Author's response to reviews

Title: Androgen-regulation of the protein tyrosine phosphatase PTPRR activates ERK1/2 signalling in prostate cancer cells

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Submitted manuscript: Androgen-regulation of the protein tyrosine phosphatase PTPRR activates ERK1/2 signalling in prostate cancer cells

Please find attached a manuscript for submission to BMC Cancer. In this we show that the Protein Tyrosine Phosphatase Receptor R (PTPRR) is an early and direct target for repression via the androgen receptor.

We anticipate this work should be of broad interest to the readership of Molecular Cancer. Prostate cancer is a major clinical burden, and is strongly connected to androgen regulation via the androgen receptor. Advanced prostate cancer often has hyper-activation of the RAS/ERK1/2 pathway, thought to be due to loss of function of key negative regulators of this pathway. PTPRR negatively regulates the RAS/ERK1/2 pathway, so our results revealing androgen repression of PTPRR directly link these two clinically important signalling pathways. Importantly, we also demonstrate that over-riding this repression by ectopic expression of PTPRR in the presence of androgens is sufficient to decrease phosphorylation of ERK1/2 and reduce both the expression of oncogenic transcription factors and proliferation of prostate cancer cells.

We look forward to hearing from you.

Yours Sincerely,

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