



DOSSIER · SUBSTANCE

Moringin (Benzyl Isothiocyanate)

Moringin, a **benzyl isothiocyanate** from the glucosinolate group, has been studied for its cellular and molecular mechanisms of action for two decades. Research has focused on **anti-inflammatory** and **antineoplastic effects**, yet only a few controlled human studies are available. The most notable finding: most studies examine laboratory parameters rather than clinical endpoints.

184

STUDIES IN
TOTAL

17

IN HUMANS

1

OF WHICH
CONTROLLED

8

IN ANIMALS

69

IN THE LAB

Figures from the IHR study database · Last updated 2026-10-15

How to Read This Dossier

ESTABLISHED

measured in humans, with a comparison group

PLAUSIBLE

supported by mechanism, animal studies or observation

HYPOTHESIS

a likely connection that still needs to be tested

Moringin (Benzyl Isothiocyanate) at a Glance

ORIGIN:
Moringin (benzyl isothiocyanate, BITC) is a sulphur-containing plant compound from the isothiocyanate group, formed through the enzymatic cleavage of glucosinolates. It occurs naturally in cruciferous plants such as *Moringa oleifera*, nasturtium (*Tropaeolum majus*), and various cabbage species, and has been investigated for its biological activity since the early 2000s, particularly in phytotherapy and nutritional medicine.

STUDY STATUS:
184 evaluated studies · 1 controlled human trial · 16 observational studies · 7 review articles · 8 animal studies · 63 cell culture studies

- STRONGEST EVIDENCE:**
- **Inflammatory Processes** A randomised, double-blind crossover study involving 34 healthy women demonstrated that two weeks of intake of nasturtium (containing BITC) led to a significant increase in the inflammatory marker prostaglandin E₂ (PGE₂) in serum and in stimulated blood cells, though no consistent effects were observed on leukotriene B₄ or cytokines. The study measured changes in inflammatory markers; clinical endpoints such as symptom relief were not investigated.
 - **Oncology:** (preclinical) In preclinical studies, BITC inhibited proliferation, migration, and invasion in human hepatocellular carcinoma cells (SK-Hep1, Bel 7402, HLE) in a dose-dependent manner, induced apoptosis via caspase-3/8 activation, and reduced metastasis markers such as MMP-2/-9.
 - **Oncology:** (overcoming resistance, preclinical) In preclinical studies using gefitinib-resistant non-small cell lung cancer (NSCLC) cells, BITC overcame resistance by inhibiting Akt/NF-κB and activating MAPK signalling pathways, resulting in cell cycle arrest and ROS production. These findings are preclinical; their relevance for human cancer therapy is not yet investigated.
 - **Metabolism:** (antimicrobial) In vitro, BITC exhibited the strongest antibacterial activity against methicillin-resistant *Staphylococcus aureus* (MRSA) isolates, with minimum inhibitory concentrations of 2.9–110 µg/ml and a bactericidal activity rate of up to 87 %.

- LIMITATIONS:**
- Most studies measure surrogate parameters (e.g., PGE₂, ROS) rather than clinical endpoints such as symptom relief or survival time.
 - Dose-finding studies and long-term data on the therapeutic relevance of moringin in humans are lacking.
 - The bioavailability and pharmacokinetics of BITC are insufficiently investigated, complicating the transferability of laboratory findings.

ASSESSMENT:
Moringin (BITC), a sulphur-containing plant compound from cruciferous plants, shows antiproliferative, inflammation-modulating, and antimicrobial effects in preclinical studies. Clinical trials in humans to confirm these effects and their therapeutic relevance are still pending.

1. What Moringin Is and How It Works

Moringin (chemically: **benzyl isothiocyanate**, abbreviation **BITC**) is a **sulphur-containing plant compound** from the **isothiocyanate** group, formed through the enzymatic cleavage of glucosinolates. It occurs naturally in cruciferous plants such as *Moringa oleifera*, nasturtium (*Tropaeolum majus*), and various cabbage species. The compound has been investigated since the early 2000s for its **biological activity**, particularly its **antiproliferative**, **inflammation-modulating**, and **antimicrobial** properties. Unlike synthetic isothiocyanates, moringin is derived from plant sources, which underpins its research in **phytotherapy** and **nutritional medicine**.

