



**Figure S1. Treatment efficiency of IVIg in a patient with pemphigus vulgaris.** Flaccid blisters formation and erosions of the tongue prior (A, B) and 7 month after IVIg treatment (C, D).

**A**

Group	number of IVIg applications	total amount of IVIg applied	length of experiment (days)	days between last treatment and analysis
A1	3	6 mg	21	5
A2	4	8 mg	35	12
A3	4	8 mg	70	47
B1	1	2 mg	35	5
B2	5	10 mg	63	5
B3	5	10 mg	77	18

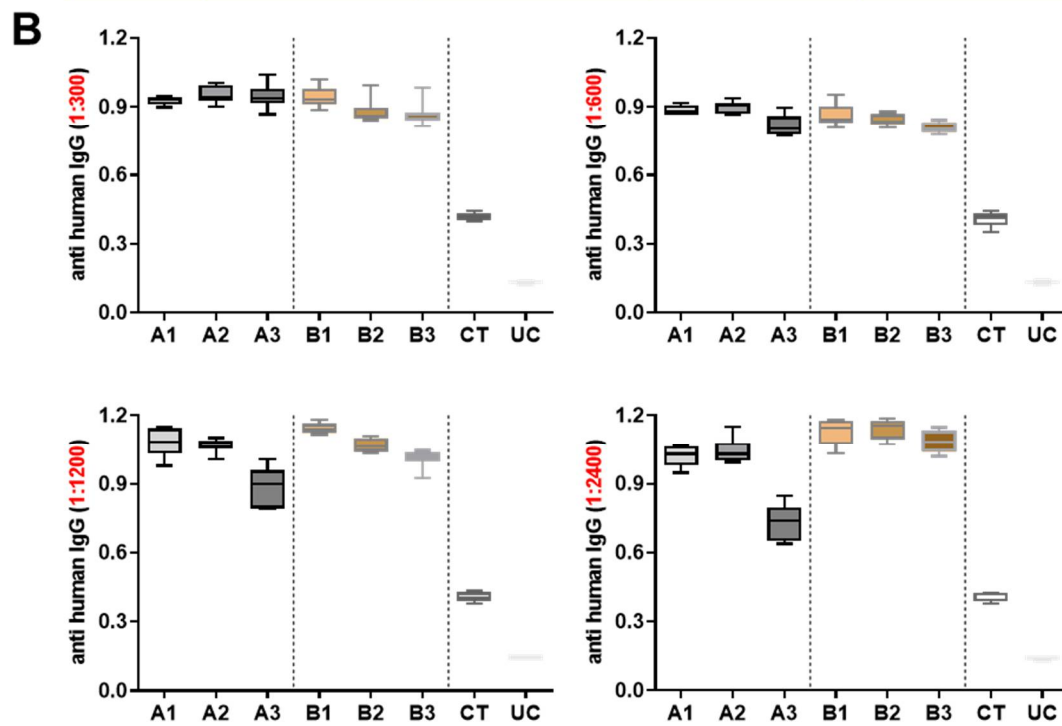
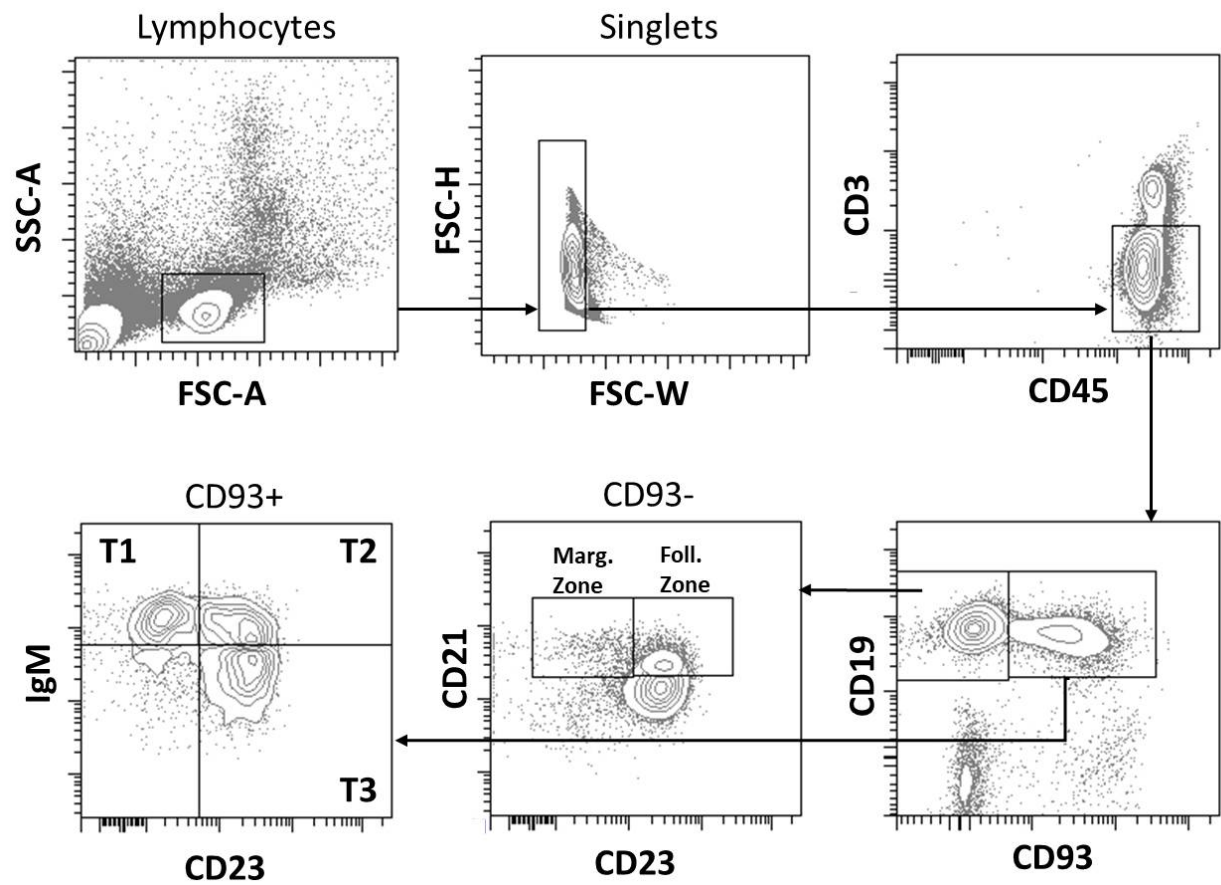


