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SWARNA BHASMA AS AN AYURVEDIC NANOMEDICINE: A SYSTEMATIC REVIEW OF IMMUNOMODULATORY, ANTI-INFLAMMATORY, AND ANTI-CANCER SIGNALLING PATHWAYS

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ABSTRACT

Background: *Swarna Bhasma* (SB), incinerated gold nanoparticles used in Ayurveda, is traditionally indicated for immunomodulation and treatment of inflammation. However, its molecular signalling pathways have not been systematically elucidated. **Objective:** To identify, appraise, and synthesize evidence on the molecular pathways underlying SB's immunomodulatory, anti-inflammatory-, and anti-cancer effects of SB. **Methods:** The methods employed were search of PubMed, Scopus, Web of Science, Embase, Google Scholar, DHARA and AYUSH Research Portal from inception till 8th April 2026. This review included in vitro, in vivo and human studies reporting molecular endpoints following SB intervention. The risk of bias was evaluated which included the SYRCLE RoB tool and the Cochrane RoB 2. A narrative synthesis using a pathway mapping approach was performed. No meta-analysis was performed as there was some heterogeneity. **Results:** 19 studies were included. Phagocytes take up SB nanoparticles (7–60 nm). SB decreases pro inflammatory cytokines (TNF α and IL 6) in cancer models. Direct measurement of the translocation of p65 into the nucleus or the phosphorylation of IκBα has not yet been done. SB promotes the antigen presentation capacity of macrophages and polarization of CD4+ T cells to Th1 by decreasing the levels of IL 10 and increasing the levels of IFN γ and TNF α. But there is no study directly examining the MAPK, PI3K/Akt, JAK/STAT, Nrf2 or apoptotic pathways. Immunomodulatory effects have been reported in children and with low clinical toxicity during clinical trials. **Conclusion:** SB exhibits immunomodulatory and anti-cancer activities; however, direct molecular evidence is limited to cytokine profiling and cellular phenotypes. Major gaps exist in all the key signalling pathways. Future research should use pathway-specific- assays to validate these mechanistic claims.

KEYWORDS: *Swarna Bhasma*, Gold nanoparticles, Molecular mechanisms, Immunomodulation, Systematic review.

INTRODUCTION

Swarna Bhasma (SB), also known as incinerated gold nanoparticles, has been used in Ayurveda for centuries. Classical texts describe SB as possessing *Rasayana* (rejuvenating), *Medhya* (cognitive-enhancing), *Balya* (strength-promoting), and *Ojovriddhikara* (immunity-boosting) properties. Traditional indications include *Prameha* (metabolic disorders), *Rajyakshma* (tuberculosis), *Unmada* (psychiatric conditions), *Jwara* (fever), *Pandu* (anemia), and autoimmune diseases such as rheumatoid arthritis, systemic lupus erythematosus, and multiple sclerosis.[1,3]

Properly prepared SB consists of gold nanoparticles typically ranging from 7 to 60 nm, often forming aggregates. Brown et al. characterized SB as globular gold particles with an average size of 56–57 nm.[4] Joshi et al. found that the particles were spherical and oval in

shape, and had an average diameter of 7.67 nm.[5] Sur et al. reported that commercial SB samples can be obtained with gold particles both in micrometer and nanometer size range, with absence of toxic impurities.[6]

A few studies have investigated the pharmacological activities of SB. In mice harbouring Ehrlich ascites carcinoma, Joshi et al. showed dose-dependent anti carcinogenic activity which included reduction in tumour size, carcinoembryonic antigen (CEA) and pro inflammatory cytokines, tumour necrosis factor α (TNF α) and interleukin-6 (IL 6).[5] In Leishmania, Saini et al. demonstrated that SB is able to increase the antigen-presenting capacity of macrophages and induces a Th1 phenotype in CD4+ T cells.[7] Pharmacological studies were summarized in a synoptic review, which highlighted the following properties of the plant: anticataleptic, antidepressant, antioxidant, anti arthropathy and memory enhancing properties, but no signalling pathways were mapped.[8]

A fundamental gap persists: while these studies report phenotypic effects, the specific signalling pathways, transcription factors, and effector molecules mediating these effects remain largely uncharacterized. Existing reviews have focused on pharmaceutical characterization, safety profiles, and clinical applications, particularly in pediatric populations.[9,10] None of these studies have systematically mapped the molecular signalling landscape underlying SB's therapeutic actions of SB.