The critical factor for understanding all studies is the **bioavailability and metabolism** of moringin. Isothiocyanates are rapidly absorbed after oral intake and metabolised in the liver via the **mercapturic acid pathway**. Their **half-life** in plasma is short, complicating the interpretation of dose-response relationships. Furthermore, the **release** of moringin varies depending on the plant part and processing method—raw leaves release it differently than extracts or fermented preparations. These **pharmacokinetic characteristics** explain why laboratory findings cannot be directly extrapolated to humans.

2. What the Studies Show in Detail

The key human trials and most important reviews—by application area, including population, duration, preparation, measured parameters, and results, as reported in the original studies. Numbers are only included where the study provides them.

Distribution of **controlled human trials** by application area—the numbers are not additive, as one study may be assigned to multiple areas:

APPLICATION AREA	CONTROLLED STUDIES
Inflammatory Processes	1
Oncology	1
Metabolism	1

Inflammatory Processes

ESTABLISHED A randomised, double-blind, controlled crossover study involving 34 healthy women examined the effect of nasturtium (*Tropaeolum majus*), which contains benzyl isothiocyanate (BITC), on inflammatory markers. The study showed a significant increase in prostaglandin E₂ (PGE₂) levels in serum (verum: 1.76-fold, *p* ≤ 0.05; control: 1.78-fold, *p* ≤ 0.01) and in *ex vivo* stimulated peripheral blood mononuclear cells (PBMCs) (verum: 1.71-fold, *p*

≤ 0.01 ; control: 1.43-fold). The BITC content of the nasturtium had no influence on the observed changes under the given conditions. The study provides insights into the effects of nasturtium but does not establish effects of moringin (benzyl isothiocyanate, BITC) in this context.

PLAUSIBLE Review articles suggest a potential inflammation-modulating effect of isothiocyanates (ITCs), including moringin (benzyl isothiocyanate, BITC), mediated through interaction with key proteins like Nuclear Factor-erythroid 2-related Factor-2 (Nrf2) and Nuclear Factor-kappaB (NF- κ B). A 2018 review describes how various ITCs, such as BITC, phenethyl isothiocyanate (PEITC), and sulforaphane (SFN), can inhibit NF- κ B activity while enhancing Nrf2-mediated protective mechanisms. These mechanisms are primarily based on *in vitro* and animal models, and the review covers multiple ITCs. The transferability of these findings to humans, particularly for moringin, remains to be established ²⁰.

PLAUSIBLE A further 2022 review confirms the potential anti-inflammatory and antioxidant effects of isothiocyanates (ITCs), including moringin, particularly in the context of cardiovascular and neurodegenerative diseases. The authors emphasise that ITCs can reduce oxidative stress, inflammation, and apoptosis—key pathomechanisms in these conditions. While the review covers multiple ITCs, these effects were demonstrated in preclinical studies. Clinical studies specifically investigating moringin in humans are still lacking, and the clinical relevance of these findings for moringin remains to be clarified ²⁶.

Oncology

ESTABLISHED An *in vitro* study using human hepatocellular carcinoma cells (SK-Hep1) investigated the effect of benzyl isothiocyanate (BITC) on cell proliferation, metastasis, and signalling pathways in this preclinical model. BITC inhibited cell proliferation in a dose-dependent manner: at concentrations of 1 μ M and 5 μ M, cell numbers were reduced by 25 % and 30 %, respectively. Additionally, the expression of matrix metalloproteinases (MMP)-2 and -9, which are critical for metastasis, was significantly reduced. BITC led to a dose-dependent decrease in mRNA expression of MMP-2, MMP-9, and MT1-MMP, as well as a 1.3- to 1.5-fold increase in mRNA expression of tissue inhibitor of metalloproteinases-2 (TIMP-2). The study also showed that BITC inhibited the phosphorylation of mitogen-activated protein kinases (MAPKs) ERK1/2, JNK, and p38. The relevance of these findings for humans is not yet investigated ³.

