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Review

Butein in health and disease: A comprehensive review

Ganesan Padmavathi[a], Nand Kishor Roy[a], Devivasha Bordoloi[a], Frank Arfuso[b], Srishti Mishra[c], Gautam Sethi[c,d,*], Anupam Bishayee[e,*], Ajaikumar B. Kunnumakkara[a,*]

[a] Cancer Biology Laboratory, Department of Biosciences and Bioengineering, Indian Institute of Technology, Guwahati, Assam 781 039, India
[b] Stem Cell and Cancer Biology Laboratory, School of Biomedical Sciences, Curtin Health Innovation Research Institute, Curtin University, Perth, West Australia 6009, Australia
[c] Department of Pharmacology, Yong Loo Lin School of Medicine, National University of Singapore, Singapore 117600, Singapore
[d] School of Biomedical Sciences, Curtin Health Innovation Research Institute, Biosciences Research Precinct, Curtin University, Western Australia 6009, Australia
[e] Department of Pharmaceutical Sciences, College of Pharmacy, Larkin Health Sciences Institute, Miami, FL 33169, USA

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ABSTRACT

Background: The risk of suffering from many chronic diseases seems to have made no improvement despite the advancement in medications available in the modern world. Moreover, the use of synthetic chemicals as medications has proved to worsen the scenario due to the various adverse side effects associated with them.

Purpose: Extensive research on natural medicines provides ample evidence on the safety and efficacy of phytochemicals and nutraceuticals against diverse chronic ailments. Therefore, it is advisable to use natural products in the management of such diseases. This article aims to present a comprehensive and critical review of known pharmacological and biological effects of butein, an important chalcone polyphenol first isolated from *Rhus verniciflua* Stokes, implicated in the prevention and treatment of various chronic disease conditions.

Methods: An extensive literature search was conducted using PubMed, ScienceDirect, Scopus and Web of Science™ core collections using key words followed by evaluation of the bibliographies of relevant articles.

Results: Butein has been preclinically proven to be effective against several chronic diseases because it possesses a wide range of biological properties, including antioxidant, anti-inflammatory, anticancer, antidiabetic, hypotensive and neuroprotective effects. Furthermore, it has been shown to affect multiple molecular targets, including the master transcription factor nuclear factor-κB and its downstream molecules. Moreover, since it acts on multiple pathways, the chances of non-responsiveness and resistance development is reduced, supporting the use of butein as a preferred treatment option.

Conclusion: Based on numerous preclinical studies, butein shows significant therapeutic potential against various diseases. Nevertheless, well-designed clinical studies are urgently needed to validate the preclinical findings.

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Abbreviations: 4E-BP1, eIF4E binding protein 1; ACE, angiotensin converting enzyme; ACTA2, alpha-actin-2; AGEs, advanced glycation end products; ALDH1A3, aldehyde dehydrogenase 1 family member A3; ALL, acute lymphoblastic leukemia; AP-1, activator protein 1; aP2, adipocyte protein 2; AQP 2, aquaporin 2; ARE, antioxidant response elements; ARF, acute renal failure; ATM, ataxia telangiectasia mutated; ATP, adenosine triphosphate; Bad, Bcl-2-associated death promoter; Bax, Bcl-2-associated X protein; BCG, Bacillus Calmette–Guérin; Bcl-2, B-cell lymphoma 2; Bcl-xL, B-cell lymphoma-extra large; C/EBPα, CCAAT-enhancer-binding protein alpha; Cdks, cyclin dependent kinases; Chk, checkpoint kinase; CML, chronic myeloid leukemia; COL1A1, collagen, type I, alpha 1; COX-2, cyclooxygenase-2; CVDs, cardiovascular diseases; CXCL12, chemokine (C-X-C Motif) Ligand 12; CXCR4, C-X-C chemokine receptor-4; DIO, diet-induced obese; DR5, death receptor 5; EGFR, epidermal growth factor receptor; eIF4E, eukaryotic translation initiation factor 4E; EMT, epithelial mesenchymal transition; EPC, endothelial progenitor cell; ERK, extracellular signal-regulated protein kinase; FAS-II, fatty acid synthase II; FOXO3a, forkhead box O3; GCL, glutamate–cysteine ligase; GSH, glutathione; HCC, hepatocellular carcinoma; HDP, human dental pulp cells; HIV, human immunodeficiency virus; HMC, human mast cells; HO-1, heme oxygenase-1; HSA, human serum albumin; HSC, hepatic stellate cells; hTERT, human telomerase reverse transcriptase; HUVECs, human umbilical vein endothelial cells; IAPs, inhibitors of apoptosis proteins; ICAM1, intercellular adhesion molecule 1; IFN, interferon; IKK, I kappa B kinase; IL, interleukin; iNOS, Inducible nitric oxide synthase; ISD, inflammatory skin diseases; JAK, Janus kinase; JNK, c-Jun N-terminal kinases; LFA-1, lymphocyte function-associated antigen 1; LPL, lipoprotein lipase; LPS, lipopolysaccharides; MAPK, mitogen-activated protein kinase; MDM2, mouse double minute 2; MDR-TB, multi drug resistant tuberculosis; MM, multiple myeloma; MMP, matrix

