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SUPPLEMENTARY INFORMATION

Somatostatin slows Aβ plaque deposition in aged *APP*$^{NL\text{-}F/NL\text{-}F}$ mice by blocking Aβ aggregation

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Supplementary Figure 1. Indel sequence of *Sst*$^{-/-}$ allele determined by Sanger sequencing.

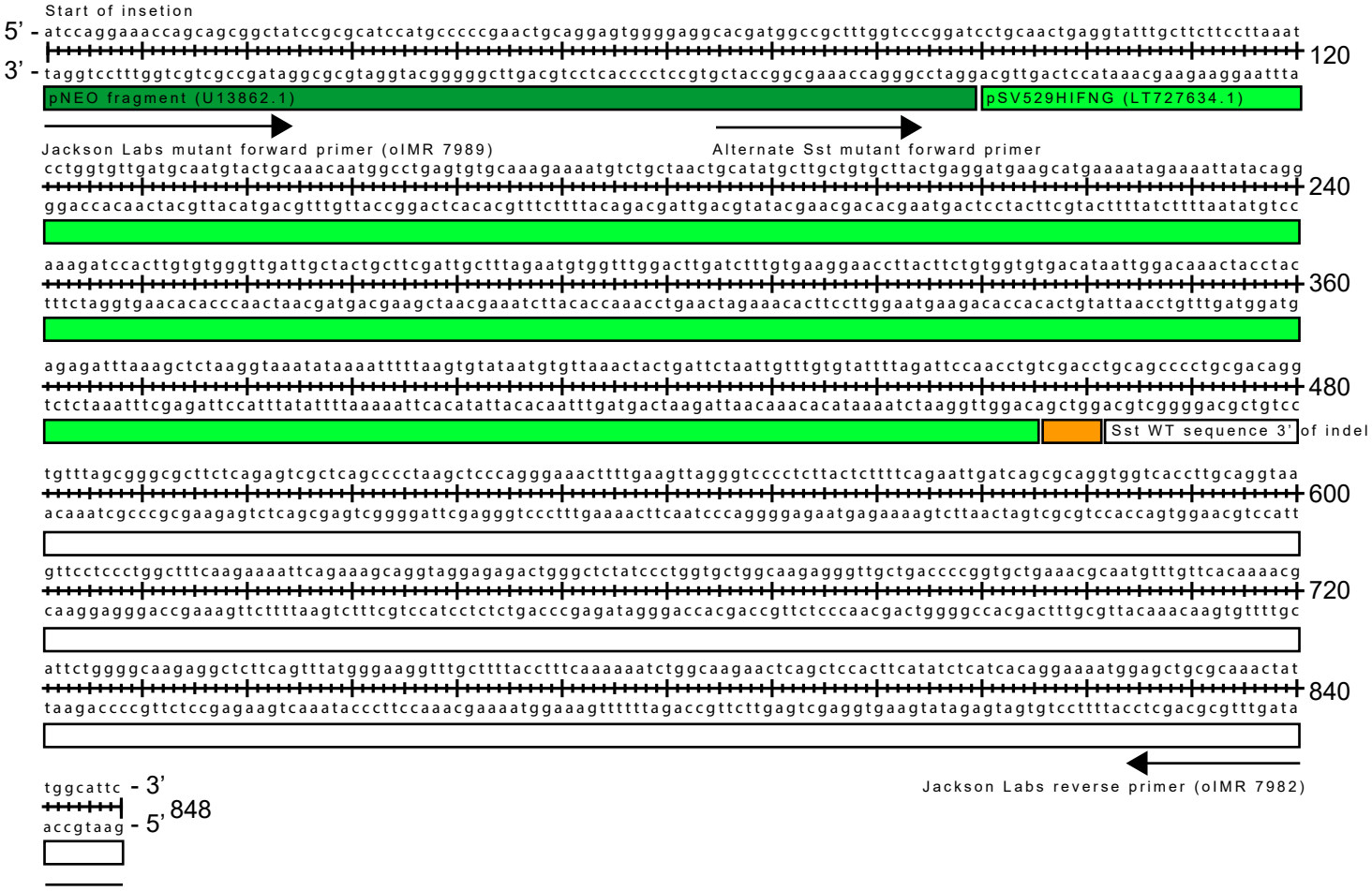
In addition to depicting the indel, the image shows the position of forward and reverse primers used in this study for genotyping.

Supplementary Figure 2. Hippocampal Aβ amyloid plaque counts in 12- or 15-month-old *App*$^{NL\text{-}F/NL\text{-}F}$ mice that express wild-type Sst levels or were Sst-deficient.