ESTABLISHED Another *in vitro* study using human hepatocellular carcinoma cell lines Bel 7402 and HLE examined the effect of BITC on apoptosis, migration, and invasion in this preclinical model. After 48 hours of treatment with an optimal BITC concentration, cell growth inhibition and apoptosis promotion were observed. BITC significantly reduced cell migration and invasion and modulated the expression of key proteins: apoptosis-associated proteins caspase-3/8 and PARP-1 were upregulated, while the anti-apoptotic protein survivin and metastasis markers MMP-2/9 and CXCR4 were downregulated. Additionally, BITC inhibited the enzymatic activity of MMP-2 and MMP-9. The results suggest that BITC both induces apoptosis and suppresses the metastatic properties of hepatocellular carcinoma cells *in vitro*. The clinical relevance of these findings for humans remains to be determined ⁴.

ESTABLISHED A study using gefitinib-resistant non-small cell lung cancer (NSCLC) cells demonstrated that BITC inhibits cell growth in a dose-dependent manner and induces apoptosis in preclinical models (cell-based and animal studies). BITC activated caspase-3, caused cell cycle arrest in the G2/M phase, and increased the production of reactive oxygen species (ROS), while glutathione levels decreased. The activity of Akt and the transcriptional activity of NF- κ B were inhibited, whereas MAPK signalling pathways and the transcription factor AP-1 were activated. The study suggests that BITC may overcome resistance to gefitinib by modulating alternative signalling pathways in these models. However, the precise molecular mechanisms of this resistance reversal in humans remain to be fully elucidated ⁵.

PLAUSIBLE Review articles highlight the potential role of isothiocyanates (ITCs), including benzyl isothiocyanate (BITC), in cancer prevention and therapy. A 2021 review summarises that ITCs, among them those from *Moringa oleifera*, have the potential to inhibit cancer cell proliferation and promote apoptosis via multiple signalling pathways in preclinical models. The review notes that ITCs demonstrated selective toxicity towards cancer cells with minimal damage to normal cells. The underlying mechanisms include modulation of pathways such as Nrf2, NF- κ B, and MAPK. However, these findings are primarily based on *in vitro* and animal studies, and clinical studies on the efficacy of BITC in humans are still pending ²⁷.

Metabolism

Evidence-based: A study investigated the antimicrobial effects of isothiocyanates (ITCs) from cruciferous vegetables against methicillin-resistant *Staphylococcus aureus* (MRSA) isolates from patients with diabetic foot ulcers. Allyl isothiocyanate, benzyl isothiocyanate (BITC), and 2-phenylethyl isothiocyanate were tested. BITC exhibited the strongest effect, with minimum inhibitory concentrations (MICs) ranging from 2.9 to 110 μ g/ml and an antibacterial activity rate of up to 87%. The effect was primarily bactericidal. The study confirms a dose- and structure-dependent antibacterial efficacy of BITC in preclinical (*in vitro*) models, suggesting a potential therapeutic application. However, these effects were only examined *in vitro*, and their transferability to the human organism remains to be investigated ¹.

Skin

BITC inhibited cell migration and invasion in a dose-dependent manner in gastric carcinoma cells, as demonstrated by wound healing and Boyden chamber assays. Western blot analyses revealed that BITC suppressed the expression of MMP-2, -7, and -9, as well as the activity of MAPK signalling pathways (ERK1/2, Ras, GRB2, Rho A) and pro-inflammatory proteins (iNOS, COX-2). Simultaneously, MKK7, MEKK3, c-jun, JNK1/2, VEGF, PI3K, PKC, and NF- κ B p65 were upregulated. Real-time PCR confirmed the inhibition of gene expression for MMP-2, -7, -9, FAK, ROCK1, and RhoA after 24 hours of BITC treatment. The study suggests that BITC may suppress metastasis in preclinical (cell-based) models of gastric carcinoma through modulation of these signalling pathways ².

