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Review Article

Chemistry and Pharmacological Properties of Morusin and Morusinol from *Morus alba*: An Overview

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ABSTRACT

Morusin or mulberrochromene is a prenylated flavone from the root bark of *Morus alba*. Structurally, the compound has three benzene rings (A–C) with one OH group at C5 of ring A, and two OH groups at C2' and C4' of ring B. There is a prenyl moiety with two methyl units at C3 of the oxygenated ring C. There is another prenyl moiety with two methyl units at C8 of ring A. Together with ring D, the 2,2-dimethyl pyran group is formed across C7 and C8. Ring D has five carbon atoms and one oxygen atom. The double bond at C2–C3 and carbonyl functional group at C4 of morusin are essential for its bioactivities. Morusinol, is another prenylated flavone with a chemical structure that is very similar to that of morusin. Exceptions are an extra OH group at C13 and a lack of the C12−C13 double bond. Morusin has been reported to exert antitumor effects on multiple types of cancer cells such as breast, liver, lung, cervical, prostate, colorectal, skin, gastric, nasopharyngeal, ovarian, pancreatic and renal cancer cells, including those of glioblastoma and osteosarcoma. The anti-cancer properties of morusinol towards melanoma and colorectal cancer cells were reported. Briefly mentioned was a case study of retraction of a paper on the selective and potent antitumor activity of morusinol towards liver carcinoma. Other pharmacological properties of morusin and morusinol were also presented. Some aspects that warrant further and more in-depth research into morusin and morusinol were suggested.

***Keywords*:** Prenylated flavones, Types of cancer, Cytotoxicity, Molecular mechanisms.

Introduction

Morus alba L. or white mulberry belongs to the family Moraceae. Endemic to central and north China, the species is now widely cultivated in east Asian countries of China, Japan and Korea.[1,2] The species is a shrub or tree (3−10 m tall) that is fast-growing and copiously produce a white latex when injured. The bark of *M. alba* is dark grey-brown, and slightly fissured with lenticels. Leaves are glossy green, alternate, broadly ovate, cordate at the base, and acuminate at the apex. Leaf margins are serrated, petioles are long and slender, and the under-surface is slightly pubescent. Leaves are variable in form ranging from entire to deeply lobed.[3,4] Trees are commonly dioecious with separate male and female plants, but may be monoecious with male and female flowers on the same plant. Flowers are inconspicuous male or female catkins. The fruit of white mulberry is green when young, turning orange to red and purplish black when ripe. The root bark, leaves and fruits of *M. alba* are shown in Figure 1. Leaves of mulberry are commonly used as fodder for silkworms and livestock, and consumed as herbal tea for lowering blood pressure.[2] In Mediterranean countries, the fruits of *M. alba* are used to produce juice, jam, liquor and canned mulberries.[5]

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The root bark of *M. alba* or Mori Cortex Radicis is known as Sang-Bai-Pi in China and Sõhakuhi in Japan.[6] White mulberry is used in traditional Chinese medicine for the treatment of cough, hepatitis, diabetes, heart diseases and other inflammatory diseases.

Chemical constituents of *M. alba* contain polysaccharides, phytosterols, tannins, alkaloids, steroids, glycosides, carbohydrates, proteins, and amino acids, as well as saponins, triterpenes, phenols, flavonoids, benzofuran derivatives, anthocyanins, anthraquinones, glycosides, vitamins and minerals.[7] Plants of *M. alba* possess a wide array of pharmacological activities. They include antibacterial, anti-inflammatory, analgesic, antipyretic, antioxidant, anti-cancer, anti-diabetes, anti-asthmatic, anti-tyrosinase, anti-depressant, gastrointestinal, respiratory, cardiovascular, hypolipidemia, anti-obesity, immunomodulatory, dermatological, pancreatic lipase inhibitory, myocardial protective, cardioprotective, antibacterial, anti-pancreatitis, hepatoprotective, renoprotective and neuroprotective properties.[3,7,8]

The uses, chemistry and pharmacology of *M. alba* are well-reviewed in recent years.[2-4,7,9] There are several reviews on the constituents of bioactive compounds in *M. alba* including morusin and its anti-cancer properties.[10-12] This overview on the chemistry, and pharmacological properties of morusin and morusinol from *M. alba* serves as an update on recent findings.

