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Review ETM

Anticancer Potential and Other Therapeutic Activities of Curcumin & Its Derivatives

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Abstract

Curcumin, a compound isolated from turmeric, is used in the management of various diseases due to its extensive medicinal properties. Curcumin, the principal polyphenol isolated from Curcuma longa (turmeric), has garnered immense scientific interest for its extensive medicinal properties, including potent anti-inflammatory, antioxidant, and anticancer activities demonstrated in preclinical models. An expanding body of research indicates broad therapeutic promise in managing diverse pathologies, from various cancers to chronic inflammatory, metabolic, and dermatological conditions. However, a significant paradox limits its clinical translation: this vast in vitro potential is almost completely nullified by catastrophic pharmacokinetic failures. This review deconstructs this "curcumin paradox." An expanding body of research indicates that curcumin may hold promise as a therapeutic agent in the management of various dermatological conditions. It possesses anticancer activity by suppressing proliferation and metastasis and by promoting cell cycle arrest or apoptosis in various cancer cells. Despite all these benefits, the therapeutic application of curcumin in clinical medicine and its bioavailability are still impaired by its poor absorption and rapid metabolism. The combination of curcumin with novel delivery systems represents a highly promising area for future applied research.

Keywords: Anticancer, anti-arthritis, antioxidants, COVID-19, psoriasis, wound care

INTRODUCTION

Curcumin is the active ingredient in the golden spice turmeric (*Curcuma longa*) [1]. Although the multicomponent nature of "curcumin" is well documented, that fact and/or the unambiguous assignment of specific structures in a particular preparation process is often unclear. The term "curcumin" will be used interchangeably with "curcuminoids" throughout this discussion. Because of its dynamic and unstable nature, curcumin is challenging to treat as a single, well-defined compound in either in vitro or in vivo settings. Nonetheless, in most studies – regardless of the exact source material, curcumin is typically referenced as the active constituent by default. It is often highlighted as the key compound of interest for therapeutic applications and serves as a lead structure in medicinal chemistry research (Table 1). Despite demonstrated efficacy in many experimental models, low bioavailability due to low absorption, rapid metabolism and elimination may limit the therapeutic effectiveness of curcumin. Curcumin has been investigated for use in the therapy and supportive care of various clinical conditions, including mucositis, rectal cancer, prostate cancer, chronic schizophrenia, and mild cognitive impairment (MCI). Biological activity of drugs, such as curcumin can be achieved by improving drug efficacy shown by clinical studies [2, 3].

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Table 1. Phytochemical composition of curcumin.

Constituent	Composition (w/w)
Curcuminoids	1-6%
Volatile oils	3-7%
Fiber	2-7%
Mineral matter	3-7%
Protein	6-8%
Fat	5-10%
Moisture	6-13%
Carbohydrates	60-70%

Researchers have found that curcumin helps fight cancer because it influences several key biological mechanisms, such as mutagenesis, oncogene expression, tumorigenesis, cell cycle regulation, apoptosis, and metastasis [4]. Therefore, improving the limitations and curcumin's activity as anticancer is extensive. Efforts have been invented to synthesize new curcumin derivatives [5].

ADME (ABSORPTION, DISTRIBUTION, METABOLISM, AND EXCRETION)

Absorption

A crucial factor in any possible treatment is absorption, particularly when it comes to oral dosage. Curcumin in several oral dosage forms has been evaluated in numerous investigations, including clinical trials. With no negative side effects, an oral dosage of up to 12g/day has been administered. Despite the fact that this high dosage was generally well tolerated, the compound's absorption is minimal.

Distribution

A compound's therapeutic value is greatly influenced by how well it spreads throughout the body. Although chemical curcumin's distribution in rats has been well investigated, human distribution has only been sporadically assessed. Variable distribution among tissue types has been documented in several mouse model studies. This significant degree of variation is probably caused by (i) variations in the way the study's dose was prepared, (ii) variations in the extraction, preparation, and detection techniques of curcumin, and (iii) a lack of specificity in the detection assay.

