PIGMENTATION

AN IN VIVO MODEL FOR POSTINFLAMMATORY HYPERPIGMENTATION

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Introduction: Post-inflammatory hyperpigmentation (PIH) is an acquired hypermelanosis. An in vivo model of acne PIH was previously established using 35% trichloroacetic acid (TCA).1

Objective: We aim to determine the minimum concentration required to cause PIH that closely resembles acne PIH.

Materials and Methods: Twenty-nine subjects (skin types I-VI) were enrolled; 20 had a history of PIH while 9 had a history of post-inflammatory erythema (PIE) upon acne resolution. At the initial visit, two acne papules were identified on the back and eight lesions were induced on the buttocks using TCA concentrations of 35%, 30%, 25%, and 20%. The acne and TCA-induced lesions were measured for erythema and pigmentation by Investigator's Global Assessment (IGA), clinical photography, and colorimetry at the initial visit as well as 24 hours, 7 days, 14 days, 28 days and 35 days after TCA application.

Results: IGA and colorimetry data for day 28 was analyzed with one-way repeated measures ANOVA. In the PIH group, there were no significant differences in IGA scores between acne, 25%, 30 % and 35% TCA-induced lesions. Higher concentrations were more likely to result in full thickness necrosis. Colorimetry L* parameter found no significant difference between acne, 30%, and 35% TCA-induced lesions. In the PIE group, there were no significant differences in IGA scores between acne, 20%, 25%, 30%, and 35% TCA-induced lesions. Colorimeter a* parameter demonstrated no significant differences between acne, 30%, and 35% TCA-induced lesions.

Conclusion: These results demonstrate that 30% TCA has the potential to induce PIH

similar to that of acne without causing necrosis. In addition, 30% and 35% TCA-induced erythema closely resembled acne-induced erythema. These results suggest that TCA-induced changes could serve as a model for the study of PIH and PIE.