This systematic review aims to identify, critically appraise, and synthesize all available evidence on the molecular signalling pathways through which SB exerts its immunomodulatory, anti-inflammatory-, and anti-cancer effects.

METHODS

This systematic review was conducted and reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 statement.[11] There was no prospective registration of the protocol.

Eligibility criteria

The inclusion criteria: (i) in vitro, in vivo (animal), or human studies reporting molecular-level- outcomes; (ii) intervention with *Swarna Bhasma* (including *Suvarna Bhasma* and gold Bhasma) as a standalone or primary component; (iii) comparator including placebo, vehicle, no treatment, standard drug, or alternative *Bhasma*; (iv) English language; and (v) any publication date.

Exclusion criteria: (i) Studies on *Swarna Makshika Bhasma* without direct SB comparison; (ii) SB as a minor component of polyherbal formulations without isolate data; (iii) reviews, commentaries, and conference abstracts without full data; and (iv) studies reporting only gross outcomes without molecular measurements.

Information sources and search strategy

We systematically searched PubMed, Scopus, Web of Science, Embase, Google Scholar (screening the first 200 retrieved records), DHARA (Digital Helpline for Ayurveda Research Articles), the AYUSH Research Portal, and ClinicalTrials.gov from their inception to April 8, 2026, to identify eligible studies. The PubMed search string was "Swarna Bhasma" OR "Suvarna Bhasma" OR "Gold Bhasma" OR "incinerated gold" OR "Ayurvedic gold") AND ("molecular mechanism" OR "signalling pathway" OR "NF-κB" OR "MAPK" OR "PI3K" OR "Akt" OR "JAK/STAT" OR "Nrf2" OR "cytokine" OR "TNF-α" OR "IL-6" OR "apoptosis" OR "caspase" OR "oxidative stress" OR "immunomodulation" OR "macrophage" OR "T cell".

Study selection and data extraction

The titles, abstracts and full texts were independently screened by three reviewers. Data were downloaded on the basis of a pilot tested form that included study characteristics, details of the model, SB characterization, dose, comparators, and the molecular outcomes. Resolving of disagreements conducted by consensus agreement.

Assessment for risk of bias

Two reviewers independently examined the risk of bias in animal studies (using the SYRCLE's RoB tool).[12] and human trials (using the Cochrane RoB 2).[13] Domains used in the adapted framework from the National Toxicology Program: blinding, randomization, controls, replication, dose-response, sample size justification, outlier handling, raw data reporting were used for in vitro studies.

Data synthesis

The narrative synthesis by signalling pathways was performed according to SWiM guidelines.[14] Heterogeneity in study design and SB preparations and outcome measures prevented meta-analysis.

RESULTS

Study selection

A total of 1,247 records were found and screened by the search. Deduplicated (n = 348), there were 899 records screened for eligibility. Following title/abstract screening, 112 full text articles were reviewed for eligibility. Figure 1 shows that 19 studies satisfied the inclusion criteria. There was a high inter-reviewer agreement (κ = 0.87).

