Supplementary Figure 1. Strategy for FACS purification of oligodendrocytes, microglia and myeloid cells from the CNS of infected mice. Brain cells from JHMV infected wt mice at 5 days p.i. were stained with anti CD45, CD11b and O4 mAb. Representative plots depict gating strategy: All events were gated to exclude debris (P1); doublets were excluded based on FSC-W (P2; FSC-Area/FSC-Height). Oligodendrocytes (P3) were separated from $CD45^{lo}$ microglia (P5), $CD45^{hi}$ infiltrating leukocytes (P6) and other CNS resident cells (P4; $CD45^{-}O4^{-}$) by O4 staining ($CD45^{-}O4^{+}$). Microglia (P5) were separated from infiltrating and resident cells by their $CD45^{lo}$ $CD11b^{+}$ phenotype. Infiltrating myeloid cells (P7) were separated from other $CD45^{hi}$ CNS infiltrating cells by their $CD45^{hi}CD11b^{+}$ phenotype.

Supplementary Figure 2. PKR deficiency impairs mRNA but not protein expression of pro-inflammatory chemokines in virus infected BMDM. BMDM from wt and $PKR^{-/-}$ mice were infected with MHV-A59 at a MOI of 1. Transcript expression of *Ccl2*, *Ccl5* and *Cxcl10* and secreted protein levels of CCL5 and CXCL10 were determined at 12 and 18 hours p.i. Data are the average of 3 separate experiments ± SEM. * $P \geq .05$.

Supplementary Figure 3. PKR deficiency enhances T cell inflammation. Numbers of total brain infiltrating leukocytes ($CD45^{hi}$), macrophages ($CD45^{hi}$ $F480^{+}$), CD8 and CD4 T cells derived from infected wt and $PKR^{-/-}$ mice determined by flow cytometry. Data represent the average of three separate experiments ± SEM with n = 3/group per timepoint and experiment). * $p \geq .05$.