Targeting Inflammation–Barrier Crosstalk: A Pre-Clinical *Ex Vivo* Study of a Cosmetic Formulation for Skin Comfort and Resilience

Authors: Layane Melody da Silva Reis Cavalcante[1], Lucia de Fatima Holanda[1], Giulia Okamoto Maciente1, Mauricio Frazilho[1], Carla De Figueiredo Presti Favaro[1], Marisa Sacuragui[1], Gabriela da Silva Leite[2], Gustavo Henrique da Silva[2], Barbara de Freitas Carli[2], Carla Monserrat Grecco Lopes[2], Ana Lucia Tabarini Alves Pinheiro[2], Kelen Fabíola Arroteia[2], Samara Eberlin[2]

Filiations: Mantecorp Skin Care, Hynova, São Paulo/SP – Brazil[1], Grupo Kosmoscience, Valinhos, SP - Brazil[2]

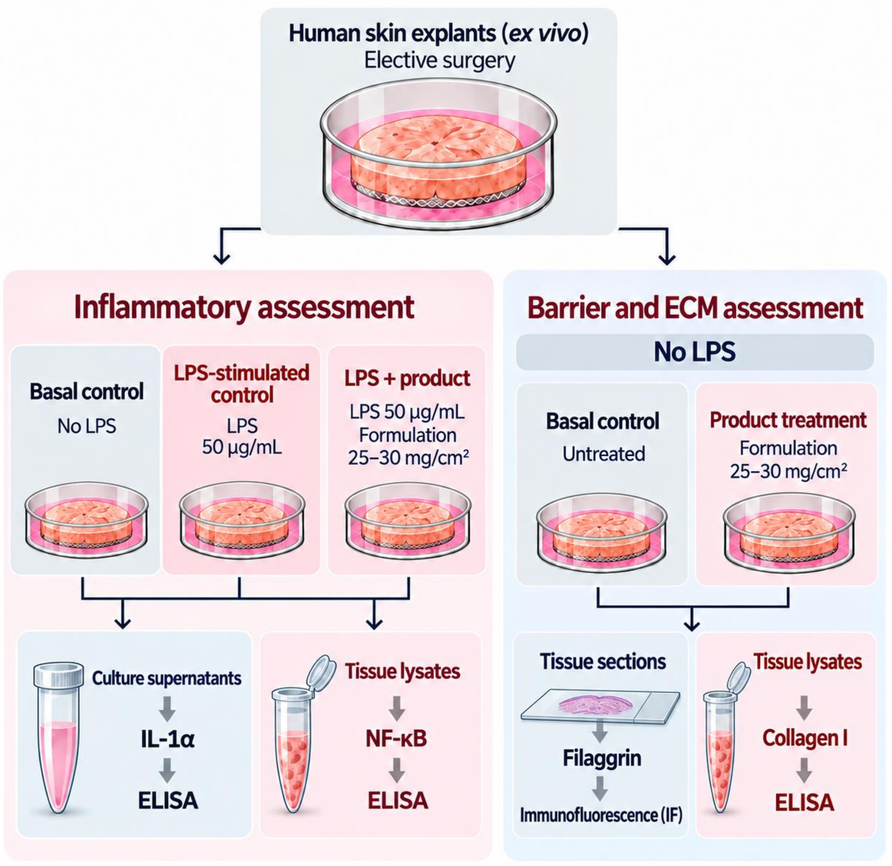
INTRODUCTION

Skin inflammation and barrier impairment are closely interconnected processes involved in skin sensitivity, discomfort, and premature aging. External aggressors can trigger inflammatory cascades, increasing interleukin-1 alpha (IL-1α) levels and activating nuclear factor kappa B (NF-κB). These responses may compromise epidermal barrier proteins, including filaggrin, and contribute to extracellular matrix degradation, affecting structural components such as collagen I. Barrier disruption may further facilitate irritant penetration, perpetuating inflammation and skin fragility. Therefore, simultaneously targeting inflammation, barrier integrity, and dermal structure represents a relevant strategy for maintaining skin homeostasis and resilience

OBJECTIVES

This study investigated the effects of a cosmetic formulation (DC-23044-28) on inflammatory mediators (IL-1α and NF-κB), the barrier-related protein filaggrin, and collagen I using human skin ex vivo models.