Figure S2. Summary of accumulative IVIg load in respective treatment groups and human IgG quantification by ELISA. (A) Group comparison regarding IVIg treatment. (B) Quantification of human IgG by ELISA in sera of treated mice at indicated dilutions displayed at OD<sub>405</sub>. Error bars in graphs represent standard deviation, mean  $\pm$ SD. n=6-8 mice per group. PBS immunized samples, control (CT). uncoated wells (UC).



**Figure S3.** Representative display of B cell gating strategy for the detection of murine splenic B cell subsets. Marg. Zone, marginal zone B cells; Foll. Zone, follicular zone B cells.

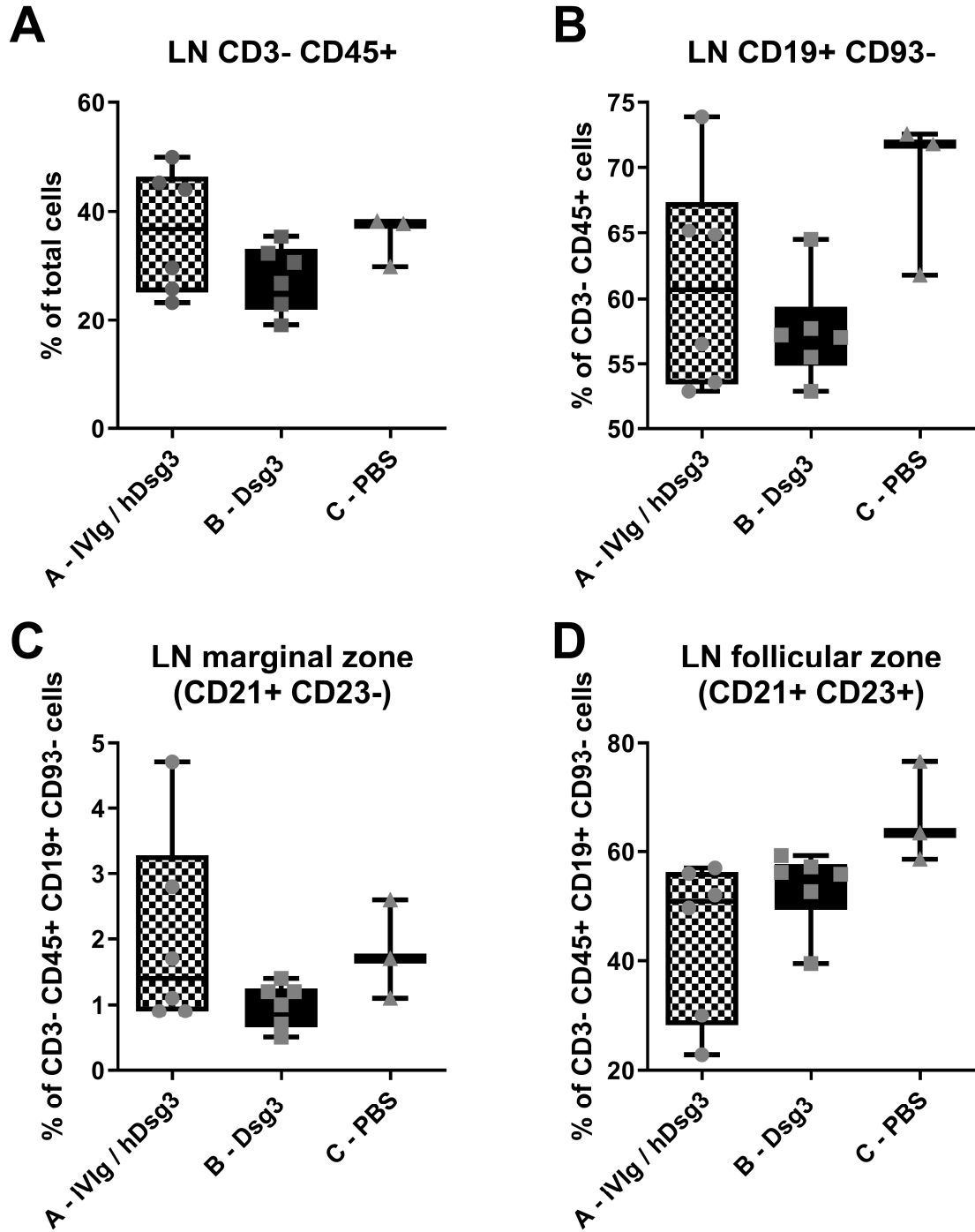


Figure S4. Representative display of experimental B cell subset analysis. Parent B cell population (CD3- CD45+ CD19+ CD93-) (A, B) was further stratified into marginal zone B cells (C) and follicular zone B cells (D). LN, lymph nodes. n=3 (PBS) - 6 mice per group.