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Prolylcarboxypeptidase may contributes to the disease process of prostate cancer

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Prolylcarboxypeptidase (PRCP) metabolizes angiotensin II (Ang II, a pressor agent), angiotensin III (Ang III, a vasoactive peptide), and des-Arg9-bradykinin (BK1-8, a known proinflammatory mediator). Prostate-cell growth involves two subtypes of bradykinin (BK) receptors, B1 and B2. It is also known that kinin receptor-mediated intracellular signaling plays an important role in promoting cell growth, migration, and invasion. B1 is a cell membrane receptor for des-Arg^{9}-BK and des-Arg^{10}- kallidin, an interaction well-known to activate inflammation. Upregulation of B1 is observed in patients with cancer. B1 expression is significantly higher than B2 in the tissue with cancer cells. It has been proposed that the B1 receptor is upregulated by its own agonists (Des-Arg^{9}-BK, des-Arg^{10}-kallidin). An alternative interpretation is that this adaptation can be attributed to the downregulation of PRCP. PRCP-dependent metabolism of angiotensin(s) – and BK1-8 signaling pathway might be involved in the pathogenesis of BPH. We hypothesized that molecular targeting of PRCP-dependent pathways can prevent prostate inflammation and subsequent cancer development. Our findings provide the first evidence that PRCP expression and activity are compromised in cultured prostate cancer cells

Biography

Shariat-Madar has completed his Ph.D from Medical University of Ohio and postdoctoral studies from The University of Michigan School of Medicine. He is the director of University of Mississippi light Microscopy Core. He has published several dozen papers in national and international journals and serving as an editor of Clinical Toxicology and Cardiovascular & Hematological Agents in Medicinal Chemistry.

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