Metabolism

Curcumin has a high potential for metabolism, in part because of its reactive nature when taken by the body. Numerous studies have investigated curcumin within human liver microsomes. Alcohol dehydrogenase is principally responsible for the elimination of the double bonds in the heptadiene dione system during phase I metabolism. Curcumin and its reduced metabolites are quickly conjugated by phase II metabolic pathways (Table 2). At the phenolic sites, glucuronides, and sulfates are the most prevalent conjugates Figure 1. It should come as no surprise that curcumin also easily interacts nonenzymatically with glutathione, most likely via a Michael-type addition. Some of the formulations being studied to increase curcumin's oral bioavailability also aim to reduce the high rate of metabolism that is seen after absorption. However, it seems that curcumin is highly susceptible to alteration by both first and second phase metabolism once it is released in *vivo*.

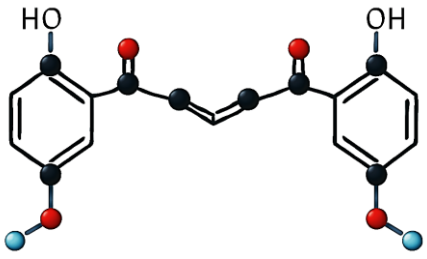

Figure 1. Molecular structure of curcumin.

Table 2. Curcumin's pharmacokinetic profile.

Barrier category	Specific mechanism	Clinical consequence
Physicochemical	Low aqueous solubility (approx. 11 ng/mL)	Negligible dissolution in the aqueous environment of the gastrointestinal (GI) tract.
	Chemical instability at alkaline pH	Pre-absorption degradation of the molecule in the small intestine.
Gastrointestinal	Poor intestinal permeability	Inefficient transport of dissolved curcumin across the intestinal epithelium.
	Intestinal first-pass metabolism	Enzymatic breakdown (e.g., glucuronidation) within enterocytes *during* the absorption process.
Metabolic	Rapid hepatic first-pass metabolism	Intensive Phase I (reduction) and Phase II (glucuronidation, sulfation) conjugation in the liver.
Overall Result	Rapid systemic elimination	High fecal excretion and sub-therapeutic plasma concentrations of *free, active* curcumin.

Excretion

Several investigations in rats explained that most of the curcumin taken orally is eliminated in the feces. Although glucuronide and sulfate metabolites have been investigated in rat plasma, very little is found in rodent urine. Regarding curcumin and its metabolites' excretion in human subjects, there are contradictory results. In one investigation, following oral treatment, curcumin was found in feces but neither the parent substance nor its metabolites were found in the blood or urine of human volunteers. In two more investigations, one or three individuals from each cohort of 12 or 15 patients had serum with curcumin or one of its metabolites, respectively. In a fourth study, curcumin was investigated in the serum of a single participant in a fourth investigation, but the conjugates of sulfate and glucuronide were found in every participant. A portion of this reporting variability, like that of absorption reports, most certainly results from variations in the source material, dosing techniques, and sample collection, preparation, and detection techniques. Curcumin may be absorbed in trace amounts and then eliminated unaltered, or it may not be absorbed at all and instead go straight to the feces if other physicochemical characteristics are taken into account (see above). The glucuronide and sulfate conjugates of metabolized curcumin are typically eliminated in urine. By the time it is eliminated, the remaining portion of any dose – whether absorbed or not – is probably broken down too much to be detected.

Toxicology

Curcumin (and its breakdown products) exhibit extensive reactivity against several human enzymes associated with chemical toxicity, including cytochrome P450s, glutathione S-transferase, and hERG channels, in addition to the therapeutic targets covered below. Each of these classes' sensitivity has significant ramifications for possible harmful side effects: Cardiotoxicity is linked to hERG channel blockage; inhibition of glutathione S-transferase (GST) and cytochrome P450 (CYP450) can result in harmful drug-drug contraindications and poor detoxification. Other than its particular enzyme toxicity, the chemical curcumin has recently been demonstrated to be an active iron chelator in vivo, causing mice fed iron-deficient diets to exhibit overt iron insufficiency. This implies that curcumin may have an impact on systemic iron metabolism, especially in those who already have a deficient iron status. Given its physicochemical characteristics and chemical composition, curcumin's demonstrated ADME effects are not unexpected. A vast area of research has been devoted to improving curcumin's pharmacokinetic characteristics as a outcome of the many studies that indicate it could be useful as a medicinal agent. According to novel research using a mouse model of colorectal cancer linked to colitis, curcumin may have a chemopreventive result connected to modifications in the microbiota of these animals.