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Introduction

Phytochemicals isolated from various plant sources and crude plant extracts have been used as nutraceuticals and medications for the treatment of several chronic diseases, such as obesity, diabetes, cardiovascular diseases (CVDs), infectious diseases, inflammatory diseases, neurological disorders and neoplasms, both in traditional and modern medicines (Aggarwal et al., 2008; Muralimanoharan et al., 2009; Roy et al., 2016). Terpenoids and polyphenols such as anthocyanins, flavonols, isoflavones, chalcones, carotenoids, vitamins, and xanthones, are some of the major bioactive phytochemicals commonly found in medicinal plants (Kaulmann and Bohn, 2014). Butein (3,4,2′,4′-tetrahydroxychalcone) belongs to the chalcone family of flavonoids and was isolated from several plants including *Toxicodendron vernicifluum*, *Semecarpus anacardium*, *Dalbergia odorifera*, and *Butea monosperma*. All these plants have been used in various systems of traditional medicine, such as Siddha, Ayurveda, Korean, Chinese, and Iranian, for curing inflammatory diseases, atherosclerosis, hepatic disorders, ulcers, gout, eye diseases, rheumatic pain, dementia, bleeding, cough, and cancers (Padmavathi et al., 2015). Butein, isolated from these plants with profound medicinal use, has inherent antioxidant, anti-inflammatory, anticancer, antinephritic, antidiabetic, and antibacterial properties (Padmavathi et al., 2015; Song et al., 2016). Like many other polyphenols, butein was also proved to have immense potential as therapeutic for the treatment of cancer, inflammatory diseases, tuberculosis, obesity, diabetes, liver fibrosis, neuropathy, and oxidative stress, with a wide range of molecular targets.

Traditional uses of butein and its source plants

As mentioned earlier, the medicinal properties of the source plants of butein have been highly explored and exploited in the traditional medicinal systems of Asian countries including China, India, Japan and Korea. For instance, crude extracts of *B. monosperma*, *Bidens bipinnata*, *Dahlia variabilis*, *D. odorifera*, *Millettia nitida* and *T. vernicifluum* rich in butein content are found to be widely used as herbal formulations in the treatment of diabetes, diarrhoea, dysentery, tuberculosis, hypertension, infectious diseases, inflammatory diseases, ischaemia, malaria, paralysis, rheumatoid arthritis etc. (Semwal et al., 2015; Padmavathi et al., 2015). Further, Qubaibabuqi- a formulation of five different herbs *viz*. *Vernonia anthelmintica* Linn., *Psoralea corylifolia* Linn., *Alpinia officinarum*, *Operculina turpethum* Linn. and *Plumbago zeylanica* Linn., is found to be used to treat vitiligo in the traditional Chinese medicine. Interestingly, the major active component of this formulation was revealed to be butein (Pei et al., 2016). Moreover,

