Table S1 Main *in vitro* assays used to assess migration and invasion in NSCLC cells

Assay	Endpoint	Information	Ref.
Migration			
Transwell migration assay (Boyden chamber)	Number of migrated cells	Single-cell migration, chemotaxis	(9-12,24-32,34-40,46,55,56,64,69,71,72,74,79,83,88,89,96,97,99,103,108,109,111,114,122,123,125,127)
Wound-healing (scratch) assay	Migration area/width	Collective migration, EMT	(4,24,25,27-32,34,39,45,46,48,50,51,53,54,56-62,65,67,68,70-73,75,76,77,79-81,83-92,94-102,104-114,116-124,126,127)
Fence assay	Migration area	Collective migration, EMT	(18)
Time-lapse cell tracking	Cell migration path	Collective or single-cell migration	(19,20,63)
Cell exclusion zone assay	Migration area	Collective migration, EMT	(19,21,22,66)
Spheroid migration assay	Migration area	Migration from cell cluster	(23)
Invasion			
Transwell invasion assay (Boyden chamber)	Number of invasive cells	Single-cell invasion through ECM	(4,11,12,25-29,31,32,34-40,45,46,48,50-55,57,59,60,62-64,67,68,71,73-77,79,80-89,92-103,105,109,110-113,115,117,120-127)
Spheroid invasion assay	Invasion area	Single or collective invasion from cluster	(21,47)
3D cell tracking	Invasion distance	Single-cell invasion	(47)
Gelatin zymography	Zymograms	MMPs activity	(26,28,40,45,46,62,77,79,91,98,102,114,119,123)

3D, three-dimensional; EMT, epithelial to mesenchymal transition; ECM, extracellular matrix; MMPs, matrix metalloproteinases

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Table S2 Overview of *in vitro* studies on migration and invasion of NSCLC cells exposed to lung carcinogens and other toxic contaminants

Group	Carcinogen/Contaminant	NSCLC cell line	Methodology (concentrations used*)	Key findings**	Ref.
Tobacco smoke	B(a)P	A549	Transwell migration and invasion assays (10 nM and 1,000 nM)	B(a)P significantly increased cell migration and invasion through up-regulating IL-8, CCL2, and CCL3 expression	(55)
		A549, YTMLC	Wound-healing assay	B(a)P increased the number of metastatic cells and TNF-α had a role in this development	(56)
			Transwell migration assay (1, 2, 5, 10 and 20 μM)		
	Cadmium	A549	Wound-healing assay (10 μM and 20 μM)	Notch1, along with HIF-1α and IGF-1R/Akt/ERK/S6K1 signalling pathways, promote malignant progression stimulated by Cd	(58)
			Transwell migration and invasion assays (2 μM)	Upregulation of HMGA2 plays an important role in Cd-enhanced migration and invasion	(37)
			Transwell migration and invasion assays (not stated)	Cd induced an increase in cell migration and invasion by promoting autophagy	(38)
			Wound-healing assay	TGIF might play a crucial role in invasion and migration of cells exposed to Cd	(57)
			Transwell invasion assay (0.5 μM and 1 μM)		
			Transwell migration and invasion assays (0.5 μM and 2 μM)	Exposure to Cd increased the expression of p-ERK, enhancing migration and invasion	(35)
	Nicotine	A549, H1299	Transwell invasion assay (100 μg/mL)	Nicotine promoted cell migration through upregulation of LINC00460	(52)
		A549	Wound-healing assay	Nicotine induced proliferation, invasion, and migration of tumor cells through the mediation of α7-nAChRs	(50)
			Transwell invasion assay (0.01, 0.1 and 1 μM)		
		A549, H1650	Wound-healing assay	ID1, after induction by nicotine, promoted migration and invasion by increasing the expression of *STMN3* and *GSPT1* genes	(51)
			Transwell invasion assay (1 μM)		
	NNK	H1299	Wound-healing assay	NNK activated the c-Src/PKCι/FAK loop, which promoted metastasis	(53)
			Cell migration assay kit		
			Transwell invasion assay (100 pM)		
		A549, H157	Wound-healing assay	Twist protein and mRNA expression were increased by NNK, and it was necessary for NNK promotion of migration and invasion	(54)
			Transwell invasion assay (2 and 5 μM)		
Air pollution	BPA	A549	Wound-healing assay	BPA can promote the *in vitro* migration and invasion via upregulation of MMPs and GPER/EGFR/ERK1/2 signals	(34)