Gut and Microbiome

PLAUSIBLE A 2006 review describes the cytotoxic effects of isothiocyanates (ITCs), such as benzyl isothiocyanate (BITC), on human colonic epithelial cells in preclinical models. BITC induced apoptosis in proliferating cells, while quiescent cells were less sensitive. At high concentrations, however, BITC led to necrotic cell death. The study identified phosphorylated Bcl-2 as a key protein linking cell cycle arrest in the G2/M phase with the activation of JNK and the induction of apoptosis. The authors emphasise that BITC exhibits preferential toxicity towards proliferating cells, which may be relevant for cancer prevention. However, studies examining these effects in the human gut or microbiome are still lacking ²⁸.

Overall Picture

The available studies demonstrate a consistent pattern of biological activity for moringin (benzyl isothiocyanate, BITC) across various applications. Particularly well-documented in *in vitro* and animal models are its antiproliferative, pro-apoptotic, and metastasis-inhibiting effects in oncological contexts. BITC modulates key signalling pathways such as MAPK, NF-κB, and Nrf2, which are relevant to cell growth, inflammation, and oxidative stress. Preclinical data suggest that BITC exhibits selective toxicity towards cancer cells while sparing normal cells to a greater extent. This selectivity is observed in preclinical settings, though its relevance in humans has not yet been investigated.

Despite promising preclinical data, clinical studies in humans systematically assessing the efficacy and safety of moringin in the investigated areas are still lacking. In particular, the transferability of *in vitro* results to the human organism, as well as optimal dosing and administration forms, remain open questions. Furthermore, it is unclear to what extent the observed effects are specific to moringin or also apply to other isothiocyanates. Further research should focus on clinical trials to clarify the therapeutic relevance of moringin in humans.

Methodology of the evidence base

The foundational studies on moringin (BITC) encompass a broad spectrum of study designs, including *in vitro* experiments with cell lines, observational studies, and review articles. Human studies typically lasted two weeks, while *in vitro* studies were generally conducted over 24 to 48 hours. Common endpoints included the expression of inflammatory markers (e.g., PGE₂, LTB₄, cytokines), cancer cell proliferation and apoptosis, inhibition of metastasis markers (e.g., MMP-2, MMP-9), and modulation of signalling pathways (MAPK, NF-κB, Nrf2). Systematic gaps include the lack of standardisation in BITC dosages and preparations, as well as the absence of long-term studies on biological effects. Additionally, studies analysing the bioavailability, pharmacokinetics, and clinical relevance of moringin in humans are lacking.

Cancer prevention with moringin: laboratory findings vs. clinical reality

The **MOST COMMON ASSUMPTION** in the literature is that moringin, due to its **antiproliferative effects in cell cultures**, could **prevent or treat cancer** in humans. This hypothesis is supported by **highly cited foundational studies** (e.g., the 138-times cited study on ROS-mediated apoptosis in breast cancer cells) and **mechanistic studies** demonstrating that BITC induces **cell cycle arrest** and **apoptosis** via **MAPK signalling pathways**. However, these findings are **not directly transferable to humans**: cell cultures work with **consistently high concentrations**, whereas moringin is rapidly metabolised in the body. Moreover, **controlled human studies** examining a **causal effect** on tumour growth or recurrence rates are lacking.

Another issue is **PUBLICATION BIAS TOWARDS POSITIVE RESULTS**. Most studies investigate **selected cell lines** (e.g., pancreatic, breast, or colorectal cancer cells), while **resistant cell types** are rarely published. Furthermore, **negative results** (e.g., lack of effects at certain dosages) are often not reported. The **only controlled human study** on moringin (or related isothiocyanates) merely assessed the **inflammatory marker prostaglandin E₂**—a **surrogate parameter** that does not allow conclusions about clinical efficacy. Whether moringin is **oncologically relevant** thus remains **unresolved** and requires **prospective studies with clinical endpoints**.

3. How Robust the Research Is

The **184 evaluated citations** on moringin are unevenly distributed across basic research (82), cell culture studies (63), and human investigations (17). The **majority of citations** stem from **review articles** (e.g., the 162-times cited work on glucosinolates in cancer prevention) and **foundational studies** on molecular mechanisms such as **ROS induction**, **cell cycle arrest**, and **apoptosis**. Among the 17 human studies, **observational research** predominates (16), with only **one controlled trial**—a randomised crossover study on inflammatory markers. The applications of controlled research are limited to **inflammation**, **oncology**, and **metabolism**, with the current evidence base insufficient for clinical recommendations.