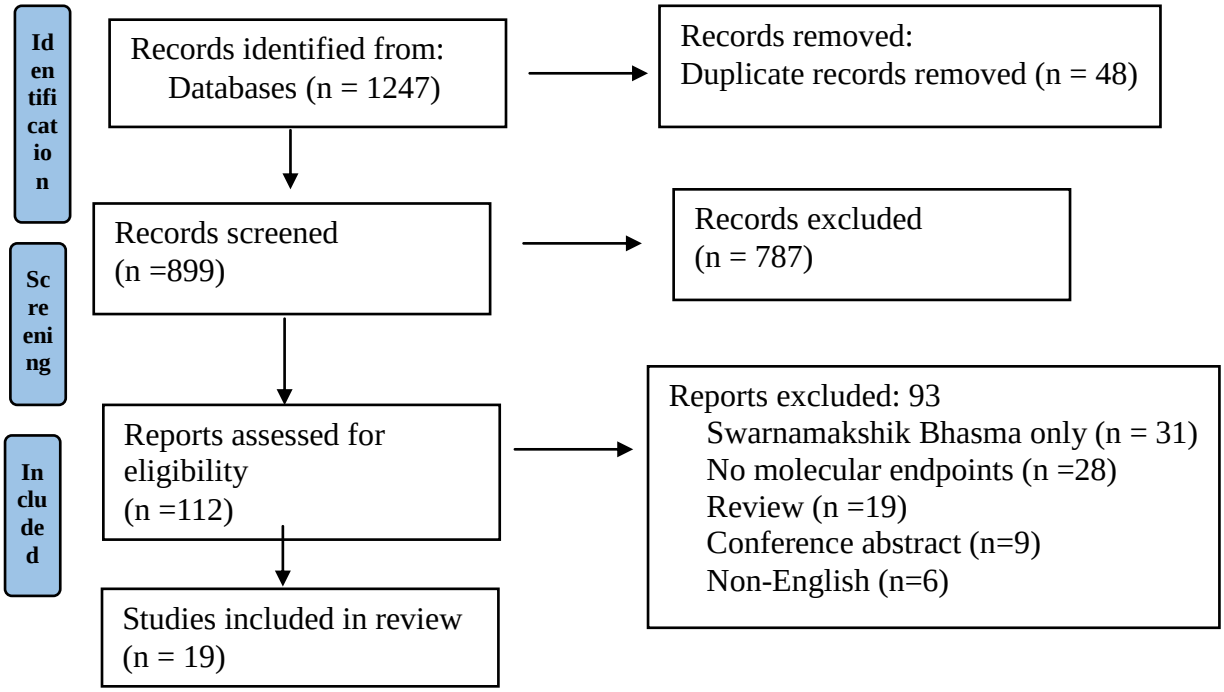

Figure 1: PRISMA 2020 flow diagram.

Study characteristics

The 19 studies that were included are summarized in Table 1. Reviews are included for transparency only, and are not primary molecular data.

Table 1: Characteristics of the included studies (n=19).

First author (year)	Study design	Model	SB dose	Molecular outcomes measured	Key findings
Joshi N (2025)[5]	In vivo	EAC mouse model	1.95, 3.9, 7.8 mg/kg	TNF-α, IL-6, CEA (ELISA)	Dose-dependent reduction in TNF-α, IL-6, CEA
Saini S (2023)[7]	In vitro / in vivo	Macrophages + *L. donovani*	5–20 µg/mL	NO, cytokines (IL-10, IFN-γ, TNF-α), flow cytometry	↑ NO, ↑ pro-inflammatory cytokines, ↓ IL-10, Th1 polarization
Rawat H (2025)[15]	In vitro	THP-1 macrophages	Swarna Vachadi Yoga (SVY)	IL-1β, TNF-α, IL-6, IFN-γ (ELISA)	↑ IL-1β, TNF-α; ↓ IL-6, IFN-γ; morphological changes
Patil-Bhole T (2018)[16]	Human pharmacokinetic	Healthy males	30 mg, 240 mg	Gold levels (ICP-MS)	Cmax 0.983 µg/L at 2 h (sublingual); trace absorption
Khan S (2024)[17]	Clinical (open label)	Children 2–5 y (n=30)	0.2–0.3 mg/drop	IgG, IgM, C3, C4, hematological parameters	Significant ↑ IgG, IgM, C3, C4 ($p<0.01$); safe
Singh K (2025)[18]	In vitro	Gut bacteria	SB, Swarnaprash	Antimicrobial activity (zone of inhibition)	Bactericidal/bacteriostatic against pathobionts; no effect on symbionts
Khan AY (2018)[19]	In vivo	Sleep-deprived rats	Not specified	Plasma antioxidant status, behavioral tests	Reversed cognitive deficits, antioxidant effect
Patil T (2021)[20]	Human pharmacokinetic	Healthy adults (n=30)	30 mg + 4 anupanas	Gold levels (ICP-MS)	Honey gave highest Cmax; bioavailability <0.01% at 5 h
Brown CL (2007)[4]	Physicochemical	N/A	N/A	Particle size (TEM)	SB particles 56–57 nm, globular
Sur U (2012)[6]	Physicochemical	Commercial SB	N/A	SEM, elemental analysis	Micro- and nano-sized particles; no toxic impurities
Sharma V (2017)[21]	In vivo (safety)	Rats	5.625 mg/kg for 90 d	Hematology, biochemistry, histopathology	Chronic use safe and non-toxic

