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Angiotensin II/BSA

Catalog Number: Y-0274B
AA Seq: DRVYIHPF-BSA
Tags: N/A
Activity: No
Endotoxin: Not analyzed
Storage: Stored at -70°C or -20°C. Avoid repeated freeze/thaw cycles.
Background: As part of the renin-angiotensin-aldosterone-system (RAAS), angiotensin II raises blood pressure by vasoconstriction, increased aldosterone release by the adrenal zona glomerulosa, sodium and water reabsorption in the proximal tubular cells, and vasopressin secretion. The direct action of angiotensin II on surrounding vessel walls is facilitated by binding to the G-protein-coupled angiotensin II receptor type 1 (AT-1) on vascular smooth muscle cells, which stimulates Ca2+/calmodulin-dependent phosphorylation of myosin and causes smooth muscle contraction that results in vasoconstriction . The RAAS is ultimately regulated by a negative feedback effect of angiotensin II on renin production by the juxtaglomerular cells of the renal afferent arteriole. Unresuscitated septic shock associated with marked hypovolemia, extracellular fluid volume depletion, decreased cardiac output, low arterial blood pressure and decreased systemic vascular resistance causes an increase in renin secretion by the juxtaglomerular cells, resulting in elevated angiotensin II plasma levels and an increased secretion of aldosterone from the adrenal cortex. Angiotensin II binding to AT-1 receptors causes dose-dependent vasoconstriction of both afferent and efferent glomerular arterioles. The most pronounced effect of angiotensin II results on efferent arterioles, resulting in reduced renal blood flow and increased glomerular filtration pressure.