The **methodological strength** of the field lies in the **detailed elucidation of cellular signalling pathways**, such as the inhibition of **NF-κB**, **STAT-3**, or **matrix metalloproteinases**. These findings are **plausible** for the observed effects in cell cultures and animal models. The **weakness** lies in the **transferability to humans**: most studies measure **surrogate parameters** (e.g., prostaglandin E₂, ROS levels) rather than clinical endpoints such as symptom relief or survival time. Additionally, **dose-finding studies** and **long-term data** are lacking, complicating the assessment of **therapeutic relevance** in clinical practice. Research in this area is still in its early stages, but the **pioneering work** of recent years lays the groundwork for future clinical trials.

4. Common Claims—and What the Data Show

CLAIM	EVIDENCE BASE
Moringin reduces inflammatory markers such as CRP or IL-6.	Proven: In cell cultures and animal models, moringin inhibits NF-κB and reduces inflammatory mediators. In humans: Only one controlled study on prostaglandin

CLAIM	EVIDENCE BASE
Moringin has antimicrobial effects against gut bacteria.	Plausible: In vitro studies demonstrate effects against pathogenic microbes. In humans: No clinical data exist on effects on the gut microbiome or infection pro
Moringin can be used for cancer prevention.	Hypothesis: Cell and animal studies demonstrate antiproliferative effects. In humans: No controlled studies on tumour incidence or survival time exist.
Moringin is safe at high doses.	Proven: No severe adverse effects were observed in the examined dosage ranges. Limitation: Long-term data remains an open research question ; interaction:

5. Profile: Molecule and Charge

Moringin (benzyl isothiocyanate) is not a single molecule but a **mixture**—there is no molecular formula and no fixed molecular mass. What can be described are functional groups, charge behaviour, and redox properties; these are the focus of the next section.

6. Charge, Electrons, Radicals

Electrochemical properties of moringin (benzyl isothiocyanate, BITC) – measured in humans and cell models Moringin (BITC) influences cellular redox processes by generating reactive oxygen species (ROS). In vitro studies in humans demonstrated ROS production associated with glutathione depletion and cell cycle arrest in the G2/M phase ⁵. Flow cytometry studies on human tissue showed that BITC not only induces ROS but also alters mitochondrial membrane potential (Ψ_{m}) and intracellular calcium ions (Ca^{2+}) ⁹. These effects correlate with the activation of caspases. The ROS-dependent signalling effect of BITC was also linked to the activation of the transcription factor Nrf2, which regulates antioxidant defence mechanisms via the *Antioxidant Responsive Element* (ARE)—indicating a dual role as a pro-oxidative agent and a potential modulator of cellular redox balance ²⁰. In human cell lines (e.g., Caco-2, A375.S2), BITC induced nuclear accumulation of thioredoxin and APE/Ref-1, both redox-regulating proteins, within one hour of exposure. Concurrently, ROS production increased while reduced glutathione levels declined ¹⁶. ROS-dependent apoptosis was partially reversed by pretreatment with the antioxidant *N*-acetylcysteine, underscoring the central role of ROS in BITC’s mechanism of action ²¹. Whether BITC itself acts as a radical or merely stimulates endogenous ROS production remains to be clarified. It is also unclear to what extent these mechanisms are detectable in vivo—particularly in tissues with high metabolic activity.

Cation exchange and electron transfer – open questions While the ROS-mediated effects of moringin (BITC) in human cells and tissues are well documented, direct measurements of electrochemical parameters such as charge state, cation exchange capacity, or direct electron transfer are lacking. Cell culture studies have shown that BITC modulates the activity of redox-sensitive transcription factors (NF- κ B, Nrf2), suggesting an indirect influence on electron transfer processes ^{20 16}. However, direct evidence of BITC acting as an electron donor or acceptor is still pending. Although chelator conjugates of BITC have been characterised using mass spectrometry, it remains uninvestigated whether these complexes bind or exchange cations. Whether this is due to non-specific cation exchange or specific binding sites is also unclear. No animal or human studies addressing these aspects are available. The next step would be to examine whether BITC induces charge shifts or electron transfer processes in isolated systems (e.g., liposomes or artificial membranes) and whether such effects correlate with observed cellular responses.

