	Bone Marrow Pop. (% Total)		Mature Pool (Absolute %)		Immature Pool (Absolute %)	
	A10ΔEC	Control	A10ΔEC	Control	A10ΔEC	Control
Myeloid	50.7	57.5	37.5	46.2	13.2	11.3
Erythroid	**38.3**	**23.2**	**33.3**	**16.8**	**9.3**	**6.3**
Lymphocytes	13.8	19.0	-	-	-	-
Mast Cells	0.0	0.0	-	-	-	-
Plasma Cells	0.3	0.3	-	-	-	-

Table S1. Bone Marrow analysis of *A10ΔEC* and control animals (n=2 for each group). Each blinded analysis was performed by manually counting 300 cells from bone marrow smears of 8-week old littermates.

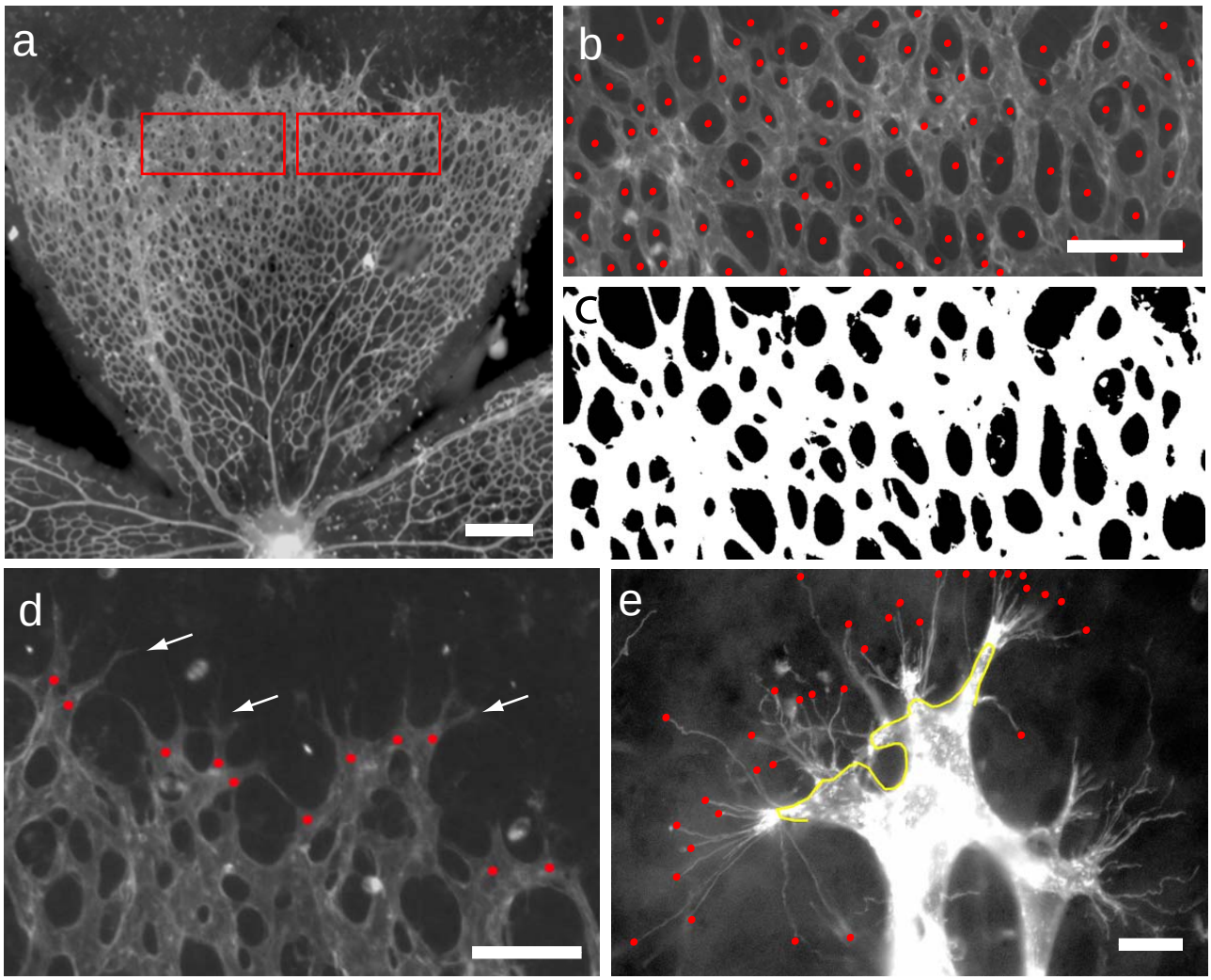

Figure S1. Quantification of retinal developmental angiogenesis. (**a**) A red outline indicates the position of a representative rectangular area (500μm x 200μm) that was used to measure vascular loops and vessel coverage adjacent to the leading edge of the developing vascular tree. (**b**) Vascular loops were quantified by counting the number of spaces between Isolectin B4-labeled vascular cells within a given rectangular area (spaces marked by red dots). (**c**) Representative example of an image used to determine endothelial cell coverage per surface area as described in materials and methods. (**d**) Red dots mark the cells that were counted to determine tip cell density at the vascular front based on morphological presence of filopodial collections (arrows). (**e**) Filopodia density at the vascular front was quantified by demarcating 100μm of the vascular front (yellow line) and counting filopodia (red dots) along that front. Scale bars for **a** = 250μm; **b,c** = 100μm, **d** = 50μm, **e** = 20 μm.

