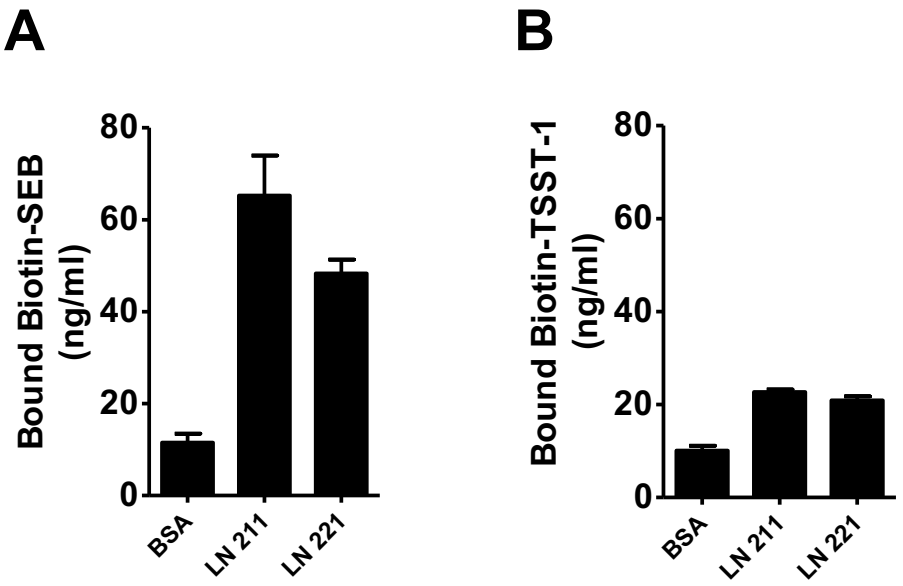

Figure S1

Figure S1. Direct binding of staphylococcal superantigens laminins.

Binding of biotinylated SEB (A) and biotinylated TSST-1 (B) to coated BSA, laminin 211 (LN211), and laminin 221 (LN221), as detected and quantified by ELISA. Results (mean ± standard deviation of triplicates) are representative of two independent experiments.

