Fabrication of Galangin-loaded polymeric nanoparticles: A propitious intervention in the management of asthma

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Nanoparticle-based drug delivery systems are developed to target alveolar macrophages associated with pulmonary inflammation. Allergic asthma is a chronic inflammatory lung disease characterized by airway hyperresponsiveness, airway inflammation and goblet cell hyperplasia to inhaled allergens and nonspecific stimuli. Peroxisome proliferator-activated receptor gamma (PPARγ), a nuclear hormone receptor is expressed in the structural and inflammatory cells of the lungs that regulates inflammatory responses in asthma pathophysiology. Galangin, a flavonoid present in Alpinia officinarum is a PPARγ agonist proven to possess anti-asthmatic property. Despite the potent therapeutic efficacy of galangin, poor aqueous solubility limits its pharmacological activity. Polymeric nanoparticles are biocompatible, safe and stable with sustained release property which are required for better therapeutic applications. Hence, nanoparticle-based approach was adopted to enhance the therapeutic efficacy of galangin, forming the rationale of the study. Activation of PPARγ may also occur by ligand-independent transcriptional activity, and conversely, the ligand may follow PPARγ-independent pathway. Therefore, our hypothesis is that galangin loaded polymeric nanoparticles (G-NPs) could enhance the anti-asthmatic effect of encapsulated galangin over free galangin via PPARγ-dependent pathway. In this study, G-NPs were prepared and characterized. In vitro drug release and hemocompatibility studies were performed. In vivo anti-asthmatic studies in ovalbumin-induced murine model were performed, wherein, G-NPs significantly ameliorated the pro-inflammatory mediators. Expression (mRNA and protein) analyses confirm the mechanistic action of PPARγ. Taken together, our findings communicate that nanoencapsulated compound exhibited better anti-asthmatic activity over free compound by suppressing the pro-inflammatory mediators via PPARγ-dependent pathway, thereby implying PPARγ as a therapeutic target for asthma.

Biography

Ms. Linda Jeeva Kumari H. is a Ph.D. scholar at the University College of Engineering (UCE), Anna University, Tiruchirappalli, Tamil Nadu, India. She has received a master's degree in biotechnology from PSG College of Technology, Coimbatore, India. Her research interests are drug delivery and pulmonary pharmacology. She has worked as a Junior Research Fellow in the Department of Science and Technology, Government of India sponsored National Facility for Drug Development for Academia,Pharmaceutical and Allied Industries, Anna University, Tiruchirappalli. She has actively participated in various international conferences and has published 11 papers in peer-reviewed journals (cumulative impact factor 32.459).

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