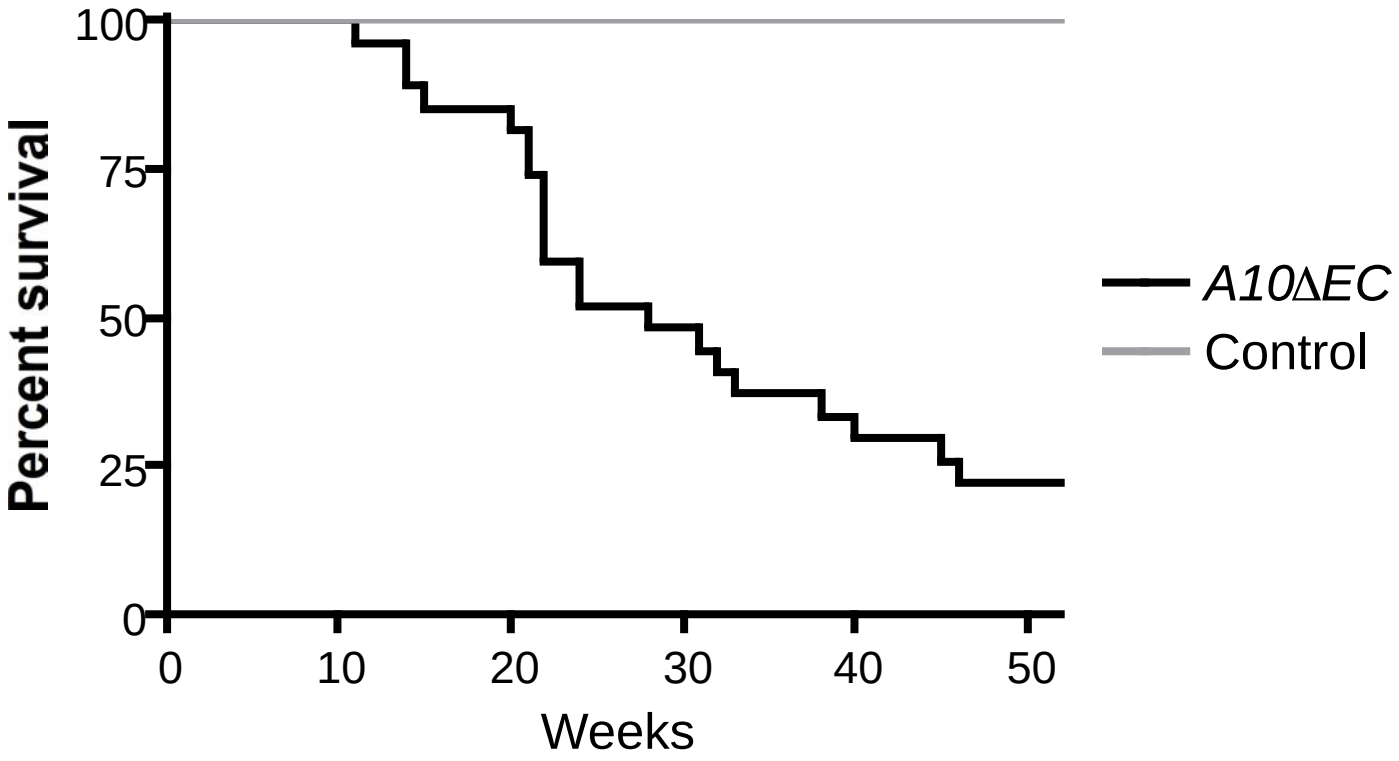

Figure S2. Kaplan Meier graph of *A10ΔEC* individuals shows decreased survival relative to littermate controls. Birth and death records of 27 male *A10ΔEC* mice and 25 control males were used to generate a 52-week survival curve. Median survival time for the *A10ΔEC* mice was ~28 weeks.

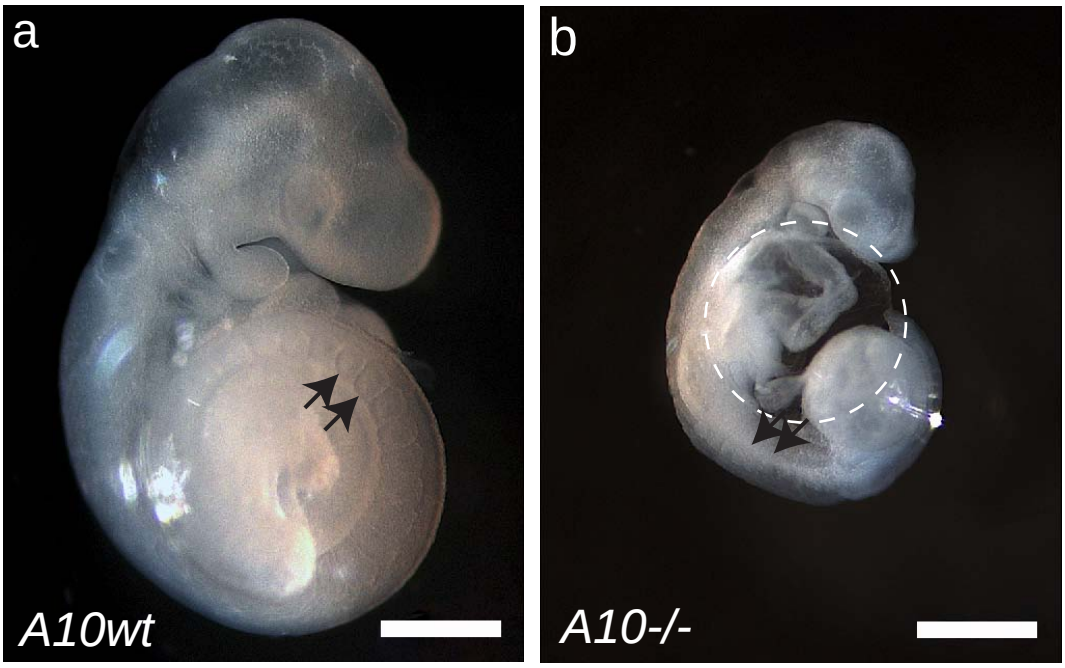

Figure S3. Cre-mediated excision of floxed *Adam10* in the germ line results in loss of function of Adam10. To test whether removal of floxed alleles of *Adam10* results in functional inactivation of Adam10, transgenic mice expressing the ubiquitously expressed *EIIa-Cre* transgene were crossed with *Adam10flox/flox* mice to inactivate Adam10 in the germline (see materials and methods for details). Comparison of a control embryo (**a**) and an *Adam10-/-* embryo (**b**) at embryonic stage E9.5, generated through timed matings, showed an enlarged heart sack (surrounded by a dotted line) and lack of somitogenesis (arrows point to somites) in the *Adam10-/-* embryo (a representative sample of three embryos is shown), which resemble the *Adam10-/-* embryos previously described by Hartmann et al., *Hum Mol Genet*. 2002;11:2615-2624. Scale bars = 500μm.

