ONLINE DATA SUPPLEMENT

Rig-Like Helicase Innate Immunity Inhibits VEGF Tissue Responses via A Type I Interferon-Dependent Mechanism

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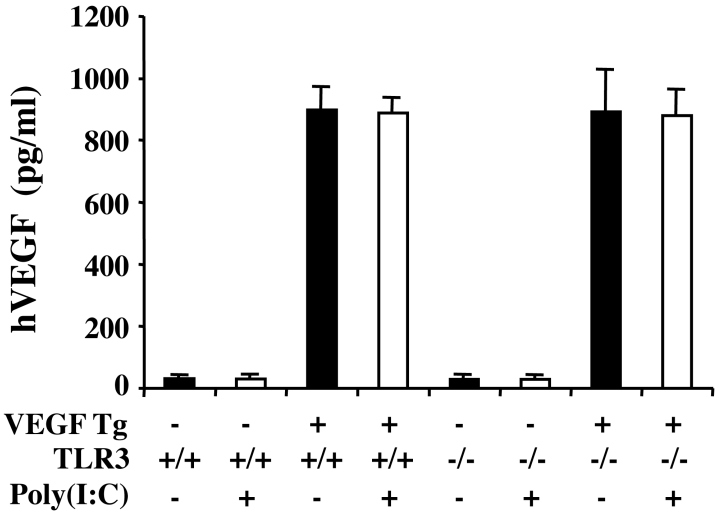
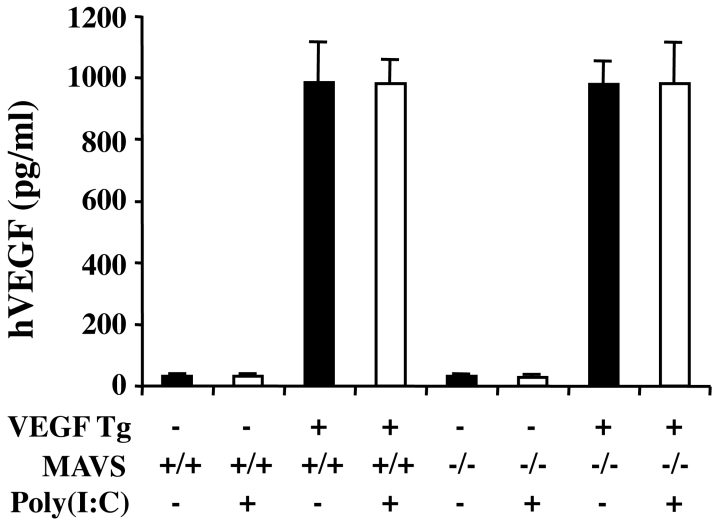
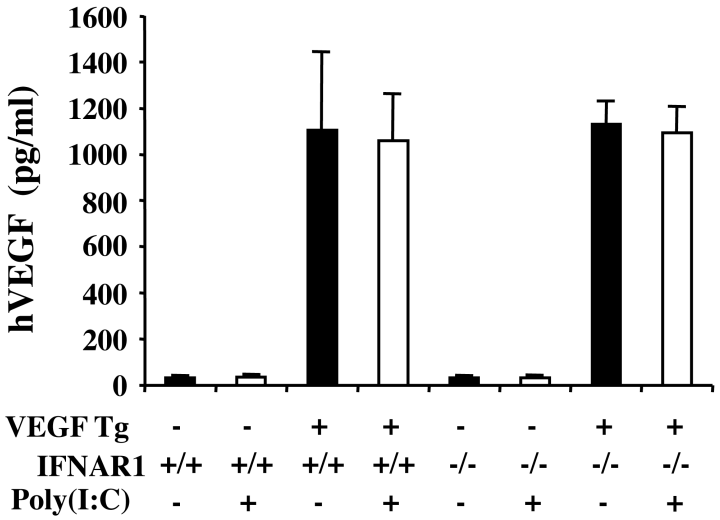
A)



