Benazepril's ACE inhibitory action hinges on a proline-like core mimicking angiotensin I's transition state, enabling competitive inhibition. A carboxylate group crucial for activity binds the active site's zinc ion; modifications reduce activity until hydrolysis restores the diacid form. An ethyl ester improves oral bioavailability via increased lipophilicity, hydrolyzing in vivo to the active benazeprilat. A phenyl group boosts ACE binding affinity through hydrophobic interactions, while the side chain and benzazepine ring influence pharmacokinetics and binding, respectively.