitro	[3]
Brain cancer	GSK-3β	GSK3β was linked with increased expression of TP53 and $p21^{Cip\text{-}1}$ in glioblastoma cells with wild-type p53 and with decreased Rb phosphorylation and expression of cyclin-dependent kinase 6, Treatment with GSK-3 inhibitor AR-A014418 sensitized GMB cells to temozolomide. GSK-3β was functioning as a tumor promoter.	Human tumor samples, in vitro studies.	[4]
Brain cancer	GSK-3β	Expression of high levels of GSK-3β was associated with poor prognosis. Treatment with a combination of temozolomide other drugs used to treat brain cancer improved prognosis. GSK-3β was acting as a tumor promoter.	In vitro, in vivo, clinical trial, 7 patients in clinical study	[5]
Brain cancer		Suppression of GSK-3β by miR-101 restored sensitivity to temozomide in brain cancer.	In vitro, in vivo	[6]

		GSK-3β was acting as a tumor promoter.		
Breast cancer	GSK-3β	GSK-3β expression was associated with MCL1 expression and inactivation. GSK-3β was acting as a tumor suppressor. High MCL1 expression was associated with poor prognosis and high P-GSK-3β (inactive) expression.	Human tumor samples (125 breast cancer), in vitro	[7]
Breast cancer	GSK-3β	High GSK-3β expression was associated with reduced distant relapse-free survival (DRFS). Tissue microarrays of 1,686 patients, low expression in 36%, high expression in 38%. GSK-3β was acting as a tumor promoter.	Human tumor samples.	[8]
Breast cancer	GSK-3β	Inhibition of GSK-3β inhibited tumor growth GSK-3β was acting as a tumor promoter.	In vitro, in vivo	[9]
Breast cancer	GSK-3β	miR-34a binding to the *PRKD1* suppressed cancer stemness through the GSK3/β-catenin signaling pathway. GSK-3 was acting as a stemness suppressor.	In vitro, in vivo	[10]
Breast cancer	GSK-3β	GSK-3 inhibition by the human THUMP domain-containing protein 1 (THUMPD1)/AKT resulted in SNAIL activation. GSK-3 was acting as a tumor suppressor.	In vitro, in vivo	[11]
Cervical cancer	GSK-3β	High expression of forkhead box M1 (FOXM1) transcription factor was associated with poor prognosis and it activated AKT and inactivated GSK-3β which resulted in higher SNAIL activity and poor prognosis. GSK-3β was acting as a tumor suppressor.	In vitro, human tumor samples	[12]
Colorectal cancer	GSK-3β	Nuclear accumulation of GSK-3β was observed in 39% (33/85) and associated with short overall survival, larger tumor size, distant metastasis and loss of membranous β-catenin. This loss was present in 37% and associated with poor survival. Nuclear expression of GSK-3β and loss of membrane β-catenin were present in CRC with worse	Human tissue microarrays	[13]

		prognosis. GSK-3β was functioning as a tumor promoter,		
Colorectal cancer	GSK-3β	GSK-3β increased NF-κB expression, inhibition of GSK-3 inhibited growth. GSK-3β was serving as a tumor promoter.	Human tumors and *in vitro* studies	[14]
Colorectal cancer	GSK-3β	CXCL5 induced ERK/ELK1/SNAIL and AKT/β-catenin, inhibited GSK-3β and promoted cancer metastasis. GSK-3β was acting as a tumor promoter.	In vitro, in vivo. chemokine ELISA arrays from CRC patients	[15]
Gastric cancer	GSK-3β	P-GSK-3β (T216, active) was expressed in 46% of cases and associated with a good prognosis. GSK-3β was acting as a tumor suppressor.	Human tissue arrays containing 281 gastric cancer specimens and in vitro studies	[16]
Gastric cancer	GSK-3β	Higher GSK-3β levels were associated with a better prognosis. GSK-3β was acting as a tumor suppressor.	Gene expression profiling in 63 tumors	[17]
Hepatocellular carcinoma	GSK-3β	S9-P-GSK-3β was over-expressed in 50% of tumor tissues and was associated with a poor prognosis. GSK-3β was acting as a tumor suppressor.	178 patients with HCC after curative partial hepatectomy	[18]
Hepatocellular carcinoma	GSK-3β	Protein arginine methyltransferase 9 (PRMT9) activation of PI3K/AKT resulted in decreased GSK-3β activity and increased SNAIL signaling. GSK-3β was acting as a tumor suppressor	In vitro, in vivo, human tumor samples	[19]
Laryngeal Cancer	GSK-3β	Suppression of miR-27a interaction with GSK-3β altered laryngeal differentiation in response to retinoic acid treatment. GSK-3β. GSK-3β was acting as a tumor suppressor.	In vitro, human tumor samples	[20]
Laryngeal Cancer	GSK-3β	Alterations in the Tat-interacting protein 30 (TIP30) tumor suppressor expression resulted in activation of AKT, inactivation of GSK-3β, deregulation of β-catenin and poor prognosis. Low TIP30 staining was observed in 43.8% of patient samples while minimal TIP30 staining in non-tumor cells was observed in	In vitro, human tumor samples, 105 laryngeal carcinomas	[21]

