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HS 3D-Seboskin model enables the preclinical exploration of therapeutic candidates for Hidradenitis Suppurativa/Acne Inversa

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Despite the rapid development in hidradenitis suppurativa (HS) research, the immediate introduction of potent therapeutic compounds in clinical trials and the lack of definitive outcome measures have led to the discontinuation of potential therapeutic compound studies. HS is a solely human disease, and therefore, the search for preclinical human models has been given priority. The 3D-SeboSkin model, a co-culture of human skin explants with human SZ95 sebocytes as a feeder layer, has been shown to prevent the rapid degeneration of human skin in culture and has been validated for HS preclinical studies. In this work, the HS 3D-SeboSkin model has been employed to characterize cellular and molecular effects of the EMA- and FDA-approved biologic adalimumab. Adalimumab, a tumor necrosis factor-α inhibitor, was shown to target inflammatory cells present in HS lesions, inducing a prominent anti-inflammatory response and contributing to tissue regeneration through a wound healing mechanism. Adalimumab inhibited the lesional tissue expression of TNF-α, IL-3, IL-15, and MCP-3 and downregulated the secretion of IL-1α, IL-5, RANTES, MCP-2, TNF-α, TNF-β, TGF-β, and IFN-γ. In contrast, IL-6 was stimulated. The compound failed to modify abnormal epithelial cell differentiation present in the HS lesions. Patients with Hurley stage II lesions exhibited stronger expression of autophagy proteins in perilesional than in lesional skin. Adalimumab modified the levels of the pro-apoptotic proteins LC3A, LC3B, and p62 in an individual, patient-dependent manner. Finally, adalimumab did not modify the NFκB signal proteins in SZ95 sebocytes and NHK-19 keratinocytes, used to study this specific pathway. The administration of the validated HS 3D-SeboSkin model in ex vivo studies prior to clinical trials could elucidate the individual pathogenetic targets of therapeutic candidates and, therefore, increase the success rates of clinical studies, minimizing HS drug development costs

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