Information used in this overview of morusin and morusinol from *M. alba* was obtained from databases notably Google Scholar Citations, ScienceDirect, PubMed and J-Stage. The search involved keywords and priority was accorded to the more recent publications ranging from 2020 to 2025. References from Google Scholar Citations were useful in downloading the Vancouver style of referencing that provides titles of papers, names of authors and journals, along with pagination and dates of publication.

Chemistry

Morusin or mulberrochromene is a prenylated flavone with a molecular formula of $C_{25}H_{24}O_6$, molecular weight of 420.5 g/mol and melting point of 214º–216ºC.[11,13] The compound was first isolated in 1976 from the root bark of *M. alba* by Nomura *et al*.[14]

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Figure 1: (L−R) Root bark, leaves and fruits of *Morus alba*

It was decades later when morusin was reported from the leaf,[15] stem bark[16] and twig[17] of the plant. The content of morusin in *M. alba* was lowest in the leaf at 40 μg/g and highest in the root bark at 867 μg/g. The branch bark content was the second highest at 270 μg/g.[18]

The chemical structure of morusin is shown in Figure 2. The compound has a flavone core comprising three benzene rings (A–C) with one OH group at C5 of ring A and two OH groups at C2' and C4' of ring B.[3,10-12,19] Morusin has two prenyl moieties. The prenyl moiety at C3 of the oxygenated ring C is unmodified and have a C12–C13 double bond and two methyl units (encircled in blue). Another prenyl moiety at C8 is fused with ring D to form the 2,2-dimethyl pyran group across C7 and C8 (encircled in green).[12] Ring D has five carbon atoms and one oxygen atom. The double bond at C2–C3 and carbonyl functional group at C4 are essential for the bioactivities of morusin, e.g., anti-inflammatory activities.

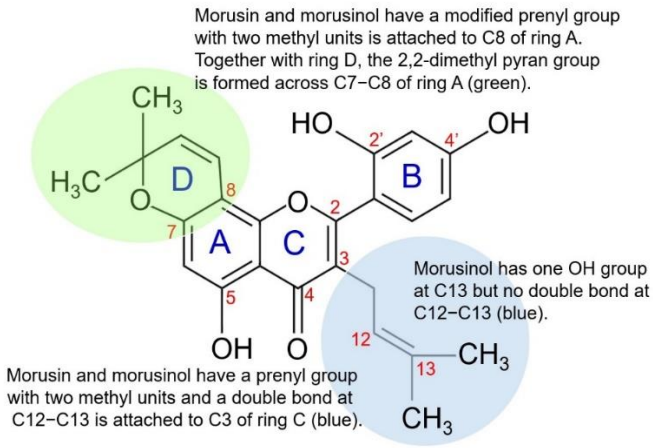

Figure 2: Molecular structures of morusin and morusinol.

Morusinol is another prenylated flavone with a molecular formula of $C_{25}H_{26}O_7$ and molecular weight of 438.5 g/mol.[13] It was first isolated from the root bark of *M. alba* by Konno *et al.*[20] Its chemical structure (Figure 2) is very similar to that morusin. It has three benzene rings, two phenyl groups at C3 and C8, a carbonyl moiety at C4, and three OH groups at C5, C2' and C4'. The phenyl groups at C3 and C8 have two methyl units each. Like morusin, the phenyl group at C8 and ring D in morusinol form the 2,2-dimethyl pyran group across C7 and C8. The C3 phenyl group of morusinol differs from morusin by an extra OH group at C13, but lacks the C12−C13 double bond.

Pharmacological Properties
Anti-cancer properties

Morusin

Morusin has been reported to exert antitumor effects on multiple types of cancer cells such as breast (4), liver (4), lung (4), cervical (3), prostate (3), colorectal (3), skin (2), gastric (1), nasopharyngeal (1), ovarian (1), pancreatic (1) and renal (1) cancer cells, including glioblastoma (3) and osteosarcoma (1) (Table 1).

