A new study reveals that renin-angiotensin system (RAS) genes within the amygdala—the brain region important for traumatic memory processing—express differently when the brain develops fearful memories, such as when people undergo traumatic stress. Researchers have found that medication may potentially be used as a pharmacological blockade of the angiotensin type 1 receptor, thereby improving components of fear memory as assessed by freezing behavior. The research team from George Washington University in Washington, D.C., will present their findings virtually at the American Physiological Society's (APS) annual meeting at Experimental Biology 2021.

Post-traumatic stress disorder (PTSD) is a strong predictor of cardiovascular disease (CVD), the leading cause of death in the U.S. The RAS gene, which is critical for blood pressure regulation, is seen as a potentially important link between PTSD and CVD. This study examined the RAS within areas of the brain responsible for processing traumatic or fear-related memories and its effects on cardiovascular regulation. The researchers hoped that it would lead to new treatment and prevention strategies for both PTSD and PTSD-related CVD risk.

Current treatment options for PTSD are limited, and the causes of PTSD-CVD risk is unclear. These preclinical findings shed light on a potential therapeutic target and extend the current understanding for the regulation of brain RAS during fear learning and memory recall processes that are impaired in PTSD.

More information: Abstract title: "Dynamic regulation of brain renin angiotensin system during fear memory reconsolidation"