TREATMENT OF VARIOUS DISEASES

Anticancer Effects of Curcumin

The imbalance between cell division and death is one of a major contributor to cancer. Uncontrolled cell growth results from cells avoiding death because apoptotic signals are absent, which can lead to many forms of cancer.

Prostate Cancer

According to recent estimates from the American Cancer Society, prostate cancer is the second most common cause of cancer-related deaths in males [6]. Curcumin is a powerful inhibitor of prostate cancer growth and induction of apoptosis both in *vitro* and *in vivo* by interrupting many cell signaling pathways, such as mitogen-activated protein kinase (MAPK), epidermal growth factor receptor (EGFR) and nuclear factor kappa B (NF-κB) [7, 8]. Recent studies have described that curcumin activates protein kinase D1 (PKD1), leading to the attenuation of β-catenin and his MAPK-mediated oncogenic signaling, thus leading to the suppression of prostate cancer Figure 2 [9].

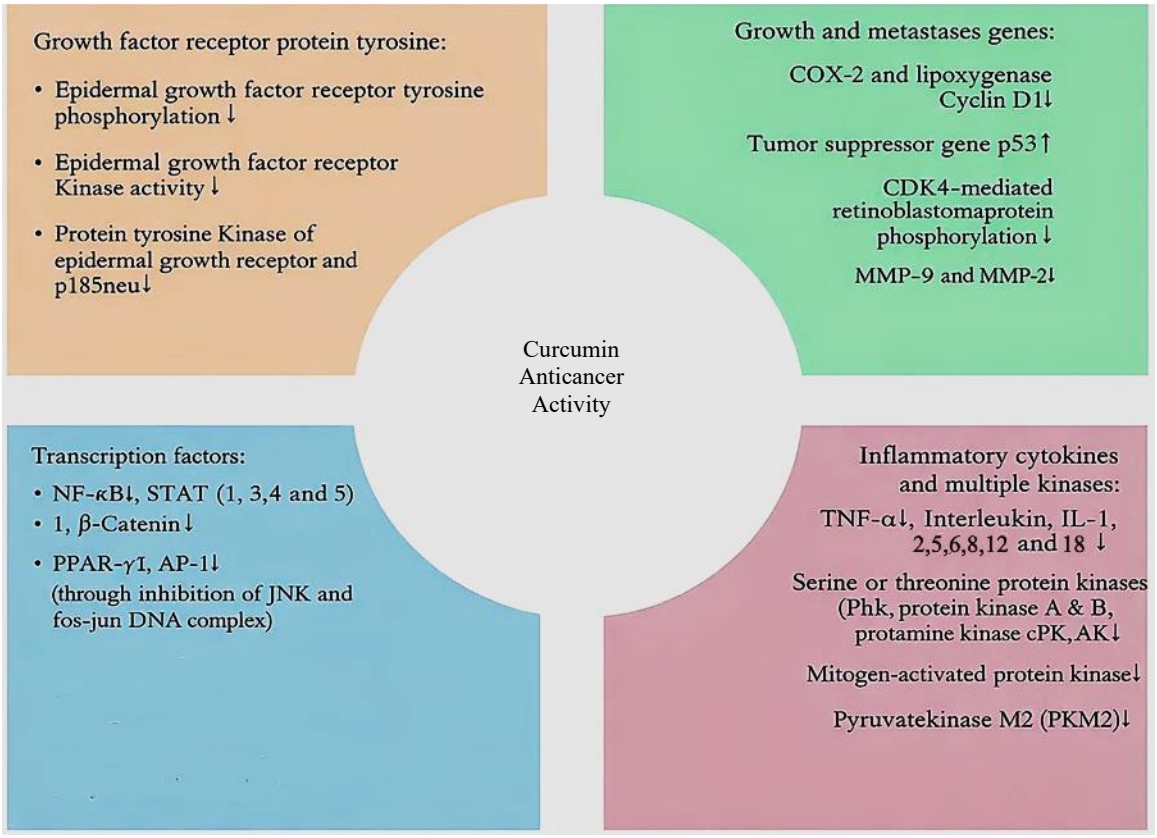

Figure 2. Curcumin's primary molecular targets in cancer cells. ↑: Increase; ↓: Decrease; MMP: Matrix metalloproteinase; AP-1: Activation protein 1.

