



Kaempferol for the treatment of asthma: A review of pre-clinical evidence on therapeutic potential and mechanism

Yu Zhao Lee^a, Fui-Ling Voon^b, Hong Yee Chung^c, Jaimah Shakwat^c, Chau Ling Tham^{d,e}, Yu-Cheng Ho^{f,**}, Ming Tatt Lee^{g,h,*}

^a Faculty of Medicine and Health Sciences, UCSI University, Kuala Lumpur 56000, Malaysia

^b Faculty of Resource Science and Technology, Universiti Malaysia Sarawak, Kota Samarahan 94300, Malaysia

^c School of Science, Monash University Malaysia, Jalan Lagoon Selatan, Subang Jaya, Selangor, Bandar Sunway 47500, Malaysia

^d Department of Biomedical Sciences, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia, Serdang 43400, Selangor, Malaysia

^e Future Foods, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia, Serdang 43400, Selangor, Malaysia

^f School of Medicine, College of Medicine, I-Shou University, Kaohsiung City 82445, Taiwan

^g Office of Postgraduate Studies, UCSI University, Kuala Lumpur 56000, Malaysia

^h School of Health Sciences, Chang-Jung Christian University, Tainan 711301, Taiwan

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ABSTRACT

Asthma remains a significant global health burden characterised by high clinical heterogeneity and immunological complexity. Despite currently available therapies, limitations like steroid insensitivity and adverse side effects persist. Furthermore, recent biologics' high costs and accessibility remain a constraint. This emphasised the need for innovative alternative treatments. Kaempferol, a naturally occurring flavanol found in various fruits, vegetables, and medicinal plants, has gained considerable attention for its multifaceted pharmacological properties. This review critically assesses existing preclinical evidence regarding the therapeutic potential and molecular mechanisms of kaempferol in experimental asthma models. Preclinical in vivo and in vitro studies consistently demonstrate that kaempferol targets three critical dimensions of asthma pathogenesis: airway inflammation, hyperresponsiveness, and airway remodelling. Mechanistically, it suppresses the infiltration of inflammatory cells and reduces the secretion of pro-inflammatory cytokines, chemokines, and IgE. These effects are achieved by modulating key signalling pathways, including NF- κ B, MAPKs, JAK/STAT, Nrf2, and NOX4-mediated autophagy. Furthermore, kaempferol alleviates smooth muscle hypercontractility and suppresses airway remodelling by mitigating subepithelial fibrosis, mucus hypersecretion, and epithelial-to-mesenchymal transition. Its translational strengths include potential steroid-sparing effects. Despite this, its clinical application is hindered by poor oral bioavailability and a lack of direct human trials. Rigorous clinical validation and advanced targeted delivery systems are required to establish kaempferol as a viable therapeutic agent for asthma management.

1. Introduction

Asthma is a prevalent chronic inflammatory airway condition impacting over 300 million individuals globally and continues to be a significant morbidity cause [1]. Asthma had been demonstrated to exhibit high clinical heterogeneity and immunological complexity, comprising many endotypes. They are defined by the major inflammatory cells involved in the pathogenesis, mainly categorised into type 2 (T2) eosinophilic asthma and non-T2 asthma phenotypes [2]. Despite

the substantial advancements in disease management afforded by inhaled corticosteroids (ICS) and bronchodilators, a significant number of patients still endure persistent symptoms, recurrent exacerbations, steroid insensitivity, or adverse effects that limit treatment efficacy [1]. The adverse effects of prolonged ICS use encompass local complications such as dysphonia and oropharyngeal candidiasis, alongside potential systemic risks like hypothalamic–pituitary–adrenal axis suppression, decreased bone mineral density, cataracts, and an increased susceptibility to respiratory infections [3]. Current biologic treatments that

* Corresponding author at: Office of Postgraduate Studies, UCSI University, No 1, Jalan Menara Gading, 56000 Cheras, Kuala Lumpur, Malaysia.

** Corresponding author at: School of Medicine, College of Medicine, I-Shou University, No.8, Yida Rd., Yanchao District, Kaohsiung City 82445, Taiwan.

E-mail addresses: r96443003@ntu.edu.tw (Y.-C. Ho), leemt@ucsiuniversity.edu.my (M.T. Lee).

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