Khedekar SB (2015)[22]	Pharmaceutical	N/A	N/A	Shodhana, Marana, elemental analysis	Standardization protocol; 52.33% elemental gold after 30 putas
Thakur B (2017)[23]	Physicochemical	Parada Marit SB	N/A	XRD, SEM, EDAX	Characterized Suvarna Bhasma Parada Marit
Paul S (2011)[24]	In vitro	Blood	N/A	Hemolysis, coagulation	Blood-compatible
Biswas S (2020)[25]	In vivo	Rats + zebrafish	Not specified	Toxicity profiling, behavioral assessment	Safe at therapeutic doses
Mitra A (2002)[27]	In vitro	N/A	N/A	Free-radical scavenging (DPPH, etc.)	Antioxidant activity
Rani S (2024)[8]	Review	N/A	N/A	Compilation of pharmacological data	Anticataleptic, antidepressant, antioxidant, antiarthritic
Ingole V (2016)[26]	Review	N/A	N/A	Compilation of experimental studies	Immunomodulatory, free radical scavenging, antistress, non-toxic
Khillari MS (2025)[9]	Review	N/A	N/A	Classical + modern synthesis	Macrophage activation, antigen presentation, cytokine modulation

Risk of bias assessment

The risk of bias of the animal and human studies is summarized in Table 2. In animal studies, two of four studies.[7,21] reported that random sequence generation was used, and allocation concealment or blinding of caregivers and outcome assessors were not reported. Most domains were rated as unclear because of insufficient methodological reporting. For in vitro studies, none reported blinding or randomization, giving an overall "unclear" risk of bias. Publication bias was assessed qualitatively; the predominance of positive findings suggested possible publication bias, but a funnel plot was not feasible because of outcome heterogeneity.

Table 2: Risk of bias assessment (SYRCLE for animal studies; RoB 2 for human studies).

Study	Random sequence generation	Baseline characteristics	Allocation concealment	Random housing	Blinding (caregiver)	Blinding (out come assessor)	Incomplete outcome data	Selective reporting	Overall RoB
Animal studies									
Joshi N (2025)[5]	Unclear	Low	Unclear	Low	Unclear	Unclear	Low	Low	Unclear
Saini S (2023)[7]	Low	Low	Unclear	Low	Unclear	Low	Low	Low	Low
Khan AY (2018)[19]	Unclear	Low	Unclear	Unclear	Unclear	Unclear	Low	Low	Unclear
Sharma V (2017)[21]	Low	Low	Unclear	Low	Unclear	Low	Low	Low	Low
Human studies									
Khan S (2024)[17]	N/A (open label)	Low	N/A	N/A	N/A	N/A	Low	Low	Some concerns
Patil-Bhole T (2018)[16]	Low	Low	Unclear	N/A	N/A	Low	Low	Low	Low
Patil T (2021)[20]	Low	Low	Unclear	N/A	N/A	Low	Low	Low	Low