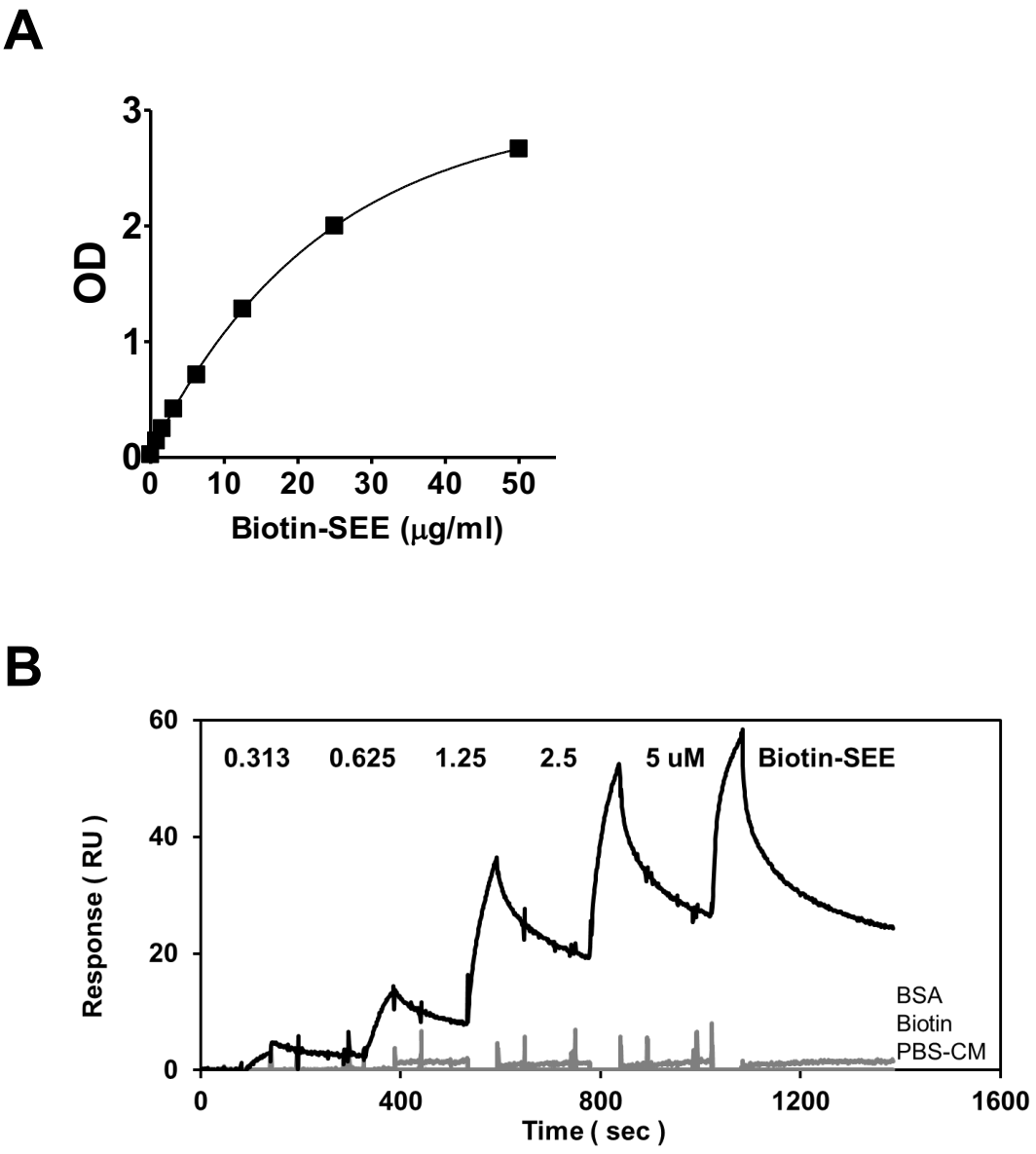

Figure S2

Figure S2. Saturable, dose-dependent binding between biotin-SEE and laminin LN221.

Technically distinct ELISA (A) and SPR (B) assays consistently show that the dose-dependent binding of biotin-SEE to immobilized LN221 approached saturation around 50 μg/mL (equivalent to 2.5 μM biotin-SEE). Under similar SPR conditions, no significant binding was observed with BSA (0 – 5 μM) or free biotin (0 – 100 μM).