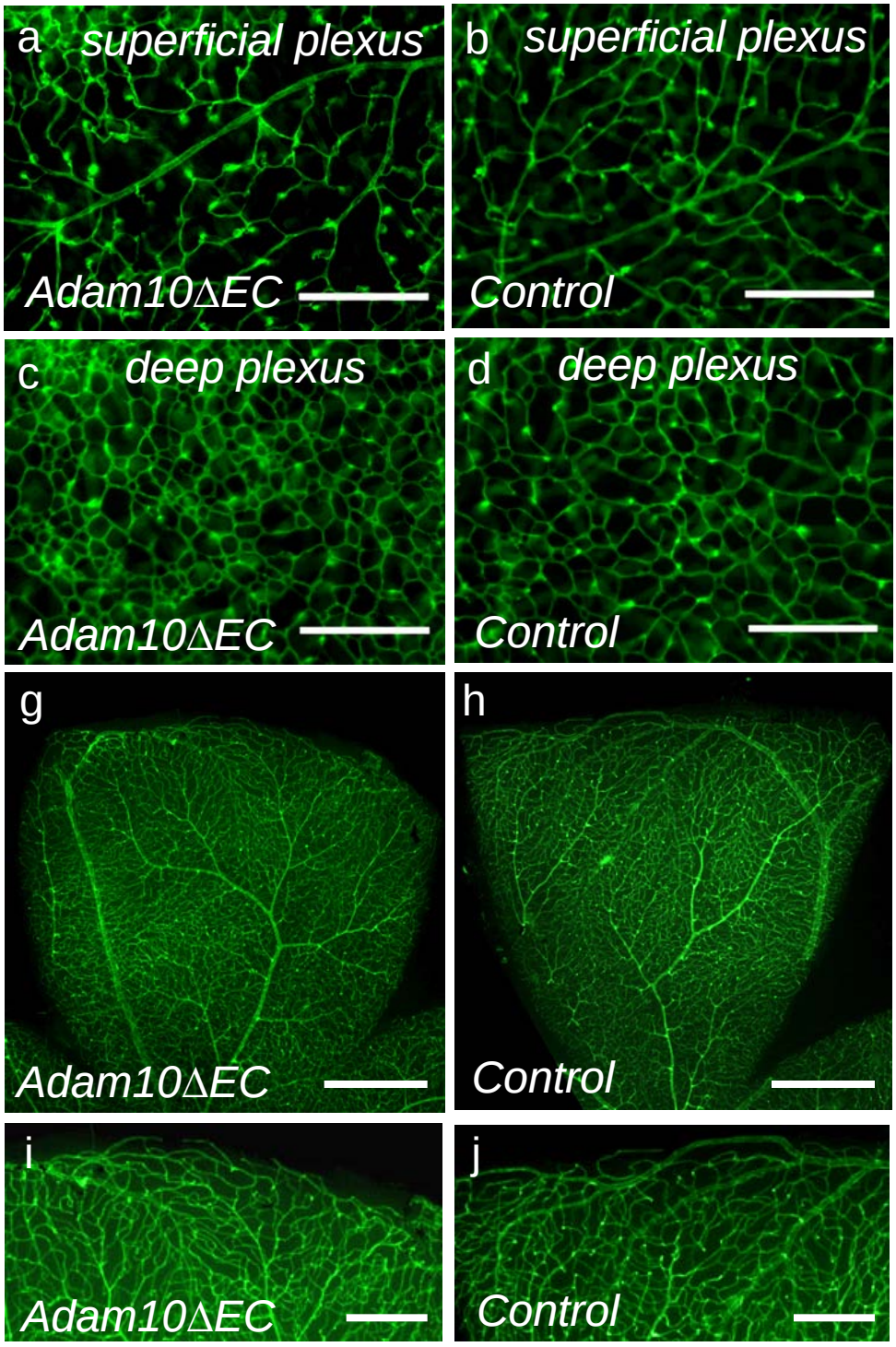

Figure S4. Retinal vessels in *A10ΔEC* and control mice remodel over time. The superficial vascular plexi (**a, b**) of p12 *A10ΔEC* and control individuals show little difference in vascular density and vessel coverage, while the deep plexi (**c, d**) demonstrate increased density and branching of vessels in *A10ΔEC* mice compared to controls. The retinal vascular tree in adult mice was indistinguishable between *A10ΔEC* mice (e, g) and controls (**f, h**). Scale bars for **a-d,g,h** = 100μm, **e,f** = 300μm.