			Transwell migration and invasion assays (10 μM)		
			Transwell invasion assay	Snail-1/Cx43/ERRγ was identified as a novel signalling pathway through which BPA promoted metastasis	(63)
			Time-lapse cell tracking (10, 50, 100, 300 nM and 10 μM)		
	gNO	A549	Wound-healing assay	gNO promoted metastasis through a mechanism involving the iNOS-dependent MMP-2 activity	(62)
			Transwell invasion assay		
			Gelatin zymography (1.0, 2.5, and 5.0 μM)		
	Oxy-PAHs	A549	Wound-healing assay	Exposure to Oxy-PAHs (9-fluorenone) induced invasion and migration of cells by the activation of EMT	(24)
			Transwell migration assay (0.16, 0.8, 4, 20 and 100 μM)		
	PM2.5	A549	Wound-healing assay	PM2.5 exposure induced ROS, which activates loc146880 expression. The lncRNA, in turn, up-regulates autophagy and promotes malignant behaviour. Both loc146880 and autophagy promoted cell migration, invasion, and EMT	(59)
			Transwell invasion assay (16 μg/cm^2)		
		A549, H1299	Wound-healing assay (50 μg/cm^2)	PM2.5 exposure induced proliferation and motility	(61)
		H1299, H520	Wound-healing assay	Cell migration, invasion, EMT and autophagy were enhanced when cells were treated with cigarette smoke extract and PM2.5 alone or in combination	(60)
			Transwell invasion assay (25 μg/cm^2)		
Other	AFB1	A549	Wound-healing assay (2.5 μM)	AFB1 promoted cell migration through upregulation of IRS2 via induction of Src phosphorylation	(65)
	Arecoline	A549, H520, H460	Cell exclusion zone assay (40 μM)	Arecoline stimulated cell migration by activating the EGFR/c-Src/FAK signalling pathway via mAChR3	(66)
	Isoflurane	A549, H1299	Wound-healing assay	Isoflurane activated the Akt-mTOR signalling pathway resulting in the promotion of cells' proliferation, migration, and invasion	(67)
			Transwell invasion assay (1 and 2%)		
	Riboflavin	A549, H3255, Calu-6	Transwell migration and invasion assays (50, 100, 200 and 400 μM)	Riboflavin at higher doses increased cell growth as well as invasion and migration	(64)

α7-nAChRs, alpha-7 nicotinic receptor; Akt, protein kinase B; AFB1, aflatoxin B1; B(a)P, Benzo(a) pyrene; BPA, Bisphenol A; CCL2, chemokine (C-C motif) ligand 2; CCL3, Chemokine (C-C motif) ligand 3; Cd, Cadmiun; c-Src, Proto-oncogene tyrosine-protein kinase Src; Cx43, connexin 43; EGFR, epidermal growth factor receptor; EMT, epithelial to mesenchymal transition; ERK, extracellular-signal-regulated kinase; ERRγ, estrogen related receptor gamma; FAK, focal adhesion kinase; gNO, nitric oxide (gaseous); GPER, G protein-coupled estrogen receptor; GSPT1, G1 To S Phase Transition 1; HIF-1α, Hypoxia-inducible factor 1-alpha; HMGA2, high mobility group A2; ID1, Inhibitor of DNA binding/Differentiation 1; IGF-1R, Insulin-like growth factor 1 receptor; IL-8, interleukin 8; iNOS, nitric oxide synthase (inducible isoform); IRS, insulin receptor substrate; lncRNA, long non-coding RNA; mAChR3, muscarinic acetylcholine receptor 3; MMP, matrix metalloproteinase; mTOR, mammalian target of rapamycin; NNK, Nitrosamine 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone; PAHs, Polycyclic aromatic hydrocarbons; PKCι, protein kinase C; PM2.5, particulate matters with less than 2.5 μm of diameter; S6K1, Ribosomal protein S6 kinase beta-1; STMN3, Stathmin like 3; TGIF, transforming growth interacting factor; TNF-α, Tumor necrosis factor α. *In some reports not all the concentrations indicated in this table were used for all the migration/invasion experiments. **Herein are presented the key findings reported by the authors related to migration/invasion and underlying mechanisms, using essentially migration/invasion related-assays. Nonetheless, some of these findings were obtained using other methodologies not provided in this table. In the original manuscript, other findings not related to migration/invasion were also reported.