Colorectal Cancer

After lung and prostate cancer, colorectal cancer is the third most prevalent type of cancer [10]. Patients identified with colorectal cancer receive chemotherapy and surgical operative removal of tumor tissue with chemotherapy, but more than half of individuals experience recurrence [11]. When curcumin is administered, it reduces M_1G levels in malignant colorectal cells without altering COX-2 protein levels [12]. Furthermore, curcumin treatment may downregulate the miR-21 gene exaggerated in colorectal cancer cells by reducing the binding of AP-1 (activator protein) to the miR-21 promoter [13]. Curcumin treatment of HCT 116 colon cancer cells decreased the formation of tumor tissue and blocked the cell cycle in the G_2/M phase via miR-21 gene regulation [14]. However, animal studies (mice) with colorectal cells showed that curcumin's ability to focus nuclear factor (NF-κB) improved the response to radiotherapy in association with curcumin [15]. In another research study, combining curcumin with ERRP, a Pan-erbB inhibitor, successfully enhanced the inhibitory activity of curcumin on colon cancer cells.

plagiarism***Breast Cancer***

Alarmingly, one of the leading causes of death for women is breast cancer [12]. Despite lumpectomy, radiotherapy, endocrine therapy and chemotherapy breast cancer recurrence rates remained high according to a meta-analysis of various research studies [16]. Therefore, more efficient treatment

strategies are still needed. Curcumin inhibited breast tumors by overexpressing the p53 gene and reducing antigen Ki-67 levels Figure 3. Other research study on MDA-MB-231 cells described that curcumin also inhibited the inflammatory cytokines CXCL1/2. Inhibition of CXCL1 and CXCL2 by curcumin leads to inhibition of the expression of many pro-metastasis genes, such as the chemotactic receptor CXCR4. Dimethyl curcumin (ASC-J9) has also been shown to be efficient against estrogen dependent breast cancer by inhibiting multiple class of steroid receptors [6, 17].