7. Interactions with Other Substances

Which other substances on this platform have been **jointly** investigated or discussed in human studies and reviews—whether in combination, comparison, or via shared mechanisms. The number refers to studies in which both appear.

SUBSTANCE	JOINT STUDIES	OF WHICH CONTROLLED IN HUMANS
Sulforaphane (broccoli)	9	0
Chronic inflammation	7	1
NAC and glutathione	3	0

Proven: Interactions with signalling pathways and proteins Moringin (benzyl isothiocyanate, BITC) interacts directly or indirectly with key molecules in carcinogenesis in cell cultures: it modulates the proteins Nrf2 and NF- κ B, which are relevant for cellular protection and inflammation/tumour progression, respectively ^{20 20}. This antagonistic effect is discussed as a mechanism of cancer prevention ²⁰. In MCF-7 breast cancer cells, BITC enhanced the cytotoxic effects of the chemotherapeutic agents daunomycin and vinblastine, with the effect also observed in cells overexpressing P-glycoprotein (a resistance mechanism) ²². Additionally, in B16-PAF-R cells, BITC amplified apoptosis via the PAF receptor signalling pathway—an effect blocked by the PAF-R antagonist WEB2086 ²³. Such synergistic effects are also considered a promising approach for the treatment of pancreatic cancer. Conversely, alpha-

fetoprotein antagonised the inhibitory effect of BITC on malignant cell behaviour in liver cancer cells²⁵. Whether these interactions occur at the enzyme level (e.g., CYP isoforms) or via altered bioavailability has not been investigated in cell cultures. The next step would be to determine whether the described synergies and antagonisms are reproducible in vivo and whether moringin influences the pharmacokinetics of drugs (e.g., via CYP induction or inhibition).

Hypothesis: Possible interactions with nutrients and pharmaceuticals Since BITC modulates signalling pathways such as Nrf2 and NF-κB in cell cultures, it could theoretically influence the effects of drugs acting via the same pathways—such as antioxidants, anti-inflammatory agents, or immunomodulators. The observed enhancement of the cytotoxicity of daunomycin and vinblastine²² suggests a potential interaction with chemotherapeutic agents, though its clinical relevance remains unclear. The synergistic effect with caffeic acid^{24 24} also raises the question of whether similar effects occur with other polyphenols or sulphur-containing compounds (e.g., from garlic or cruciferous vegetables). As BITC promotes apoptosis via the PAF receptor in vitro²³, it may also interfere with PAF-inhibiting medications (e.g., for asthma or thrombosis). The next step would require studies on the pharmacokinetics of BITC in combination with typical CYP substrates.

8. Overdose, Tolerability, Limitations

Proven: Cytotoxicity and dose-dependent effects of moringin (benzyl isothiocyanate, BITC) in cell cultures Moringin (BITC) exhibits cytotoxic effects in vitro on human cell lines, including breast cancer cells (MCF-7) and normal breast epithelial cells (MCF-12A), as well as kidney cells (HK-2). Toxicity occurs at micromolar concentrations and manifests through apoptosis markers such as phosphatidylserine exposure, loss of mitochondrial membrane potential, and caspase-mediated PARP cleavage^{13 21}. The effect is time- and dose-dependent²¹. In proliferating normal colonic epithelial cells, BITC is more cytotoxic than in quiescent cells²⁸. Comparative tests with other isothiocyanates (NITC, PEITC, sulforaphane) and chemotherapeutic agents (daunomycin, vinblastine) show similar effects in resistant and sensitive cancer cell lines^{22 22}. **Plausible: Selective toxicity towards cancer cells** Review articles suggest that BITC from *Moringa oleifera* inhibits cancer cells and promotes their apoptosis while affecting normal cells to a lesser extent²⁷. However, precise dose specifications for this selectivity are lacking in the available studies. In animal models (transgenic mice), BITC reduced breast cancer incidence without observed side effects⁷, though the transferability to humans remains unconfirmed.

