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ARB drugs and inhibition of adrenal beta-arrestin-1-dependent aldosterone production: Implications for heart failure therapy

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Abstract

Aldosterone mediates many of the physiological and pathophysiological/cardio-toxic effects of angiotensin II (AngII). Its synthesis and secretion from the zona glomerulosa cells of the adrenal cortex, elevated in chronic heart failure (HF), is induced by AngII type 1 receptors (AT_1Rs). The AT_1R is a G protein-coupled receptor, mainly coupling to $G_{q/11}$ proteins. However, it can also signal through βarrestin-1 (βarr1) or -2 (βarr2), both of which mediate G protein-independent signaling. Over the past decade, a second, $G_{q/11}$ protein-independent but βarr1-dependent signaling pathway emanating from the adrenocortical AT_1R and leading to aldosterone production has become appreciated. Thus, it became apparent that AT_1R antagonists that block both pathways equally well are warranted for fully effective aldosterone suppression in HF. This spurred the comparison of all of the currently marketed angiotensin receptor blockers (ARBs, AT_1R antagonists or sartans) at blocking activation of the two signaling modes (G protein-, and βarr1-dependent) at the AngII-activated AT_1R and hence, at suppression of aldosterone in vitro and in vivo. Although all agents are very potent inhibitors of G protein activation at the AT_1R, candesartan and valsartan were uncovered to be the most potent ARBs at blocking βarr activation by AngII and at suppressing aldosterone in vitro and in vivo in post-myocardial infarction HF animals. In contrast, irbesartan

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