		25.7% of samples. GSK-3β was acting as a tumor suppressor.		
Leukemia	GSK-3β and GSK-3α	Genetic deletion of GSK-3β in mice led to myelodysplastic disease syndrome (MDS), subsequent deletion of GSK-3α led to AML. Different roles of GSK-3α and GSK-3β in MDS progression into AML. GSK-3α and GSK-3β were acting as tumor suppressors.	Gene knock out studies in mice, gene profiling.	[22]
Leukemia	GSK-3α and GSK-3β	GSK-3 stimulated acute lymphoblastic leukemia with mixed-lineage leukemia gene (MLL) growth by destabilization of the cyclin-dependent kinase inhibitor p27(Kip1). GSK-3 promoted growth, GSK-3 was acting as a tumor promoter.	In vitro, in vivo, in human AML patients	[23]
Leukemia	GSK-3α	GSK-3α was a target in AML. GSK-3α was serving as a tumor promoter.	Chemical small molecule screening, in vitro, in vivo	[24]
Leukemia (AML)	GSK-3α and GSK-3β	GSK-3α and GSK-3β phosphorylation leading to their inhibition correlated with poor prognosis. S21-P-GSK3α and S9-P-GSK-3β positively correlated with phosphorylation of AKT, BAD, and P70S6K, and negatively correlated with β-catenin and FOXO3A. GSK-3α and GSK-3β were serving as tumor suppressors	In vitro, human patient samples, reverse phase protein analysis (RPPA) in a cohort of 511 AML patients	[25]
Leukemia (Natural Killer Cells cytotoxic to AML)	GSK-3β and GSK-3α	(GSK-3β) expression was elevated in AML-NK cells and decreased their activity as NK cells. Inhibition of GSK-3 restored NK cytotoxicity by increasing TNF-α production. GSK-3 was serving as a tumor suppressor.	In vitro, in vivo	[26]
Lung cancer	GSK-3β	High levels of TGFβ induced integrin β3/AKT, inhibited GSK-3β activity, and induced SNAIL activity and promoted metastatic potential. GSK-3β was acting as a tumor suppressor.	In vitro, in vivo, clinical data base	[27]

