

Peroxisome Proliferator Activated Receptor- Gamma (PPAR γ) in Ovarian Cancer Cells

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Background and Objectives: PPAR agonists have been used against different cancers and in each, growth inhibition is seen. However, little is known about PPAR γ in ovarian cancer. Since PPAR γ is known to inhibit growth, we propose a major role of PPAR γ in ovarian cancer initiation and progression.

Methods: Ovar3 were plated at 60-90% confluence, treated in serum free media for 24 hours with thiazolidinedione (TZDs): Rosiglitazone(Rosi), Ciglitazone(CGZ) and Troglitazone(TGZ) as well as PMA(Phorbol Ester) and hCG(Human chorionic gonadotropin) . Real time PCR, Westerns, proliferation(MTS and BrdU), cell cycle and annexin assays were performed.

Results: TZD treatments increase the expression of PPAR γ , Rosi by four fold, while CGZ and TGZ by five and twenty three fold respectively. PMA and hCG did not affect expression. Rosi increases protein levels expression. There was a decrease in proliferation at higher concentrations of TZDs in Ovar3 and other cells. A higher fraction of cells treated with CGZ and TGZ are in the G0/G1 phase. Annexin V assay indicates a slight increase in apoptotic cells after 4 hours of treatment with CGZ and TGZ.

Discussion and Conclusion: Our findings demonstrate that PPAR agonists stimulate PPAR expression and decreases cell proliferation suggesting that targeting PPAR γ may be a therapeutic approach in the treatment or prevention of ovarian cancer.

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