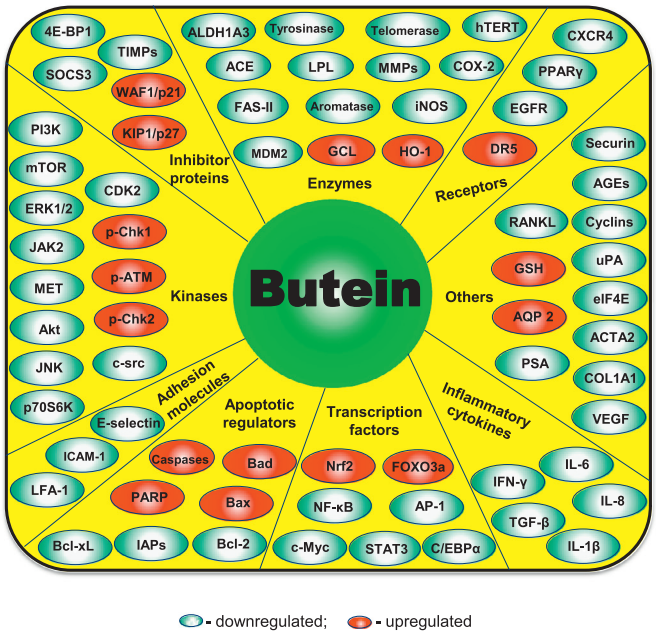

Fig. 1. Molecular targets of butein.

several decades of research on pure butein evidenced that butein with the other major active component of these medicinal plants is responsible for their enormous biological activities. Therefore, the prime aim of this review is to summarize the studies available on the effect of butein against various chronic diseases with an understanding on underlying molecular mechanisms of action.

Database and literature search strategies

For the literature review, we used standard search strategies involving the exploration of online databases, such as PubMed which comprises biomedical literature from MEDLINE, Science direct, Scopus and Web of Science™ core collections using key words, followed by evaluation of the bibliographies of relevant articles.

Molecular targets of butein

Extensive research over the past few decades has revealed butein to be a potent multitargeted flavonoid. As depicted in Fig. 1, butein was shown to alter the expression and activity of several genes, transcription factors, enzymes, and proteins involved in important cellular processes essential for tumor initiation, progression, and chemoresistance (Padmavathi et al., 2015). The major molecular target affected by butein treatment in most of the diseases investigated is the transcription factor nuclear factor κB (NF-κB). Butein was shown to prevent the activation of NF-κB by inhibiting IκB kinase (IKK) and hampering its nuclear translocation, ultimately leading to the deregulation of its downstream molecules. This butein-mediated inactivation of NF-κB was found to induce apoptosis and inhibit proliferation, cell cycle progression, angiogenesis, invasion, metastasis, and chemoresistance of various cancers (Chua et al., 2010; Jang et al., 2012; Khan et al., 2012; Moon et al., 2010a; Moon et al., 2010b; Pandey et al., 2007; Zhang et al., 2008). Besides cancer, NF-κB inhibition also attenuated cytokines and lipopolysaccharide (LPS)-induced oxidative stress and inflammation, increased glucose tolerance and decreased fat accumulation, ameliorated liver fibrosis by preventing hepatic stellate cell (HSCs) activation, and suppressed tumor-induced osteoclastogenesis (Benzler et al., 2015; Hayashi et al., 1996a; Kojima et al., 2015; Lee et al., 2004; Rasheed et al.,

metalloproteinase; MPM, malignant pleural mesothelioma; mTOR, mammalian target of rapamycin; NF-κB, nuclear factor-kappa B; NO, nitric oxide; Nrf2, nuclear factor erythroid 2 [NF-E2]-related factor 2; OXPHOS, oxidative phosphorylation; PaCa, pancreatic cancer; PARP, poly-(ADP-ribose) polymerase; PDGF, platelet-derived growth factor; PDGFRB, platelet-derived growth factor receptor beta; PKC, protein kinase C; PMA, phorbol 12-myristate 13-acetate; PPARγ, peroxisome proliferator-activated receptor γ; PSA, prostate-specific antigen; RANKL, receptor activator of nuclear factor-kappa B ligand; ROS, reactive oxygen species; RVS, *Rhus verniciflua* Stokes; SCI, spinal cord injury; SOCS3, suppressor of cytokine signaling 3; STAT3, signal transducer and activator of transcription 3; TB, tuberculosis; tBHP, *tert*-butylhydroperoxide; TGF-β, transforming growth factor beta; TIMP, tissue inhibitor of metalloproteinase; TNF-α, tumor necrosis factor alpha; TRAIL, tumor necrosis factor-related apoptosis-inducing ligand; uPA, urokinase plasminogen activator; VEGF, vascular endothelial growth factor; VSMCs, vascular smooth muscle cells; α-SMA, α-smooth muscle actin.

* Corresponding authors.
E-mail addresses: phcgs@nus.edu.sg (G. Sethi), abishayee@ULarkin.org, abishayee@gmail.com (A. Bishayee), kunnumakkara@iitg.ernet.in (A.B. Kunnumakkara).

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