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Table S3 Overview of *in vitro* studies on migration and invasion of NSCLC cells exposed to natural bioactive compounds

Group	Natural bioactive compound	NSCLC cell line	Methodology (concentrations used)*	Key findings**	Ref.
Polyphenols non-flavonoids	Curcumin	801D	Wound-healing assay Transwell invasion assay (10 μM)	Low toxicity levels of curcumin suppressed migration and invasion through inhibition of Rac1/PAK1 signalling pathway and MMP-2/9 expression	(68)
		A549 H226	Transwell migration assay (5 and 10 μM)	Curcumin suppressed proliferation and migration via inhibition of EGFR and the TLR4/MyD88 pathway	(69)
	Ephemeranthol A	H460	Wound-healing assay (10, 50 and 100 μM)	Ephemeranthol A suppressed migration and EMT by decreasing N-cadherin, vimentin, and Slug as well as inhibiting the activation of FAK and Akt	(70)
	Honokiol	A549, H460, H226, H1299	Wound-healing assay Transwell migration and invasion assays (5, 10 and 20 μM)	Honokiol suppressed migration by inhibition of PGE_2 and COX-2, leading to the inactivation of the β-catenin signalling pathway	(71)
		A549, H460	Wound-healing assay Transwell migration assay (30 μM)	Honokiol inhibited migration and EMT by targeting c-FLIP, resulting in the suppression of N-cadherin and Snail	(72)
		A549	Transwell migration assay (45 μM)	miR-148a-5p and miR-148a-3p are potential biomarkers of honokiol-treated cells and, consequently, inhibited proliferation and migration, and induced apoptosis	(9)
	Mangiferin	A549, H460, H520	Wound-healing assay Transwell migration assay (25 μg/mL)	Mangiferin inhibited migration, regulated EMT by upregulating the expression of PER1, mediated LPS-induced NLRP3 inflammasome expression, and the production of inflammatory cytokines	(30)
	Phoyunnanin E	H460, H292, A549	Wound-healing assay Transwell invasion assay (1, 5 and 10 μM)	Phoyunnanin E inhibits the motility of cells via the suppression of EMT, migratory-associated integrins αv and β3, and FAK/Akt signals which in turn suppress downstream migratory proteins	(73)
	Resveratrol	A549	Transwell migration and invasion assays (50 μM)	Resveratrol inhibited proliferation, migration, invasion, and promoted apoptosis by inhibiting the expression of STAT-3	(74)
			Wound-healing assay Transwell invasion assay (25 μM)	Resveratrol-induced Rad9 expression (mediated by DNA damage and ROS), significantly suppressed proliferation, migration, and invasion, and activated cellular senescence	(75)
	Rottlerin	A549	Wound-healing assay Transwell invasion assay (1 and 3 μM)	Rottlerin hampered migration and invasion by inhibiting the expression of TAZ	(76)
Flavonoids	Acacetin	A549	Wound-healing assay Transwell invasion assay Gelatin zymography (1, 2.5 and 5 μM)	Acacetin inhibited migration and invasion by preventing p38a phosphorylation via the MKK3/6 and/or the MLK3 signaling pathways. Additionally, it inhibited NF-kB and AP-1, causing suppression of MMP-2/9 and u-PA expression	(77)
	Atalantraflavone	A549, 95D	Cell Migration Assay Kit (10, 25 and 50 μM)	AFL suppressed NSCLC progression by inhibiting migration through Twist1	(78)
	Anthocyanins	A549	Transwell migration and invasion assays Gelatin zymography (25, 50 and 100 μM)	Anthocyanins decreased the expression of MMP-2, u-PA, TIMP-2, and PAI, causing the inhibition of migration and invasion in a dose-dependent manner	(40)