Morusin inhibited HT-29 colorectal, Hep3B liver and MCF-7 breast cancer cells with IC_{50} values of 6.1, 8.5 and 12.7 μM, respectively.[21] Morusin inhibited the proliferation or viability of MDA-MB-231 and MCF-7 breast cancer cells, and A549 lung cancer cells with IC_{50} values of 3.2, 3.4, and 3.1 μM, respectively, after 24 h of incubation.[22] Against prostate cancer cells of DU145, M2182, PC3 and LNCaP, the IC_{50} growth inhibition of morusin was 26, 22, 20 and 22 μM, respectively.[23] When tested against normal epithelial prostate cells of RWPE-1, inhibition was significantly weaker at 43 μM. Morusin from the leaf also possesses cytotoxic properties with IC_{50} values of 0.6, 7.9 and 9.2 μM against HeLa cervical, MCF-7 breast, and Hep3G liver cancer cells, respectively.[15] Against P-388 leukemia cells, the IC_{50} value of morusin was 10 μM.[24] Morusin with a prenyl unit at C7 and C8 might have attributed to its stronger cytotoxicity than kuwanon C (14 μM) and 5,7,2',4'-tetrahydroxy-3-methoxy-flavone (37 μM).[24,25] In another study on the cytotoxicity of morusin loaded in niosomes, cytotoxicity showed 21.5% (MDA-MB-453), 26.3% (HT-29), 35.9% (PANC-1) and 46.3% (SKOV-3) mortality of cancer cells after 48 h of treatment.[26] When tested against liver cancer cells, the IC_{50} values of morusin were 4.90 and 4.20 μg/ml against HepG2 and Hep3B, respectively.[27] Against LO2 normal liver cells, cytotoxicity was weak at 12.5 μg/ml. Morusin promoted the apoptosis, and inhibited the growth and migration of MDA-MB-453 breast and HCT116 colon cancer cells.[28] Morusin inhibited angiogenesis, tumor progression and tumor migration, and triggered apoptosis, cell cycle arrest and autophagy in breast, liver, lung, cervical, prostate, colorectal, glioblastoma, gastric, nasopharyngeal, ovarian and pancreatic cancer cells (Table 1).

This review showed that the anti-cancer properties of morusin involved the following types of cancer and molecular mechanisms:

1. Promoted adipogenic differentiation and lipo-apoptosis mediated by peroxisome proliferator-activated receptor γ (PPARγ) and CCAAT/enhancer-binding protein beta (C/EBPβ),[32] increased Bcl2-associated X protein (Bax) and decreased Survivin expression,[33] modulated phosphoinositide 3-kinase (PI3K)/protein kinase B (Akt) signaling,[34] and targeted epidermal growth factor receptor (EGFR) and SRC[35] in breast cancer cells.
2. Induced apoptosis *via* angiogenesis inhibition,[27] signal transducer and activator of transcription 3 (STAT3), and nuclear factor-kappa B (NF-κB) pathways,[29] mitogen-activated protein kinase (MAPK),[30] and exerted antitumor effect *via* activated adenosine monophosphate-activated protein kinase (AMPK)[31] activation in liver cancer cells.
3. Induced apoptosis *via* suppression of EGFR/STAT3,[36] and down-regulated the expression of cyclooxygenase-2 (COX-2) and vascular epithelial growth factor (VEGF),[37] AMPK activation,[38] extracellular signal-regulated kinase (ERK) and PI3K)/Akt signaling[39] in lung cancer cells.

Table 1: Anti-cancer activities of morusin from *Morus alba* against different cancer cells.