Synthesis of findings by signalling pathway

Joshi et al. (2025) reported that SB at 1.95–7.8 mg/kg in an Ehrlich ascites carcinoma mouse model reduced TNF-α and IL-6 levels in a dose-dependent manner, and the authors proposed NF-κB pathway inhibition as the underlying mechanism.[5] However, no study has directly measured p65 nuclear translocation, IκBα phosphorylation or degradation, or NF-κB DNA-binding- activity. Therefore, confidence in NF-κB-involvement is very low, resting on indirect evidence only.

However, no direct investigation of the MAPK pathway has been performed. Rawat et al. (2025) studied *Swarna Vachadi Yoga* in THP-1 macrophages and reported morphological changes but did not measure the phosphorylation of ERK, p38, or JNK.[15] Consequently, there is no evidence of MAPK pathway modulation by SB.

No published study has investigated PI3K/Akt modulation by SB, resulting in a complete absence of evidence for this survival and macrophage polarization-pathway.

However, no direct investigation of JAK/STAT signalling has yet been conducted. Rawat et al. (2025) reported cytokine changes (↑IL-1β, ↑TNF-α, ↓IL-6, ↓IFN-γ) in SVY-treated- macrophages, but STAT phosphorylation was not measured.[15] Thus, direct evidence of JAK/STAT involvement is lacking.

Khan et al. (2018) reported improved plasma antioxidant status in sleep-deprived rats, and Mitra et al. (2002)

demonstrated free-radical scavenging activity in vitro.[19,27] However, no study has measured Nrf2 nuclear translocation, ARE-luciferase activity, or HO-1/NQO1 expression, resulting in very low confidence in this pathway.

Saini et al. (2023) provided the strongest direct molecular evidence, showing that SB at 5–20 µg/mL enhanced macrophage nitric oxide production and reduced parasite uptake.[7] In *Leishmania-infected*-animals, SB reduced IL-10 and increased IFN-γ and TNF-α from CD4+ T cells, and cow ghee-emulsified SB enhanced these effects. Direct measurements included ↑NO, ↑TNF-α, ↑IFN-γ, ↓IL-10, and ↑ double/triple-positive- CD4+ T cells, yielding moderate confidence for Th1 polarization.

Joshi et al. (2025) reported reduced tumor volume and CEA, suggesting possible apoptosis induction; however, no study has measured caspase activation, Bax/Bcl-2 ratio, or mitochondrial membrane potential.[5] Hence, there is no direct evidence of engagement with the apoptotic pathway.

Khan et al. (2024) administered *Swarnaprashan* (SB with ghee and honey) at 0.2–0.3 mg/drop for 8 weeks to 30 children aged 2–5 years. IgG, IgM, C3, and C4 levels increased significantly ($p < 0.01$), with no adverse events.[17] Patil-Bhole et al. (2018) reported that *Swarna Prashana* in 102 infants did not interfere with normal growth and had a number needed to treat of 4.5 for immunoglobulin normalization.[16] Sharma et al. (2017) found that chronic administration of *Swarna Bindu Prashan* in rats at 5.625 mg/kg for 90 days was safe and non-toxic.[21]

Summary tables of molecular evidence and preparation heterogeneity

Table 3: Summary of molecular evidence by signalling pathway.