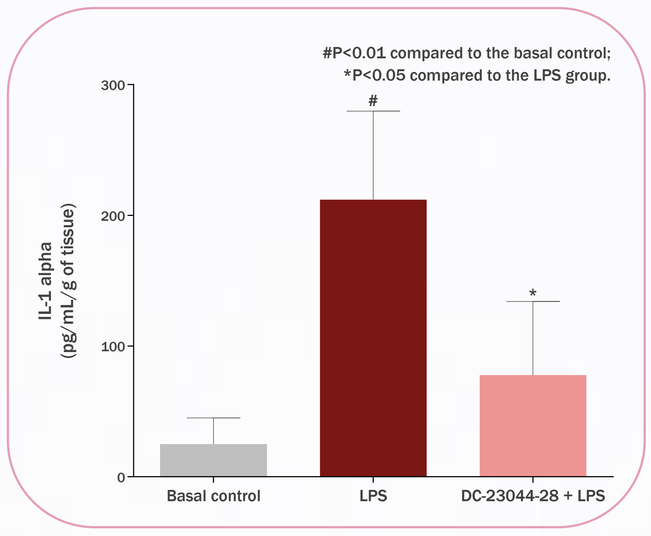
MATERIALS AND METHODS



RESULTS

743.05% ↑ **IL-1α** 29.98% ↓

63.27% ↑ **NF-κB** 22.42% ↓



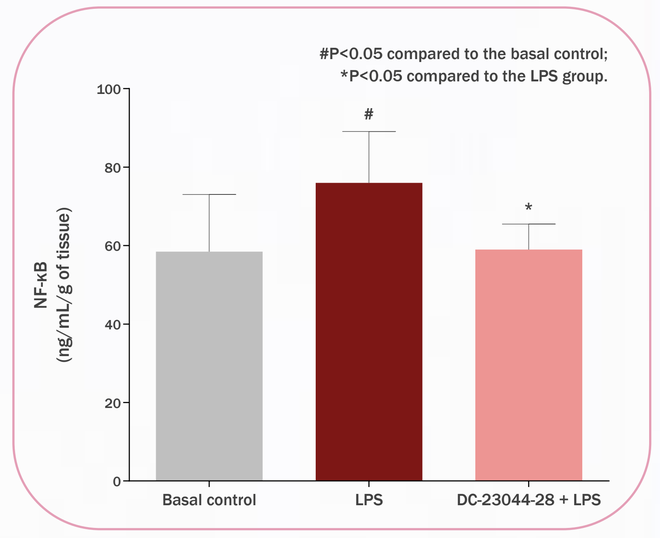
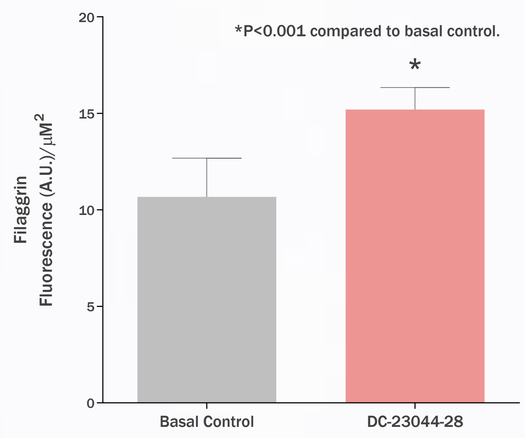
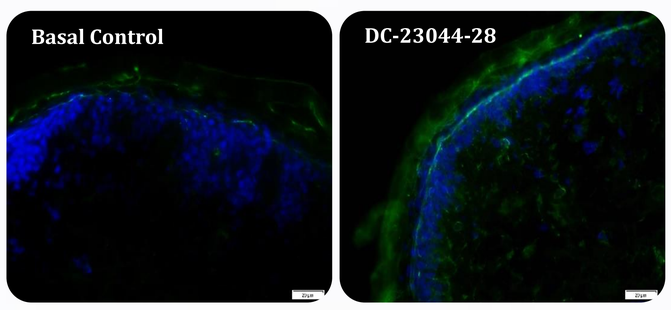


Figure 1: Effects of the evaluated product DC-23044-28 on IL-1α (left) and NF-κB (right) production in human skin culture exposed to inflammatory stress, quantified by ELISA. Data represent the mean ± standard deviation of 3 replicates (ANOVA, Bonferroni).

