

Enhancing Theoretical Understanding of the Onset of Type 1 Diabetes

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Introduction

In type 1 diabetes, also known as autoimmune diabetes, the body’s own immune system attacks and destroys insulin-producing pancreatic beta cells, leading to an insulin shortage and causing symptoms [1]. Currently, the causes and cures are largely unknown [2]. However, previous experiments have shown that in NOD (non-obese diabetic) mice, a standard model for diabetic research, the level of T cells (a specific type of immune cell) fluctuates cyclically in the weeks leading up to the appearance of symptoms [[3] cited in [4]]. To better understand the mechanism underlying these oscillations, Mahaffy and Edelstein-Keshet constructed a mathematical model of the immune–pancreas system. One parameter in the model is the level of pancreatic beta cells, which slowly decreases over time as the disease progresses; at a certain level, the fluctuations described experimentally appear [4].

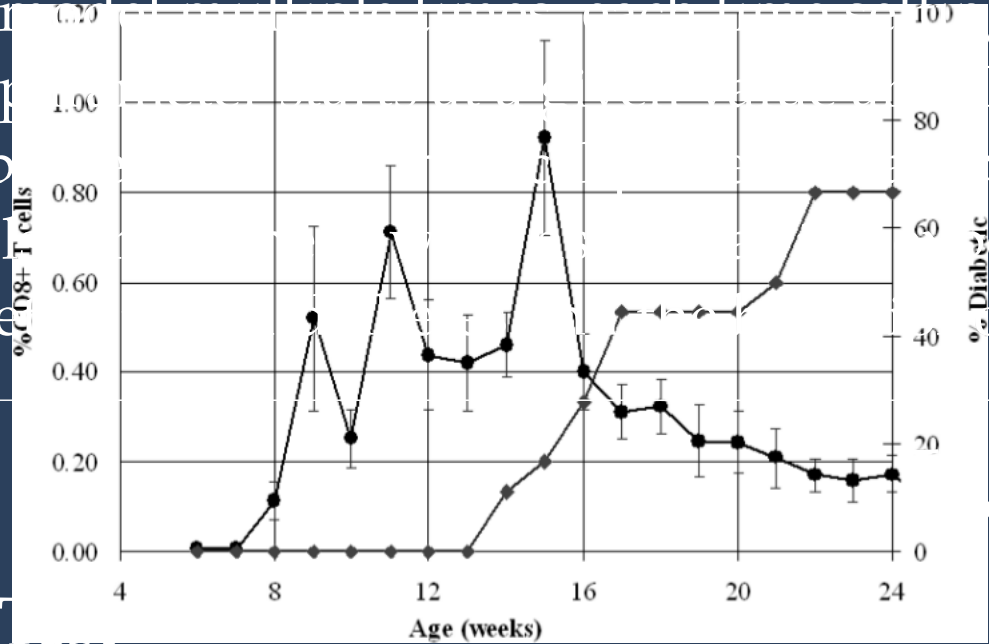
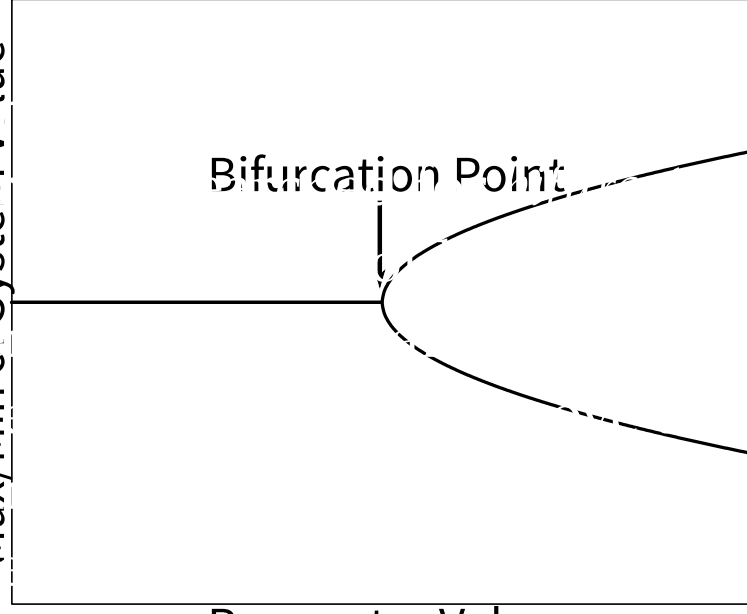
Original paper [4] studied the model’s behavior at various constant parameter values [4]; in particular, it showed that, as a parameter value changes, or qualitative changes in behavior that occur when a parameter reaches a certain value. For instance, a system may remain constant at one parameter value; if the parameter value changes, the system may then oscillate between two defined values (see diagram) [5]. This model was used to study the onset of type 1 diabetes by applying research demonstrating that in certain systems, slowly varying parameters can change the qualitative nature of the system [6]. This model was used to study the onset of type 1 diabetes, as the original paper explicitly stated that the parameter should continuously slowly fall. To summarize: for the original *static* analysis, the authors re-ran the model multiple times, each time setting the parameter to a fixed value. For the *continuous* analysis here, the parameter continuously decreases over time. Thus, this experiment will help us better understand and apply the theoretical results of Mahaffy and Edelstein-Keshet to the experimental results they cited in predicting and understanding the onset of the immune system in this disease.

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