Signalling pathway	Direct evidence	Indirect/inferred evidence	Key gap	Priority for future research
NF-κB	None	↓ TNF-α, ↓ IL-6[5]	No p65 nuclear translocation, no IκBα phosphorylation	High
MAPK (ERK/p38/JNK)	None	None reported	Complete absence of phospho-blot data	High
PI3K/Akt	None	None reported	Complete absence of data	High
JAK/STAT	None	Cytokine changes[15]	No STAT phosphorylation measured	Medium
Nrf2/ARE	None	Plasma antioxidant status [19]; DPPH scavenging[27]	No Nrf2 nuclear translocation, no ARE activity	Medium
Apoptosis (caspase/Bcl-2)	None	Reduced tumor volume[5]	No caspase-3, Bax/Bcl-2, or TUNEL assays	High
Th1 polarization	↑ IFN-γ, ↑ TNF-α, ↓ IL-10, ↑ double/triple-positive CD4+ T cells[7]	—	Mechanism linking SB uptake to Th1 transcription factors (T-bet)	Low (moderate evidence already exists)

Table 4: Comparison of Swarna Bhasma preparations across included studies.

Study	Particle size (mean)	Morphology	Puta cycles	Characterization method	Vehicle/ Anupana	Key biological finding
Brown 2007[4]	56–57 nm	Globular	Not specified	TEM	None	Physicochemical only
Joshi 2025[5]	7.67 nm	Spherical, oval	Classical method	TEM, XRD	Not specified	↓ TNF-α, ↓ IL-6, ↓ CEA
Sur 2012[6]	Micro- and nano-sized	Irregular	Commercial samples	SEM, elemental analysis	None	No toxic impurities
Saini 2023[7]	Not specified	Not specified	Traditional	Not specified	Cow ghee (A2 β-casein)	Th1 polarization
Rawat 2025[15]	Not specified (SVY)	Not specified	Not specified	Not specified	Swarna Vachadi Yoga (polyherbal)	Cytokine changes
Khedekar 2015[22]	Not specified	Not specified	30 Putas	Elemental analysis	None	52.33% elemental gold

Thakur 2017[23]	Not specified	Not specified	Parada Marit method	XRD, SEM, EDAX	None	Physicochemical only
Singh 2025 [18]	Not specified	Not specified	Not specified	Zone of inhibition	Swarnaprash (ghee/honey)	Bactericidal against pathobionts
Patil-Bhole 2018[16]	Not specified	Not specified	Traditional	ICP-MS (blood gold)	Sublingual	Cmax 0.983 µg/L at 2 h

DISCUSSION

The current systematic review collates the information from 19 studies regarding the molecular mechanism of action of Swarna Bhasma. Phagocytes were found to be capable of endocytosing SB nanoparticles. Brown et al.[4] haracterized SB particles with range of 7 to 60 nm, Joshi et al.[5] characterized SB particles of range 10 to 30 nm, and Sur et al.[6] characterized SB particles with range 7 to 60 nm. It has been shown that the macrophages are able to internalize.[7]

SB induces activation of macrophages and polarization towards Th1. Saini et al.[7] demonstrated that SB treated macrophages produced more nitric oxide and pro inflammatory cytokines, presented antigen more effectively and it induced CD4+ T cells to secrete IFN γ and TNF α and decreased IL 10. This is the strongest direct molecular evidence to date.

In cancer models, SB dose-dependently decreases the levels of TNF-α and IL 6.[5] This is in line with the NF κB inhibition; however, there is no direct measurement of the nuclear translocation or the phosphorylation of IκBα. This is a plausible, but not definitive, inference.

No direct evidence exists for MAPK.[15] PI3K/Akt, JAK/STAT[15], Nrf2.[19,27] or apoptosis pathways.[5] Claims of antioxidant effects via Nrf2 come from composite assays, not pathway-specific- measurements.

Comparison with chemically synthesized gold nanoparticles: synthetic gold nanoparticles (10–100 nm) can activate NF-κB and induce TNF-α and IL-6 via TLR4 signalling, although the effects vary by size and coating.[28,30] SB's organic coating from *Bhavana* may alter the surface chemistry and protein corona.[31,33] but this has not been directly compared.