Lung cancer	GSK-3α	CREB induced GSK-3α which promoted lung cancer cell growth. GSK-3α was acting as a tumor promoter.	In vitro, in vivo, human tumors	[28]
Lung cancer	GSK-3α and GSK-3β	Tivantinib was initially thought to be a c-MET inhibitor. Subsequently, GSK-3α and GSK-3β were determined to be targets of tivantinib in lung cancer cells. GSK-3α and GSK-3β were acting as tumor promoter	In vitro	[29]
Lung cancer (non-small cell)	GSK-3α and GSK-3β	GSK-3β levels were elevated in 41% of human NSCLC samples and led to increased proliferation in comparison to normal tissues. GSK-3β was acting as a tumor promoter.	In vitro, in vivo, 29 human tumor specimens	[30]
Melanoma	GSK-3α	Elevated expression of GSK-3α in 72% of samples, but not GSK-3β. 80% of tumors expressed elevated levels of catalytically active phosphorylated GSK-3α (Y279-P-GSK-3α), but not phosphorylated GSK3β (Y216-P-GSK-3β). Inhibition of GSK-3α induced apoptotic death to retard tumorigenesis. GSK-3α was acting as a tumor promoter.	In vitro, in vivo, 39 human tumor samples.	[31]
Melanoma	GSK-3β	Neuron navigator 2 (NAV2) inhibited GSK-3β which increased β-catenin and SNAIL activity. GSK-3β was acting as a tumor suppressor.	In vitro, in vivo, human tumor samples	[32]
Myeloma	GSK-3α and GSK-3β	Treatment with Thiadiazolidinone (TDZD; a GSK-3 non-competitive inhibitor) resulted in Forkhead transcription factors (FOXO3a) activation. TDZD induced apoptosis in primary myeloma cells but not in normal CD34 cells. GSK-3 was acting as a tumor promoter.	In vitro, human myeloma cells, primary hematopoietic cells	[33]
Neuroblastoma	GSK-3β	Inhibition of GSK-3β with 9-ING-41 suppressed growth via inhibition of XIAP. GSK-3β was acting as a tumor promoter.	In vitro, in vivo	[34]
Oral Cancer	GSK-3β	AKT and GSK-3β expression was associated with a poor prognosis. Phosphorylated	Human tumor specimens (118 patient samples	[35]

		GSK-3β (inactive) was associated with cervical lymph node (CLN) metastasis. GSK-3β was acting as a tumor suppressor.	and normal controls).	
Oral squamous cell cancer	GSK-3α and GSK-3β	Links between GSK-3α and GSK-3β and cyclin D1 and TP53. Inactive GSK-3β was expressed at higher levels than inactive GSK-3α. Inactive GSK-3β was detected at increased percentages in older patients (40->70 years old) than younger patients (<40 years old). GSK-3β was acting as a tumor suppressor.	In vitro, 179 human patient samples	[36]
Osteosarcoma	GSK-3β	The P2X7 receptor promoted PI3K/AKT and β-catenin activity and inhibited GSK-3β. GSK-3β was acting as a tumor suppressor.	In vitro, in vivo, human tumor samples	[37]
Ovarian cancer	GSK-3β	GSK-3 expression was associated with increased tumor growth, poor prognosis and chemoresistance. GSK-3 was functioning as a tumor promoter.	In vitro, in vivo, 71 human tumor samples.	[38]
Ovarian cancer	GSK-3β	Constitutively active GSK-3β induced entry into the S phase, increased cyclin D1 expression and facilitated the proliferation of ovarian cancer cells. GSK-3 inhibition prevented the tumor formation of the tumor in nude mice. GSK-3 was acting as a tumor promoter.	In vitro, in vivo	[39]
Pancreatic cancer	GSK-3α and GSK-3β	GSK-3 promoted NF-κB activity. GSK-3β may have been the more important isozyme in regulating in NF-κB. GSK-3β was acting as a tumor promoter.	Human tumors and in vitro studies.	[40]
Pancreatic cancer	GSK-3β	Inhibition of GSK-3 activity caused stabilization of β-catenin activity. GSK-3β expression was a strong prognosticator in PDAC. High expression of GSK-3β was associated with better survival. PDAC Patients with GSK-3β expression > than the third quartile (Q3) had a 46% reduced risk of dying of	Immuno-fluorescence on human tumor microarray from 163 patients.	[41]

