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**Angiotensin receptor blockade resolves cancer induced wounding**

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The lifecycle of Cancer (solid tumours) can usefully be simplified into two phases of the disease; the earlier phase where abnormal growth is a prerequisite for carcinogenesis and the later phase, malignancy, where abnormal growth cannot continue without harnessing the body's wound response. Induced wounding in the stroma provides many benefits to Cancer progression including destruction and remodelling of competing healthy tissue, growth of new blood vessels and immune suppression by tasking infiltrating lymphocytes to participate in the wound response. Furthermore, a review of laboratory and clinical evidence clearly demonstrates that this Cancer induced wounding is critically dependent upon up-regulation of the Angiotensin II type 1 (AT1) receptor.

Classically associated with the cardiovascular system, expression of AT1 occurs through activation of systemic mechanisms of sensing oxidative, hypoxic and sheer stresses. Activation of AT1 by Angiotensin II leads to the production of a host of pro-inflammatory mediators including cytokines, chemokines, adhesion molecules and other factors such as TGF-beta, VEGF and Matrix metalloproteinases.

Early clinical studies utilising Angiotensin Receptor Blockade in Hormone Refractory Prostrate Cancer have demonstrated increased patient survival and significant benefits in pain management. Logically, this approach holds great promise as a means of halting the progression of all solid tumours and in addition should greatly enhance the effectiveness of immune therapy strategies.