Type 2 immunity evolved to ensure epithelial barrier integrity and protect against parasitic helminths and noxious environmental substances. When dysregulated, type 2 immunity becomes type 2 inflammation, which is a principal driving force of several inflammatory diseases, such as atopic dermatitis and asthma.

Immune dysregulation in such diseases is often highly complex and involves many different cell types and inflammatory mediators. However, clinical studies of targeted therapies suggest that only a few components play a clinically significant role.

**Biologics that inhibit type 2 molecules**
Targeting type 2 via IgE, cytokines and their receptors

**JAK inhibitors targeting JAK signaling pathways**
Simultaneous inhibition of type 1, 2, and 3 cytokines
Selectivity is dose-dependent

Studies of drugs targeting type 2 immune mediators helped clarify the biological mechanisms that underlie type 2 immunity and that provide therapeutic advances for type 2 inflammatory diseases.

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