Mahin Khatami, J Cell Sci Ther 2011, 2:4
doi: http://dx.doi.org/10.4172/2157-7013.S1.01

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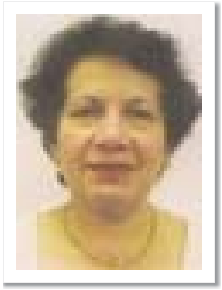

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Unresolved inflammation: Loss of immune and architectural integrity in susceptible target tissues and inflammatory diseases or cancer

Unresolved inflammation was defined as loss of balance between two biologically opposing arms, termed 'Yin' (apoptosis, pro-inflammatory, growth-arresting or 'tumoricidal') and 'Yang' (wound healing, post-inflammatory, growth- promoting or 'tumorigenic') responses of acute inflammation. Unresolved or persistent inflammation could create an 'immunological chaos' or 'immune tsunami' in susceptible affected tissues by inappropriate co-expression of apoptotic and wound healing mediators that would cause damage to cellular components and functions (e.g., altered chromosomal and genetic function, inappropriate expression of cellular and extracellular components, proteins or receptor molecules or defective membrane function). Inflammation-induced alterations in immune dynamics were suggested as fundamental basis for initiation and progression of a wide range of acute (rapid, severe) inflammatory diseases (e.g., sepsis, meningitis, pneumonia), including perhaps drug-induced cancer cachexia and anorexia or a wide range of age-associated chronic autoimmune and neurodegenerative diseases as well as cancer.

Our earlier discoveries (1980's) that were established on experimental models of acute and chronic ocular inflammatory diseases are suggestive of the first evidence for a direct association between inflammation and tumorigenesis. Recent extension of these studies and integration of data led to a first report on inflammation-induced identifiable developmental stages of immune dysfunction that included changes in clinical, histopathological and immunological findings (e.g., tissue edema, tearing, vascular hyperpermeability, neovascularization and angiogenesis, loss of mast cell function-'leaky' MCs , activation of macrophages, site-specific antibody profiles, histamine and prostaglandin synthesis, involvement of goblet cells, thickening and/or thinning of epithelial tissues) that led to induction of massive lymphoid hyperplasia in conjunctival-associated lymphoid tissues (CALTs). Extension, confirmations and validations of these ground-breaking studies are potentially essential steps toward systematic identification of developmental stages of inflammation-induced immune response dysfunction that would lead to carcinogenesis. Inflammation is likely a common denominator in the genesis and progression of chronic diseases and cancer. Promotion of inherent capacity of immune system (immune surveillance) is suggested as key strategies in appropriate designs of clinical trials and effective therapeutics.

Biography

Dr. Mahin Khatami immigrated to USA in 1969 after training in Chemistry (BS) and Science Education (MS) in Iran. She received her MA in Biochemistry from SUNY at Buffalo (1977) and Ph.D. in Molecular Biology from the University Of Pennsylvania (UPA, 1980). Her Postdoctoral framings were in physiology, protein chemistry and immunology at UVA, Fox Chase Cancer Center & UPenn. She became A Faculty of Medicine at Dept. Ophthalmology-UPA until 1992; and in collaboration with a team of scientists, under direction and support of John H Rockey, MD, Ph.D., she quickly earned her supervisory responsibilities on two major projects; cell/molecular biology of diabetic retinopathy/maculopathy and experimental models of acute and chronic inflammatory diseases. As a junior Faculty, she was perhaps a most productive scientist in the country as she published 39 scientific articles and over 60 abstracts in conference proceedings in the first decade of her academic career. Since 1998, at NCI/NIH, extension of her earlier discoveries on immunobiology of inflammatory diseases became closely relevant to her duties as Program Director-HAS for developing concepts for molecular diagnosis ,prevention and therapy of cancer for large clinical Trials (Prostate-Long-Colorectal- Ovarian) and designs of cohort clinical studies. Dr. Khatami has lectured internationally; served as scientific judge; consultant to pharmaceutical companies; research advisor; member of professional societies; editorial member ships & reviewer activities; symposia organizer; president of graduate women In Science, Washington Chapter. Before retiring in 2009, her position title was Assistant Director for Technology Program Development, Office of Technology and Industrial Relations and Program Director-IMAT, Office of Director, NCI/NIH. She is currently Book Editor on Inflammatory Diseases.