Cancer type, cell line, effect and molecular mechanism	Reference
Liver cancer	
H_{22}: Morusin inhibited tumor growth in mice with transplanted hepatocarcinoma. The injection of 40 mg/kg/day of morusin had a tumor suppression effect (46%) similar to that of 5-FU (57%), a cancer drug.	17
HepG2 & Hep3B: Morusin exhibited potent antitumor cell activity *in vitro* and *in vivo* through apoptosis induction and angiogenesis inhibition. The mechanism involved the suppression of the IL-6/STAT3 signaling pathway.	27
SK-Hep1: Morusin inhibited tumor cell progression by suppressing STAT3 and NF-κB, reduced the activity of MMP-2 and -9, and decreased lung colonization of tumor cells in nude mice.	29
Bel-7402: Morusin induced apoptosis of cells by increasing the expression of P-ERK1/2 and P-JNK, and up-regulating the mitochondrial and MAPK pathways.	30
Hep3B & Huh7: Morusin increased cell cytotoxicity, and displayed antitumor activity *via* AMPK-mediated G1 arrest and anti-glycolysis of cells.	31
Breast cancer	
MCF-7 & MDA-MB-231: Morusin inhibited cell apoptosis, adipogenic differentiation, and lipo-apoptosis *via* C/EBP*β* and PPAR*γ*. The administration of 5 and 10 mg/kg of morusin into mice inoculated with MCF-7 cells resulted in tumor inhibitory rates of 46.5% and 64.1%, respectively.	32
MCF-7, MDA-MB-231, MDA-MB-157 & MDA-MB-453: Morusin induced cell apoptosis by inducing Bax (pro-apoptotic) and suppressing Survivin (anti-apoptotic). Morusin was not cytotoxic to normal human breast epithelial cells (MCF10A).	33
MDA-MB-231, BT549 & Hs578T: Morusin promoted apoptosis and inhibited cell migration by modulating the PI3K-Akt pathway. Morusin also increased the expression of cleaved-PARP, and decreased the expression of p-PI3K and p-Akt.	34
MCF-7, MDA-MB-231, MDA-MB-157 & MDA-MB-453: Morusin suppressed cancer cell growth by targeting EGFR, SRC and MAPK1.	35
Lung cancer	
H1299, H460 & H292: Morusin induced apoptosis of cells by suppressing EGFR/STAT3 activity. The anticancer effects of morusin in erlotinib-resistant H1975 cells suggest that it can be used for treatment of advanced lung cancer cells with acquired resistance to EGFR TKI.	36
A549: Morusin induced apoptosis and suppressed migration of cells by down-regulating the expression of COX-2 and VEGF, suggesting its ability to check lung tumor angiogenesis.	37
H1299 & H460: Morusin induced cell apoptosis and autophagy *via* AMPK activation, suggesting that the combined treatment with morusin and autophagy inhibitor might be an effective therapy for chemo-resistant lung cancer cells.	38
A549 & NCI-H292: Morusin induced cell apoptosis, and autophagy. PI3K/Akt suppression, and ERK and JNK activation contributed to apoptosis and autophagy. The increased intracellular ROS generation might be a possible mechanism of action of morusin.	39
Cervical cancer	
HeLa: Morusin inhibited cell growth and migration, and induced cell apoptosis. Mechanisms involved in apoptosis were attenuation of NF-κB activity, decrease in Bcl-2, and increase in Bax and caspase-3.	40
HeLa: When used with 3-MA (autophagic inhibitor), apoptosis induced by morusin is stronger than morusin alone. Overall, autophagy induced by morusin enhanced cell survival by inhibiting cell death.	41
HeLa: Morusin exerted stronger cell apoptosis when used with SG. Morusin activated RACK1 while SG reduced caspase-3 activation. Overall, SG suppressed the antitumor capacity of morusin.	42
Prostate cancer	
DU145, M2182, PC3, LNCaP & RWPE-1: Morusin induced cell death by inactivating STAT3 signaling and activating SHP1, a tyrosine phosphatase.	23
DU145 & PC3: Morusin induced apoptosis and anti-Warburg effect *via* ROS-mediated inhibition of FOXM1/c-Myc signaling.	43
PC3 & 22Rv1: Morusin induced apoptosis and G2/M cell cycle arrest, suppressed TGF-*β* cell migration and invasion, and inhibited EMT *via* inhibition of the Akt/mTOR signaling pathway. Morusin also attenuated tumor growth in a xenograft murine model.	44

