

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

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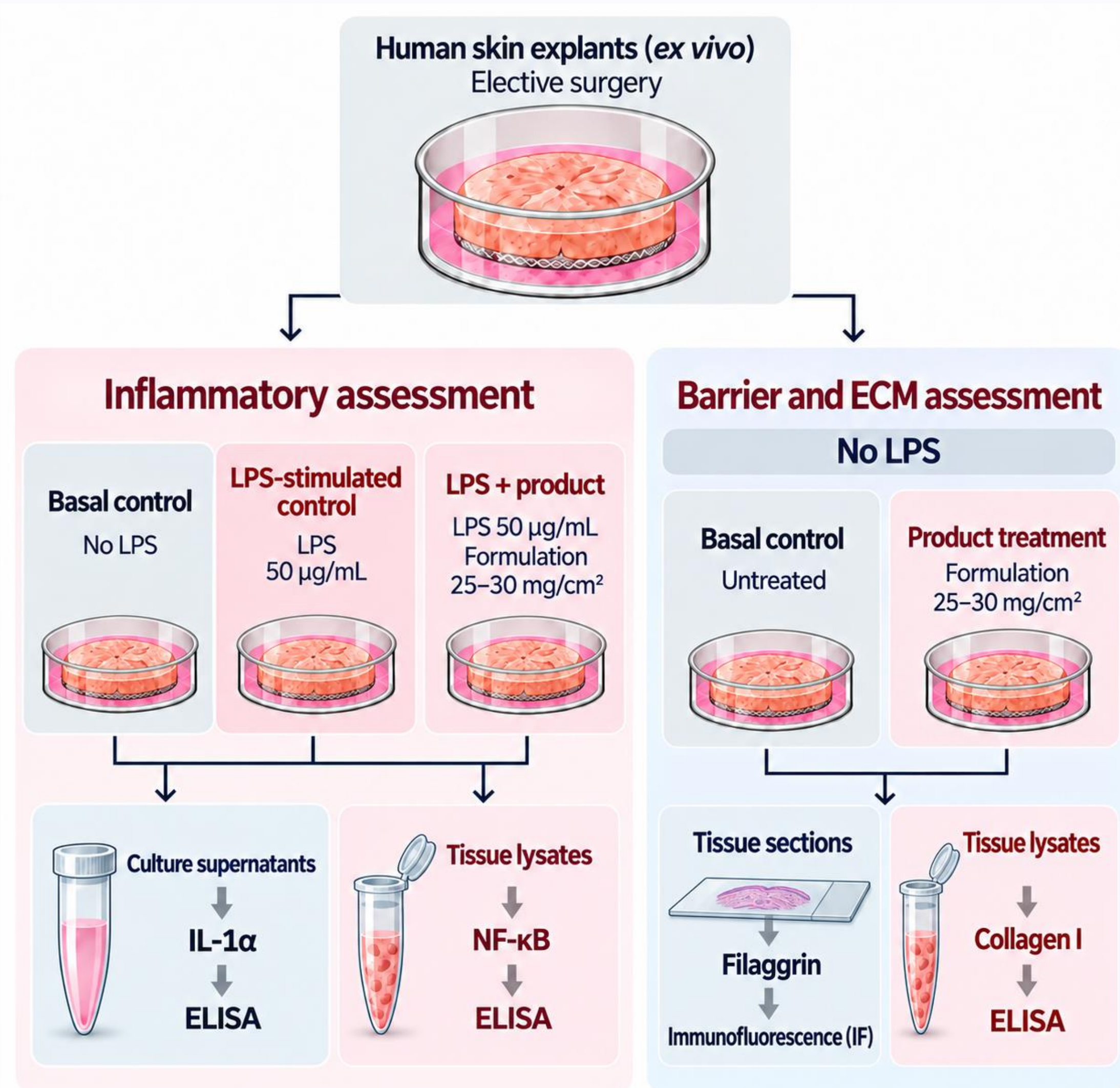
INTRODUCTION

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-1 α) levels and activating nuclear factor kappa B (NF- κ B). 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-1 α and NF- κ B), the barrier-related protein filaggrin, and collagen I using human skin *ex vivo* models.

