body mounts an immune response against the body's own chromatin. In this response, the chromosomal proteins serve as the autoantigen, while the DNA component of the chromatin serves as an immune adjuvant that stimulates toll-like receptors, for example, TLR7 (18,19). Regarding T cells, in SLE it is the case that $CD8^+$ T cells lose their ability to kill target cells, because these T cells lack perforin. Because of the lack of perforin, SLE results in an increase in bacterial, viral, and fungal infections (20). The viral infections include those from Epstein–Barr virus and cytomegalovirus. Moreover, the $CD8^+$ T cells are less effective because they receive suboptimal activation from $CD4^+$ T cells (21). Regarding this point, in SLE $CD4^+$ T cells have reduced expression of IL-2. IL-2 is a cytokine that supports the differentiation and survival of $CD8^+$ T cells. FDA has provided guidance for trial design and endpoints for clinical trials on SLE (22).

4. *Asthma*

Asthma has an allergic component called "extrinsic asthma" and a nonallergic component called "intrinsic asthma" (23). The disease is characterized by a process called airway remodeling. Airway remodeling has the histological features of epithelial shedding, basement membrane thickening, smooth muscle hypertrophy, mucosal hyperplasia, and neovascularization. A number of autoantigens have been identified in asthma, but it is unclear how immune response against these autoantigens contributes to the pathology of the disease. These autoantigens include collagen V, bronchial epithelial cytokeratin, epithelial group factor receptor, activin A type 1 receptor, and alpha-catenin (24). The pathology of asthma is mediated by Th2-type cytokines, IL-4, IL-5, IL-9, and IL-13. The immune cells most responsible for the pathology of asthma are eosinophils. The following demonstrates that IL-5, IL-13, and IL-4, each contribute to the pathology of asthma. The contribution of IL-5 to asthma pathology is demonstrated by the fact that administering an anti-IL-5 antibody (mepolizumab) to patients with severe asthma results in reductions in eosinophil counts, as well as improvements in lung function, as measured by the forced expiratory volume (FEV_1) test (25). The role of IL-13 in the pathology of asthma is demonstrated by the fact that administering an

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