

SUPPLEMENTAL MATERIAL

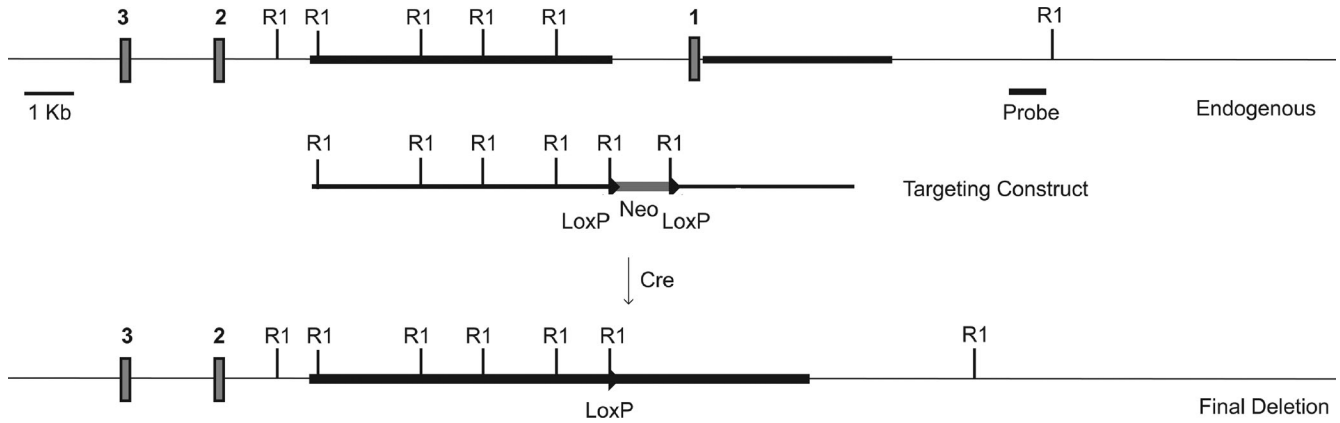
Cortez et al., <http://www.jem.org/cgi/content/full/jem.20130904/DC1>

Figure S1. *Crtam*^{-/-} mice. A targeting construct designed to replace exon1 of *Crtam* (including the initial translation start site) with a floxed neomycin resistance cassette was electroporated into E14.1 (129P2Ola/Hsd) ES cells. One correctly targeted clone was identified and confirmed by Southern blot analysis using multiple enzyme digests with 5', 3', and internal probes. The clone was injected into C57BL/6 blastocysts and chimeras bred to CMV-Cre tg mice to excise the neomycin resistance cassette. The mutation was backcrossed until the genetic background was >99% C57BL/6, facilitated by genome-wide SSLP typing at 10cM intervals (done by the Speed Congenics Facility of the Rheumatic Diseases Core Center). Heterozygote mice were intercrossed to generate *Crtam*^{-/-} mice. Mice were genotyped by PCR using the following oligonucleotides: *Crtam* 66121 (5'-TGTCACCAACACCTCGTTCA-3'), *Crtam* 65821 (5'-CCATTTACTCTAAGTCCTTACCTTG-3'), *Crtam* 63951 (5'-TATCTACCTTGGCATTTCCTAGG-3'), WT product = 301 bp, and *Crtam* deletion product = 357 bp. Exons are represented by gray boxes; the position of the 3' external probe used for initial Southern blot screening of DNA digested with EcoRI is indicated; R1 = EcoRI.