MATERIALS AND METHODS



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RESULTS

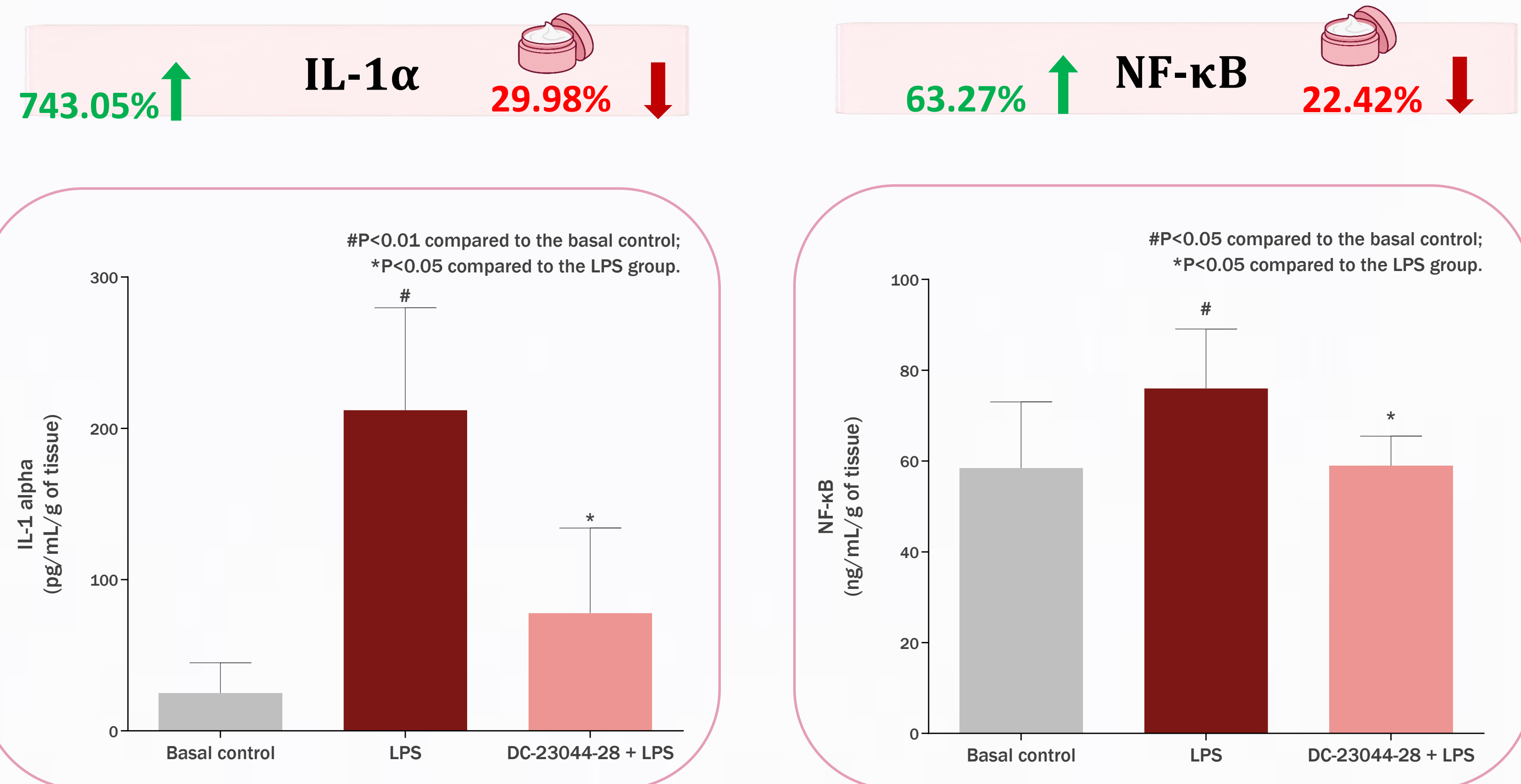


Figure 1: Effects of the evaluated product DC-23044-28 on IL-1 α (left) and NF- κ B (right) production in human skin culture exposed to inflammatory stress, quantified by ELISA. Data represent the mean $\pm$ standard deviation of 3 replicates (ANOVA, Bonferroni).

