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autoimmunity prevalence in CME carriers with that of type 1 diabetic patients; and (ii) a prospective, long-term follow-up study of islet autoimmunity in CME carriers over several years, and (iii) a follow-up study to determine whether this putative association is related to the presence of antibodies directed to beta-cell antigens. Another consideration for this study was the influence of the antibodies on disease progression. Within one year of testing, 2 carriers of type 1 diabetes developed autoantibodies and progressed to diabetes. In general, the presence of antibodies directed to GAD or insulin do not necessarily predict progression of disease in type 1 diabetes ([@B36]). However, a prospective study performed in the U.K. examined islet antibodies of CME carriers, and antibody status was reported in conjunction with diabetes status. Of the three CME carriers with diabetes, two had antibodies, but none developed diabetes for longer than 1 year. The mean follow-up period was also shorter than in our study, and islet autoantibody levels increased over time. The third CME carrier did not have antibodies,

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developed diabetes after 1 year of follow-up, and antibodies disappeared after 2 years of follow-up ([@B37]). Another cross-sectional study in the U.S. compared CME carriers with type 1 diabetic patients and found that the presence of antibodies predicted future diabetes in CME carriers more than two years from the baseline autoantibody testing ([@B9]). To be certain of the accuracy of the diagnosis of type 1 diabetes among study participants with anti-GAD antibodies, we verified positive test results by RIA. We found that the levels of anti-GAD antibodies by RIA were strongly correlated with the levels of anti-GAD antibodies by EIA. It is not known which assay is more precise. The EIA is less specific but gives a more quantitative reading of the level of autoantibodies. In a study of a single islet antibody (IgG to the 64-kD protein of the insulinoma antigen 2), antibodies

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