		H1299	Wound-healing assay Transwell migration and invasion assays Gelatin zymography (10, 20 and 40 μM)	P3G inhibited invasion, motility, and secretion of MMP-2/9, and u-PA. These inhibitory effects might occur due to the inactivation of ERK 1/2 and AP-1 signalling pathways	(79)
			Wound-healing assay Transwell migration and invasion assays (6.25 μM of individual anthocyanidins or their equimolar mixture 3.12, 6.25 and 12.5 μM)	The combination of anthocyanidins synergistically inhibited cell growth, invasion, and migration, and promoted cell-cycle arrest and apoptosis when compared to individual anthocyanins	(25)
	Artonin E	A549, H460, H292, H23	Wound-healing assay Transwell invasion assay (0.05, 0.1, 0.25, 0.5 μg/mL)	Artonin E inhibited migration and invasion via suppression of activated FAK, downstream-activated Akt, and CDC42	(80)
	BIO-A	A549	Transwell invasion assay (20, 40 and 80 μM)	BIO-A inhibited proliferation through down-regulating Ki-67 and VEGF, induced apoptosis by activation of Caspases-3 and 9, and suppressed cell migration by downregulating MMP-2 and VEGF	(82)
	Cycloartobiloxanthone	H460	Wound-healing assay Transwell invasion assay (1, 5 and 10 μM)	Cycloartobiloxanthone inhibited migration and invasion by suppressing several migratory-regulated mechanisms including FAK and CDC42 signaling, decreasing integrin α5, αv, and β3 levels, and inhibiting EMT	(81)
	Deguelin	H292	Wound-healing assay Transwell migration and invasion assays (0.5, 1.5 and 2.5 μM)	Deguelin inhibited migration and invasion through the inhibition of Ras, PKC, Akt, and NF-κB signaling pathway resulting in the down-regulation of MMP-2/9 and uPA	(83)
		H23, H1299, A549	Wound-healing assay Transwell invasion assay (200 and 500 nM)	Deguelin inhibits cell migration and invasion and by suppressing CtsZ expression and its downstream FAK/Src/Paxillin signaling	(84)
	ECG	A549	Wound-healing assay Transwell migration and invasion assays Gelatin zymography (10, 30 and 50 μM)	ECG suppressed TGF-β-induced EMT and invasion of cells by reducing expression levels of fibronectin, p-FAK, MMP-2 and u-PA	(46)
	EGCG	A549	Wound-healing assay Transwell invasion assay (5, 10 and 20 μM)	EGCG inhibited TGF-β-induced EMT via downregulation of phosphorylated Smad2 and ERK1/2	(85)
	Fisetin	A549	Wound-healing assay Transwell migration and invasion assays Gelatin zymography (1, 5 and 10 μM)	FIS suppressed adhesion, migration, and invasion via inhibition of ERK1/2 and downregulation of MMP-2 and u-PA at both protein and mRNA expression levels	(28)
			Wound-healing assay Transwell invasion assay (10 and 40 μM)	FIS suppressed proliferation, migration, adhesion, and invasion	(48)
			Wound-healing assay Transwell invasion assay (10 μM)	The combination of FIS and paclitaxel significantly reduced cancer cell migration and invasion through a marked rearrangement of actin and vimentin cytoskeleton and the modulation of metastasis-related genes	(86)
		A549, H1299	Wound-healing assay Transwell invasion assay (5 and 10 μM)	FIS significantly inhibited migration, invasion and EMT through up-regulation of E-cadherin, ZO-1 and downregulation of vimentin, N-cadherin and MMP-2	(4)
	Genistein	A549, H460	Wound-healing assay Transwell invasion assay (20 and 40 μM)	Genistein inhibited migration and invasion	(87)
	Hesperidin	A549, H460, H1975	Wound-healing assay Transwell migration and invasion assays (25, 37.5, 50 and 62.5 μg/mL)	Hesperidin inhibited the migratory and invasive capabilities by mediating the SDF-1/CXCR-4 signalling pathway	(88)
