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LETTER TO THE EDITOR

Is 37LRP cell surface receptor for PSP94?

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Dear Editor,

This has reference to the commentary 'Growth inhibition properties of the putative prostate cancer biomarkers PSP94 and CRISP-3' by van Eynde *et al.* published online in your journal in 2010.[1] PSP94 and CRISP-3 are both secretory proteins. Identifying cell surface-binding sites for both these molecules and understanding the downstream signaling would unravel their role(s) in prostate cancer. Though existence of cell surface binding sites for PSP94 has been indicated,[2,3] till date the identity of the 'cell surface' receptor for PSP94 is not reported in literature. van Eynde *et al.* in their commentary state that PSP94 attaches to cell membranes and one of its binding partners is 37LRP (laminin receptor precursor). The reference which they have cited has reviewed the original study by Annabi *et al.*[4] which proposes the involvement of 37LRP in binding to a 15-mer peptide representing amino acids 31–45 of PSP94 (PCK3145) to cell surface and not the PSP94 protein *per se*. These experiments were conducted on HT1080 cell line which is a human fibrosacoma cell line and similar studies in prostate cell lines have not yet been reported. Annabi *et al.*[5] in another study have also reported that transfection of HT1080 with 67LR plasmid increases cell adhesion to PCK3145 peptide. 67LR is mature form of the laminin receptor present on the cell surface. 37LRP is a 37-kDa precursor protein which is predominantly cytoplasmic. Studies distinguishing between the expression of 37LRP and 67LR on prostate cancer cell surface are also not available. More importantly, the native PSP94 protein binding to prostate cell surface *via* 37LRP is yet to be demonstrated.

van Eynde *et al.* further quote that binding of PSP94 to cell surface receptors inhibits MMP9 secretion and VEGF signaling. These activities again are reported only for the PCK3145 peptide and not the native protein as per the cited study by Lamy *et al.*[6] Extrapolating the studies carried out on a peptide and assigning a function to native protein without experimental evidence can be misleading. In our opinion, these facts should be taken into account while commenting on PSP94 and its receptor interactions.

1 van Eynde A, Litovkin K, Bollen M. Growth inhibition properties of the putative prostate cancer biomarkers PSP94 and CRISP-3. *Asian J Androl*; e-pub ahead of print 22 November 2010; doi: 10.1038/aja.2010.120.
2 Phadke M, Vijayalakshmi S, Sheth AR. Evidence for the presence of specific receptors for inhibin in human prostate. *Indian J Exp Biol* 1982; **20**: 419–20.
3 Yang JP, Baijal-Gupta M, Garde SV, Fraser JE, Finkelman MA *et al.* Identification of binding proteins for PSP94 in human prostate adenocarcinoma cell line LNCaP and PC3. *Prostate* 1998; **35**: 11–7
4 Annabi B, Currie JC, Bouzeghrane M, Dulude H, Daigneault L *et al.* Contribution of the 37-kDa laminin receptor precursor in the anti-metastatic PSP94-derived peptide PCK3145 cell surface binding. *Biochem Biophys Res Commun* 2006; **346**: 358–66.
5 Annabi B, Bouzeghrane M, Currie JC, Dulude H, Daigneault L *et al.* Inhibition of MMP-9 secretion by the anti-metastatic PSP94-derived peptide PCK3145 requires cell surface laminin receptor signaling. *Anticancer Drugs* 2006; **17**: 429–38.
6 Lamy S, Ruiz MT, Wisniewski J, Garde S, Rabbani SA *et al.* A prostate secretory protein94-derived synthetic peptide PCK3145 inhibits VEGF signaling in endothelial cells: implication in tumor angiogenesis. *Int J Cancer* 2006; **118**: 2350–8.

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