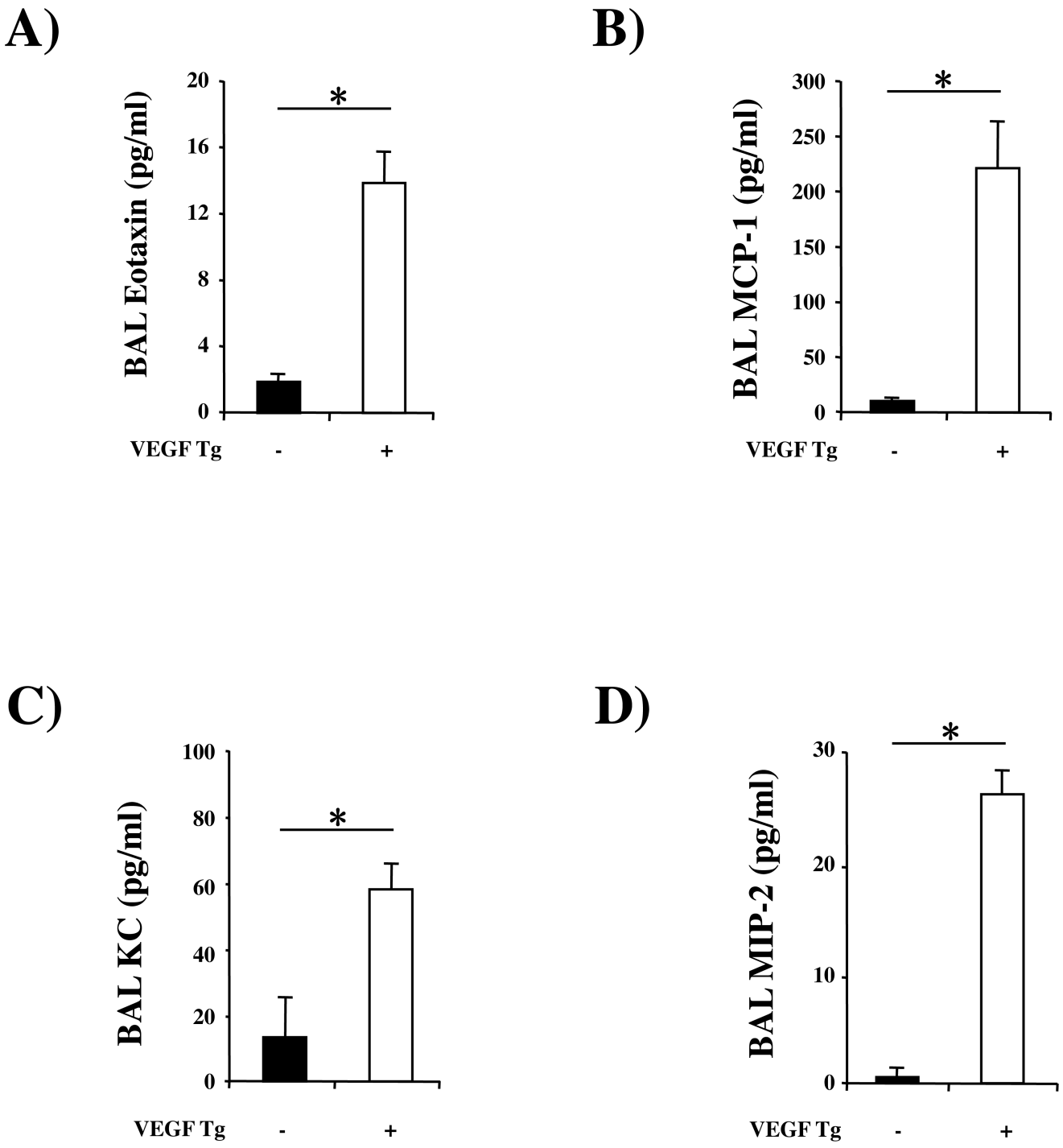
B)



C)

