Prostate-specific antigen (PSA), considered only an established marker for the detection of prostate cancer, has been identified as a member of the human kallikrein family of serine proteases and now, it is known that PSA is not specific to prostate, semen, and gender. Increased PSA serum levels have been reported also in cardiovascular patients and both elevated as well as diminished PSA have been reported during acute myocardial infarction (AMI). Preliminary observations have concluded that when elevation of prostate-specific antigen occurs during AMI, it seems to relate to a higher occurrence of major adverse cardiac events and that coronary lesions are frequent and often more severe than when a diminution of PSA occurs. Large studies need to be done to confirm these preliminary results but the journey of PSA could be longer than expected.

Prostate-specific antigen (PSA) has been identified as a member of the human kallikrein family of serine proteases, and it has been considered only an established marker for the detection of prostate cancer. Such sources including other malignant and non-malignant non-prostatic diseases are also known to be associated with increased PSA serum levels and now, it is known that PSA is not specific to prostate, semen, and gender. Prostate-specific antigen kallikrein does not seem to have kinin-generating activity. The inactive precursor form of PSA, proPSA, is converted rapidly to active PSA by human kallikrein 2, which has also bradykinin-generating activity. Human kallikrein 2 also activates the single-chain urokinase-type plasminogen activator and forms a complex with plasminogen activator inhibitor-1 too. Other proteases also seem to be involved in the formation of active PSA. It has been suggested that PSA may be an inducer of apoptosis, a negative regulator of growth, an inhibitor of angiogenesis, and a key cytokine that both initiates and terminates tissue repair, and its sustained production underlies the development of tissue fibrosis, particularly after MI. Notably, PSA levels have been reported declined by a statistically significant extent after initiation of statin treatment and regulation of PSA expression has been also reported by an angiotensin II receptor blocker with peroxisome proliferator-activated receptor gamma ligand like action.

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The opinions expressed in this article are not necessarily those of the editors’s of the European Heart Journal or of the European Society of Cardiology

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