Glioblastoma WJ1 & WJ2: Morusin suppressed the growth of stem cells *via* stemness attenuation, adipocyte trans-differentiation, apoptosis induction, and PPARγ up-regulation *in vitro* and *in vivo*. Morusin was cytotoxic against GSC (3.9 μg/ml), but not against normal liver cells (38 μg/ml).	45
U87MG, U373MG & T98G: Morusin induced TRAIL sensitization of cells by suppressing the STAT3 pathway and reducing Survivin, XIAP, EGFR and DR5.	46
U87 & GI1: Morusin and CTX loaded in PLGA nanoparticles induced cell proliferation and apoptosis *via* ROS generation, enhanced caspase activity, and inhibition of MMP activity. CTX is a peptide derived from scorpion venom.	47
GL261, U87 & U251: Morusin reduced the migration of cells *via* the CCL4-CCR5 axis.	48
Colorectal cancer HT-29: Morusin induced apoptosis of cells by activation of caspases, suppression of NF-κB activity and reduction of the inhibitory effect of XIAP.	49
HCT116 and SW480: Morusin induced cell apoptosis *via* increase in PARP and caspase 3, and suppression of ZNF746 and c-Myc activity.	50
HCT116: Morusin inhibited the growth of sphere-forming cells *via* induction of cell cycle arrest, inactivation of Akt pathway, and followed by suppression of β-catenin signaling.	51
Skin cancer A375 & A2058: In combination with MAPK pathway inhibitors, morusin possessed more potent antitumor activity against BRAF-mutant melanoma cells than MAPK pathway inhibitors alone both *in vitro* and *in vivo*. Inhibition involved activation of the STAT3/SOX2 pathway.	52
A375 & MV3: Morusin displayed potent antitumor activity through induction of cell apoptosis and cell cycle arrest. Morusin also inhibited cell proliferation, migration, and invasion through activation of p53-mediated pathways. Cytotoxicity of morusin in IC_{50} value was 4.6 and 9.7 μM, respectively.	53
Gastric cancer MKN45 & SGC7901: Morusin inhibited cell proliferation and tumor growth both *in vitro* and *in vivo* by promoting cell cycle arrest and down-regulating c-Myc.	54
SNU-1 & AGS: Morusin suppressed cell stemness characteristics by inhibiting HIF-1α accumulation and nuclear translocation.	55
Nasopharyngeal cancer HONE-1, NPC-39 & NPC-BM: Morusin inhibited cell migration and invasion by suppressing the expression of MMP-2 and by down-regulating the ERK1/2 pathway.	56
Ovarian cancer A2780, SKOV-3 & HO-8910: Morusin induced paraptosis-like cell death both *in vitro* and *in vivo* through mitochondrial calcium overload and dysfunction. Morusin also increased ROS generation and induced ER stress.	57
Pancreatic cancer AsPC-1, BxPC-3, MIA PaCa-2 & PANC-1: Morusin induced apoptosis and inhibited invasion of cells by down-regulating STAT3 signaling. The latter was mediated through the suppression of JAK1, JAK2 and c-Src kinases.	58
Renal cancer 769-P, 786-O & OSRC-2: Morusin inhibited cell proliferation and migration, and induced apoptosis and cell cycle arrest at G1 phase *via* suppression of the MAPK signaling pathways. Results of nude mouse xenograft confirmed that morusin inhibited tumor growth *in vivo*.	59
Osteosarcoma U2OS & HOS: Morusin suppressed proliferation, induced apoptosis, and reduced migration and invasion of cells *via* inhibition of the PI3K/Akt signaling pathway and the expression of MMP-2 and -9.	60
Gall-bladder cancer GBC-SD & NOZ: Morusin reversed EMT in cells by regulating STAT3/HIF-1α signaling.	61

