Cassia tora seed extract suppresses heat-induced lipogenesis via inactivation of JNK/p38 MAPK signaling in human sebocytes

Hyuk Chul Kwon *, Tae Yang Kim, Chun Mong Lee, Kwang Sik Lee, Kun Kook Lee
Songpa R&D Center, Coreana Cosmetics CO., Ltd, 6, Samgok 2-gil, Seonggeo-eup, Seobuk-gu, Cheonan-si,
Chungcheongnam-do, 331-833, Republic of Korea

* corresponding author. E-mail address: gurcjf@coreana.co.kr (H.-C. Kwon)

Coréana

ABSTRACT

Heat induced by infrared (IR) radiation from sun exposure increases skin temperature and can lead to thermal and photo-aging. However, little is known about the relationship between heat induced by IR radiation and lipid biosynthesis in human sebocytes. In this study, we found that peroxisome proliferator-activated receptor (PPAR) γ , sterol regulatory element-binding transcription factor (SREBP)-1, and fatty acid synthase (FAS) were upregulated and the c-Jun N-terminal kinase (JNK)/p38 signaling pathways were activated in human sebocytes following heat exposure. Treatment with Cassia tora seed extract suppressed this up-regulation of PPAR γ , SREBP-1, and FAS expression and also suppressed the increase in squalene and triglyceride levels. These findings provide evidence that IR radiation can stimulate sebum production; Cassia tora seed extract can reverse this effect, and may therefore be useful for treating skin disorders such as acne vulgaris.

METHODS

For the treatment, cells were maintained in culture medium without FBS (i.e., serum starved) for 6 h and then moved for 30 min to incubators set at 37°C (control), 41°C, or 44°C (heat shock). Culture media were replaced with fresh media without FBS. To investigate expression of gene and signaling pathway, we performed western blotting. Lipid levels were assessed by Nile red staining, Squalene and triglyceride assay.

OBJECTIVES

The molecular basis for the relationship between heat and sebum production is not known in human sebocytes. To address this question, this study investigated the expression of factors involved in lipid biosynthesis in human sebocytes exposed to heat.

ACKNOWLEDGMENTS

This research was supported by the Ministry of Trade, Industry & Energy, Korea Institute for Advancement of Technology through the Encouragement Program for the Industries of Economic Cooperation Region.

CONCLUSIONS

- Heat-shock of human sebocytes *in vitro* induces PPAR γ , SREBP-1, and FAS levels
- Squalene and triglyceride levels were increased in heat shocked human sebocytes
- CTSE reversed heat shock effect on gene and lipid levels
- The JNK and p38 pathways were activated by heat-shock and this was reversed by CTSE
- These results suggest that an increase in skin temperature caused by excessive sun exposure can promote skin disorders such as acne vulgaris and that CTSE is a candidate agent for the treatment of this condition

RESULTS

1. FAS, SREBP-1, and PPAR γ , are upregulated by heat in human sebocytes.

2. Inhibition of lipid synthesis by *Cassia tora seed extract* (CTSE) in heat-treated human sebocytes

3. Heat induces lipid synthesis in human sebocytes via inactivation of JNK/p38 signaling.

REFERENCES

- Acne and sebaceous gland function, *Clin. Dermatol*
- Factors affecting excretion in man the rate of sebum, *J. Soc. Cosmet. Chem*
- The effect of local temperature variations on the sebum excretion rate, *Br. J. Dermatol*
- Heat shock-induced matrix metalloproteinase (MMP)-1 and MMP-3 are mediated through ERK and JNK activation and via an autocrine interleukin-6 loop, *J. Invest. Dermatol.*