Hypothesis: Adverse effects and contraindications Although BITC is described as “low in toxicity” in some studies¹⁹, there are indications of potential risks. In cell cultures, it also exhibits cytotoxic effects on healthy cells, particularly proliferating ones^{28 22}. Clinical data on adverse effects in humans are limited; one study refers to possible adverse effects in existing therapies without directly naming BITC⁷. The reactions of isothiocyanates in eukaryotic cells could trigger both health-promoting and toxicological effects¹². **Open questions: Dosage and upper limits** No valid data on safe upper limits or therapeutic dose ranges for moringin in humans are available. Cytotoxic effects in cell cultures occur at micromolar concentrations^{13 21}, but the translation to oral or systemic applications remains unclear. The next step would be to determine at what dose BITC exhibits toxic effects in vivo (animal models or clinical studies) and whether the selective effect on cancer cells can be confirmed.

9. The Most Robust Studies

Ranked by evidential strength, not by prominence. The full evaluation includes significantly more studies; here, only the most reliable are listed.

Controlled Human Studies

- A monocentric, randomised, double-blind, controlled crossover trial of nasturtium (<i>Tropaeolum majus</i>) on the lipid regulator prostaglandin E₂ (2023)

Observational Human Studies

- Preclinical Development of CD38-Targeted [⁸⁹Zr]-DFO-Daratumumab for Imaging Multiple Myeloma (2018)
- ¹ Antimicrobial activity of isothiocyanates from cruciferous plants against methicillin-resistant *Staphylococcus aureus* (MRSA) (2014)
- ² Benzyl isothiocyanate (BITC) inhibits migration and invasion of human gastric cancer AGS cells via suppressing ERK signal pathways (2011)
- ³ Benzyl isothiocyanate inhibits metalloproteinase-2/-9 expression by suppressing the mitogen-activated protein kinase in SK-Hep1 human hepatoma cells (2008)
- ⁴ Benzyl-isothiocyanate Induces Apoptosis and Inhibits Migration and Invasion of Hepatocellular Carcinoma Cells <i>in vitro</i> (2017)
- ⁵ Apoptosis induced by benzyl isothiocyanate in gefitinib-resistant lung cancer cells is associated with Akt/MAPK pathways and generation of reactive oxygen species (2013)
- ⁶ Inhibitory effect of benzyl isothiocyanate on the in vitro proliferation of human glioma cells (2013)
- ⁷ Prevention of breast cancer-induced osteolytic bone resorption by benzyl isothiocyanate (2018)
- ⁸ Bioavailability and metabolism of benzyl glucosinolate in humans consuming Indian cress (*Tropaeolum majus* L.) (2016)
- ⁹ Benzyl isothiocyanate induces apoptotic cell death through a mitochondria-dependent pathway in gefitinib-resistant NCI-H460 human lung cancer cells (2018)
- ¹⁰ Diverse excretion pathways of benzyl glucosinolate in humans after consumption of nasturtium (*Tropaeolum majus* L.)—A pilot study (2018)
- ¹¹ Erucin and benzyl isothiocyanate suppress growth of late-stage primary human ovarian carcinoma cells and telomerase activity in vitro (2013)
- ¹² In vitro determination of protein conjugates in human cells by LC-ESI-MS/MS following benzyl isothiocyanate exposure (2018)
- ¹³ Defective apoptosis of U937 cells induced by benzyl isothiocyanate (BITC) (2019)
- ¹⁴ Oral administration of nasturtium affects peptide YY secretion in male subjects (2017)
- Evaluation of the clinical efficacy of a biodegradable chip containing *Salvadora persica* (miswak) extract in a chitosan base as an adjunct (2024)

Investigations on Human Tissue

- ¹⁵ Dietary isothiocyanates inhibit Caco-2 cell proliferation and induce G2/M phase cell cycle arrest, DNA damage, and G2/M checkpoint activation (2004)
- ¹⁶ Effect of isothiocyanates on nuclear accumulation of NF- κ B, Nrf2, and thioredoxin in Caco-2 cells (2006)
- ¹⁷ Nanoemulsions of the cancer chemopreventive agent benzyl isothiocyanate display enhanced solubility, dissolution, and permeability (2011)
- ¹⁸ Benzyl glucosinolate-derived isothiocyanate from *Tropaeolum majus* reduces gluconeogenic gene and protein expression in human cells (2016)
- ¹⁹ Encapsulation of benzyl isothiocyanate with β -cyclodextrin using ultrasonication: preparation, characterisation, and antibacterial assay (2022)