		pancreatic cancer. GSK-3β was acting as a tumor suppressor.		
Prostate Cancer	Both	GSK-3α and GSK-3β were detected at higher levels in 25/79 and 24/79 tumor samples respectively, in comparison to normal prostatic tissue. GSK-3α was elevated in low Gleason sum score tumors while GSK-3β was expressed in high Gleason tumors, and both isoforms correlated with high expression of the androgen receptor (AR). Treatment with a GSK-3 inhibitor suppressed proliferation. GSK-3 was functioning as a tumor promoter.	In vitro, in vivo and in 79 human tumor samples	[42]
Renal Cell Carcinoma	GSK-3β	miR-199a downregulated GSK-3β and suppressed growth of RCC. GSK-3β was acting as a tumor promoter.	Human tumor samples and in vitro.	[43]
Renal Cell Carcinoma	GSK-3β	miR-203a targeting GSK-3β was detected at high levels in RCC and associated with a poor prognosis. miR-203a was overexpressed in 27 of 40 (68%) RCC patient samples. GSK-3β was acting as a tumor suppressor.	In vitro, 40 RCC tumor samples.	[44]
Thyroid carcinomas	GSK-3α and GSK-3β	Junctional adhesion molecule A (JAM-A) was downregulated in anaplastic thyroid carcinomas and resulted in increased GSK-3α, GSK-3β, and TP53 phosphorylation.	Human tissue arrays	[45]
Tongue (oral) cancer	GSK-3β	GSK-3β was detected at lower levels in 39% of patient samples in comparison to normal epithelial cells and was associated with reduced survival. In contrast, cyclinD, a target of GSK-3β was detected at higher levels in 65.9% of samples and was associated with a poor prognosis. GSK-3β was acting as a tumor suppressor	41 Human tissue samples, immunohisto-chemistry.	[46]

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Table S2. Examples of Preclinical Studies with GSK-3 Inhibitors and Nutraceuticals/Natural Products Involving Cancer Models

Molecule	Result	Reference
GSK-3 Inhibitors		
Lithium chloride	Lithium chloride inhibited GSK-3 which suppressed proliferation in Eca-109 human esophageal cancer cells. GSK-3 was functioning as a tumor promoter.	[1]
AR-A014418	Treatment with GSK-3β inhibitor AR-A014418 sensitized GMB cells to temozolomide. GSK-3β was functioning as a tumor promoter.	[2]
BIO	BIO induced apoptosis, cell cycle arrest in glioblastoma cells.	[3]
Tideglusib, AZD1080, and BIO	These GSK-3 inhibitors suppressed GSK-3 mediated phosphorylation of substrates involved in proliferation such as c-MYC in KRAS-dependent tumors.	[4]
ABC1183	ABC1183 inhibited GSK-3α and GSK-3β. ABC1183 inhibited the growth of a numerous cancer cell lines by decreasing cell survival by inducing G_2/M arrest by altering GSK-3 and WNT/β-catenin signaling.	[5]
SB21673	SB21673 inhibits GSK-3α and GSK-3β. c-JUN degradation was enhanced by SB21673 and breast cancer tumorigenesis was inhibited.	[6]
SB216763, GSK inhibitor XIII, and AR-A014418	SB216763 and the GSK inhibitor III suppressed AR-transcriptional activity as well as AR expression in prostate cancer cells. In contrast, AR-A014418 stimulated proliferation.	[7]
Lithium chloride, SB216763, and GSK-3 IX (BIO)	Treatment of MLL LSC with GSK-3 inhibitors resulted in reversion of MLL LSCs to a pre-LSC stage and reduced their growth.	[8]
GSK-3 IX (BIO) and SB216763	Inhibition of GSK-3 suppressed maintenance of MLL leukemia.	[9]
GSK3-IX	The GSK-3α and GSK-3β inhibitor GSK3-IX inhibited MLL leukemia maintenance and growth.	[9]
GS87	GS87 is a novel GSK-3 inhibitor that was isolated upon screening for more optimal effective inhibitors that induce AML differentiation. GS87 inhibits both GSK-3α and GSK-3β.	[10]
Thiadiazolidinone (TDZD)	TDZD is a non-competitive inhibitor of GSK-3. Treatment of human myeloma cells with TDZD resulted in apoptosis in primary myeloma cells but not in normal CD34 cells.	[11]
Combination of GSK-3 inhibitors with immunotherapy		
SB415286 and CD8+ CTLs	GSK-3 inhibitor treatment of CD8+ T cells inhibited TBX21 (T-bet) expression and decreased PD-1 expression and increased cytolytic T cell responses.	[12]
LY2090314, tideglusib, SB415286 GSK-3 inhibitors and NK cells	Treatment of NK cells with GSK-3 inhibitors LY2090314, tideglusib or SB415286, increased TNF-α levels and cytotoxicity towards AML cells.	[13]
SB216763 and GMB-specific CAR-T cells	Treatment with GSK-3 inhibitor of antigen specific CAR-T cells lowered PD-1 expression and promoted long term survival, memory and tumor elimination.	[14]
Enzastaurin	Enzastaurin was initially developed as a PKC-β inhibitor. One of its targets is GSK-3. It has been examined in clinical	[15]

