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EXTRACT OF *Cassia fistula* L. STALK (CFS) MODULATES HEALING PROCESS THROUGH INCREASED PROLIFERATION OF KERATINOCYTES

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INTRODUCTION

Cassia fistula L., belongs to the Fabaceae family, is a common species of semi-arid region of Brazil. Previous studies have reported that *C. fistula* has several pharmacological activities and bioactive compounds identified were phenolic acids. Studies have shown effects the extract of *C. fistula* flowers and stem in intense antioxidant and hypoglycemic activity. The aim of this study was to evaluate the effect of *Cassia fistula* stem extract (CFS) on human keratinocytes (HaCaT) proliferation, as well as its role in the *in vitro* wound healing process.

MATERIAL AND METHODS

HaCaT keratinocytes were plated in 96-well plate with DMEM supplemented with 10% fetal bovine serum and incubated at 37ºC with 5% CO_2. After 24 hours, CFS (100–400µg/mL) or vehicle (water) was incubated and cell viability and proliferation were evaluated by MTT and SRB assay. The absorbance was measured at 570nm. To evaluate the wound healing *in vitro*, the Scratch assay was performed, which was evaluated after 12, 24, 48 and 72 hours of treatment. The E-cadherin expression was evaluated by flow cytometer. One-way ANOVA determined comparisons between multiple groups and student t-test compared mean differences.

RESULTS

Treatment with CFS significantly stimulated increased HaCaT proliferation at all doses (100 - 400 µg/mL). These results corroborate with Scratch assay, where the concentrations of 100 and 200 µg/mL stimulated celll migration by completely closing the scratch area after 72 hours with or without mitomycin (proliferation inhibitor). Expression of E-cadherin, a glycoprotein in membrane important to cell-cell adhesion, was significantly reduced at doses of 100 and 200 µg/mL, as compared to the control group. This low expression is essential for cell migration, which is characteristic of the proliferative phase of wound healing, event necessary for the complete regeneration of the injured tissue.

CONCLUSIONS

These results suggest that CFS increases HaCaT proliferation and migration, and a possible mechanism of action is by decrease of E-cadherin expression. Therefore, CFS represents a potential therapeutic strategy of wound healing.

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