Electronic supplementary material
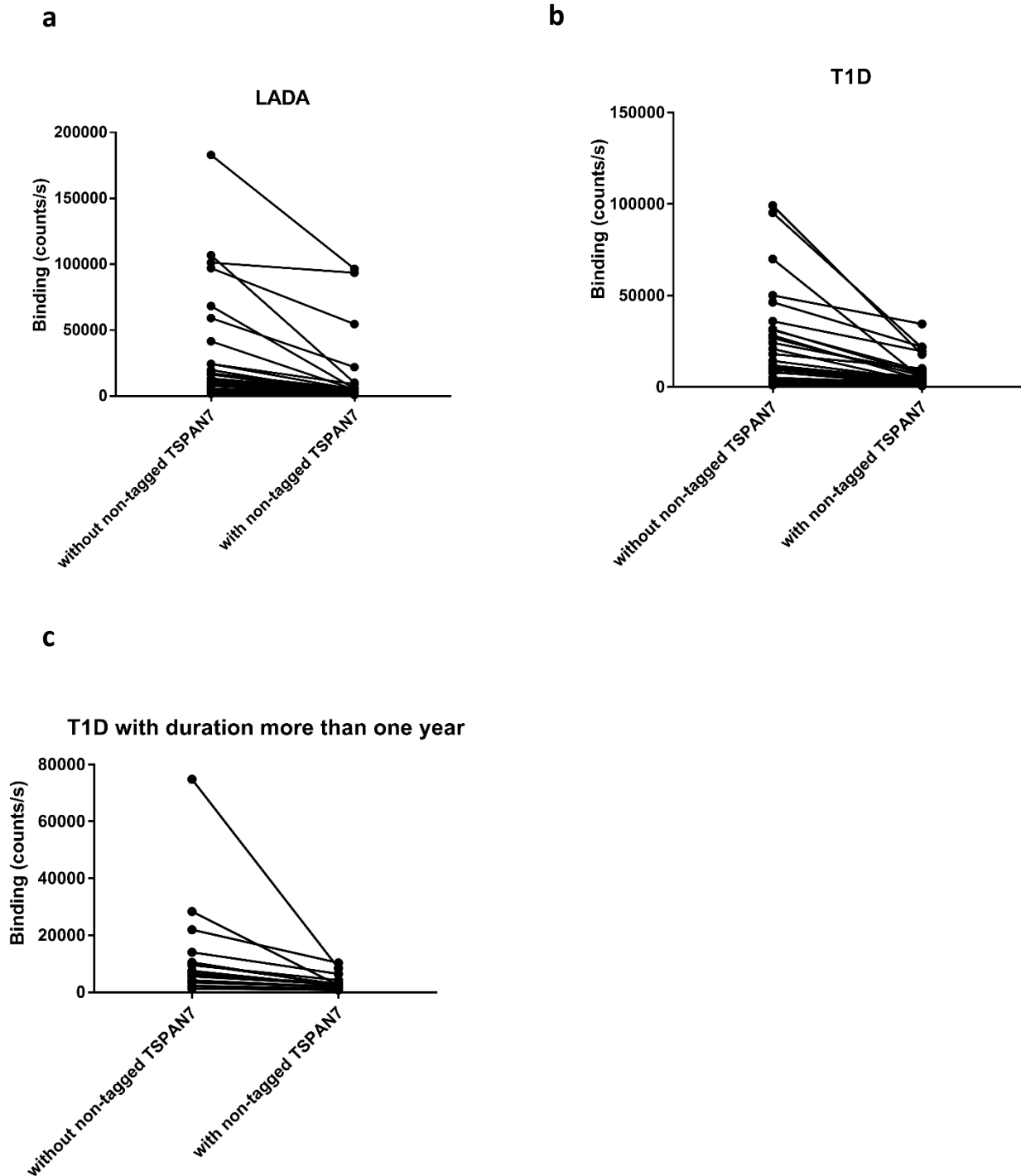
ESM Table 1 Baseline data of individuals with LADA

