The IL-36 pathway and generalized pustular psoriasis

The interleukin-36 (IL-36) pathway plays an important role in inflammation. IL-36 cytokines are expressed by, and act upon, various types of cells – such as keratinocytes, epithelial cells and immune cells – and work together in balance to regulate the inflammatory response.1

**IL-36 PATHWAY ACTIVATION**

IL-36 agonists bind to the IL-36 receptor to activate the pathway and stimulate the inflammatory response, including the recruitment and activation of immune cells.2–4

**IL-36 PATHWAY INHIBITION**

The IL-36 receptor antagonist (IL-36RA) binds to the IL-36 receptor to block signaling and suppress the inflammatory response.3,4

**DYSFUNCTION OF THE IL-36 PATHWAY: a key driver of generalized pustular psoriasis**

Uncontrolled inflammatory signaling, resulting from IL-36 receptor antagonist (IL-36RA) dysfunction or over-expression of IL-36 agonists can lead to autoinflammatory skin diseases, such as generalized pustular psoriasis (GPP).2,5,7–9

Boehringer Ingelheim’s randomized, placebo-controlled clinical trial program targeting the IL-36 pathway has advanced scientific knowledge in GPP.16

The Effisayil™ clinical trial program involves the largest and broadest patient population ever studied globally in GPP, leading to the first specific treatment approved for GPP flares across multiple countries and regions.16,17

**Definitions:**

- **Agonists:** molecules that bind to a receptor to activate a biological response
- **Antagonists:** molecules that bind to a receptor or a cytokine to block or inhibit a biological response
- **Cytokines:** molecules involved in cell signaling and the immune response

References:


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