

Effect of Vascular Endothelial Growth Factor 165_b and (VEGF165_b) KIT-ligand/Stem Cell Factor (SCF) on Porcine Primordial Follicle Viability *In vitro*

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Vascular endothelial growth factor (VEGF) is well known for its ability to regulate angiogenesis where it has been reported of playing a crucial role in metastasis of tumors. Stem cell factor (SCF) plays a crucial role in cell viability. Growth of a mammalian oocyte starts with an avascular structure called 'primordial follicle' and after the ovulation this avascular structure transforms into a heavily vascular corpus luteum. Primordial follicle activation has been regulated by many intrinsic and extrinsic factors of animals many of which have not been studied completely. It has been observed that the VEGF and its isomers have regulatory effects on the formation of the vascular bed in developing follicles and it supports the activation of the dormant follicles. The objective of the current study was to determine the effect of anti-angiogenic VEGF_{165b} and SCF on porcine primordial follicle development *in vitro*. VEGF_{165b} is known to be an anti-angiogenic factor where SCF is well known for its ability to enhance cell migration, proliferation and cell survival. The tissue samples were treated with VEGF_{165b} with 0ng/ml, 0.1, 1.0, 10.0 ng/ml and day 0 (Negative control) samples were fixed instantly in 10% neutral buffered formalin and each treatment had 6 replicates. Separate three trials were conducted in order to observe the rest of the effects in each treatment. All the treatments were supplemented with 10ng/ml of SCF to stabilize the cell survival. All the treated tissues were subjected to 72hrs of incubation under 5% CO₂ with the humidified atmospheric conditions at 37.5°C and followed by a histological assay to obtain the preliminary data. Out of three different dose regimes in VEGF_{165b} treated tissues, 0.1ng/ml has shown 65.15% of follicle viability. It was numerically the highest recorded viable follicle count whereas 1.0ng/ml and 10.0ng/ml treatments reported 39.61% and 20.20% follicle viability respectively. Although, 0.1ng/ml VEGF_{165b} showed the highest viable follicle count, it was lower than the viability showed in previous study with 0.1ng/ml VEGF_{165a} (92.6%). It was proposed under natural conditions these VEGF isomers and SCF were in an equilibrium which regulated the angiogenesis and anti-angiogenesis. In conclusion it was evident that VEGF_{165b} did not support the follicle viability alone, even with the presence of SCF that stabilized the cell viability. Further studies are necessary for understanding the exact role of VEGF_{165b} and SCF combination effect on cellular viability which may bring new insights into cancer therapy.

Keywords: follicle activation, KIT ligand, VEGF_{165b}, anti-angiogenic factor