INTRODUCTION

To assess the regulatory mechanism of sulforaphane ~~for~~ in activated HMC-1 cells, we investigated the ~~inhibition~~ inhibitory effect of sulforaphane on PMACI-induced NF-κB activation. ~~Because~~ Since the blockade of NF-κB is linked with anti-inflammatory ~~effect~~activity, we postulated that sulforaphane and WE mediate ~~its~~ their effects at least partly via ~~inhibiting~~ inhibition of NF-κB activation. ~~The p~~Pretreatment with WE or sulforaphane mitigated ~~the~~ PMACI-induced NF-κBp65 levels in the nuclear protein extract (Fig. 7A upper). As an inhibition marker of NF-κB activation, ~~the~~ phosphorylation of IκBα in cytosolic protein extracts was analyzed. ~~As a result, we show~~Our results show that WE and sulforaphane mitigated ~~the~~ PMACI-induced IκBα phosphorylation (F~~f~~ig. 7A lower). Protein levels of PARP and GAPDH ~~did~~ were not affected by any treatment in the nuclear and cytosolic protein extracts.

Finally, we ~~wished to identify~~identified ~~if~~ whether sulforaphane could regulate MAPK~~s~~ phosphorylation ~~because~~ since inflammatory cytokine levels secreted from ~~the origin of~~ HMC-1 cells are known to be regulated via MAPK~~s~~ signaling pathways [7]. ~~The s~~Stimulation with PMACI induced significant increases in the phosphorylat~~ed~~ion levels of p38, JNK, and ERK MAPKs compared to those of untreated cells (Fig. 7B). However, ~~the~~ pretreatment with sulforaphane or WE ~~mitigates~~ mitigated the elevated phosphorylation ~~the phosphorylated~~ levels of p38, JNK, and ERK MAPKs ~~increased~~ by PMACI stimulation (Figure 7B).