Natural killer cells can be activated or inhibited through receptors expressed on T cells that modulate T cell antigen receptor complex-dependent responses. One such receptor is NKG2D, which interacts with major histocompatibility complex class I (MICA) polypeptide A or B polypeptide ligands. Although these ligands are absent from most normal cells, they can be induced through stress (e.g., epithelial tumors, microbial infections, and certain autoimmune lesions). Drs. Spies and Groh have developed a method to prevent ERp5 modulation of immune cell function/development by inhibiting ERp5 function when expressed on immune cells. Targeting downregulation of ERp5 is therapeutically applicable to both autoimmune diseases as well as MIC-related cancers (e.g., those cancers associated with elevated levels of soluble MIC).

Applications
- Inflammatory disorders
- Autoimmune disease
- Tumors expressing membrane-bound MIC

Advantages
- Cell-specific targeting of activated T-cells, dendritic cells, and other immune cells
- Cell surface accessibility

Market Overview
In 2017, the global autoimmune disease therapeutics market was valued at USD 109.83 billion. However, with a CAGR of 4.2%, the market is projected to reach USD 153.32 billion by 2025. Several key factors are driving this growth, including wide availability of advanced therapeutics, a strong pipeline of late-stage drugs, and an increase in autoimmune disease prevalence (e.g., rheumatoid arthritis). Although the market has multiple segments, the anti-inflammatory category held the largest share in 2017 and is due to have the greatest CAGR.