Figure S5

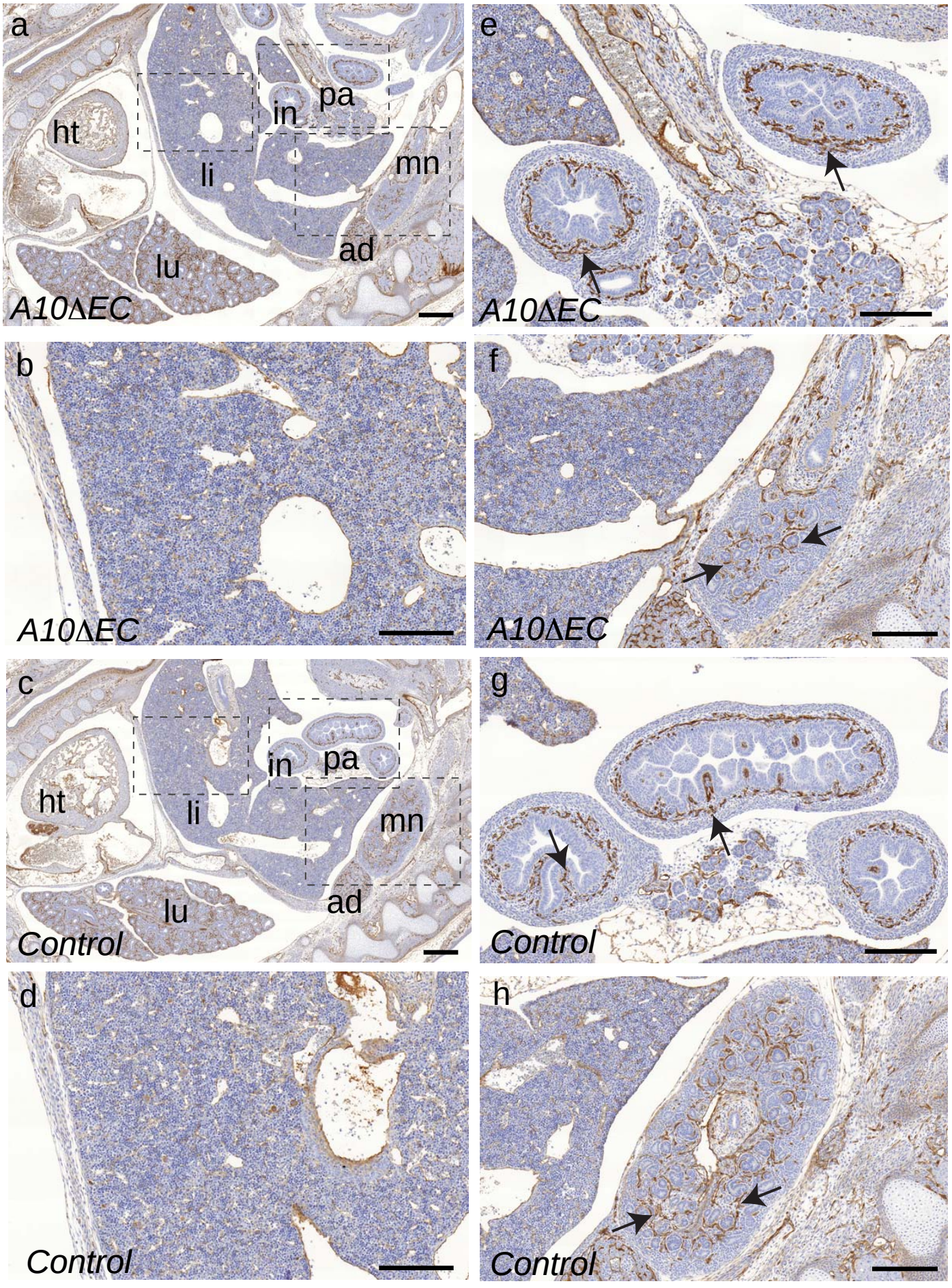
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Figure S5. Sections of an E14.5 *A10ΔEC* or control embryo stained with the endothelial cell marker MECA-32 show no evident differences in the distribution of endothelial cells at this stage of development. The staining pattern of *A10ΔEC* embryos at E14.5 with MECA-32 (**a**) was comparable to that in controls (**c**) in a low magnification overview of the thorax and abdominal section (ht = heart, li= liver, in = intestine, pa = pancreas, mn= mesonephros, ad = adrenal gland, lu= lung). Enlarged views of the liver and diaphragm (**b,d**) intestine (**e,g,** arrows point to MECA-32-positive cells), and the kidney (**f,h**, arrows point to developing glomeruli) are shown for A10ΔEC embryos (**b,e,f**) or controls (**d,g,h**). Scale bars for **a-h** = 250μm.