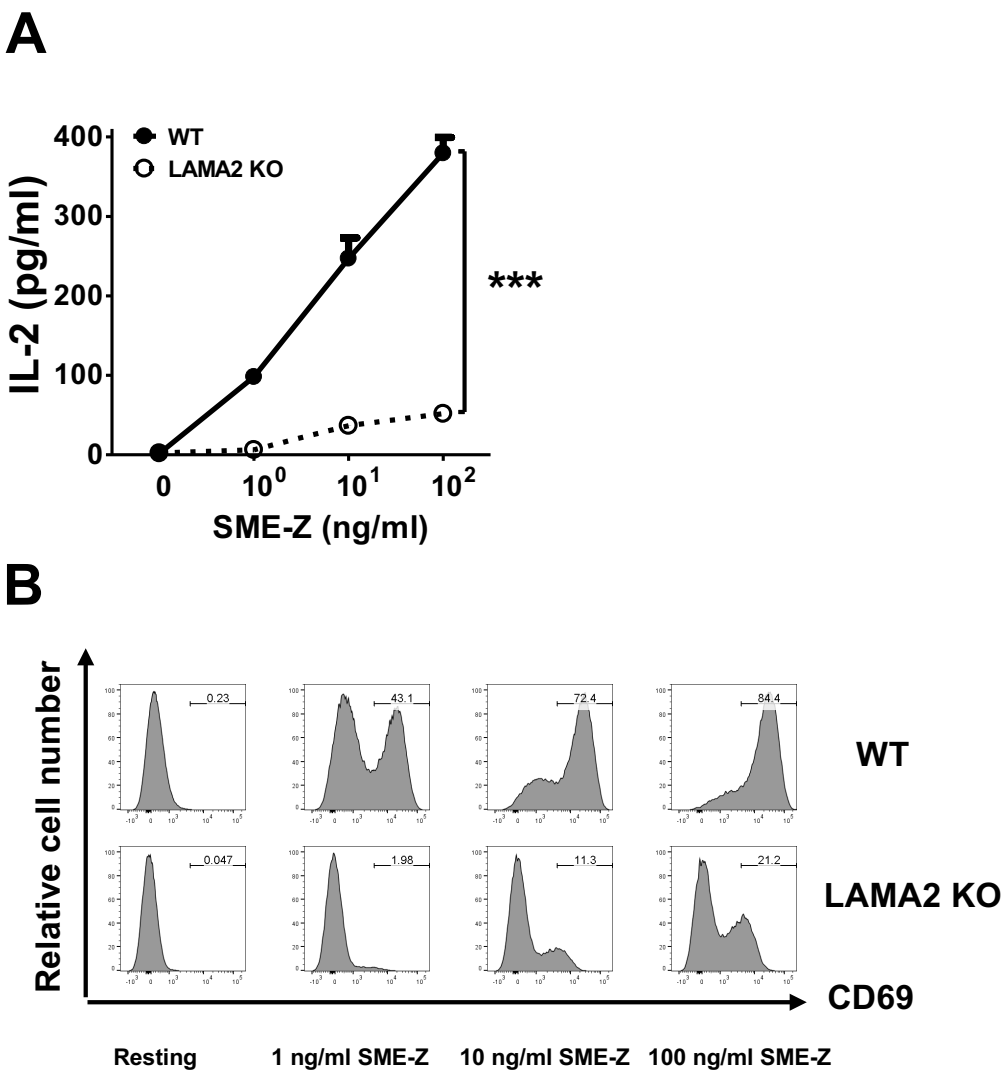

Figure S3

Figure S3. LAMA2 is involved in SME-Z-induced T cell activation in LCK-deficient T cells.

(A) Quantification of IL-2 in culture supernatants of wild-type and LAMA2 knockout JCaM1.6 T cells stimulated with LG2 cells and indicated concentrations of SME-Z. Results (mean ± standard deviation of triplicates) are representative of three independent experiments. (B) JCaM1.6 T cells stimulated as of (A) were subjected to flow cytometry analysis using Alexa Fluor 647-labelled anti-CD69 antibody as a marker of T cell activation. Data are representative of two independent experiments. Statistical analysis was performed using the two-way ANOVA. ***: $p<0.001$.

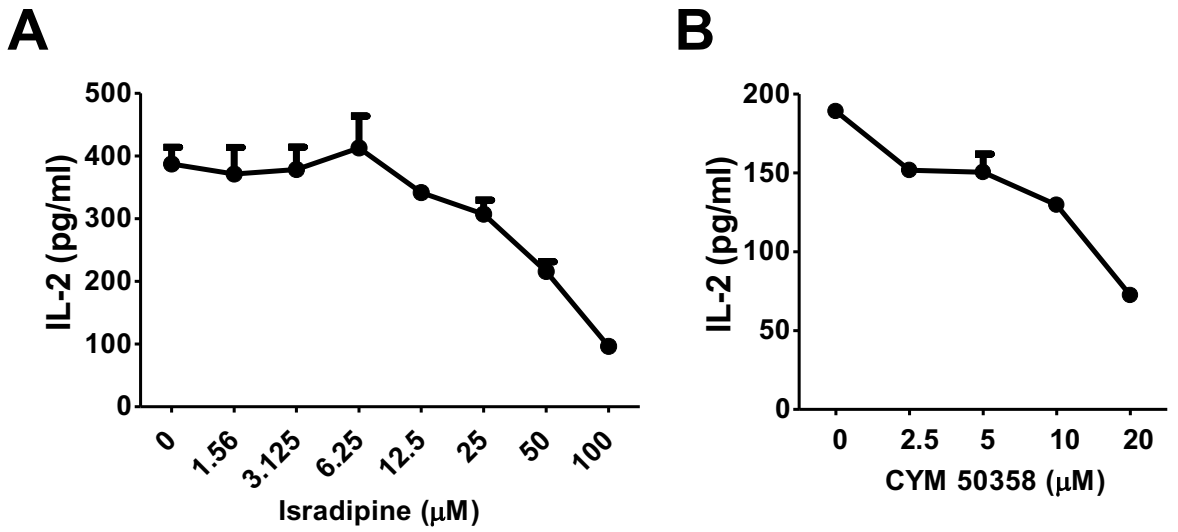

Figure S4

Figure S4. VGCC antagonist and S1PR4 antagonist down-regulate T activation in LCK-deficient T cells responding to superantigens. IL-2 accumulation in cultured supernatants of JCaM1.6 T cells and LG2 cells stimulated with 1 ng/ml of SEE for 8 h in the presence or absence of indicated concentration of israpidine (VGCC antagonist) (A) or CYM 50358 (S1PR4 antagonist) (B). Results (mean ± standard deviation of triplicates) are representative of two independent experiments.