

Effect of angiotensin II, ATP, and ionophore A23187 on potassium efflux in adrenal glomerulosa cells

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Angiotensin II stimulus on perfused bovine adrenal glomerulosa cells elicited an increase in ^{86}Rb efflux from cells previously equilibrated with the radioisotope. When ^{45}Ca fluxes were measured under similar conditions, it was observed that Ca and Rb effluxes occurred within the first 30 s of the addition of the hormone and were independent of the presence of external Ca. The ^{86}Rb efflux due to angiotensin II was inhibited by quinine and apamin. The hypothesis that the angiotensin II response is a consequence of an increase in the K permeability of the glomerulosa cell membrane triggered by an increase in cytosolic Ca is supported by the finding that the divalent cation ionophore A23187 also initiated ^{86}Rb or K loss (as measured by an external K electrode). This increased K conductance was also seen with 10^{-4} M ATP. Quinine and apamin greatly reduced the effect of ATP or A23187 on ^{86}Rb or K release in adrenal glomerulosa cells. The results suggest that Ca-dependent K channels or carrie