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Vascular Dysfunction and Posttraumatic Stress Disorder: Examining the Role of Oxidative Stress and Sympathetic Activity

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Purpose

The physiological manifestations of posttraumatic stress disorder (PTSD) have been associated with an increased risk of cardiovascular disease (CVD) independent of negative lifestyle factors. The goal of the study was to better elucidate the mechanisms behind the increased CVD risk by examining peripheral vascular function, a precursor to CVD in young, otherwise healthy adults. Moreover, this study sought to determine the role of oxidative stress and sympathetic nervous system (SNS) activity in PTSD-induced vascular dysfunction.

Methods

Subjects

Thirty-five subjects, fourteen individuals with PTSD (PTSD; *n*=14, 10 women, 6 men; age 24 ± 1 years) and twenty-one healthy controls (CTRL; n=21, 15 women, 9 men, 22 ± 1 years), participated in the study.

Study visits

The PTSD group participated in two visits, consuming either a placebo or antioxidant cocktail (AO - vitamins C and E and alpha-lipoic acid (ALA)) prior to their visits, in a randomized order.

Sympathetic Nervous System Activity Measures

To assess sympathetic nervous system activity, heart rate variability (HRV) was recorded using a 3-lead ECG system. The low frequency (LF) to high frequency (HF) ratio was analyzed to evaluate the sympathovagal balance.

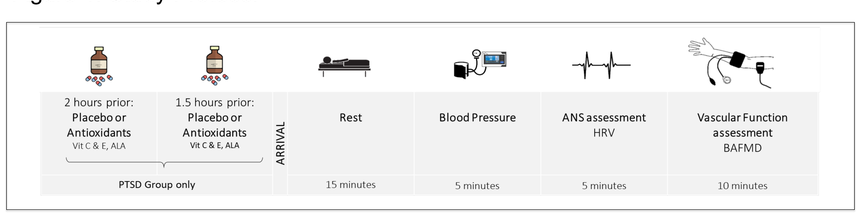
Vascular Function Measures

Peripheral vascular function was assessed in the brachial artery (BA) of the right arm via the reactive hyperemia-induced flow mediated dilation (FMD) technique utilizing a Doppler ultrasonography system. BA and arm microvascular function were determined by percent change of diameter from baseline normalized for shear rate (FMD/Shear), and blood flow area under the curve (BF AUC), respectively.

Statistical analysis

Student's t-test was conducted for within group and between group comparisons.

Figure 1. Study Protocol



Results

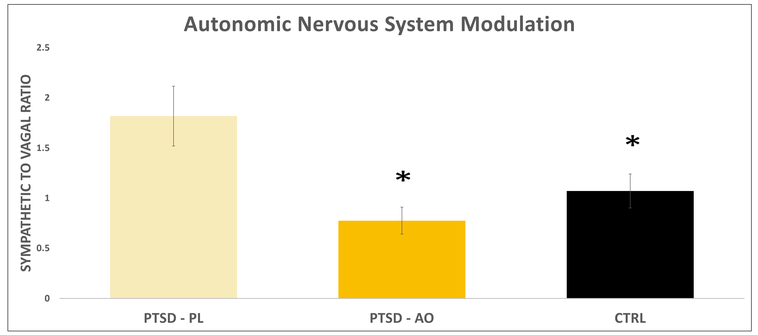


Figure 2: Sympathetic to Vagal Ratio. Data expressed as mean ± SEM
* - significantly different from PTSD-PL. `

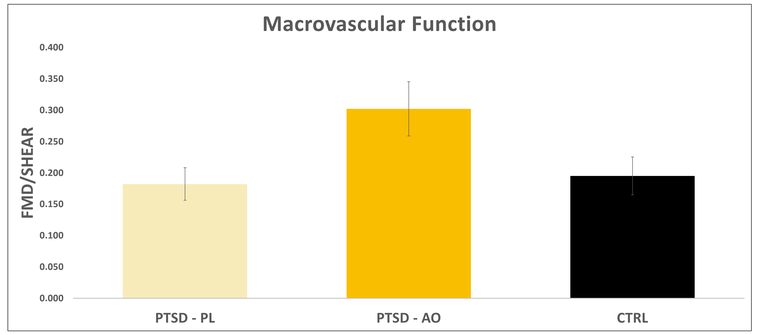


Figure 3: FMD/Shear. Data expressed as mean ± SEM.

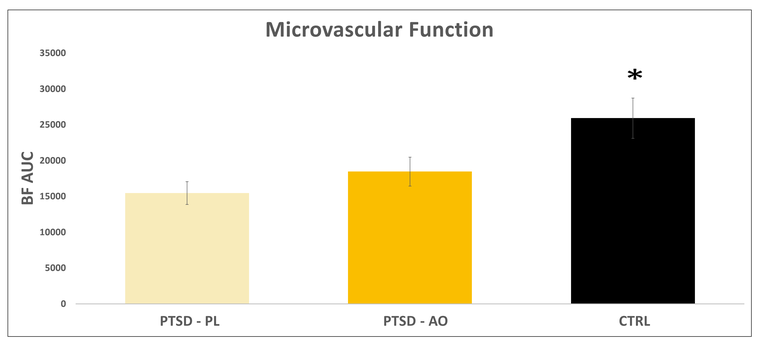


Figure 4: FMD BF AUC. Data expressed as mean ± SEM.
* - significantly different from PTSD-PL.

Table 1. Subject Characteristics (Mean ± SEM)

Variable	PTSD (n=14)	CTRL (n=21)
Sex	10 Female, 4 Male	15 Female; 6 Male
Age (years)	24 ± 1	22 ± 1
Height (cm)	166 ± 3	168 ± 2
Weight (kg)	64 ± 1	71± 3
BMI	23 ± 2	25 ± 3
BF (%)	26 ± 3	24 ± 2

Table 2. Vascular Function Measures (Mean ± SEM)

Variable	PTSD (PL)	PTSD (AO)	CTRL
Symp to Vagal Ratio	0.776 ± 0.134	0.776 ± 0.134	1.071 ± 0.169
FMD/Shear	0.182 ± 0.026	0.302 ± .043	0.195 ± 0.030
BA BF AUC	15468 ± 1593	18461 ± 2016	25915 ± 2814

Results/Conclusion

- SNS activity was significantly higher (p = 0.03) in the PTSD group when compared to the controls but was significantly reduced (p = .003) following the AO supplementation to which the difference between PTSD and control group was no longer significant (p=.20).
- Brachial artery function was not different between groups, but microvascular function (evaluated as BA BF AUC) was significantly lower in the PTSD group when compared to controls. Following the acute AO supplementation, BF AUC was augmented to which it was no longer significantly different (p = 0.13) when compared to the control group.
- Taken together, PTSD had higher sympathetic activity and lower microvascular function when compared to controls. Following AO supplementation, individuals with PTSD reported attenuated microvascular dysfunction and SNS activity to similar levels as the control group.
- Therefore, modulation of oxidative stress, via an acute AO supplementation, improved microvascular dysfunction in individuals with PTSD, potentially by reducing the substantial SNS activity associated with this disorder.

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