Peer Reviewed Alzheimer's Research Program (PRARP)

Each year, the Department of Defense's office of the Congressionally Directed Medical Research Programs (CDMRP) assesses scientific opportunities to advance research in specific areas. The investigators supported by individual programs are making significant progress against targeted diseases, conditions, and injuries. This list is not intended to be a full representation of accomplishments, but rather a sampling of the broad portfolio of research and advances resulting from congressional appropriations.

Year	PRARP Research Contributions	Additional Information and Hyperlinks
2011	Novel discovery of peptides which block NF-κB activation/neuroinflammation, blocking further damage to the neurons in mice with Alzheimer's disease-like pathology.	• Rangasamy SB, Corbett GT, et al. 2015. Intranasal delivery of NEMO-binding domain peptide prevents memory loss in a mouse model of Alzheimer's disease. J Alzheimers Dis 47(2):385-402.
2011	Discovery of mutations in the ATP-binding cassette, subfamily A (ABC1), member 7 (ABCA7) gene that may represent a risk factor for late-onset Alzheimer's disease in African Americans.	• Cukier HN, Kunkle BW, et al. 2016. ABCA7 frameshift deletion associated with Alzheimer disease in African Americans. Neurol Genet 2(3):e79.
2011	TBI in older Veterans was associated with a 60% increase in the risk of developing dementia over 9 years after accounting for competing risks and potential confounders. The results suggest that TBI in older Veterans may predispose toward development of symptomatic dementia and raise concern about the potential long-term consequences of TBI in younger Veterans and civilians.	• Barnes DE, Kaup A, et al. 2014. Traumatic brain injury and risk of dementia in older veterans. Neurology 83(4):312-9.
2012	Preliminary evidence (in mice) that increased Retinoid X receptor (RXR) activity improves cognitive deficits and amyloid clearance. This process involves APOE isoforms, Rho GTPases, and Wnt signaling. The increased Wnt signaling may be beneficial to several essential neurological processes, such as neuronal cell differentiation, plasticity, and regulation of neuroinflammation.	• Nam KN, Mounier A, et al. 2016. RXR controlled regulatory networks identified in mouse brain counteract deleterious effects of Aβ oligomers. Sci Rep 6:24048. • Gyoneva S, Kim D, et al. 2015. Ccr2 deletion dissociates cavity size and tau pathology after mild traumatic brain injury. J Neuroinflammation 12:228.
2013	Development of a new strategy for characterizing toxic tau species associated with Alzheimer's disease and traumatic brain injury.	• Tian H, Davidowitz E, et al. 2015. Isolation and characterization of antibody fragments selective for toxic oligomeric tau. Neurobiol Aging 36(3):1342–55. • Williams S, Schulz P, and Sierks MR. 2015. A sensitive phage-based capture ELISA for sub-femtomolar detection of protein variants directly from biological samples. Biotechnol Prog 31(1):289-98.