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Article Review

A critical review of the article by Zongwen et.al, 2012 titled ‘Human Chorionic Gonadotropin b induces migration and invasion via activating ERK1/2 and MMP-2 in human prostate cancer DU 145 cells’.

Clear signs were found that acted as proof and evidence that hCGb was highly expressed in both Mrna and protein levels versus empty vector transfected DU 145 cells. Either hCGb Mrna or protein amount was little in the control cells under these experimental conditions. (Zongwen et.al, 2012) performed a research on the existence and changes in levels of both Mrna and proteins in DU 145 cells. To ensure quality and validity in research ingredients, diversity was ensured. HCGb standards were purchased from Abcam (Hong Kong), the construct pVSneo-hCGb containing hCGb cDNA was purchased from La Jolla, CA whilst restriction enzymes including Sall, XhoI, EcoRI, BamHI, HindIII and T4 DNA and crystal violet was purchased from St. Louis (Zongwen et.al, 2012).

(Zongwen et.al, 2012) after conducting transfection, found the presence of established stable cell line overexpressing HCGb in DU 145 CELLS. However the article fails to account for the fact that majority of the studies on human prostate cells have come up with results that show these cells to be Androgen receptor negative (Fatouma et.al, 2006). Some studies including the one by (Zongwen et.al, 2012) have provided evidence that DU and PC-3 cells do contain AR Mrna. The article does not mention this underlying contradiction that needs to be addressed to clarify scientific queries. (Fatoumah et.al, 2006) also claimed that after excessive immunoblotting it was found that the relative levels of the AR mRNA and protein that were

detected in DU-145 and PC-3 cell lines were lower than the LNCaP, an AR-positive cell line (Fatoumah et.al, 2006).

(Anthony et.al, 2004) also reported that Even after down-regulation of bcl-2 protein expression by either one of these strategies, the cellular phenotype induced by subsequent G3139 treatment (inhibition of cellular growth and the generation of reactive oxygen species) was essentially identical to that induced in mock-infected or wild-type DU145 cells in which bcl-2 protein expression had not been down-regulated previously (Espy et.al, 2006). This leads to the conclusion that DU 145 cells are not necessarily associated with prolife phenotype and related proteins(Anothony et.al, 2004). This creates further problems in terms of the research results for (Zongwen et.al, 2012).

Also, using new transfection reagents is a very critical decision. Although this might be a step essential to ensure that cells are kept in a protective environment, it also needs to be made sure that the new reagent is not toxic and is not creating optimization issues. The toxicity of the reagent may tamper with the final research results (Goncalves et.al, 2014). To ensure validity and consistency it is recommended to check for authenticity of results on various methods including Oligonucleotide Transfections, siRNA Transfections and Western Blot Analysis that are relevant to protein check in DU 145 cells in one way or the other (Goncalves et.al, 2014) found considerable differences in results once these methods were also used.

(Espy et.al, 2006) conducted a research on the real time PCR equipment and found that whether or not researchers found protein in DU cells also depended on the use of nucleic acid probe formats that support features like excitation and detection wavelengths aand relative thermocycling times (Srikanth et.al, 1999).

To summarize (Zongwen et.al, 2012), it is clear that the researcher should have used the existing theory and should have tried to build on that rather than setting outright to find the existence of protein. Also efforts could have been made to compare Lncap levels with MRNA. No information is provided on the details of the new reagent that might have affected the final results followed by the argument that other techniques including western blot analysis could have been used.

References

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