Abbreviations: Akt = protein kinase B, AMP = adenosine monophosphate-activated protein, AMPK = adenosine monophosphate-activated protein kinase, Bax = Bcl-2-associated X protein, Bcl-2 = B-cell lymphoma 2, BRAF = v-raf murine sarcoma viral oncogene homolog B1, CCAAT-enhancer binding protein β (C/EBPβ), CCL4 = chemokine CC motif ligand 4, CCR5 = chemokine receptor 5, C kinase-1 = RACK1, COX-2 = cyclooxygenase-2, CTX = chlorotoxin, DR5 = death receptor 5, EGFR = epidermal growth factor receptor, EMT = epithelial-mesenchymal transition, ER = endoplasmic reticulum, ERK = extracellular signal-regulated kinase, FOXM1 = Forkhead box protein M1, 5-FU = 5-fluorouracil, HIF-1α = hypoxia-inducible factor-1α, IL-6 = interleukin 6, JAK = Janus kinase, JNK = c-Jun N-terminal kinase, 3-MA = 3-methyladenine, MAPK = mitogen-activated protein kinase, MMP = matrix metallopeptidase, MOR = morusin, mTOR = mammalian target of rapamycin, NF-κB = nuclear factor-kappa B, PARP = poly (ADP-ribose) polymerase, PI3K = phosphoinositide 3-kinase, PLGA = polylactic-co-glycolic acid, PPARγ = peroxisome proliferator-activated receptor γ, RACK1 = C kinase-1, ROS = reactive oxygen species, SG = stress granules, SOX2 = SRY-box transcription factor 2, STAT3 = signal transducer and activator of transcription 3, TGF = transforming growth factor, TKI = tyrosine kinase inhibitor, TRAIL = tumor necrosis factor-related apoptosis-inducing ligand, VEGF = vascular epithelial growth factor, XIAP = X-linked inhibitor of apoptosis protein, and ZNF746 = zinc finger protein 746.

4. Induced apoptosis *via* attenuation of NF-κB activity,[40] autophagy,[41] and activation of C kinase-1 (RACK1),[42] in cervical cancer cells.
5. Inactivated STAT3 signaling,[23] inhibited Forkhead box protein M1 (FOXM1)/c-Myc,[43] epithelial-mesenchymal transition (EMT), and protein kinase B/mammalian target of rapamycin (Akt/mTOR)[44] in prostate cancer cells.
6. Suppressed the growth of stem cells *via* stemness attenuation, adipocyte trans-differentiation, apoptosis induction, and peroxisome proliferator-activated receptor γ (PPARγ) up-regulation,[45] and induced tumor necrosis factor-related apoptosis-inducing ligand (TRAIL) expression of cells by regulating EGFR and death receptor 5 (DR5) in glioblastoma cells.[46] Morusin also inhibited glioblastoma cell growth *via* reactive oxygen species (ROS) generation, enhanced caspase activity, and inhibition of matrix metallopeptidase (MMP) activity,[47] and reduced cell migration *via* the chemokine CC motif ligand 4 (CCL4)-chemokine receptor 5 (CCR5) axis.[48]
7. Induced apoptosis *via* activation of caspases and suppression of NF-κB activity,[49] suppression of ZNF746 and c-Myc activity,[50] and inhibited the growth of sphere-forming cells *via* the inactivation of Akt pathway[51] in colorectal cancer cells.
The molecular mechanisms of morusin against other cancer cells i.e., skin,[52,53] gastric,[54,55] nasopharyngeal,[56] ovarian,[57] pancreatic[58] and renal[59] cancer including osteosarcoma[60] are summarized in Table 1. Colorectal and skin cancer cells are represented by two studies each, while gastric, nasopharyngeal, ovarian, pancreatic, renal and gall-bladder cancer cells are represented by one study each.

Morusinol
Unlike morusin that has anti-cancer effects towards a wide array of cancer cells, morusinol has only a few studies reporting its anti-cancer properties. From the root bark of *M. alba*, the IC_{50} cytotoxicity of morusinol against THP-1 monocytic leukemic cells was 4.3 μM.[62] Against NCM460 normal colon epithelial cells, the cytotoxicity of morusin was very weak at 56.2 μM. The IC_{50} cytotoxicity of morusinol against A375 and MV3 melanoma cells was 18.0 and 10.0 μM, respectively.[63] Against a panel of HCT116, HCT15, SW480, SW620 and LOVO colorectal cancer cells, the IC_{50} values of morusinol were 13.8, 16.2, 12.3, 21.5 and 17.5 μM, respectively.[64] When tested against a panel of colorectal, pancreatic and gastric cancer cells (two types each), strongest IC_{50} cytotoxicity was 6.0 μM and 9.5 μM against ASPC1 pancreatic and SW620 colorectal cancer cells, respectively.[13]