	Hydroxysafflor yellow A	A549, H1299	Wound-healing assay Transwell migration and invasion assays (5, 10 and 20 μM)	HYSA inhibited migration, invasion, and EMT by suppressing PI3K/Akt/mTOR and ERK/MAPK signaling pathways	(89)
	Luteolin	A549	Wound-healing assay (50 μM)	Luteolin disrupted cell migration through the prevention of stress fiber formation	(90)
	Morin	A549	Transwell migration assay (50 μM)	Morin decreased cell viability, colony formation, and migration rate through the downregulation of miR-135b that directly targets and represses CCNG2	(10)
	Myricetin	A549	Wound-healing assay Gelatin zymography (50 and 100 μM)	Myricetin inhibited migration by reducing MMP-2/9 expressions via inhibition of the FAK/ERK signaling pathway	(91)
	Quercetin	A549, HCC827	Wound-healing assay Transwell migration and invasion assays (10, 25 and 50 μM)	Quercetin inhibited metastasis by suppressing the Snail-mediated EMT	(31)
	Scutellaria Flavonoids	A549, H1299	Transwell invasion assay (80 μM)	Baicalein significantly inhibited cell invasion and EMT by upregulating the mRNA and protein expression of E-cadherin and downregulating the Twist1 and Vimentin expression	(93)
			Wound-healing assay Transwell invasion assay (baicalin 200 μM, baicalein 10 μM and wogonin 40 μM)	Baicalin, baicalein, and wogonin activated Rap1-GTP binding and dephosphorylated Akt and Src by suppressing a7nAChR, consequently triggering inhibition of Id1 and thus blocking proliferation, EMT, and angiogenesis	(94)
		A549, 95D	Wound-healing assay Transwell invasion assay (4, 8, 16 μM)	OA inhibited the invasion and migration of tumour cells by suppressing Snail via inhibition of the ERK/GSK-3β signalling pathway	(92)
	Sotetsuflavone	H1650	Wound-healing assay Transwell invasion assay (64 and 128 μM)	Sotetsuflavone was able to inhibit proliferation, migration, and invasion	(95)
Bibenzyls	Chrysotobibenzyl	H460, H292	Wound-healing assay Transwell migration and invasion assays (1, 5, 10 and 50 μM)	Chrysotobibenzyl inhibited cell migration via depletion of Cav-1, integrins β1, β3, and αv and also suppressed EMT	(27)
	Gigantol	H460, H292	Wound-healing assay Transwell migration and invasion assays (5, 10 and 20 μM)	Gigantol suppressed the migratory behaviour through a Cav-1-dependent pathway.	(29)
		H460	Wound-healing assay Transwell migration and invasion assays (1, 5, 10 and 20 μM)	Gigantol suppressed EMT, resulting in a reduction of migration	(96)
	Moscatilin	H23	Wound-healing assay Transwell migration and invasion assays (0.25, 0.5 and 1 μM)	Moscatilin inhibited cell migration and invasion through attenuation of endogenous ROS	(97)
	Riccardin D	A549	Wound-healing assay Transwell invasion assay Gelatin Zymography (2.5, 5, 10 and 20 μM)	The ability of invasion and migration was suppressed upon exposure to riccardin D, and MMP-2/9 levels were significantly decreased	(98)
	TDB	H292	Wound-healing assay Transwell migration and invasion assays (0.5, 1 and 5 μM)	TDB reduced cell migration and invasion by decreasing migration-regulated proteins, including integrins αv, α4, β1, β3, and β5, as well as downstream signalling proteins, such as pFAK, Rac1-GTP, and CDC42	(39)
Terpenes	Actein	A549, 95D	Wound-healing assay Transwell migration and invasion assays (20 and 40 μM)	Actein suppressed cell migration and invasion	(99)
