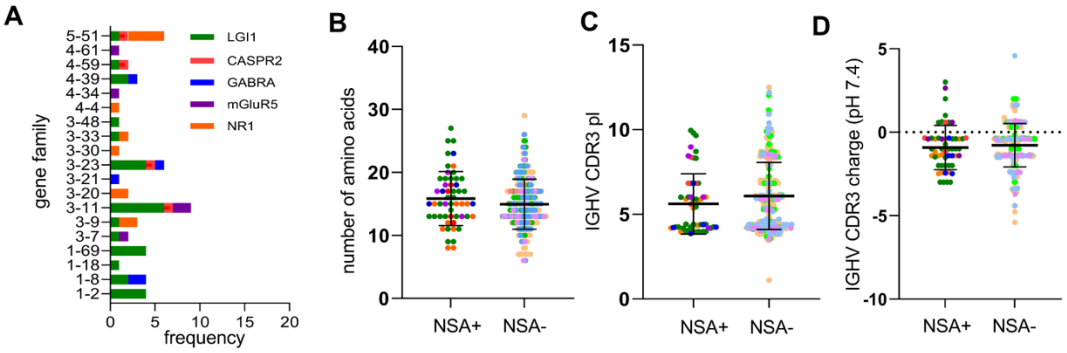
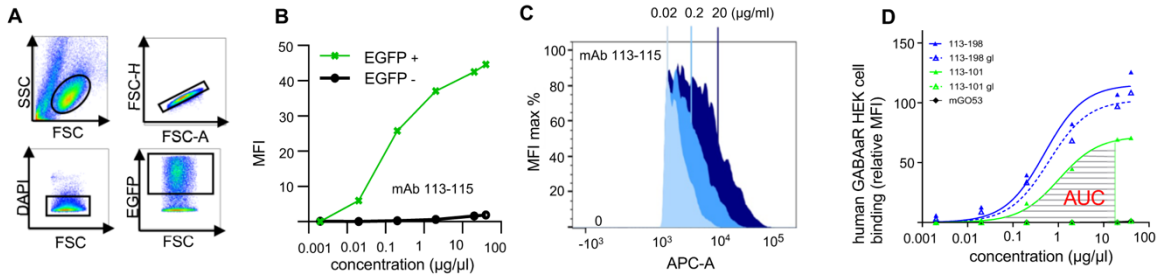
eFigures

eFigure 1



Characterization of mAbs. IGHV gene family accumulation was not observed in the different encephalitis subgroups (**A**). Characteristics of mAb CDR3 regions such as IGHV CDR3 length (**B**), isoelectric point (pI) (**C**) and charge at pH 7.4 (**D**) showed no differences between NSA+ and NSA- mAbs. Each point represents one mAb.

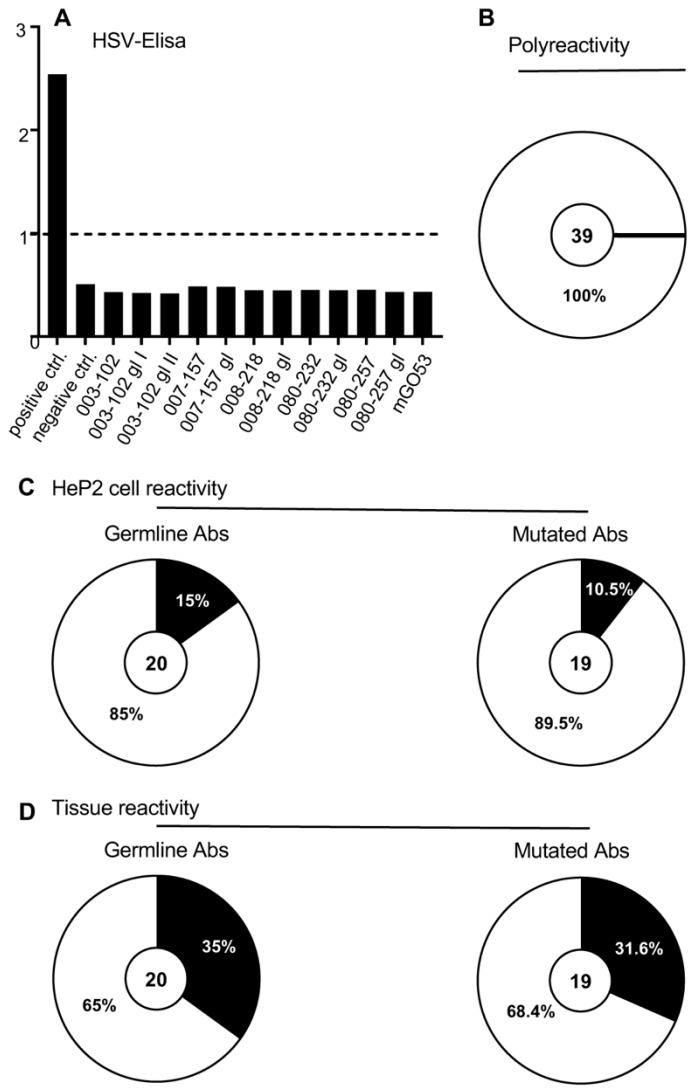
eFigure 2



Quantification of antibody binding strength. FACS gating strategy included selection of the respective HEK cell populations by size (FSC) and granularity (SSC), duplets (FSC-A vs FSC-H) as well as dead cells (DAPI) were excluded and MFI of EGFP+ (co-transfected agent) was measured (**A**). The signal of co-transfected EGFP correlated with the NSA expression, exemplarily depicted for $GABA_AR$ expression. Raised primary concentrations (113-115) led to

strong increase of secondary antibody signal within EGFP+ but not EGFP- cells (**B**). The concentration-dependent right shift of secondary fluorescence intensity is shown for $GABA_AR$-reactive mAb 113-115 (vertical lines representing MFI) (**C**). Nonlinear regression models for one site specific binding of concentration-dependent MFI values were used to model binding, here shown by representative binding curves of $GABA_AR$ reactive mAbs 113-101 and 113-198 and their reverted counterparts (**D**). FSC = forward scatter; SSC = side scatter.

eFigure 3



Cross-reactivities of mAbs. NMDAR-reactive maturated and germline mAbs did not react with HSV in an HSV ELISA (**A)**, horizontal dotted line displays the threshold for a positive result.

None of the mAbs showed polyreactivity against selected autoantigens (**B**), several mutated and germline mAbs reacted with HEp-2 cells (**C**) and different types of murine tissue (**D)**, percentages of reactive mAbs are marked black in the pie charts.