Table S1. Preclinical anticancer activities and molecular mechanisms of α-mangostin in other cancer types not included in Table 1. Note that "–" indicates unavailable data.

Cancer Type	Preclinical Type	Preclinical Model	Treatment Period (Dosage)	Half-Maximal Inhibitory Concentration (IC_{50})	Therapeutic Effect	Molecular Mechanism	Reference
HCC	*In vitro*	Human anoikis-resistant or parental hepatocellular carcinoma cell (HepG2)	24 h (0 - 20 μM)	In adherent (parental) cells: 5.5 μM (CI not available) In suspended (anoikis-resistant) cells: 14 μM (CI not available)	Induced apoptosis; Abrogated metastasis phenotype of both anoikis-resistant (suspension) and the parental (adherent) HepG2 cells; Suppressed EMT; Promoted anoikis in both cells	↑activity of cleaved caspases-3 and pro-apototic proteins - Bax, BimEL and t-Bid and ↓activity of pro-caspases – 8,9 and anti-apoptotic proteins – c-FLIP and Mcl-1; ↓expression of MMP-2 and MMP-9; ↑level of E-cadherin and ↓levels of N-cadherin, vimentin, integrin αv and integrin β1; Downregulated of the PI3K/AKT and ERK pathways - ↓phosphorylation of AKT and ERK	[1]
GBC	*In vitro*	Human gall bladder cancer cells (GBC-SD and NOZ)	24, 48, and 72 h (0 - 16 μM), combined with gemcitabine (0, 10^{-3}, 10^{-2}, 10^{-1}, 1,	5 μM (CI not available)	Suppressed proliferation and colony formation; Induced apoptosis;	↑BAX expression and ↓PCNA and Bcl-2 expression; ↓SERB1, FASN and ACC	[2]

			10^1, 10^2 and 10^3 μM)		Inhibited lipogenesis	expression and ↑p-AMPK expression	
GBC	*In vivo*	Male BALB/c nude mice xenografted with NOZ cells	Daily for 4wks AM: (2 mg/kg) + gemcitabine (40 mg/kg)	-	Potentiated gemcitabine-induced inhibition of tumor growth	-	[2]
OR	*In vitro*	Human oral squamous cell carcinoma (HOSCC) cells (HSC-2, HSC-3, HSC-4, Ca9-22 and SAS)	24 h (20 μM), combined with recombinant human TRAIL (100 ng/ml)	-	Suppressed proliferation; Induced apoptosis; Caused cell-cycle arrest at S/G2/M-phase	↑caspase-9,-3 and 7 expression and release of cytochrome c from mitochondria	[3]
OR	*In vitro*	Human tongue squamous cell carcinoma (SCC25)	24 h (1.95 – 259 μg/ml), coated in mucoadhesive film	152.5 μg/ml (CI not available)	Reduced HPV-16 pseudovirus-infected cells; Inhibited inflammation	-	[4]
GC	*In vitro*	GC cell line SGC7901 and CDDP-resistant GC cells (SGC7901/CDDP)	24 or 48 h (15, μM) + cisplatin (CDDP) (2 μg/ml)	48 h: SGC7901: 12.86 (CI not available) μMSGC7901/CDDP: 13.69 μM (CI not available)	Enhanced sensitivity of SGC7901/CDDP to CDDP; Induced apoptosis; Enhanced CDDP-induced autophagy; Modified cell proliferation, autophagy and apoptosis	↑BAX, cleaved caspase-3 and -9; ↑LC3-II/I and Beclin1, while↓p62; ↓EB13 and p-STAT3/STAT3 ratio	[5]
GC	*In vitro*	Human gastric adenocarcinoma cell lines (BGC-823 and SGC-7901)	Cell viability: 6, 12, 18, 24, and 48 h (0 – 10 μg/ml); Apoptosis: 6, 18, 24 h (7 μg/ml)	-	Inhibited cell proliferation; Induced apoptosis	Release cytochrome c and AIF – mitochondrial dysfunction; Inhibited STAT3 activation, ↓p-	[6]

						STAT3, Bcl-xL and Mcl-1 ↓ pro-inflammatory factors – IL-1b, IL-4 and IL-8 expression and ↑ anti-inflammatory factor – IL- 10; ↑pro-apoptotic factors - Bax, Bad, cleaved caspase-3 and cleaved PARP expression and ↓anti-apoptotic factors - Bcl-2 and Bal-xl expression; ↑LC3, LC3-II and Beclin1 expression and ↓p62 and LC3-I expression; Suppressed p-PI3K, p-AKT and p-mTOR expressions	
SC	*In vivo*	ICR female mice with DMBA/TPA-induced skin tumors	Once daily until death (5 and 20 mg/kg)	-	Inhibited skin tumorigenesis and inflammation; Accelerated apoptosis; Promoted autophagy in skin tumors; Inhibited tumor proliferation		[7]

(The upward and downward arrows represent increased (↑) and decreased (↓) levels of specific molecules or expressions, respectively. The numbers in the "Therapeutic Effect" column correspond to the same numbered mechanisms in the "Molecular Mechanism" column.

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