TACSM Abstract

Voluntary Exercise Reduces Alzheimer's-like Pathology After Inflammation in Mice

Marielle K. Weintraub[2], Jordon D. White[2], Stephanie M. Turner[1], Samantha L. Hodges[2], Amanda L. Morin[2], Lindsey N. Sadler[2], Gary W. Boehm[2], Michael J. Chumley[1]

[1]Dr. Michael J. Chumley Laboratory, Department of Biology; Texas Christian University; Fort Worth, TX

[2]Dr. Gary W. Bohem Laboratory, Department of Psychology; Texas Christian University; Fort Worth, TX

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Advisor/Mentor: Chumley, Michael (m.chumley@tcu.edu) & Bohem, Gary (g.boehm@tcu.edu)

ABSTRACT

Current global statistics estimate that 44.4 million people are afflicted with dementia, and that 50%-75% of these patients suffer from Alzheimer's disease (AD; Prince et al. 2013). AD, a progressive disorder categorized by neuronal and behavioral deterioration, is the 6th leading cause of death in America (Alz facts and figure 2012). One hallmark pathology of AD is the presence of amyloid-beta (Aβ) in the brain, which can limit cell-to-cell communication, leading to cognitive deficits, and neuronal cell death. Although the exact origins of this disease still remain unknown, one possible catalyst of AD pathology is inflammation. Our lab has previously shown that 7 consecutive peripheral injections of a bacterial mimetic led to systemic inflammation, increased levels of Aβ in the brain, and cognitive dysfunction (Kahn et al., 2012; Weintraub et al., 2013). Currently there are very few effective treatments that diminish AD symptomology. One documented way to decrease inflammation without the use of pharmaceuticals is through regular physical exercise (Cho et al., 2003; Cotman & Berchtold, 2002; Cotman et al., 2007). The present study tested the hypothesis that voluntary exercise would decrease the level of brain Aβ following inflammation. Interestingly, we found that two weeks of voluntary wheel running after inflammation led to a reduction of Aβ when compared to sedentary recovery. These results indicate that exercise may be an effective modality to reduce AD-like pathology, and that these effects appear to be facilitated by higher versus lower levels of exercise, as measured by total distance run.