

Supplemental Figure 1. Co-injection of granulocytes precipitates blister induction by antibodies to type VII collagen in neonatal mice. Three doses of rabbit antibodies to type VII collagen or control antibody (10 mg/body weight/day) were given subcutaneously 24 h apart to neonatal mice. After another 24 h, 50 ng of recombinant IL-8 and C5a were administered intradermally along with the 4th dose of antibodies and, subsequently, 5 x 10⁶ murine granulocytes were injected subcutaneously into the back of the mice. (a) Blister on the back of a neonate treated with pathogenic rabbit antibody to type VII collagen, but not in (b) a littermate injected with normal rabbit IgG (Insets show magnifications of injection sites). Histologic analysis of murine skin revealed (c) extensive subepidermal cleavage and a neutrophil-rich inflammatory infiltrate at the dermal-epidermal junction in the neonate injected with antibodies to type VII collagen (x200). In contrast, (d) a dermal and subcutaneous infiltration of neutrophils, but no significant recruitment of leukocytes at the dermal-epidermal junction and no subepidermal splits were seen in a new-born mouse treated with control antibody (x200). (c) Immunofluorescence analysis of lesional skin from a diseased mouse shows (e) rabbit IgG and (g) murine C3 deposits at the epidermal basement membrane, whereas in a mouse injected with control rabbit IgG, no deposits of (f) rabbit IgG or of (h) murine C3 are detected (x400).

Supplemental Table 1. EBA patients' autoantibodies show weaker binding to mouse skin than to human skin

Sera	IF microscopy ^A	
	Human skin	Murine skin
EBA 1	160	160
EBA 2	320	20
EBA 3	160	0
EBA 4	160	0
EBA 5	10	10
SA2953 ^B	160	20,480
SA2954 ^B	5,120	20,480

^ASerum reactivity to the DEJ was determined by IF microscopy using sections of non-split human and murine skin. Sera were titrated to end-point for IgG reactivity. ^BImmune rabbit serum.