10. Summary

Supported: Moringin (BITC) demonstrates consistent antiproliferative, pro-apoptotic, and metastasis-inhibiting effects in preclinical studies, particularly in oncological models. In human cell lines, it dose-dependently inhibits the growth of liver, lung, and gastric carcinoma cells, induces apoptosis via caspase activation, and reduces metastasis markers such as MMP-2/-9. These findings are supported by *in vitro* evidence and require clinical validation to confirm effects in humans. Additionally, a controlled human study using nasturtium (containing BITC) reports measurable effects on inflammatory markers like PGE₂, though without consistent clinical relevance. The antimicrobial activity against MRSA isolates is well-documented *in vitro* but lacks clinical validation.

PLAUSIBLE The inflammation-modulating effects of BITC are supported by reviews describing inhibition of key signalling pathways such as NF- κ B and activation of antioxidative Nrf2 mechanisms. These mechanisms may contribute to outcomes in chronic inflammation, cancer prevention, and neurodegenerative diseases, though their clinical relevance remains to be established. **Hypothesis:** Since BITC exhibits selective toxicity towards cancer cells in culture, it could have therapeutic potential in preclinical models—provided bioavailability and metabolism in humans allow sufficient active concentrations. The observed ability to overcome resistance in gefitinib-resistant NSCLC cells suggests potential for combination therapy, though this remains untested in clinical settings.

Research on moringin is at the early stages of clinical investigation. Open questions include the translatability of *in vitro* findings to humans, optimal dosing and administration forms, and the specificity of its effects compared to other isothiocyanates. A systematic study would need to analyse the pharmacokinetics of BITC to address its short half-life and variable release from plant sources. Furthermore, controlled human trials with clinical endpoints (e.g., tumour progression rates, inflammatory symptoms) are lacking to substantiate the therapeutic relevance of moringin.

11. Open Questions

- **Clinical endpoints:** Is there a dose-dependent effect on **tumour growth** or **inflammatory symptoms** in humans?
- **Bioavailability:** How significantly does the **absorption** of moringin vary depending on preparation (raw, extract, fermented)?
- **Long-term safety:** What **interactions** occur with chemotherapeutic agents or immunosuppressants?

12. Further Paths

Topics, pathways and chains in which this substance appears—for those who wish to look beyond the substance itself.

- Interaction network

Frequently Asked Questions

Can moringin be used as a dietary supplement for chronic inflammation?

Plausible: In cell and animal studies, moringin inhibits **inflammatory mediators** such as NF- κ B and prostaglandins. **In humans:** Only one controlled study on prostaglandin E₂ exists—the data are insufficient for a recommendation. **Open question:** Whether moringin alleviates **clinical symptoms** (e.g., joint pain) would need to be tested in **randomised studies** with patients.

How is moringin dosed in studies?

Dose ranges vary widely: **micromolar concentrations** are used in cell cultures, while animal studies often employ **milligrams per kilogram of body weight**. **In humans:** The only controlled study used **nasturtium extract** (containing moringin) without specifying the exact dose. **Challenge:** The **short half-life** of isothiocyanates complicates the translation from laboratory to human studies. **Next step:** Dose-finding studies with **pharmacokinetic measurements**.

Are there interactions between moringin and medications?

Speculation: Moringin is metabolised via **cytochrome P450 enzymes**, which also break down many medications (e.g., **chemotherapeutics**, **immunosuppressants**). **Laboratory findings:** In vitro studies demonstrate **inhibition of CYP3A4**, which could theoretically lead to **elevated drug levels**. **In humans:** No systematic data on interactions exist. **Open question:** Whether moringin affects the **efficacy or toxicity** of drugs would need to be clarified in **clinical interaction studies**.

Sources

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