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Title: The Extended Renin-Angiotensin System – A Promising Target For Traumatic Brain Injury Therapeutics

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COMMENTS TO AUTHORS

Following TBI, activation of angiotensin II type I receptor (AT1R) can promote inflammation, generate reactive oxygen species, increase glial proliferation, and reduce cerebral blood flow. Thus, it stands to reason that blockade or countersignaling of AT1R would reduce damage in the traumatic penumbra. Pre-clinical studies have demonstrated efficacy for Angiotensin II receptor blockers (ARBs) in reducing pathological sequelae of TBI. In this article the authors briefly highlight various RAS modulators and select eRAS ligands with potential as neurotherapeutic agents against TBI. Although it is not new, it is interesting and clinically meaningful and worth sharing.