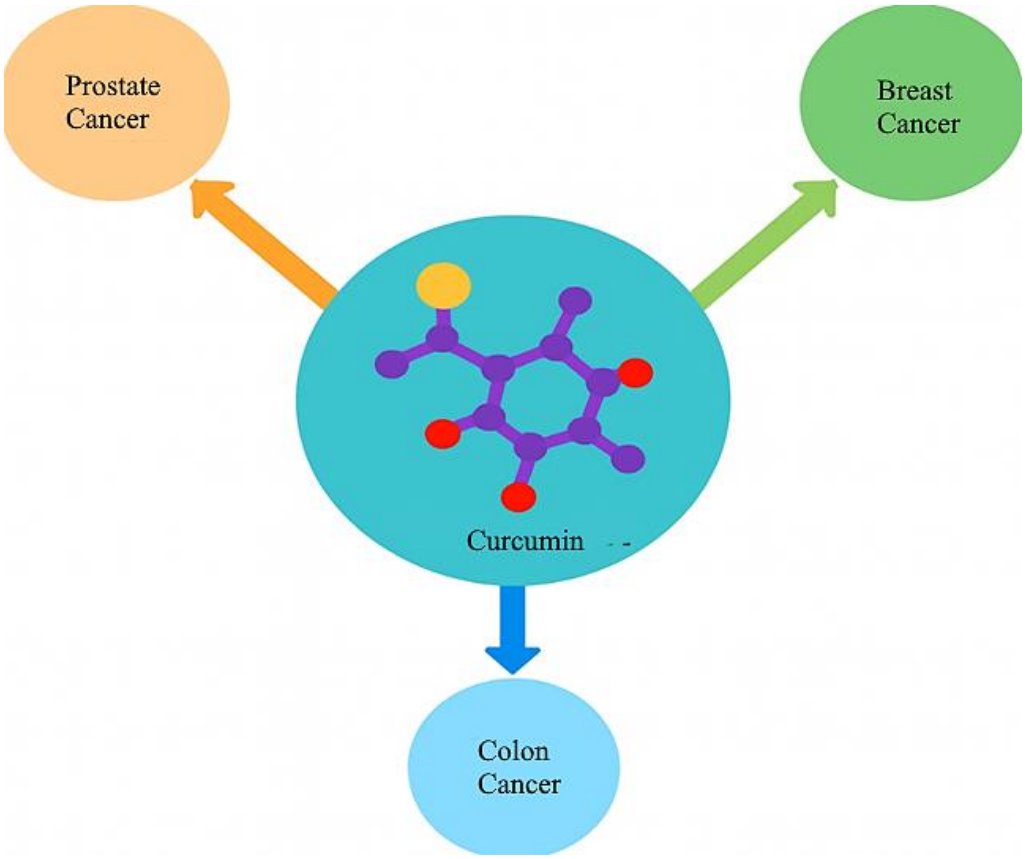

Figure 3. Distinct types of cancer inhibited by curcumin.

Pancreatic Cancer

The efficiency of curcumin in the management of pancreatic cancer was assessed in a non-randomized, open-label, phase II clinical trial supervised by Dhillon et al. In this research [18, 19], for eight weeks, patients with histologically diagnosed pancreatic cancer received treatment with a mixture of curcuminoids (curcumin, bisdemethoxycurcumin, and desmethoxycurcumin) at a dose of 8g daily. Patients had not received chemotherapy or radiation therapy in the 4 weeks before and during the study period. Tumor response (according to classical criteria for response assessment in solid tumors), tumor markers and serum cytokine concentrations were evaluated after 8 weeks. Based on this result, they described that combination therapy of curcumin and gemcitabine for pancreatic cancer is possible; however, curcumin dosages should not be more than 8g per day [20].

Head and Neck Cancer

In a research study, head, and neck squamous cell carcinoma (HNSCC) patients were used to evaluate the effects of curcumin on reducing IκB kinase β (IKKβ) activity and controlling proinflammatory cytokines. Patients were instructed to chew a curcumin tablet (2mg) and samples of saliva were taken both before and after chewing the tablet to measure IKKβ activity and concentrations of salivary cytokines interleukin (IL)-6 and IL-8. Curcumin caused a reduction in IKKβ activity in salivary cells of HNSCC patients [21, 22].

Arthritis

The main types of arthritis are osteoarthritis (OA), rheumatoid arthritis (RA), and gouty arthritis. Osteoarthritis, the most common joint disease, is a degenerative joint disease that involves inflammation. Osteoarthritis is more common in people over 50 and in women. Inflammation of cartilage, subchondral bone, and synovium may all play important roles in the progression of osteoarthritis [22]. Curcumin reduces joint inflammation and relieves pain symptoms, mainly due to its anti-inflammatory and chondroprotective properties [23]. Curcumin stimulates the chondroprotective transcriptional regulator Cbp/p300 interacting transactivator with ED-rich tail 2 (CITED2) and suppresses the mRNA expression of pro-inflammatory mediators IL-1β and TNF-α, MMPs 1, 3, and 13, and ADAMTS5 in primary cultured chondrocytes [24, 25].

Atherosclerosis

Atherosclerosis is a prevalent cardiovascular condition with a complicated pathogenesis [26] closely linked to coronary heart disease, hypertension, hyperlipidaemia, and other cardiovascular ailments. It is a disease described by mild chronic inflammatory condition of the arterial wall, which is the reason of multiple cardiovascular and cerebrovascular disorder. Macrophages are a key component in atherosclerosis [27]. Both proinflammatory (M1) and anti-inflammatory (M2) macrophages control homeostasis in healthy tissues [28]. M1 macrophages predominated in atherosclerotic plaques and the associated inflammatory cytokines IL-6, iNOS, and IL-1β were elevated, exacerbating the atherosclerotic lesions [29]. Curcumin modulates macrophage polarization and plasticity by influencing the TLR4/MAPK/NF-κB signaling pathway and has a positive effect on reducing atherosclerosis [30]. Furthermore, curcumin supplementation could reduce the activation of NF-κB in the aorta and the concentration of IL-1β and TNF-α in the aorta and serum [31].

