Author's response to reviews

Title: The endogenous soluble VEGF Receptor-2 isoform suppresses lymph node metastasis in a mouse immunocompetent mammary cancer model

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Title: The endogenous soluble VEGF Receptor-2 isoform suppresses lymph node metastasis in a mouse immunocompetent mammary cancer model

Dear Editor,

I would like to thank you for acceptance. In addition, we really appreciate Referee 3 for excellent suggestions and comments.

We have made a number of changes (indicating with red) in line with the Referee-3’s suggestions, as detailed below.

Thank you very much for your efforts on our behalf.
Sincerely yours,

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Reviewer: Bronislaw Pytowski

Thank you very much for excellent comments to improve our manuscript.

1. Page 5, line 2 from bottom: this protein

2. Page 13, line 1: was

3. Page 15, lines 10-14: The sentence has been divided: “The inhibition of metastasis in these groups may simply be a reflection of the suppressed tumor growth and cell proliferation. However, this therapeutic benefit is apparent because conventional therapies are often insufficient to eradicate metastatic breast cancer.”

4. Page 15, lines 15-18: The sentence has been changed as suggested by the referee as follows: “The prolonged survival, reduced tumor volume, and suppression of metastasis after pesVEGFR-2 therapy suggests that esVEGFR-2 may potentially represent a novel therapy for cancer treatment.”

5. Page 16, lines 7-5 from bottom: The sentence has been changed as suggested by the referee as follows: “Thus, treatment with pesVEGFR-2 that primarily inhibits lymphangiogenesis may be ineffective in this experimental setting.”

6. Page 17, lines 3-1 from bottom: The sentence has been changed as follows: “On a related note, sVEGFR-3 cannot only bind VEGFR-3 but also acts as trap for VEGF-C, which blocks VEGFR-3 signaling”

7. Page 18, lines 11-14: The sentence has been changed as follows: “Brideau et al. reported that J4 transgenic mice overexpressing endostatin (driven by the keratin K14 promoter) in epidermal basal cells exhibited inhibited angiogenesis and lymphangiogenesis in skin tumors induced by a carcinogen followed by a tumor promoter agent”