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Supplementary Fig. 1: Analysis of PDGFR- β staining in different stage Lewis lung carcinomas grown in wild type and in Ang-2-deficient mice. Tumors were harvested 7 days (top), 12 days (middle) or 21 days (bottom) after tumor inoculation. Tumor sections were double-stained for the endothelial cell marker CD31 (green) and for the mural cell marker PDGFR- β . Vessel coverage was calculated as the percentage of PDGFR- β -positive vessels compared to the number of CD31-positive vessels. CD31 and PDGFR- β co-localized in >90% of all analyzed samples. High power analysis revealed that CD31 staining and PDGFR- β staining could clearly be separated in some vessels, whereas it distinctly co-localized in many tumor microvessels. Co-localization of both markers was consequently interpreted to reflect PDGFR- β expression by angiogenic endothelial cells rather than *bona fide* mural cell staining. PDGFR- β was therefore considered a questionable mural cell marker and excluded from all further analysis.

Nasarre et al. Supplementary Fig. 2



Supplementary Fig. 2: Growth of B16F10 melanoma in wild type and in Ang-2-deficient mice. Tumor growth dissociated during early tumor growth when the tumors had grown to 0.2-0.4 cm³ (**A**). At later time points tumor growth rates were similar in wild type and in Ang-2-deficient mice as evidenced by parallel or even converging growth curves of log transformed tumor volume data (**B**). Total tumor weight was reduced in Ang-2-deficient mice, albeit not significantly (**C**).

Nasarre et al. Supplementary Fig. 3



Supplementary Fig. 3: Effect of Ang-2 deficiency on microvessel density, vessel diameter and perfusion (A) and on mural cell recruitment and maturation (B) in B16F10 melanomas. B16F10 melanomas were subcutaneously injected in wild type and in Ang-2-deficient mice and harvested when the first tumor had grown to 2 cm³. MVDs and vessel diameters were quantitated in CD31-stained tissue sections. Perfusion was assessed on the basis of FITC-lectin perfusion labeling. For mural cell coverage analysis, tumor sections were double-stained for the endothelial cell marker CD31 and for the mural cell markers Desmin (top), NG-2 (middle), and α -SMA (bottom). Vessel coverage was calculated as the percentage of Desmin-, NG2-, and α -SMA-positive vessels compared to the number of CD31-positive vessels.