	studies with various cancer types, often in combination with bevacizumab.	
SB415286 or LiCl and TRAIL	Inhibition of GSK-3 enhanced the induction of apoptosis mediated by TRAIL in gastric cancer cells.	[16]
Combination of GSK-3 inhibitors with chemotherapy		
CHIR99021 and paclitaxel	Effects of combination of the GSK-3 inhibitor CHIR99021 and paclitaxel on lung cancer.	[17]
SB415286, RO 318220, lithium chloride and paclitaxel	SB415286 inhibits both GSK-3α and GSK-3β. RO 318220 inhibits PKC and GSK-3. More mitotic arrest was observed when GSK-3 inhibitors were combined with paclitaxel than in the absence of the GSK-3 inhibitors.	[18]
LY2090314 and nab-paclitaxel	LY2090314 suppressed TAK1 levels. LY2090314 plus nab-paclitaxel combined treatment increased the survival of mice in orthotopic pancreatic tumor models.	[19]
AR-A01441, TDZD-8, 9-ING-41 and Camptosar	AR-A01441, TDZD-8, and 9-ING-41 suppressed neuroblastoma growth, 9-ING-41 was most effective. The combination of 9-ING-41 and Camptosar was effective in suppressing tumor growth of xenografts.	[20]
9-ING-41, 9-ING-87 and irinotecan	Treatment with GSK-3 inhibitors and the chemotherapeutic drug irinotecan reduced drug resistance in a breast cancer PDX model.	[21]
AR-A014418 and gemcitabine	GSK-3 inhibitor suppressed some of the genes induced by gemcitabine that are involved in drug resistance of PDAC cells.	[22]
Combination of GSK-3 inhibitors with other inhibitors or agonists		
9-ING-41 and either chloroquine and bafilomycin	9-ING-41 have been examined either by itself or in combination with autophagy inhibitors chloroquine and bafilomycin on RCC lines	[23]
lithium chloride, SB216763, inhibitor IX (BIO) and NF-κB inhibitors PDTC parthenolide, or BAY 11-7082 and chemotherapeutic drugs.	Combining GSK-3, NF-κB inhibitors and certain chemotherapeutic drugs resulted in increased osteosarcoma death both in vitro and in animal xenograft studies.	[24]
AR-A014418 and Troglitazone	Treatment of prostate cancer cells with GSK-3 inhibitor and PPAR agonist suppressed NF-κB activity increased cell death.	[25]
6BIO and AR-ASO	6BIO improved the targeting of antisense oligonucleotide (ASO) inhibitor and resulted in increased inhibition of AR signaling.	[26]
AR-A014418, 5-chloro-2,4-dihydroxpyridine (CDHP) and 5FU	GSK-3β inhibitor AR-A014418 induced head and neck cancer stem cells [CD44 (high)/ESA (low)] to undergo mesenchymal-to-epithelial transition (MET) back to CD44 (high)/ESA (high) cells. Furthermore, this combined treatment induced the cells to differentiate.	[27]
Inhibitors originally developed to target other signaling molecules which also target/inhibit GSK-3 activity		
Tivantinib	Tivantinib was initially developed as a c-MET inhibitor but it was subsequently determined to target GSK-3α and GSK-3β in lung cancer cells.	[28]
GDC-0941	GDC-0941 is a PI3K inhibitor. It increased the sensitivity of GBM cells to radiotherapy and reduced chemoresistance to temzolomide.	[29]
AktX. Lithium chloride	AktX is an AKT inhibitor. The effects AktX and lithium chloride on brain cancer cells were determined. AktX	[30]

