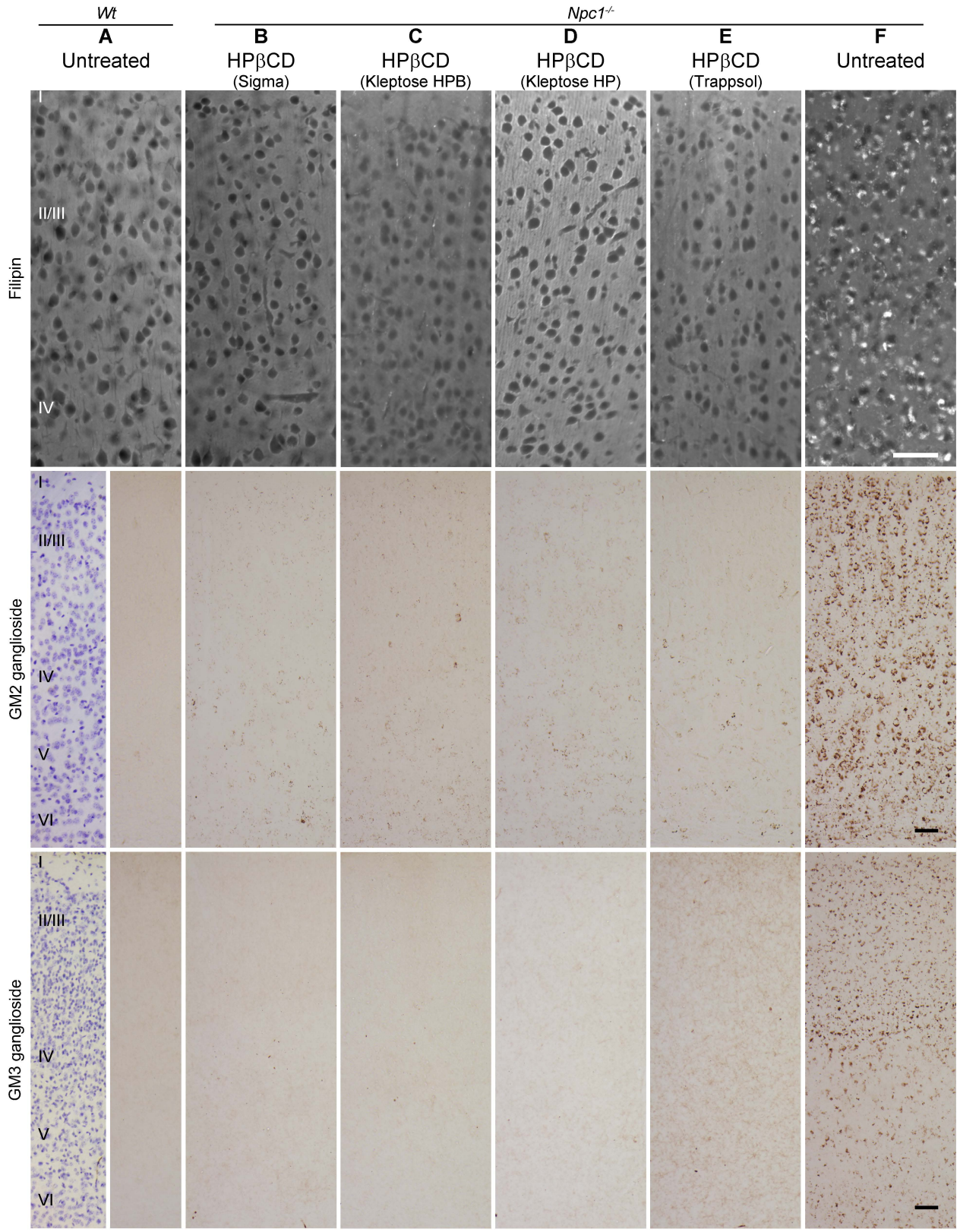

Supplementary Figure 1. UC and GM2 and GM3 ganglioside accumulation in brain cells of 3-week-old mice treated with different commercial preparations of HPβCDs. Top row: Sample fluorescence photomicrographs of dorsomediolateral neocortex from untreated *Wt* mouse (A) and CD-treated (B-E) and untreated (F) *Npc1*$^{-/-}$ mice stained with filipin to detect UC. Virtually all neurons show positive cytoplasmic staining of UC within the cell body in untreated *Npc1*$^{-/-}$ mice (F). In contrast, *Wt* mice show no filipin-positive distinct puncta (A). Nuclei appear as dark holes. All HPβCD commercial preparations tested show similar highly effective reduction of UC storage (B-E). Middle row: Sample brightfield photomicrographs of dorsomediolateral neocortex stained by immunoperoxidase to detect GM2 accumulation. Dark brown puncta of GM2 immunoreactivity are evident throughout dorsal neocortical neurons in untreated *Npc1*$^{-/-}$ mice (F) in contrast to *Wt* mouse neurons which remain essentially unstained (A). The four commercial preparations of HPβCD lead to equivalent reduction in GM2 storage. Bottom row: Sample brightfield photomicrographs of dorsal neocortex stained by immunoperoxidase to detect GM3. Dark brown puncta of GM3 immunoreactivity are evident within neurons of the dorsal neocortex from untreated *Npc1*$^{-/-}$ mice (F), though less abundant than GM2, and absent in *Wt* mouse cortex (A). Like for UC, all commercial preparations of HPβCD appear to reduce GM3 storage to an equivalent degree. Nissl stain is depicted in left half of *Wt* panels for GM2 and GM3 staining to visualize cortical layers which are marked by roman numerals. Scale bars = 50 μm.