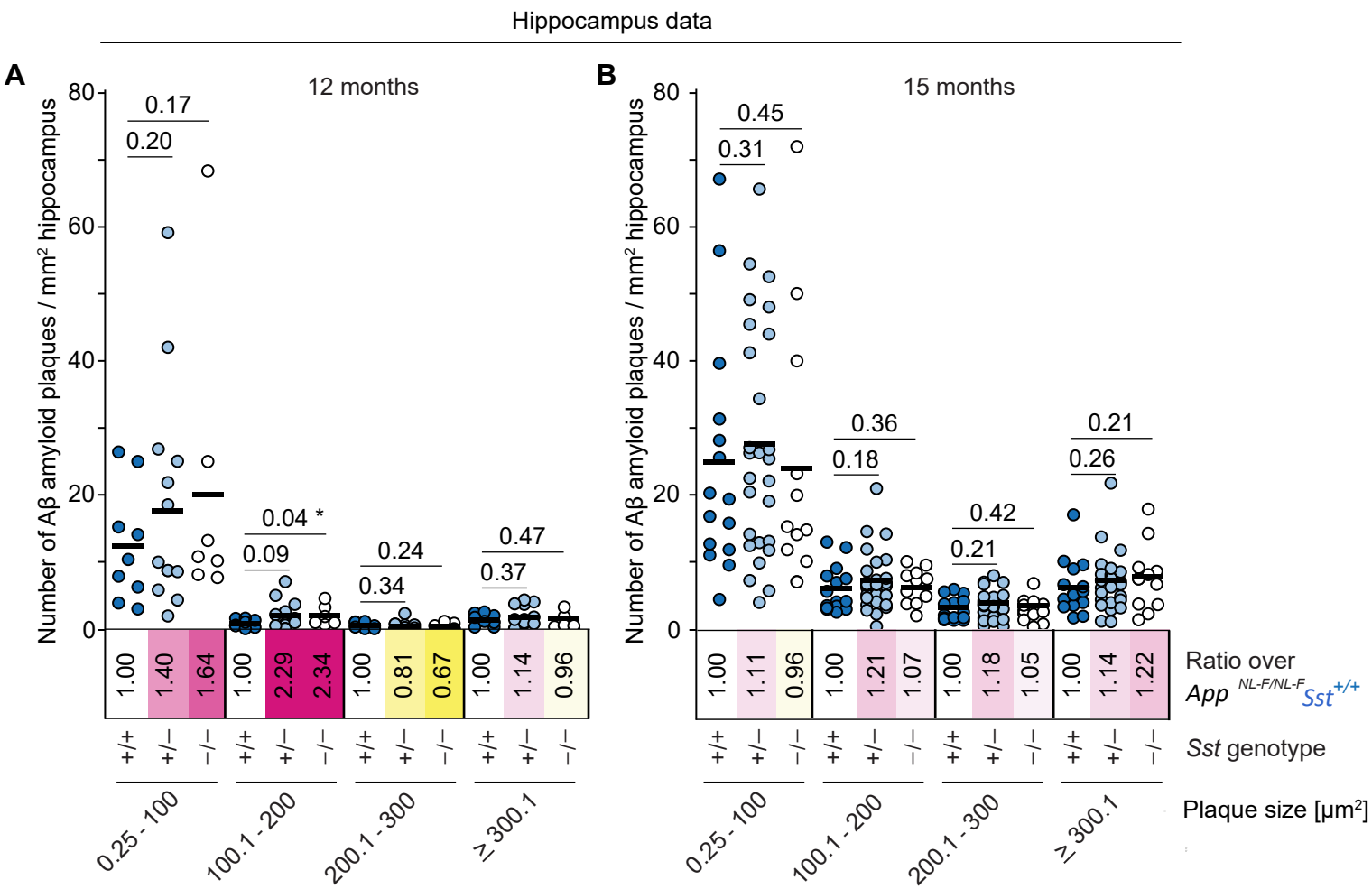
Hippocampal Aβ amyloid plaque densities increased between 12- and 15-month-old *App*$^{NL\text{-}F/NL\text{-}F}$ mice. Differences in hippocampal Aβ amyloid plaque densities were observed when comparing *App*$^{NL\text{-}F/NL\text{-}F}$ mice that expressed wild-type Sst versus Sst-deficient mice. More specifically, a trend toward higher Aβ plaque densities of small sizes (0.25-200 µm) was observed in 12-month-old *Sst* gene-deficient mice, echoing the increase in Aβ amyloid plaque densities observed in the cortex of *Sst* ablated *App*$^{NL\text{-}F/NL\text{-}F}$ mice (**Fig. 3F**).

Supplementary Figure 3. Original images of Coomassie stains and western blots.

Supplementary Figure 1



Supplementary Figure 2



Supplementary Figure 3