

Preface

Type 1 diabetes



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Guest Editor

In *Brave New World*, Aldous Huxley envisioned in vitro fertilization and test tube babies as the norm for a society that had the technology to change the course of normal human biologic function. We who work in the field of type 1 diabetes mellitus hope to find similar scientific and technological means of fundamentally altering the course of this disease either by preventing it, reversing it, or finding new and better ways to treat it.

The field of type 1 diabetes mellitus has changed dramatically over the last 20 years and continues to change at a furious pace. Understanding of the autoimmune nature of this disorder is progressing on genetic and environmental fronts, allowing the identification of possible causative agents including enteroviral infections, antigens such as insulin, and other proteins that may influence the development of the T cell repertoire under the limitations imposed by the genetic background (eg, HLA) of patients. Animal models of disease have helped to define some of the pathways and the antigens that may underlie human disease and possible therapies that have been and will be used in immune intervention trials. Improvements in the understanding of T cell biology as well as improved technology are beginning to suggest ways of detecting primary reactivity to target antigens and the ability to monitor responses during immune intervention trials to change the course of patients with prediabetes, newly diagnosed disease, and those undergoing islet transplantation to reverse hyperglycemia.

The Edmonton protocol has proved that islet transplantation can be performed nearly as successfully as pancreas transplantation. Although a fantastic advance, as Drs. Sutherland, Gruessner, and Hering point out,

discovering modifications to this protocol that will result in immunologic tolerance could eventually make this a treatment for all with this disease. Finding new sources of islet tissue, whether from stem cells or other precursor cells, is crucial because even with successful islet transplantation regimens, there will not be enough pancreatic islets for all those who would need them.

Technologies to help monitor glucose continuously, whether noninvasive or invasive, along with insulin pump systems, offer the possibility for a closed-loop, artificial pancreas, which may offer a technological alternative to the immunologic therapies discussed above. Lastly, better treatments for those with type 1 diabetes mellitus have included recognizing concurrent illness, developing a better understanding of the mechanisms underlying diabetes complications, and developing new insulins and other biologic agents that will help to lower blood sugars, improve risk factors, and thus minimize future diabetes complications. Obviously, not all of these approaches and efforts will be wholly successful, but the scope and breadth of the work in the field and covered in this issue suggests important changes in how patients with type 1 diabetes mellitus will be treated and cared for in the future.

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