

Supplementary Figure 1. No obvious pathology is observed in the colon of diseased hCD1Tg/HJ1Tg/ApoE^{-/-} mice. Colon samples were fixed in 10% formalin and laid out in Swiss rolls for paraffin embedding. Paraffin sections were cut and stained with H&E to examine the tissue architecture. Scale bars represent 100 μ m.



Supplementary Figure 2. hCD1Tg/HJ1Tg/ApoE^{+/+} and hCD1Tg/HJ1Tg/ApoE^{-/-} mice have comparable numbers of FoxP3⁺ regulatory T cells. Cells isolated from the (A) cervical lymph nodes and (B) dermis were stained intracellularly with Foxp3 and gated on CD45⁺TCRb⁺. Subsequently, the CD4 and Foxp3 positive Treg cell population was examined (n=3-5). ***P < 0.005; **P < 0.01; *P < 0.05; n.s. = not significant. Statistical analysis was performed using One-Way ANOVA followed by Bonferroni post-hoc test.



Supplementary Figure 3. CD1b is expressed on a subset of dermal dendritic cells. (A) Dermal cells were isolated and stained with mAbs against CD45, CD11b, CD11c, and CD1b. CD1b expression on CD11b⁺CD11c⁻, CD11b⁺CD11c⁺ and CD11b⁻CD11c⁺ populations were analyzed by flow cytometry. (B) Epidermal cells were isolated and stained with mAbs against CD45, CD207, CD11c and CD1b. CD1b expression on CD207⁺CD11c⁺ population (Langerhans cells) was analyzed by flow cytometry by gating on CD45⁺ cells. (C) CD1b expression on hair follicle stem cells and keratinocytes was determined by gating on CD45⁻ cells and then on integrin α 6 and β 1 double positive cells. Subsequently, CD34⁺ Sca-1⁻ hair follicle stem cells or CD34⁻ Sca-1⁺ epidermal keratinocytes were output for CD1b expression. Data shown are representative of at least 2 experiments for all figure panels.



Supplementary Figure 4. Fatty acids do not show preferential accumulation in the skin of hCD1Tg/HJ1Tg/ApoE^{-/-} mice and reactivity of lipids to a second mouse CD1b-autoreactive hybridoma. Total lipid was extracted from skin and liver tissues of indicated mice, weighed and analyzed by gas chromatography. (A) Fatty acid species in the skin of diseased hCD1Tg/HJ1Tg/ApoE^{-/-} and healthy hCD1Tg/HJ1Tg/ApoE^{+/+} mice (n=3) were quantified. (B) Ratio of fatty acids from diseased over healthy mice in the skin and liver. (C) Polar phospholipids (PL), cholesterol (Chol) and fatty acid mixtures (FA1: saturated and monounsaturated and FA2: polyunsaturated) were loaded on to CD1b protein and incubated with LN1-7 T cell hybridoma for 24 h. IL-2 in the supernatant was measured by ELISA. Data are representative of at least 2 experiments. **P < 0.01; *P < 0.05. Statistical analysis was performed using Student's t-test.

Name	Sequence (5'-3')
For genotyping	
HJ1α F	TGACACCTGCTCAGTTCTTGTGC
HJ1α R	TAGCTTGTTCCCTGCACTTGG
CD1c F	CAGAAACTGCAGAAACAGCA
CD1c R	GATGGGTGAAAAGAGGTGAAA
	TGTGACTTGGGAGCTCTGCAGC
ApoE Common	
	GUGUUUGAUTGUATUT
II -18Rα R	TGGTGGCTGTTTCATTCCTGT
IL-12R61 F	TGCCGCTACTTCTCCTCAG
IL-12R61 R	ACTTCATGGTTCGGTTCCCAA
P*	
TLR9 F	ATGGTTCTCCGTCGAAGGACT
TLR9 R	GAGGCTTCAGCTCACAGGG
S100a7 F	GTA CTC AGG TCA TGG TTC TG
S100a7 R	GGT ATT CAA GCA AGG TAT CAC
S100a8 F	GGA GTT CCT TGC GAT GGT GAT
S100a8 R	TCC TTG TGG CTG TCT TTG TGA
β-actin F	CTT CTT TGC AGC TCC TTC GTT
β-actin R	AGG AGT CCT TCT GAC CCA TTC
U -174 F	
II -17A R	
IL-22 F	ATG AGT TTT TCC CTT ATG GGG AC
IL-22 R	GCT GGA AGT TGG ACA CCT CAA
GM-CSF F	GGC CTT GGC AGC ATG TAG AGC
GM-CSF R	GGA GAA CTC GTT AGA GAC GAC TT
TNF-α F	CCA CCA CGC TCT TCT GTC T
TNF-α R	GGC ACC ACT AGT TGG TTG T
IL-6 F	CTT GGG ACT GAT GCT GGT GAC A
IL-6 R	GCC TCC GAC TTG TGA AGT GGT A
IGBT R	GGCC TTA GTT TGG ACA GGA TCT G
IL-23 F	CCA TTA GGA CTT GTG CTG TTC T
IL-23 R	CCA AGG GCT CGA GAC TTT ATT C
IFN-γ F	TCA AGT GGC ATA GAT GTG GAA GAA
IFN-γ R	TGG CTC TGC AGG ATT TTC ATG
HJ1Vβ F	ATC TCT TCC CGG TGC TGA TT
HJ1Vβ R	TCT GGT TCC TGA GCC AAA AT

Supplementary table 1: Oligonucleotide primers used in the study