COVID-19

Coronavirus disease 19 (COVID-19/2019-nCoV) is an infectious ailment, because of the intense acute respiration syndrome coronavirus 2 (SARS-CoV-2) [32]. In an open label nonrandomized medical trial, oral nano-formula of curcumin with dose of eighty mg two times day by day ought to substantially fasten the decision time of COVID-19-precipitated symptoms, enhance oxygenation, and decrease sanatorium live time in evaluation with manage group [33]. In addition, orally administered curcumin with piperine as adjuvant remedy in COVID-19 remedy ought to notably lessen morbidity and mortality, enhance the medical symptoms. The medical manifestations of COVID-19, such as acute respiration misery syndrome (ARDS), common pneumonia, and multi-organ failure.

ARDS is the main purpose of COVID-19 mortality, overall because of cytokine hurricane syndrome [34]. Patients with COVID-19 confirmed excessive tiers of inflammatory cytokines (TNF, IL-1β, IL-6, IL-8), colony stimulating factors (G-CSF and GM-CSF) and inflammatory chemokines (MCP1, IP10, and MIP1α) [22]. Thus, suppressing the accelerated inflammatory reaction that happens in COVID-19 can be beneficial in stopping the intensity of the ailment. Curcumin, a herbal compound with anti-inflammatory effect, ought to as an adjuvant drug in COVID-19 remedy [35]. In addition to anti-inflammatory effect, curcumin also can play an antiviral position through inhibiting SARS-CoV-2 access into cells and inhibiting viral proliferation [18]. Curcumin has a whole lot of pharmacological outcomes and excessive safety, which makes it an adjunctive drug for the remedy of COVID-19 [36–38].

Psoriasis

Psoriasis (PsO) is a chronic inflammatory, multisystemic, and multifactorial disorder that affects approximately 3% of the world's population. The thick silvery plaques observed clinically are the result of uncontrolled proliferation of keratinocytes. Curcumin can suppress excessive production of TNF-α by activated macrophages [39–41]. Curcumin has been shown to directly attach to the receptor binding site of TNF-α through covalent and non-covalent interactions and block subsequent TNF-dependent activation of NF-κB [42–46]. Additionally, curcumin is a noncompetitive inhibitor of phosphorylase kinase (PhK), a serine/threonine-specific protein kinase [47].

In this research, decreased PhK levels in plaque samples examined with curcumin 1% alcohol gel and other conventional topical treatments were associated with decreased keratinocyte transferrin receptor (TRR) expression, parakeratosis severity, and epidermal CD8+ T cells were associated with decreased density [48–52]. These preliminary observations may suggest that drugs capable of inhibiting PhK activity, such as curcumin, may be evaluated effective candidates for topical management of psoriasis Figure 4 [53].