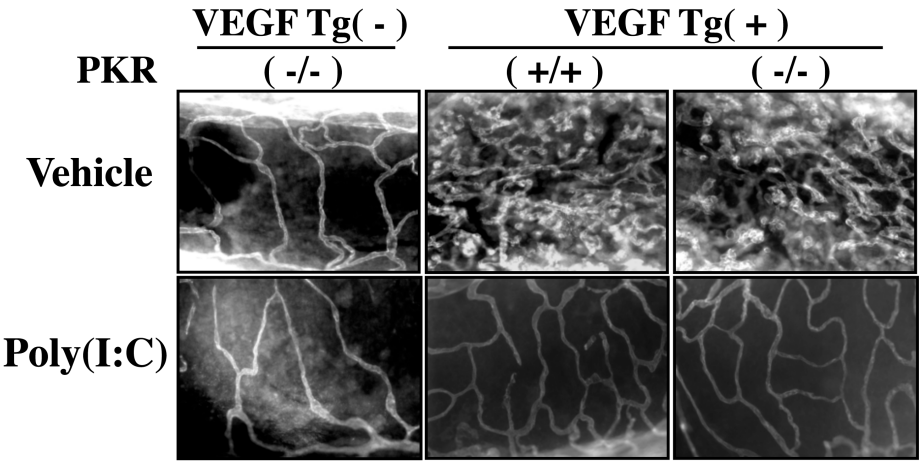


<u>Supplement Figure 1</u>. Effects of Poly(I:C) on the production of transgenic VEGF. WT (VEGF-) and Tg+ mice with wild type genetic backgrounds (+/+)or the noted null (-/-) mutations of TLR3, MAVS or IFNAR1 were treated with dox and Poly(I:C)(+) or its vehicle control Poly(I:C)(-) for 2 weeks. The levels of BAL hVEGF were then assessed by ELISA. The noted values represent the mean +/- SEM of a minimum of 4 mice each.



Supplement Figure 2. Chemokines in the BAL from VEGF Tg+ mice. WT (VEGF Tg-) and VEGF Tg+ mice were treated with dox for 2 weeks. The levels of the noted chemokines in BAL were evaluated by ELISA. The noted values represent the mean +/- SEM of a minimum of 4 mice. (*P<0.05)

A)



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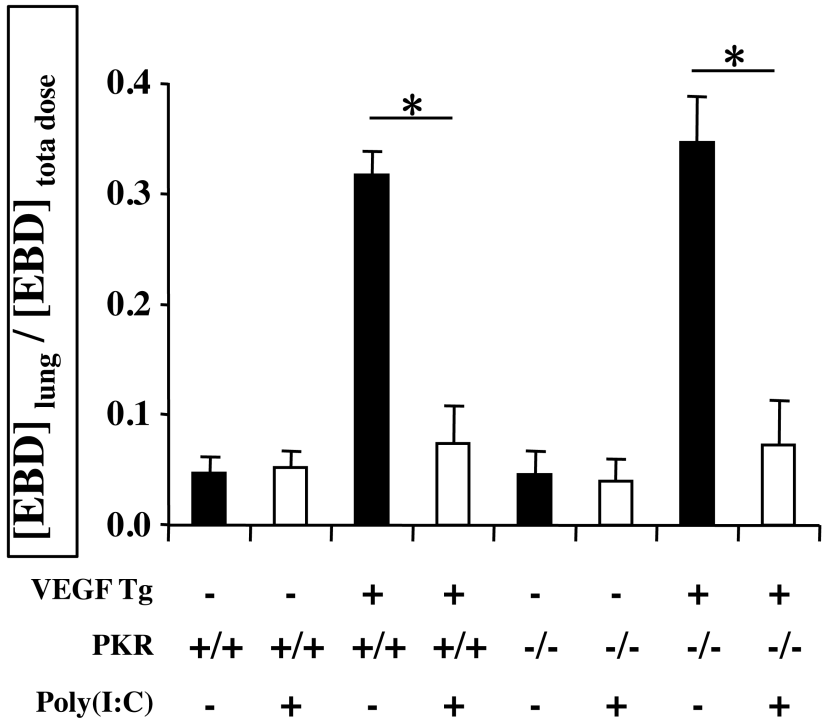

Supplement Figure 3. Role of PKR in Poly(I:C) regulation of VEGF responses. WT (VEGF Tg-) and VEGF transgenic (VEGF Tg+) mice were bred with mice with wild type (+/+) or null mutant PKR loci. This generated transgene negative mice with wild type PKR loci (VEGF Tg-/PKR$^{+/+}$), transgene negative mice with null PKR loci (VEGF Tg-/PKR$^{-/-}$), transgenic mice with wild type PKR loci (VEGF Tg+/PKR$^{+/+}$), and transgenic mice with null PKR loci (VEGF Tg+/PKR$^{-/-}$). These mice were treated with Poly(I:C) (30 μg) or vehicle (Poly(I:C)-), transgene activation was accomplished with dox 24 hours later, and treatment with Poly(I:C) or vehicle and dox were continued for 2 weeks. Angiogenesis (A) and dye extravasation (B) were assessed.