in a rheumatic disease which has proved resistant to all forms of treatment. Indeed my colleagues and I instituted a similar trial of anti-thymocyte in combination with other immunosuppressive drugs which was reported in a conference proceedings [2]. However, the initial improvement was not sustained in a longer follow-up than the original mean of 3.7 yr. We also encountered a high incidence of serum sickness induced by anti-thymocyte globulin and one patient (not included in the report) died from an acute renal crisis during the initial period of steroid treatment. Perhaps the most interesting outcome of such trials is the poor response of scleroderma to intensive immunosuppression compared with other ‘autoimmune’ rheumatic diseases, thereby raising questions about current concepts of immune mechanisms in scleroderma.

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Reply

We appreciate Dr Denman’s comments about our study of immunosuppressive therapy in diffuse scleroderma, and the reference to his earlier work. We would agree that the effects of strong immunosuppression in scleroderma are disappointing compared with the effects seen in other rheumatic diseases. The evidence supporting a role for autoimmune mechanisms contributing to this condition is, however, quite strong. One possibility would be that the autoimmune process is important in the initiation of the disease, but by the time there is extensive skin and organ fibrosis a great deal of irreversible damage has occurred, so that immunosuppression has only a modest effect at this stage. The situation in type I diabetes could be analogous where there is unquestionable evidence of an autoimmune pathogenesis, but immunosuppressive therapy is futile by the time islet cell destruction and insulin deficiency has developed.

We would therefore like to emphasize the importance of early diagnosis and prompt treatment with appropriately targeted therapy.

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