	suppressed AKT and increased GSK-3β expression and inhibited glioma cell proliferation.	
Zidovudine	Zidovudine is an anti-viral drug. Treatment of drug resistant pancreatic cells with zidovudine resensitized the cells to gemcitabine. Zidovudine suppressed the AKT/GSK-3/SNAIL pathway.	[31]
Doxazosin	Doxazosin is an antihypertensive drug. It was observed to inhibit PI3K/AKT signaling in GBM by upregulation of active GSK-3β and TP53. Treatment with doxazosin was associated with low neurotoxicity.	[32]
Erlotinib, SU11274, XAV939, everolimus	EGFR, c-MET, WNT, mTORC1 blocker treatments in various combinations overcame drug resistance of NSCLC cells.	[33]
miR-101, temozomide	Suppression of GSK-3β by miR-101 inhibits GSK-3β expression and restored sensitivity to temozomide in brain cancer cells.	[34]
Nutraceuticals/Natural Products which may alter GSK-3 activity		
Curcumin	Curcumin suppressed Syk activity which inhibited AKT and induced GSK-3 activity and inhibited B lymphoma growth.	[35]
Curcumin and Tetrahydrocurcumin	Curcumin induced GSK-3 activity and inhibited WNT/β-catenin signaling and suppressed azoxymethane-induced colon carcinogenesis.	[36]
Berberine	Berberine inhibited AKT which resulted in GSK-3 activity in melanoma cells treated with alpha melanocyte stimulating hormone (α-MSH). Berberine suppressed induction of microphthalmia-associated transcription factor (MITF) and tyrosinase activity.	[37]
Berberine and lapatinib	Combining berberine with the dual EGFR and HER receptor inhibitor lapatinib decreased lapatinib-resistance of breast cancer cells. Treatment with berberine and lapatinib induced higher levels of ROS and increased GSK-3 activity and decreased c-MYC levels.	[38]
Resveratrol	Resveratrol increased GSK-3 activity which suppressed WNT/β-catenin signaling and decreased invasion and migration in breast cancer cells.	[39]
Apocynin	The effects of apocynin and resveratrol on pancreatic cancer cells were mediated by decreased levels of phosphorylated GSK-3β and ERK1/2 present in the nucleus.	[40]
Microsclerodermin A	Microsclerodermin A inhibited NF-κB activity in PDAC. Potential involvement of GSK-3.	[41]
Caffeine	Caffeine inhibited JB6 mouse epidermal cells proliferation by suppression of AKT and activation of GSK-3.	[42]
Indirubin	Indirubin inhibited GSK-3 and cyclin dependent kinase activity in leukemia cells. Indirubin may have competed for the ATP binding sites in the kinase domains of the proteins.	[43]
Tetrandrine	Tetrandrine inhibited AKT which resulted in GSK-3 activation in colon cancer cells.	[44]
Differentiation-inducing factor-1	Differentiation-inducing factor-1 inhibited AKT and induced GSK-3 activity in colon cancer cells which resulted in apoptosis.	[45]

Dioscin	The effects of dioscin on proliferation were examined with osteosarcoma cells. Dioscin inhibited AKT activity which resulted in GSK-3 activation.	[46]
Nimbolide	Nimbolide inhibited PI3K activity in oral cancer cells which resulted in increased GSK-3 activity and inhibition of cytoprotective autophagy.	[47]
Oridonin	Oridonin increased GSK-3 expression which resulted in c-MYC degradation and growth inhibition and apoptosis in leukemia cells.	[48]
Apicidin	Apicidin resistance in HCC may result from decreased GSK-3 activity and increased WNT/β-catenin activity.	[49]
Wogonin	Wogonin inhibits cell growth and induces apoptosis by inhibiting the expression of GSK-3β in lung cancer cells.	[50]
Sulforaphane	Sulforaphane treatment resulted in induction of miR-19 and suppression of GSK-3β and increased WNT/β-catenin expression.	[51]
Butyrate	Butyrate induced ROS and miR-22/SIRT-1 pathway in hepatic cancer cells which resulted in suppression of AKT, increased PTEN and GSK-3 and apoptosis.	[52]
Ursolic acid	Treatment of ovarian carcinoma cells with ursolic acid resulted in inhibition of GSK-3 and induction of apoptosis	[53]
Gambogenic acid	Gambogenic acid stimulated GSK-3 activity and inhibited growth in GBM cells.	[54]

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