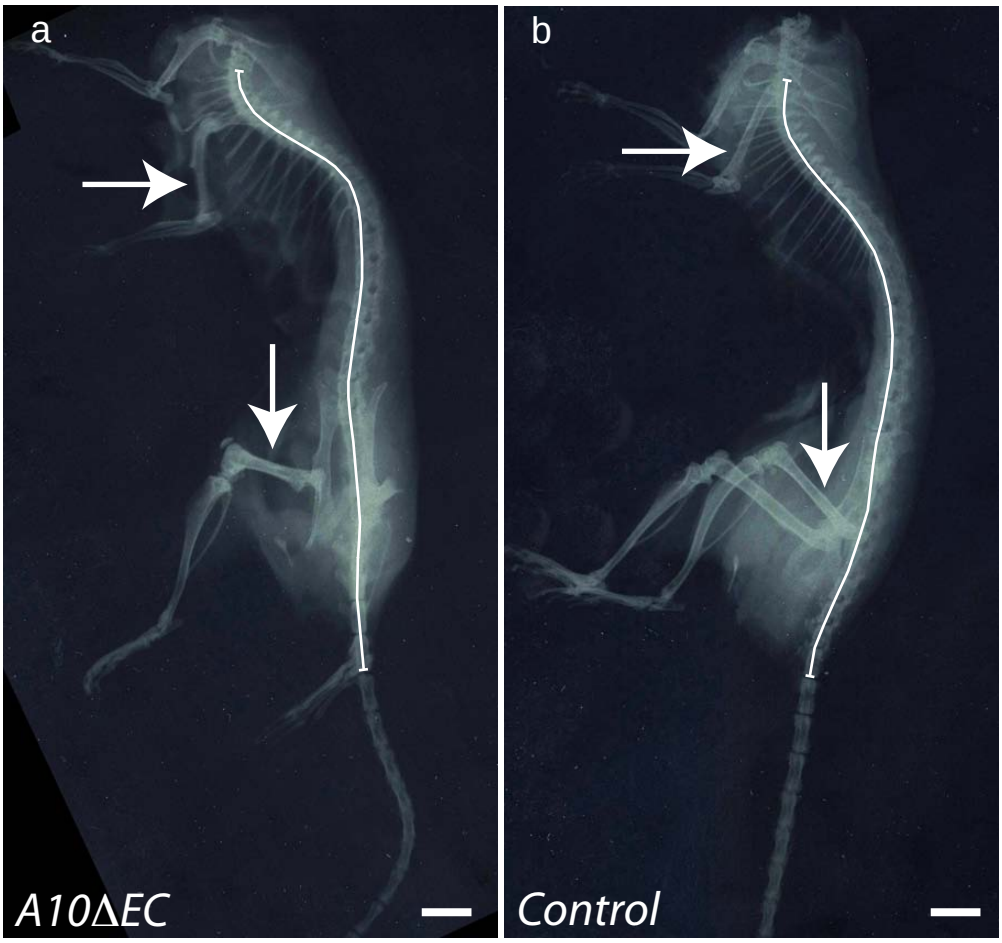

Figure S6. X-rays of *A10*ΔED and control mice
X-rays of an *A10*Δ*EC* (**a**) and control (**b**) mouse show shortened femurs and humeri in the mutant mouse compared to the control mouse (pointed by arrows), while the distance from thoracic vertebra 1 to caudal vertebra 2 was comparable (solid line, *A10*Δ*EC* mice: 64.8 ± 1.3 mm, n = 2; control: 64.0 ± 0.5 mm, n = 2). Two-tailed Student's t-test: p= 0.47, no significant difference. Scale bars = 5mm.