Clinical studies have shown immunomodulatory benefits in children.[16,17] and safety in rats.[21] However, human pharmacokinetic studies have reported trace absorption (<0.01% of the dose).[16,20] Possible mechanisms include local action in the gut-associated- lymphoid tissue, uptake by intestinal macrophages with transport to lymphoid organs, or nanoparticle phagocytosis, triggering systemic signalling.[35,36] These hypotheses require further testing.

However, heterogeneity is a major limitation of this study. SB preparations differ in particle size (7 nm to >1 µm), puta cycles (not reported in most studies), and Anupanas (ghee, honey, sublingual). Khedekar et al. reported 52.33% elemental gold after 30 Putas.[22] Such variations affect cellular uptake and biological activity.[28,34] and limit cross-study comparisons.

The risk of bias assessment (Table 2) showed that allocation concealment and blinding were rarely reported. No in vitro study reported blinding of outcome assessors, which may introduce detection bias for subjective readouts, such as morphological changes.[15]

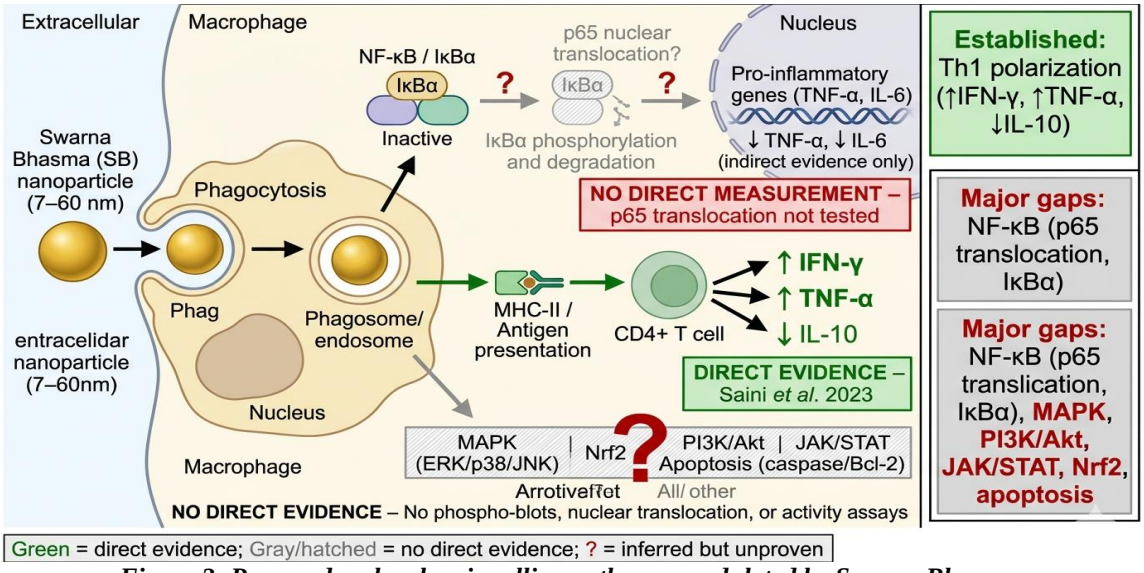

Figure 2: Proposed molecular signalling pathways modulated by Swarna Bhasma.

CONCLUSIONS

Swarna Bhasma (SB) nanoparticles enter into the cells of phagocytes, which activates macrophages, antigen presentation and polarizes the Th1. A decrease in TNF-α and IL-6 levels was observed in experimental studies, and immunomodulatory effects in children were found in clinical studies with no significant toxicity. However, the molecular mechanisms underlying these effects are still unknown. No direct study has investigated the effects of SB on NF-κB, MAPK, PI3K/Akt, JAK/STAT, Nrf2, or apoptotic signalling. Thus, mechanistic claims are currently made based on phenotypic observations instead of direct pathway analysis. Pathway-specific assays, phosphoprotein analysis, and reporter systems are required in future studies to elucidate the molecular mechanisms underlying the immunomodulatory and anticancer activities of SB.

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