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Differentiation of bone marrow cells in arthritic mice with decreased complement activity

There is evidence that complement components induce cell migration in mesenchymal stem cells and regulate cytokine production in osteoblastic cells thus playing a regulatory role in normal bone formation. The aim of the present study was to investigate the involvement of complement system in the differentiation of bone marrow cells in complement-depleted model of rheumatoid arthritis (RA). Arthritis was induced by intraarticular injection of zymosan in cobra venom factor (CVF)-treated mice depleted of functional complement. The expression of different markers by bone marrow [1], on fibroblasts (CD29), mesenchymal cells (CD105), dendritic cells (CD14, CD86), osteoclasts (CD265), cells expressing Dectin1 (CD369) and megakaryocytes (CD62P) was determined by flowcytometry. The lack of functional complement activity at the point of arthritis initiation (day 3) lead to an increase of fibroblast and megakaryocyte populations, to a decrease of mature and dectin1 positive populations, while the number of mesenchymal cells was not changed, all compared to arthritic mice. Immunohistochemical staining showed that low complement activity diminished arthritis-induced generation of megakaryocytes and platelets in BM. Chronic inflammation during erosive conditions such as rheumatoid arthritis, leads to dysregulated differentiation and prolif?ration of bone cells, inflammation of synovial membrane and bone marrow, and degradation of cartilage and bone. Present results point that the lack of functional complement changed the ratio between different cell populations that can be used for determining the development and stage of rheumatoid arthritis and can help finding of new therapeutic approaches.

Editorial **Published Date:-2018-10-15 00:00:00**

Endogenous Ligands of Toll Like Receptors: A Danger Signal to the Brain Memory at High Altitude

Sojourn to high altitude may affect various human systems if proper acclimatization not followed. If acclimatization failed, sojourners may suffer with high altitude sickness such as acute mountain sickness (AMS), high altitude pulmonary edema (HAPE) and high altitude cerebral edema (HACE). Although a sojourner’s tolerance to high altitude hypoxia varies according to differences in physiology and physical conditioning. Acute mountain sickness may cause headache, insomnia, dizziness, nausea, vomiting and fatigue. While HACE is more serious stage where brain swelling occurs and it is potentially fatal. A sojourner with HACE may experience confusion, amnesia, delusions, and loss of consciousness. Staying in high altitude (above 9000 feet) environment poses low oxygen supply (hypobaric hypoxia) to the different body organs including brain.

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Expression of C-type Natriuretic Peptide and its Specific Guanylyl Cyclase-Coupled Receptor in Pig Ovarian Granulosa Cells

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Background: C-type natriuretic peptide (CNP) was isolated from porcine brain and is a 22-amino acid peptide which belongs to the natriuretic peptide (NP) family. Even though this peptide shares structural similarity to other endogenous NPs including atrial natriuretic peptide (ANP) and brain natriuretic peptide (BNP) its receptor selectivity is different from other NPs. The present study was undertaken to investigate the expression of C-type natriuretic peptide (CNP) and its specific guanylyl cyclase (GC)-coupled receptor in the granulosa cells of the pig ovarian follicle.

Results: Specific 125I-[Tyr0]-CNP(1-22) binding sites were localized in the granulosa cell layer of the ovarian follicle with an apparent dissociation constant (Kd>) and a maximal binding capacity (Bmax) of 1.41±0.39 nM and 2.75±0.65 fmol/mm2 respectively. Binding of 125I-[Tyr0]-CNP(1-22) to these sites was also prevented by atrial natriuretic peptide (ANP(1-28)), brain natriuretic peptide (BNP(1-26)) and des[Gln18,Ser19,Gly20, Leu21,Gly22] ANP(4-23) (C-ANP). Production of 3',5'-cyclic guanosine monophosphate (cGMP) by particulate GC in the granulosa cell membranes was stimulated by natriuretic peptides (NPs) with a rank order of potency of CNP(1-22)>>BNP(1-26)>ANP(1-28). HS-142-1, a selective antagonist of the two recognized GC-coupled NPRs, inhibited CNP(1-22)-stimulated cGMP production in granulosa cell membranes in a dose-dependent manner. Also mRNAs for all three recognized NPRs were detected in granulosa cells using reverse transcriptase-polymerase chain reaction (RT-PCR). Serial dilution curves of granulosa cell extracts were parallel to the standard curve of synthetic CNP.

Conclusion: These results indicate that CNP and its specific receptor are expressed in the granulosa cells of the pig ovary, and suggest that CNP may be a local autocrine and/or paracrine regulator via activation of its specific GC-coupled receptor, NPR-B.

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[The Immunitary role in chronic prostatitis and growth factors as promoter of BPH]

In the actual medical therapy of BPH, we can see: antibiotics, alpha blockers, 5-ARI, fitotherapeutics/natural products (Serenoa repens) with different which display clinical activities and other molecules such as FANS (local or systemic dosage forms) cortisones and others. Relationship between immune systems and chronic prostatitis are strictly involved in BPH progression. A vicious cycle that involve chronic flogosis, tissue remodeling, grow factors, inhibition of apoptosis, and other phenomena. Observing BPH pathogenesis under an immunologic point of view make possible to search new pharmacological strategies, to improve actual therapy.

The aim of this work is to observe some relevant literature in our opinion related the management of BHP and its progression under a pharmaceutical and immunological point of view. A deep knowledge in the pharmaceutical properties of some molecules (antimicrobials, anti-phlogosis agents, Anti-androgenic agents, alpha blockers, 5-ARI and other treatments, techniques, interventions or instruments) can help the physicians to pick the right choice.