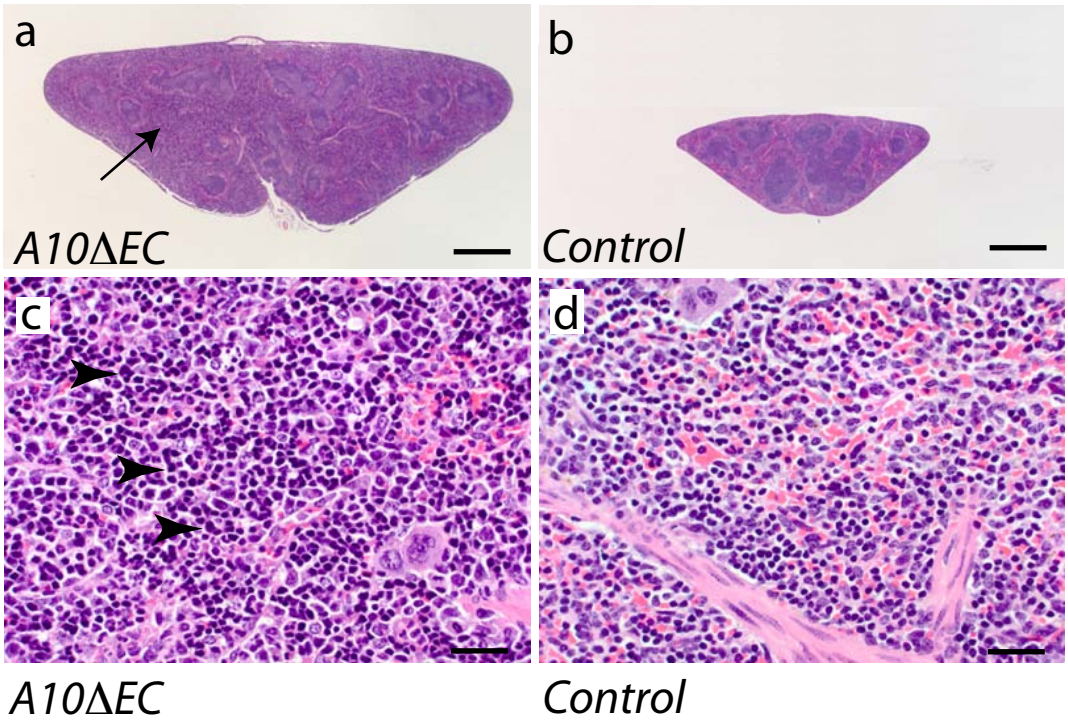

Figure S7. Histopathology of the Spleen. Histological analysis at low (**a, b**) and high (**c, d**) magnification reveals splenomegaly and expansion of red pulp (arrow in **a**) with predominance of hematopoietic precursors (arrowheads in **c**) in *A10ΔEC* individuals, while the white pulp appeared normal in both populations. Scale bars for **a,b** = 1000μm; **c,d** = 50μm.

