The continuing evolution of targeted therapy for inflammatory skin disease

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Treatment of inflammatory skin disease has evolved from non-specific suppression of immune cells to increasingly precise targeting and modulation of immune mechanisms at all levels. This has led to dramatic treatment successes and deepened understanding of the pathophysiology. The cycle of in vitro studies, animal models, clinical trials, and case series of non-primary indications is a feedback loop that informs and guides the design of ever better disease models and therapeutic targets. Not only are we constantly discovering new molecules driving skin inflammation, we have also found that psoriasis and other autoimmune conditions are driven by distinct mediators occurring in early and late phases, which could be an opportunity for phase-specific or multipronged interventions. The deeper our mechanistic understanding, the more likely we will be able to discover subtle strategies to reprogram each patients' immune cells without having to dampen or eliminate their protective effects against pathogens and tumors. Lastly, ongoing genomic studies might soon confirm interesting genetic markers for predictive personalized medicine, the earliest currently being evaluated in psoriasis such as HLA-Cw6 and TNFAIP3. Taken together, the continued evolution of immune therapies in skin will potentially allow an unprecedented form of medicine that is not bent on silencing the pathogenic mechanism, but rather aims at using subtle interventions to shepherd the immune cell swarm back on the correct path.
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