	Alisol B	A549	Wound-healing assay Transwell invasion assay (6 and 9 mM)	Alisol B suppressed cell migration and invasion through the inhibition of the PI3K/AKT/mTOR pathway	(101)
	Betulin	H460	Wound-healing assay Transwell invasion assay Gelatin zymography (11 and 30 μM)	Betulin suppressed migration and invasion by inhibiting MMP-2/9	(102)
	Frondoside A	LNM35	Wound-healing assay Transwell invasion assay (0.1 and 0.5 μM)	Frondoside A inhibited cell migration, invasion and angiogenesis	(100)
	Nagilactone E	A549	Transwell migration and invasion assays (2 and 4 μM)	NLE inhibited TGF-β1-stimulated cell migration and invasion by suppressing Smad2 and Smad3, thus suppressing EMT	(103)
	Triptolide	A549, H460, H358	Transwell migration and invasion assays (10 nM)	Triptolide altered the expression of microRNAs involved in cellular movement and decreased migration and invasion by reducing FAK expression which impaired its downstream signalling	(36)
	Ursolic acid	H1975	Wound-healing assay (25 μM)	Ursolic acid induced apoptosis, and inhibited cell migration and proliferation by negatively regulating the β-catenin/TCF4/CT45A2 signalling pathway	(104)
Alkaloids	Daurinoline	A549	Wound-healing assay Transwell invasion assay (5, 10 and 20 μM)	Daurinoline inhibited the proliferation, migration, invasion, and EMT phenotype of chemo-resistant cells by reversing EMT and Notch-1	(105)
	Krukovine	A549, H460	Wound-healing assay (5, 7.5, 10 and 20 μM)	Krukovine suppressed migration by preventing the phosphorylation of ERK, AKT, PI3K, mTOR, C-RAF, and p70sk6k	(106)
	Oxymatrine	A549	Transwell migration and invasion assays (1 mg/mL)	OMT inhibited cancer progression and metastasis by upregulation of miR-520 and downregulation of VEGF	(11)
			Wound-healing assay (1.5 and 2 mg/mL)	OMT inhibited cell migration	(107)
Steroids	Bufalin	H460	Transwell migration and invasion assays Gelatin zymography (25, 50, 100 and 200 nM)	Bufalin inhibited invasion and migration by suppressing NF-kB and MMP-2/9	(26)
			Wound-healing assay Transwell migration and invasion assays (2.5, 5 and 10 nM)	Under sub-lethal concentrations, bufalin significantly inhibited cell adhesion, migration, and invasion of gefitinib-resistant H460 cells	(109)
		A549	Wound-healing assay Transwell migration assay (50 nM)	Bufalin suppressed TGF-β-induced EMT and migration by downregulating TβRI and TβRII	(108)
	Cardenolides	A549	Wound-healing assay Transwell invasion assay (10 and 50 nM)	DGX and CON demonstrated anti-proliferative activity and decreased migration and invasion by suppressing MMP-2/9 and p-FAK expression	(110)
	Ophiopogonin B	A549	Wound-healing assay Transwell migration and invasion assays (2.5, 5 and 10 μM)	OP-B significantly reduced invasion and migration through inhibition of EphA2/Akt and the corresponding signaling pathway	(111)
Others	Coix polysaccharides	A549	Wound-healing assay Transwell invasion assay (200 and 300 μg/mL)	Coix polysaccharides inhibited migration and invasion by downregulating the expression of S100A4	(112)
	Cordycepin	PC9, H1975	Wound-healing assay Transwell invasion assay (10, 20 and 40 μM)	CD can affect proliferation, migration, invasion, cell cycle, and apoptosis through the activation of AMP-activated protein kinase and subsequent inhibition of mTOR	(113)
	Dihydroaustrasulfone alcohol	A549	Wound-healing assay Transwell migration assay Gelatin zymography (20, 30 and 40 μg/mL)	Dihydroaustrasulfone alcohol inhibited invasion and migration through suppression of the ERK1/2 signalling pathway resulting in a decrease in MMP-2/9 activities	(114)
