Type 1 diabetes (T1D) is the result of the cell mediated destruction of pancreatic beta cells. The presence of autoantigen-specific autoantibodies is considered to be rather an epiphenomenon but in contrast to cell reactivity autoantibodies can be routinely detected. We analyzed 40 T1D patients and 11 healthy control. The relationship between the patient’s autoantibody profile and peripheral blood mononuclear cell (PBMC) cytokine production was examined. Concretely the production of IL-17 and IFN-gamma following stimulation with three different concentrations of autoantigens GAD65 and IA2 was detected by ELISPOT. The ratio of basic cellular populations in PBMCs was measured by flow cytometry. Using the generalised linear mixed statistical model, we demonstrated a significant interaction between the patient’s autoantibody profile and mode of stimulation (p<0.001 for both measured cytokines). An increased production of IL17 was found in patients with high IA2 autoantibodies compared to patients with low levels of autoantibodies (p=0.005) and healthy controls (p<0.001) regardless of the mode of stimulation. The titre of IA2 autoantibodies positively correlates with the proportion of Tc (p<0.001) and negatively with Th (p=0.016) lymphocytes so the high titre of IA2 autoantibodies was related to increased production of IL17 and an increased proportion of Tc lymphocytes. Our results show that a patient’s autoantibody profile reflects the type of her/his cellular immune response which can have a potential practical impact. Supported by national projects: MZCR through the conceptual development of research organisation 00064203 (Motol University Hospital, Prague), IPL 699 001, IGA MZCR NT/11407-5 and GACR P302/10/1679.