

**Table 6. Interfering Medications and Their Effects on Aldosterone and Renin**

<b>Effect on Renin or Aldosterone</b>	<b>Medication</b>
Lower renin	<p><math>\beta</math>-Blockers, central acting alpha 2 agonist, <math>\alpha</math> methyl dopa, NSAIDs</p> <p>Combined estrogen and progesterone-containing OCPs and HRT decrease DRC</p>
Raise renin	<p>MRAs, diuretics including ENaC inhibitors, ARBs, ACE inhibitors, SGLT2 inhibitors</p> <p>Combined estrogen and progesterone-containing OCPs and HRT increase PRA</p> <p>Drospirenone blocks the MR and thus increases PRA and DRC</p>
Lower aldosterone	ARBs, ACE inhibitors, $\beta$ -blockers, central alpha 2 agonist, $\alpha$ methyl dopa
Raise aldosterone	<p>Diuretics*, MRAs</p> <p>Combined estrogen and progesterone-containing OCPs and HRT</p> <p>Drospirenone</p>

\*By promoting natriuresis, diuretics (including MRAs) may induce a rise in aldosterone secondary to a rise in renin/angiotensin II. In the case of thiazide or loop diuretics, however, this may be mitigated by the development of hypokalemia (which inhibits aldosterone production).

ACE, angiotensin-converting enzyme; ARB, angiotensin II–receptor blocker; CCB, calcium-channel blocker; DRC, direct renin concentration; HRT, hormone-replacement therapy; MRA, mineralocorticoid antagonist; OCP; oral contraceptive; PRA, plasma renin activity; SGLT2, sodium-glucose cotransporter 2.; ENaC – Epithelial Sodium Channel inhibitor