Response to Comment on Suri et al. on Diabetes Reversal in NOD Mice

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Faustman et al. present no new information to explain why three independent laboratories failed to reproduce their previous results implicating spleen cell transdifferentiation in the reversal of murine type 1 diabetes. Modulation of the immunological process in nonobese diabetic (NOD) mice has been accomplished by many laboratories using different protocols and does not represent a novel finding in their work.

The major finding of Kodama et al. (1) that donor spleen cells can give rise to new β cells, should not be confused with the reversal of diabetes in NOD mice after treatment with adjuvant and allogeneic cells. Many experiments in the past 15 years have shown the great susceptibility of the NOD mouse to interventions that prevent or reverse the disease (7). For example, an elegant report by Chatenoud et al. showed that treatment of newly diabetic NOD mice with monoclonal antibody to CD3 resulted in permanent reversal of the disease (8). Indeed immunomodulation, including using Freund’s adjuvant, arrests an active diabetic process in the mouse (9–11). Although we remain puzzled by the differences between our results and those in (1), we are reassured by the findings of two other laboratories of the reversal of autoimmune diabetes in the absence of splenocyte-derived differentiation of new β cells (2, 3). We hope these points clarify the scientific questions raised and answered in our studies (2–4) so that the field continues to move forward.

References and Notes

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