BIOGRAPHICAL SKETCH

Provide the following information for the Senior/key personnel and other significant contributors.

NAME: Strain, Jeremy

eRA COMMONS USER NAME: JSTRAIN:

POSITION TITLE: Postdoctoral Research Associate

EDUCATION/TRAINING (Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable.)

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>Completion Date MM/YYYY</th>
<th>FIELD OF STUDY</th>
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</thead>
<tbody>
<tr>
<td>University of Texas at Dallas, Richardson, TX</td>
<td>BA</td>
<td>2010</td>
<td>Neuroscience</td>
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<tr>
<td>University of Texas at Dallas, Richardson, TX</td>
<td>PhD</td>
<td>2015</td>
<td>Cognitive Neuroscience</td>
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A. PERSONAL STATEMENT

After choosing a career in neuroscience my primary focus evolved into utilizing multimodality neuroimaging techniques to map patterns of disease progression. During my graduate training at the University of Texas at Dallas I studied neuroimaging correlates of cognitive sequelae in former NFL athletes. Some of the work we produced were the first comprehensive examinations of concussions in this population. We identified behavioral brain relationships that associated with history of concussion, spurred two publications (Strain et al., 2015; Strain et al., 2016) and an invitation to speak at an international conference. Within this same population, I also initiated a project that focused on somatic depression symptoms that correlated with decreased white matter structural integrity (Strain et al., 2013). This paper helped highlight the emotional disturbances that plagued this population and brought the topic to the forefront at academic conferences.

During my time at Washington University my primary focus has involved detecting subtle inflammatory changes in aviremic HIV+ patients. Utilizing novel neuroimaging techniques that can in-vivo isolate correlates of inflammation in HIV+ patients. The technique, diffusion basis spectral imaging (DBSI), can deconvolve the diffusion signal into various meaningful anatomical correlations. This concept was the basis for a paper, that is currently under review, that compared levels of inflammation derived from DBSI between aviremic HIV+ and HIV-. The findings from the initial study laid the foundation for the aims proposed in this grant. Combining this novel approach with other imaging modalities is ideal for observing disease severity in HIV patients that have already attained viral suppression.


virologically well controlled HIV+ patients. *JAIDS*. 76:423-430. PMCID: PMC5659935


**B. POSITIONS AND HONORS**

**Positions and Employment**

2015 - Postdoctoral Research Associate, Washington University in Saint Louis, Neurology, Radiology, BME, Saint Louis, MO

**C. Contribution to Science**

**Neuroinfectious diseases:**

1. My first experience with neuroinfectious disease was evaluating longitudinal data to follow the prognosis of individuals infected by West Nile Virus at different stages. This led to an interest in the effects of infection on cognition. This introductory project evolved into a desire to understand the neurobiology of HIV and the neuroanatomical mechanisms that disrupt cognition in that population. Residual inflammation is a known entity in people living with HIV (PLWH). However methods of detecting inflammation within the brain are secondary with current methods that involve invasive blood draws to determine inflammation activity. Utilizing diffusion based techniques that isolate restricted patterns of diffusion we provided evidence of global inflammation throughout the brain in PLWH. We showed that standard DTI metrics can be influenced by this inflammation and that no white matter changes were observed when the impact of cellularity was included. In a secondary project we sought to examine biomarkers of AD in an HIV population to determine if the degenerative cognitive deficits present in that population correspond with PET tau markers. We observed no relationship between cognitive deficits or effects of HIV that associated with tau implicating a separate pathology from AD.


**Traumatic Injury:**
2. When evaluating traumatic injury chronic neurobehavioral sequelae from concussions range from good recovery to cognitive dysfunction and mood symptoms to post-concussive syndromes. Traumatic brain injury has been reported as a risk factor for dementia but most individuals who experience a concussion suffer from no symptoms. The rational behind who develops these cognitive or mood impairments is unknown. I have utilized various neuroimaging markers to determine anatomical changes associated with symptom presentation.

**Neuroimaging Correlates of Cognitive Decline:**
3. The techniques I have developed from infectious diseases and traumatic injury have also been applied to understanding brain behavior relationships in numerous other diseases. Recently, I utilized positron emission tomography to identify the spatial topography of tau pathology in Alzheimer’s Disease (AD) and how it relates to white matter damage. Both demyelination and wallerian degeneration co-occur in this population and we sought to determine whether this loss in structural integrity associated with tau accumulation. I was also pivotal in an intervention project that assessed neuroimaging correlates of strategic training to bolster/preserve cognitive reserve to counteract declining performance in normal aging and AD. In addition to AD, I have been involved in several collaborative projects involving patients with Multiple Sclerosis to understand what factors contribute to disruption of the BOLD signal and white matter changes.


### Additional Information: Research Support and or Scholastic Performance

### Ongoing Research Support

**Career Fellowship Grant A2018817F** Strain (PI) 08/01/18-08/01/20

White matter hyperintensity localization and severity in association with PET tau in Alzheimer Disease. This grant focuses on comparing and contrasting autosomal dominant AD with sporadic AD on associations of white matter injury in relation to phosphorylated tau from PET imaging.