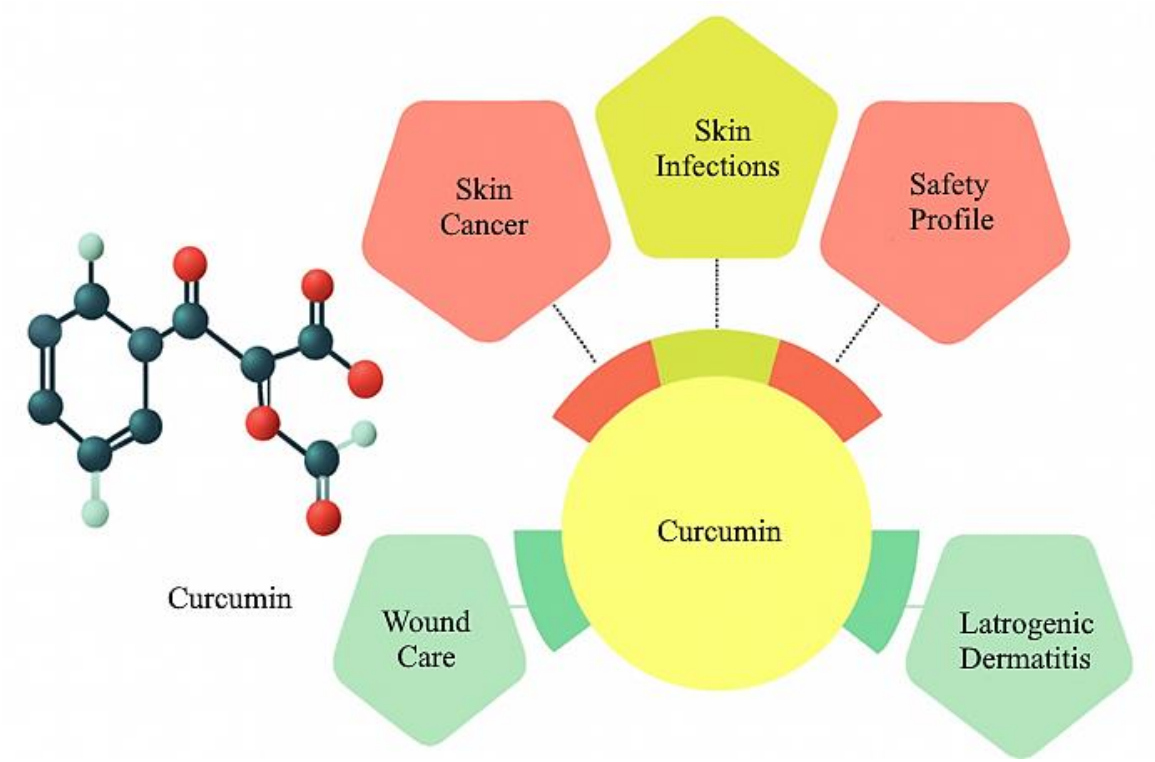

Figure 4. Use of curcumin in the treatment of various skin disorders.

Wound Care

Wound healing management is a pharmacological challenge with important economic impact on healthcare systems worldwide, and its costs are increasing rapidly [54, 55]. Wound healing is a complicated and progressive process that involves a series of cellular and molecular events. Briefly, it can be explained into three stages: (i) homeostasis and inflammation, (ii) proliferation with the formation of granulation tissue, and (iii) remodeling with the formation of new epithelium and scar formation [40, 56]. During the inflammatory phase, significant numbers of neutrophils are recruited to the injury site and release proteases, reactive oxygen species (ROS), and inflammatory mediators, such as TNF-α and IL-1 [41, 57–62]. As explained above, curcumin may reduce inflammation by inhibiting nuclear factor-κB (NF-κB) and suppressing TNF-α expression, as well as by interfering with LPS signaling. Additionally, curcumin exerts anti-inflammatory property by acting on other signaling mechanism, such as: peroxisome proliferator-activated receptor gamma (PPAR-γ) and myeloid differentiation protein 2-TLR4 co-receptor (TLR4-MD2) [63].

CONCLUSIONS

Turmeric is a medicinal plant that dates back 4,000 years to the Vedic civilization of India and is very commonly used in natural medicine and other therapeutic systems. Accumulating evidence has confirmed that curcumin may modulate phenomena involved in inflammatory, proliferative, and infectious diseases of the skin. Curcumin-related derivatives are a new platform for chemotherapy, and it is clear that curcumin derivatives can improve therapeutic efficacy and overcome the above limitations. Curcumin has excellent anti-inflammatory properties, regulating NF-κB, MAPK, AP-1, JAK/STAT, and other signaling pathways and inhibiting the production of inflammatory mediators. Bioavailability and therapeutic effects

(anti-inflammatory) of curcumin have been improved to some extent by structural modification of curcumin, formulation evaluation and combination therapy. Various research studies explain Curcumin has significant anticancer effects against various types of cancers (Prostate, breast, colorectal, pancreatic, head, and neck cancers) [64]. Additionally, its therapeutic potency and safety in cancer patients alone or in combination therapy with other anticancer drugs has been demonstrated in several human clinical trials [48, 58, 65]. Curcumin disrupts various cellular pathways, including MAPK, EGF, NF-κB, PKD1, COX-2, STAT3, TNF-α and IKKβ. However, the use of curcumin as an anticancer agent is mainly limited by its low aqueous solubility, responsible for low cellular uptake, low bioavailability and poor chemical stability [54–57, 63, 66]. Various methods have been taken to overcome these limitations, including structural modifications and the aid of drug delivery systems. The possibility of using curcumin in combination with conventional drugs or formulating novel drug delivery systems represents a promising area for future application-oriented research.

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