The anti-cancer properties of a commercial morusinol (99.7% purity) purchased from DESITE, Chengdu, China, were tested against A375 melanoma cells.[63] Results showed that morusinol significantly inhibited melanoma growth by inducing cell cycle arrest at G0/G1 phase, apoptosis, and DNA damage. Morusinol was found to be a potent CHK1 inhibitor. The *in vivo* study affirmed the antitumor activity of morusinol using a xenograft mouse model. Overall, morusinol was found to be promising for treating malignant melanoma.[63]

The anti-cancer properties of morusinol (>98% purity) purchased from Lemeitian Medicine, Chengdu, China, was tested against HCT116 and HCT15 colorectal cancer cells.[64] Morusinol inhibited cell proliferation, apoptosis, and induced autophagy *via* promotion of forkhead box O3 (FOXO3) nuclear accumulation, which suppressed sterol regulatory element binding transcription factor 2 (SREBF2). Results of tumor xenograft in mice showed that morusinol significantly impeded tumor growth. It was suggested that morusinol can serve as an anticancer drug for treating colorectal cancer cells.[64]

A group of scientists from the Department of Pathology, Shanghai East Hospital, China, published a paper on the antitumor activity of morusinol against SK-HEP-1 liver cancer cells in 2019.[65] Reported for the first time, the antitumor activity of morusinol involved autophagy, G2/M cell cycle arrest, inhibition of cell invasion and migration, and targeting of the Ras/MEK/Erk pathway. Two years later in 2021, the journal (Medical Science Monitor Journal) retracted the 2019 paper because of a breach of publishing guidelines as figures were not original and manipulated.[66]

Other Pharmacological Activities

Morusin
Besides its anti-cancer properties, morusin from *Morus* species has been reported to possess many other bioactivities. These bioactivities include antinociceptive,[67] anti-osteoarthritis,[68] anti-inflammatory,[68,69] tyrosinase,[69] anti-bacterial,[70,71] anti-HIV,[72] anti-diabetes,[73] neuro-protective,[74,75] anti-obesity,[76] platelet aggregation,[77] anti-nephritis[78] and anti-mycoplasma pneumonia[79] properties. Lifespan extension,[80] inhibition of post-menopausal osteoporosis,[81] inhibition of idiopathic pulmonary fibrosis,[82] anti-tuberculosis[83] and anti-pseudo allergic reactions[84] are new pharmacological findings of morusin. The compound is found to be inactive in the inhibition of α-glucosidase,[85] β-secretase and acetylcholinesterase (AChE)[84] and phospodiesterase-4 (PDE-4).[87]

Morusinol
Other pharmacological properties of morusinol from *Morus* species include inhibition of arterial thrombosis,[88] inhibition of TXB_2 formation,[88] modulation of platelet activation,[88,89] and antibacterial activity.[90] The antibacterial activity of morusinol is markedly less than that of morusin.[91] This has been attributed to the modified C3 prenyl group in the former. Morusinol did not show any inflammatory activity based on inhibitory effect on lipopolysaccharide (LPS)-activated nitric oxide (NO) production in RAW264.7 cells.[92]

Conclusion

Some terminological aspects of the anti-cancer effects of morusin are worthy of further investigation. They include lipo-apoptosis, paraptosis, cytoprotective autophagy, cytotoxic autophagy, anti-Warburg effect and adipocyte trans-differentiation. The structure-activity-relationships (SAR) of morusin and morusinol with respect to anti-cancer properties are poorly studied and need more in-depth studies. The potential applications of morusin and morusinol in the treatment of cancers present new domains for research scientists. The clinical potentials of morusin pose exciting research through clinical studies. The pharmacokinetics of morusin and morusinol, and their synergistic activity in combination with other drugs or bioactive compounds require further analysis with regard to their health-promoting benefits to humans.

Conflict of Interest

The author declares no conflict of interest.

Author's Declaration

The author hereby declares that the work presented in this article is original and that any liability for claims relating to the content of this article will be borne by him.

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