	Esculetin	A549	Transwell invasion assay (5 and 20 μM)	Esculetin inhibited the proliferation and regulated EMT via the downregulation of vimentin and Snail, and up-regulation of E-cadherin	(115)
	Evodiamine	A549	Wound-healing assay (4 and 8 μM)	Evodiamine suppressed cell migration	(116)
	Ganoderan	A549	Wound-healing assay Transwell invasion assay (0.25, 0.5 and 1 mg/mL)	Ganoderan inhibited migration, invasion and EMT via the ERK signalling pathway	(117)
	Glycyrol	A549	Wound-healing assay (2.5, 5 and 7.5 μM)	Glycyrol suppressed migration	(118)
	Goniothalamin	H1299	Wound-healing assay Gelatin Zymography (1, 2, 5 and 10 μg/mL)	GTN attenuated cell migration and caused a reduction in the activity levels of MMP-2/9 due to its DNA-damaging effect	(119)
	Isothiocyanates	A549, SK-MES-1	Wound-healing assay Transwell invasion assay (20 μM)	PEITC suppressed migration and invasion by regulating MMP-2 and induced autophagy as well as suppressed the JAK2/STAT3 pathway	(121)
		A549	Wound-healing assay Transwell invasion assay (5, 10 and 15 μM)	SFN-Cys inhibited invasion via microtubule-mediated Claudins dysfunction, but SFN-NAC inhibited invasion via microtubule-mediated inhibition of autolysosome formation	(120)
		H1299, 95D, 95C	Transwell migration and invasion assays (1, 1.5 and 5 μM)	Sulforaphane inhibited migration and invasion by hampering EMT, through a decrease in the levels of miR-616-5p, which resulted in suppression of the GSK3β/β-catenin signalling pathway	(12)
		A549, H1299	Wound-healing assay Transwell migration and invasion assays (1, 2 and 3 μM)	Sulforaphane inhibits migration and invasion via ERK5 activation, resulting in an increase in the expressions of E-cadherin and ZO-1 and a decrease in N-cadherin and Snail1	(122)
	Magnolin	A549, H1975	Wound-healing assay Transwell migration and invasion assays Gelatin Zymography (15, 30 and 60 μM)	Magnolin suppresses cell migration and invasion by targeting the ERKs/RSK2 signaling pathway. It also modulated EMT marker proteins such as N-cadherin, E-cadherin, Snail, Vimentin, and MMPs	(123)
	Methylene chloride extracts of *Morus alba*	H1299, H460, A549	Wound-healing assay Transwell migration and invasion assays (5, 10 and 25 μg/mL)	MEMA inhibited the migratory activity of cells through blockage of Src/STAT3-mediated EMT	(32)
	Renieramycin M	H460	Wound-healing assay Transwell invasion assay (0.5 and 2.5 μM)	Renieramycin M decreased migration and invasion	(124)
	Salidroside	A549	Transwell migration and invasion assays (800 μM)	Salidroside inhibited migration and invasion by decreasing the expressions of MMP-2, RhoA, ROCK1, and vimentin	(125)
	Sodium new houttuyfonate	A549, H1299	Wound-healing assays Transwell invasion assays (0.1, 0.2 and 0.4 mM)	SNH decreased cell metastasis and invasion by suppressing EMT progression	(126)
	Δ^9-Tetrahydrocannabinol	A549, SW-1573	Wound-healing assay Transwell migration and invasion assays (5, 10 and 15 μM)	THC treatment inhibits EGF-induced cell motility and invasion	(127)
	Thymoquinone	A549	Wound-healing assay Transwell invasion assay Gelatin zymography (10, 20 and 40 μM)	TQ suppressed the proliferation, migration, and invasion by inhibiting PCNA, cyclin D1, MMP-2/9	(45)