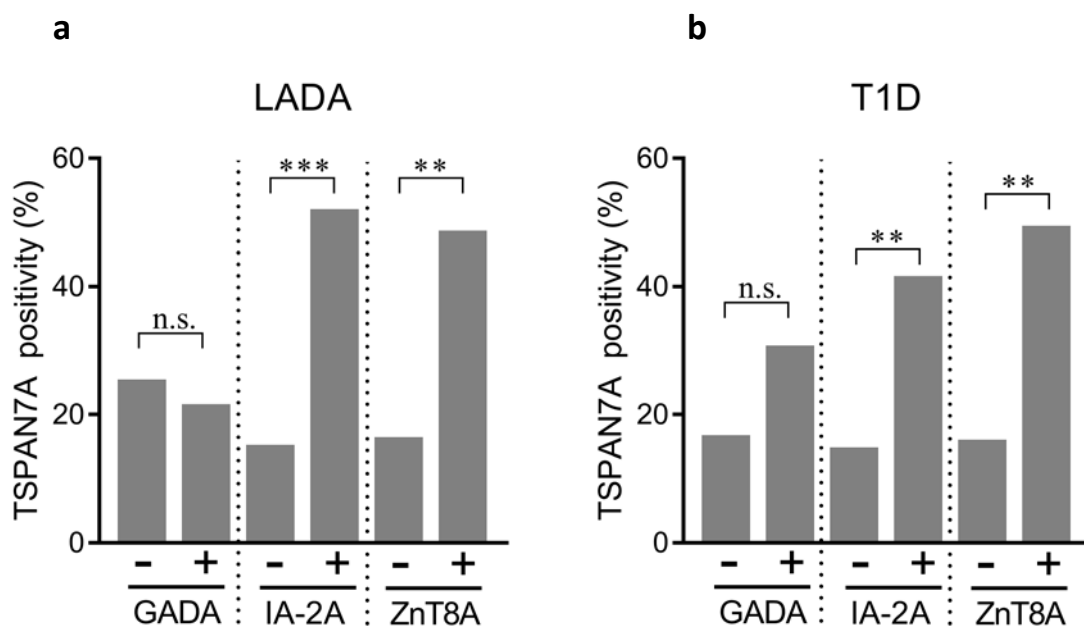|	LADA without TSPAN7A (n=41)	LADA with TSPAN7A (n=12)
Male sex, % (n)	57.1 (24)	50 (6)
Age, years	47.8±12.4	46.7±10.3
Duration, months	6 (2.5-20)	8 (2-12.8)
BMI, kg/m^2	24±3.6	22.9±2.3
GADA titer, IU/mL	125.8 (52.2-689.5)	432.1 (34.5-822.4)

Data are expressed as % (n), mean±SD, and median (25th–75th percentile). LADA: latent autoimmune diabetes in adults; BMI: body mass index; GADA: glutamic acid decarboxylase antibodies. GADA, glutamic acid decarboxylase antibodies; TSPAN7A, tetraspanin 7 autoantibodies.

ESM Fig. 1

a

b

c

ESM Fig.1 Competition assay in the absence versus presence of non-tagged TSPAN7 in the LIPS assay. Each line represents the change in CPS caused by the addition of non-tagged TSPAN7 of each person. TSPAN7A, tetraspanin 7 antibodies; LIPS, luciferase immunoprecipitation system assay; T1D: type 1 diabetes; LADA: latent autoimmune diabetes in adults;

ESM Fig. 2

a

b

ESM Fig.2 TSPAN7A prevalence are associated with IA-2A and ZnT8A in LADA (a; n=173) and T1D (b; n=158), but not with GADA. In LADA: 21.2% vs 25% TSPAN7A positive in GADA-pos and GADA-neg individuals, respectively, $p>0.05$; 51.6% vs 14.8% TSPAN7A positive in IA-2A-pos and IA-2A-neg individuals, respectively, $p<0.001$; 48.3% vs 16% TSPAN7A positive in ZnT8A-pos and ZnT8A-neg patients, respectively, $p<0.01$; In T1D: 30.3% vs 16.3% TSPAN7A positive in GADA-pos and GADA-neg individuals, respectively, $p>0.05$; 41.2% vs 14.4% TSPAN7A positive in IA-2A-pos and IA-2A-neg individuals, respectively, $p<0.01$; 49% vs 15.6% TSPAN7A positive in ZnT8A-pos and ZnT8A-neg patients, respectively, $p<0.01$; LADA: Latent autoimmune diabetes in adults; T1D: type 1 diabetes; GADA: glutamic acid decarboxylase antibodies; IA-2A: protein tyrosine phosphatase autoantibodies; ZnT8A: zinc transporter 8 antibodies; TSPAN7A: tetraspanin 7 antibodies; n.s.: not significant; vs versus. **$p<0.01$, ***$p<0.001$.