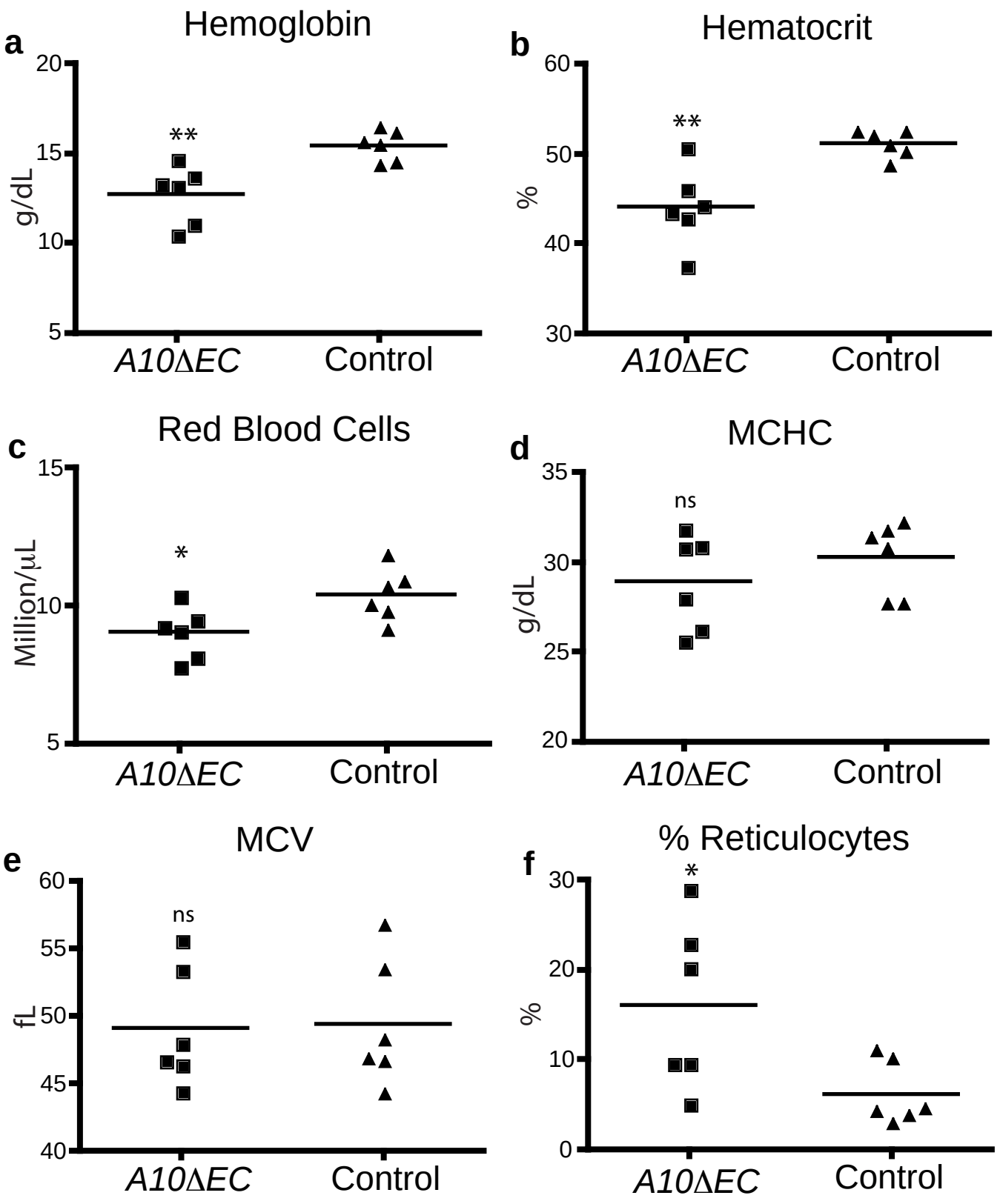

Figure S8. Erythroid Hematology of *A10ΔEC* and control mice. Hematologic analysis of hemoglobin (**a**), hematocrit (**b**), red blood cell concentration (**c**), mean corpuscular hemoglobin concentration (**d**), mean corpuscular volume (**e**), and percent reticulocytosis (**f**) of blood from *A10ΔEC* and control mice revealed lower values of hemoglobin, hematocrit, RBC concentration, but an increase in reticulocytosis in mutant individuals. Additional analysis of monocytes, lymphoctes, eosinophils, basophils, neutrophils, and platelets revealed no statistical difference between mutant and control individuals (not shown). *A10ΔEC* n=4, controls n=4. * = $p \leq 0.05$ and ** = $p \leq 0.01$ in a two-tailed student's t-test.