α7-nAChRs, alpha-7 nicotinic receptor; AFK, atalantraflavone; Akt, protein kinase B; AP-1, activation protein 1; BIO-A, Biochanin A; Cav-1, Caveolin 1; CCNG2, Cyclin-G2; CD, Cordycepin; CDC42, cell division control protein 42; c-FLIP, Cellular Fas-associated death domain-like IL-1 beta-converting enzyme inhibitory protein; CON, convallatoxin; COX-2, cyclooxygenase-2; C-RAF, RAF proto-oncogene serine/threonine-protein kinase; CT45A2, cancer/testis antigen family 45 member A2; CtsZ, Cathepsin Z; CXCR-4, C-X-C chemokine receptor type 4; DGX, digitoxigenin monodigitoxoside; ECG, Epicatechin-3-gallate; EGCG, Epigallocatechin-3-gallate; EGFR, epidermal growth factor receptor; EMT, epithelial-to-mesenchymal transition; EphA2, Ephrin type-A receptor 2; ERK 1/2, extracellular signal-regulated kinases 1 and 2; FAK, focal adhesion kinase; FIS, fisetin; GSK-3β, glycogen synthase kinase 3 beta; GTN, Goniothalamin; HYSA, Hydroxysafflor yellow A; Id1, inhibitor of differentiation 1; JAK2, Janus kinase 2; MAPK, mitogen-activated protein kinase; MEMA, Methylene chloride extracts of Morus alba; MHMM-41, (3E,5E)-3-[(1H-indol-3-yl)methylene]-5-(3-hydroxy-4-methoxybenzylidene)-1-methylpiperidin-4-one (MHMM-41); MKK3/6, Mitogen-activated protein kinase kinase; MLK3, Mitogen-activated protein kinase kinase; MMPs, matrix metalloproteinases; mTOR, mammalian target of rapamycin; MyD88, Myeloid differentiation primary response 88; NF-kB, nuclear factor kappa B; NLE, nagilactone E; NLRP3, NLR family pyrin domain containing 3; OA, oroxylin A; OMT, Oxymatrine; OP-B, Ophiopogonin B; P3G, Pelargonidin-3-O-glucoside; PAI, plasminogen activator inhibitor; PAK1, Serine/threonine-protein kinase; PCNA, Proliferating cell nuclear antigen; PDGF-BB, Platelet-derived growth factor; PEITC, Phenethyl isothiocyanate; PER1, period circadian protein homolog 1 protein; pFAK, focal adhesion kinase; PGE2, prostaglandin E2; PI3K, Phosphoinositide 3-kinase; PKC, protein kinase C; Rac1, Ras-related C3 botulinum toxin substrate 1; Rap1, Repressor Activator Protein 1; RhoA, Ras homolog family member A; ROCK1, Rho Associated Coiled-Coil Containing Protein Kinase 1; ROS, reactive oxygen species; RSK2, ribosomal S6 kinase-2; S100A4, S100 calcium binding protein A4; SDF-1, stromal cell-derived factor 1; SFN-Cys, sulforaphane-cysteine; SFN-NAC, sulforaphane-N-acetyl-L-cysteine; SNH, Sodium new houttuyfonate; Src, Proto-oncogene tyrosine-protein kinase Src; STAT-3, signal transducer and activator of transcription 3; TAZ, transcriptional co-activator with PDZ-binding motif; TβRI/II, TGF-β receptor I/II; TCF4, T-cell factor 4; TDB, 4,5,40-trihydroxy-3,30-dimethoxybibenzyl; (TGF)-β1, transforming growth factor; THC, Δ9-Tetrahydrocannabinol; TIMP-2, tissue inhibitor of matrix metalloproteinase-2; TLR4, Toll-like receptor 4; TQ, Thymoquinone; u-PA, urokinase plasminogen activator; VEGF, Vascular endothelial growth factor; ZO-1, Tight junction protein 1. *In some reports not all the concentrations indicated in this table were used for all the migration/invasion experiments. **Herein are presented the key findings reported by the authors related to migration/invasion and underlying mechanisms, using essentially migration/invasion related assays. Nonetheless, some of these findings were obtained using other methodologies not provided in this table. In the original manuscript, other findings not related to migration/invasion were also reported.

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