

SUPPLEMENTAL MATERIAL

Kennedy et al., <http://www.jem.org/cgi/content/full/jem.20140455/DC1>

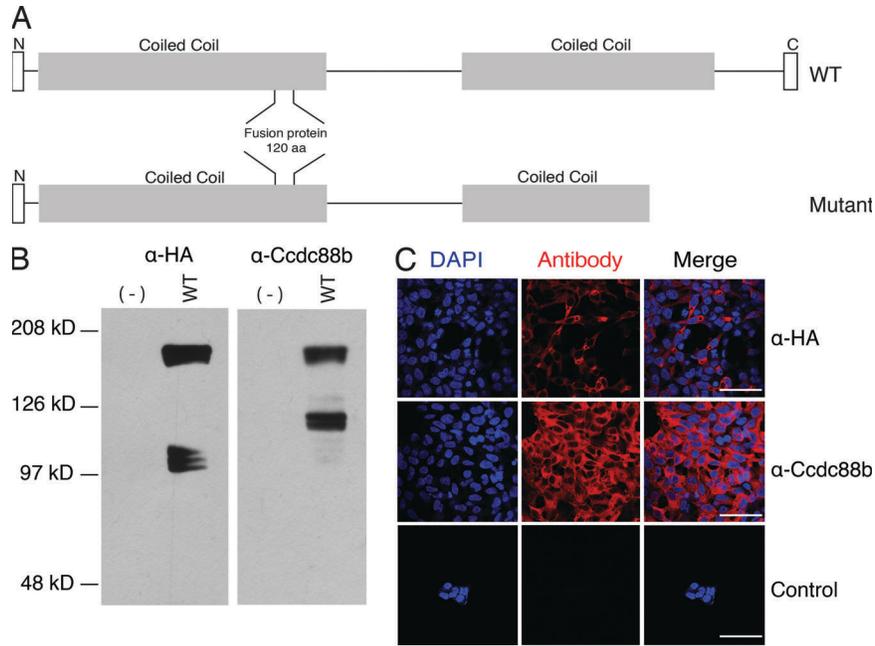


Figure S1. Production and characterization of anti-CCDC88B antibody. (A) Schematic representation of CCDC88B protein showing the N-terminal microtubule binding domain (N), two central CCDs, the C-terminal organelle-binding domain (C), and the position of the segment used as immunogen (fused to GST). (B) Immunoblotting analysis of total cell extracts from HEK293T untransfected control cells (–) and HEK293T cells stably expressing a full-length WT CCDC88B (WT) modified by the addition of an in-frame HA epitope tag (HA; indicated), and analyzed with anti-HA (α-HA) or anti-CCDC88B hyper-immune serum (α-CCDC88B). Molecular mass markers are identified to the left of the blots. (C) Untransfected control cells (bottom row), as well as HEK293T cells transfected with full length HA-tagged CCDC88B cDNA (top and middle rows) were stained with either anti-HA (top row) or anti-CCDC88B antiserum (middle row) and examined by immunofluorescence. The last column is a merge of images from the first and second columns. Bars, 50 μm.

Table S1, provided as an Excel file, shows the score for candidate genes at the human 11q13 locus associated with susceptibility to multiple inflammatory diseases. ChIP-seq of major myeloid inflammatory mediators (Irf8, Irf1, Stat1, and NF-κB [p65]), and RNA-seq before and after IFN-γ stimulation were used to compute an inflammatory score. This score is used to evaluate the possibility of a gene being implicated in inflammatory conditions.