Filaggrin 42.47% ↑

Collagen I 5.81%





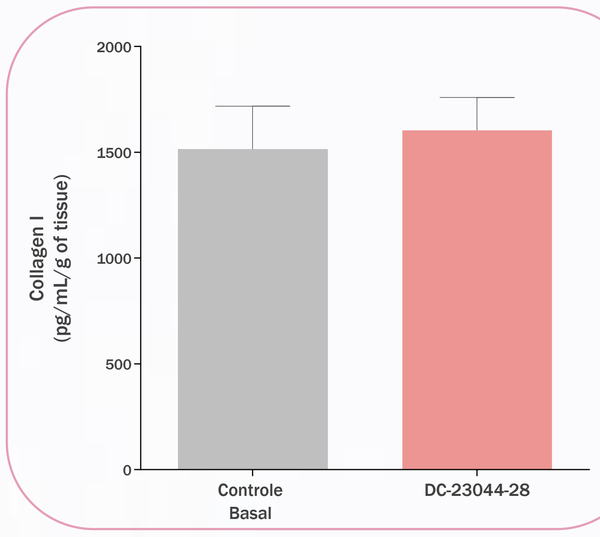


Figure 2: Effects of the evaluated product DC-23044-28 on filaggrin (left) and type I collagen (right) production in human skin culture, quantified by ELISA. Data represent the mean ± standard deviation of 3 replicates (Student's t-test).

DISCUSSION

The combined modulation of inflammatory markers and structural proteins indicates a dual mechanism of action. The reduction of IL-1α and NF-κB suggests attenuation of early inflammatory signaling, while the increase in filaggrin supports barrier reinforcement. These findings are particularly relevant for sensitive or environmentally stressed skin, where inflammation and barrier dysfunction coexist. In conclusion, the investigated formulation demonstrated significant anti-inflammatory activity and enhancement of epidermal barrier-related proteins in human skin ex vivo models. This integrated biological response supports its potential to improve skin comfort, resilience, and overall skin quality. Further clinical studies are warranted to confirm these effects in vivo.

REFERENCES

1. Isaac C, Ribeiro PMA, Ladeira SL, Conduta JRA, Tutihashi RMC, Ferreira MC. Alterações no processo de reparo fisiológico. Rev Bras Cir Plást. 2011;26(3):428–34.
2. Barnes JP, Adcock IM. Antiinflammatory actions os steroids: molecular mechanisms. Trends Pharmacol Sci 14;12, 436-441, 1993.
3. Blackwell TS, Christman JW. The role of nuclear fator kappa B in cytokines gene regulation. AM J Respi Cell Mol Biol 17:1, 3-9, 1997.
4. Christman JW, Lancaster LH, Blackwell TS. Nuclear Factor kappa B: a pivotal role in the systemic response syndrome and new target for therapy. Int Care Med 24:11, 1131-1138, 1998.
5. MerikaM, Williams AJ, Cheng G, Collins T, Thanos D. Recruitment of CBP/p300 by the IFNβ enhaceosome is required for synergistic activation of transcriptional. Mol Cell 1:2, 436- 441, 1993.
6. Dimitrakova E, Kostov I. Studies on the level of proinflammatory cytokines IIL-1a, IL-1b, IL, TNF-a in pregnant women with acute pyelonephritis. Akush Ginekol (Sofiia). 2011;50(2):3-6.

Contact Info

E-mail:
analucia@kosmoscience.com
barbara@kosmoscience.com

Website:
www.kosmoscience.com

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