

# X ISNIM CONGRESS & III SIPNEI CONGRESS

## **NPY MODULATES HUMAN ADIPOSE STROMAL ADIPOSE CELLS CHEMOTAXIS, ANGIOGENESIS AND WOUND HEALING**

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NPY is a linear polypeptide with 36 amino acid residues, it exerts pleiotropic activities ranging from regulation of cardiovascular and neuroendocrine functions to stimulation of food intake and obesity, by activating multiple Gi/o-protein coupled receptors, designated Y1 through Y5. NPY is able to induce immune activation or suppression, depending on a myriad of factors such as the activated Y receptors and cell types involved. Both sympathetic nerves and immune cells are capable to produce NPY, which has a protective buffering function against the immune challenges exerted by low-level inflammation and contributes to resilience against environmental, inflammatory, and oxidative stress. Our results indicate that adipose-derived stromal cells (ASCs) are stimulated by NPY present in platelet lysate and in fact they express its Y1, Y2 and Y5 receptors, as detected by western blots and immunofluorescence assays. NPY induces angiogenesis and the blocking of NPY through selective Y1, Y2 and Y5 antagonist receptors significantly reduces the angiogenesis ability.

Furthermore, difficult wounds treated by leuco-platelet concentrate (LPC) showed a higher healing percentage than wounds treated with traditional therapies. Immunohistochemistry analysis showed that abundant VEGF was present near the leuco-platelet infiltrated and that abundant newly formed capillaries, characterized by a cubic, "reactive endothelium" were present near the site of LPC infiltration. It is known that NPY stimulates the expression of VEGF and its ability to promote the formation of new capillaries, in vitro as well as in vivo may be related to the challenge VEGF production challenge.

These results suggest that, depending on the specific environment, NPY may exerts positive or on the contrary negative effects. Its proangiogenic activity may promote tissue trophism in ischemic conditions improving wound healing, but may be detrimental when favoring neoangiogenesis within neoplastic tissues.