PM10-Induced Cytotoxicity and Pro-Inflammatory Cytokine Expression in Human Skin Cells: Protective Effects of *Wolffia* Extract

Juwainee Madardam
Khon Kaen University (KKU), Thailand
Thailand Institute of Scientific and Technological Research (TISTR), Thailand

Thanchanok Muangman
Thailand Institute of Scientific and Technological Research (TISTR), Thailand

Kanyanat Kaewiad
Thailand Institute of Scientific and Technological Research (TISTR), Thailand

Theerapak Natekuerkoon
Université Marie et Louis Pasteur, France

Céline Viennet-Steiner
Université Marie et Louis Pasteur, France

Nisachon Jangpromma
Khon Kaen University (KKU), Thailand

Abstract

Particulate matter with a diameter of ≤10 μm (PM10) is a major air pollutant known to induce skin inflammation and cellular damage. PM10 exposure can trigger cytotoxicity and stimulate the production of pro-inflammatory mediators in skin cells, contributing to pollution-related skin disorders. This study investigated the cytotoxic and inflammatory effects of PM10 on human skin cells and evaluated the protective effects of *Wolffia* extract. Human keratinocytes (HaCaT) and human dermal fibroblasts (HDFa) were used as in vitro skin models. Cells were exposed to various concentrations of PM10 to assess cytotoxicity, and 150 μg/mL PM10 was selected to induce inflammation. Pro-inflammatory cytokines and mediators, including TNF-α, IL-6, IL-8, and PGE2, were quantified. PM10 significantly increased cytotoxicity and elevated levels of TNF-α, IL-6, IL-8, and PGE2 in both HaCaT cells and fibroblasts. Treatment with *Wolffia* extract markedly reduced PM10-induced inflammatory mediator production without causing cytotoxic effects. These findings indicate that PM10 induces inflammatory responses in human skin cells, while *Wolffia* extract demonstrates protective and anti-inflammatory potential against pollution-induced skin damage.

Keywords

Particulate matter 10, Inflammation, Cytotoxicity, Human skin, Human keratinocytes (HaCaT), human dermal fibroblasts (HDFa), *Wolffia* extract.