Filaggrin

42.47% ↑

Collagen I

5.81%

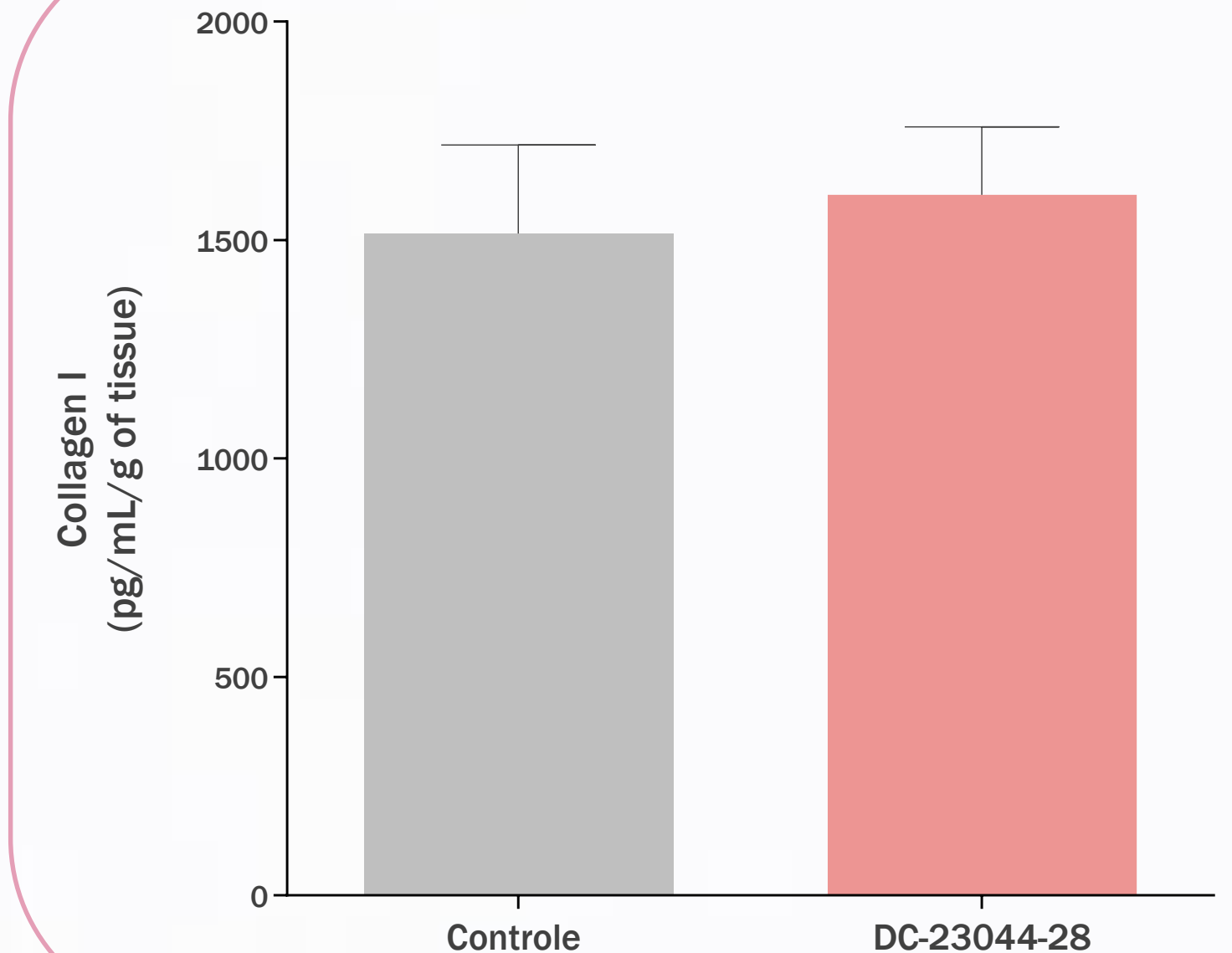
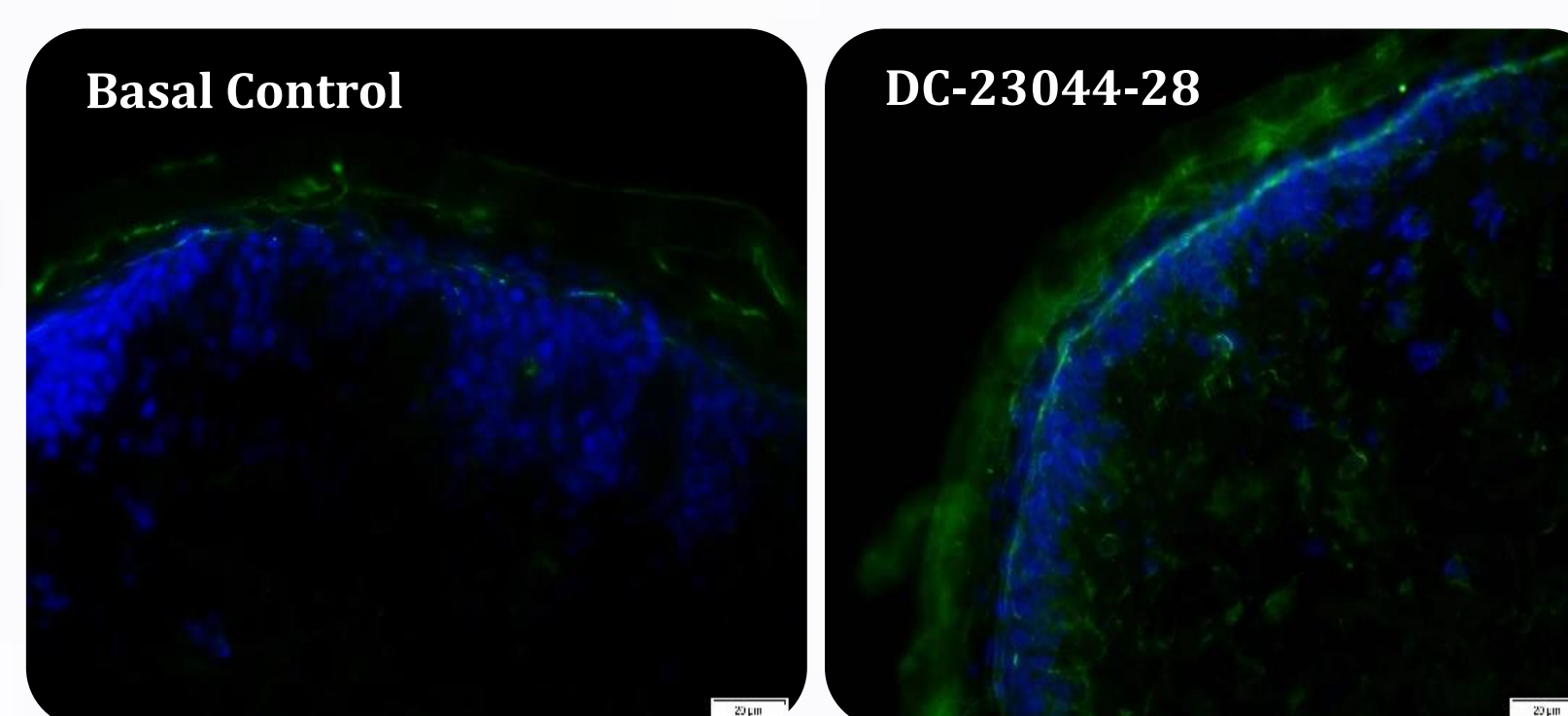
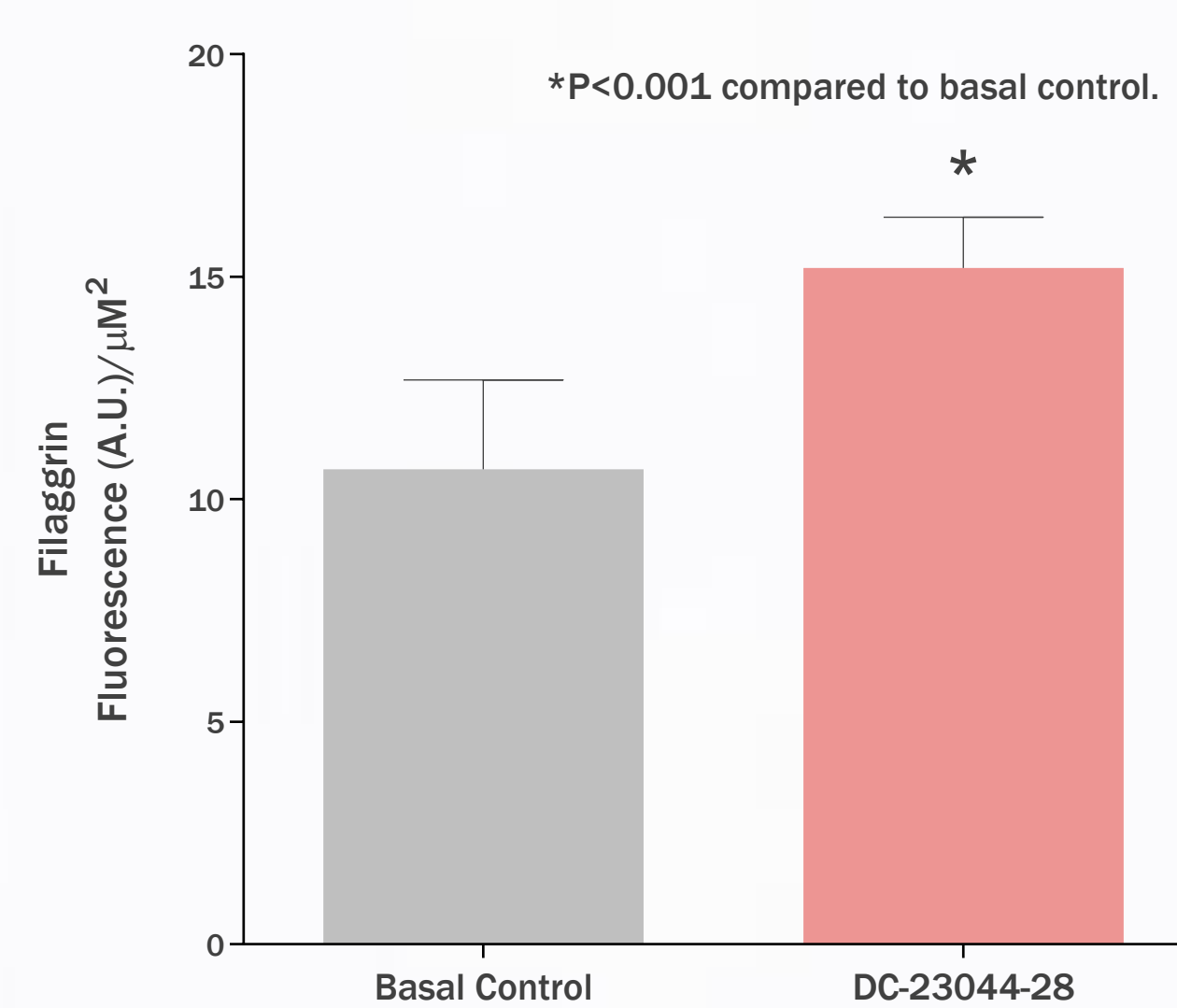
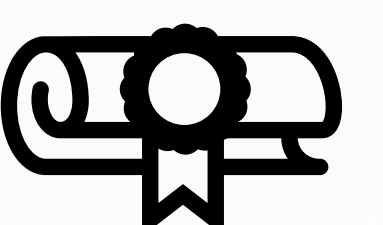


Figure 2: Effects of the evaluated product DC-23044-28 on filaggrin (left) and type I collagen (right) production in human skin culture, quantified by ELISA. Data represent the mean $\pm$ standard deviation of 3 replicates (Student's t-test).

DISCUSSION

The combined modulation of inflammatory markers and structural proteins indicates a dual mechanism of action. The reduction of IL-1 α and NF- κ B 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*.



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