DURING the past decade a wealth of information concerning the pathogenesis of Type I  
diabetes has become available. Two spontaneous animal models of the disease have been  
discovered and characterized (the Biobreeding rat and the non-obese diabetic mouse); the  
importance of a gene or genes in the major histocompatibility complex in Type I diabetes of  
human beings, of mice, and of rats has been appreciated; and the prognostic importance of  
selected assays for islet-cell antibodies has been defined. T-cell abnormalities that precede  
diabetes have been discovered. Evidence has suggested that progressive loss of first-phase  
insulin secretion precedes diabetes, and immunologic . . .