

# Rapid Reduction in Posttraumatic Stress Disorder Symptom Severity after Three Fascial Counterstrain Manual Treatments: A Proof-of-Concept Study

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## ABSTRACT

### Introduction:

Posttraumatic stress disorder (PTSD) is associated with hyperactivity of the sympathetic nervous system (SNS) and elevated levels of central and peripheral pro-inflammatory biomarkers. Fascial Counterstrain (FCS) is a manual therapy technique purported to reduce inflammation and neuroexcitation by decreasing the concentration of pro-inflammatory mediators in the interstitial tissues. We hypothesized that a short course of FCS treatment that specifically targets the interstitium surrounding the SNS will result in a decrease in the severity of PTSD symptoms.

### Materials and Methods:

Twenty-four consenting volunteers with a baseline Clinician-Administered PTSD Scale for DSM-5 (CAPS-5) score  $\geq 11$  were randomized to either an FCS treatment group ( $n=13$ ) or waitlist control group ( $n=11$ ) after obtaining informed consent as approved by Solutions IRB. Changes in PTSD symptom severity were assessed after three 1.5-hour FCS treatments in 11 subjects in the treatment group and compared to 11 waitlist controls using a piecewise linear mixed-effects regression model. Changes in self-reported depression, anxiety, somatic symptoms, life satisfaction, and subjective measure of overall improvement were also assessed.

### Results:

Three 1.5-hour FCS treatments administered over an average duration of 19.5 days [95% CI (15.2-23.8)] resulted in a significantly greater reduction in PTSD symptom severity in the FCS treatment group compared to the waitlist control group [CAPS-5 scores: 18.7-point decrease, 95% CI (8.7-28.7) vs. 1.3-point decrease, 95% CI (0.7-1.9); main effect for FCS treatment:  $P < .001$ ; FCS group vs. control group comparison:  $P = .0017$ ]. Compared to waitlist controls, the FCS treatment group also had statistically significant decreases in self-reported depression, anxiety, and somatic symptoms in addition to increases in self-reported overall improvement and life satisfaction.

### Conclusion:

This proof-of-concept study is the first to demonstrate that a short course of FCS manual therapy targeting the SNS resulted in a rapid and statistically significant reduction in clinician-rated PTSD symptom severity, as well as patient-reported depression, anxiety, and somatic symptoms. Large, randomized studies in PTSD should compare the effectiveness of FCS manual therapy to evidence-based psychotherapies or pharmacotherapies to evaluate its potential as a rapid, cost-effective, and scalable public health intervention.

### Clinical Trial Registration:

IRB Registration # IORG0007116.

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## INTRODUCTION

Posttraumatic stress disorder (PTSD) and associated comorbid mental health and medical disorders can negatively impact force readiness.<sup>1</sup> Despite the substantial burden to individuals, families, and communities, only a minority of individuals with PTSD receive treatment,<sup>2</sup> and the rates of dropout from treatments are high.<sup>3</sup> In addition, the rates of clinical response and remission are low, and PTSD symptoms persist despite completion of evidence-based treatment.<sup>4</sup> The annual economic burden of PTSD in the United States was assessed at \$232.2 billion, with estimates of \$19,630 per individual with PTSD<sup>5</sup> further supporting the urgent need for innovative, rapidly effective treatment options that are not only well tolerated, but broadly accepted by individuals with PTSD.

Hyperactivity of the sympathetic nervous system (SNS)<sup>6</sup> in PTSD together with a dysregulated hypothalamic pituitary adrenal (HPA) axis,<sup>7</sup> attenuated parasympathetic responses, and increased levels of pro-inflammatory biomarkers<sup>8</sup> have been proposed as a potential explanation for the increased risk of cardiovascular disease,<sup>9</sup> chronic pain,<sup>10</sup> and autoimmune disorders<sup>11</sup> in these patients.

Pharmacological<sup>12</sup> and non-pharmacological<sup>13</sup> interventions that decrease activity of the SNS have been explored as potential treatment options for PTSD. Stellate ganglion blockade (SGB) decreases central and peripheral sympathetic tone and has been used to treat sympathetically mediated regional pain syndromes, chronic fatigue, long COVID, and ventricular tachyarrhythmias.<sup>14</sup> Stellate ganglion blockade has also shown promise as an adjunctive treatment in patients with refractory PTSD.<sup>15</sup>

Fascial Counterstrain (FCS) is a manual therapy for pain and somatic dysfunction that is based on Strain-Counterstrain osteopathic principles,<sup>16</sup> and may have potential therapeutic application in PTSD. Although the physiological mechanisms underlying FCS have not been fully elucidated, the targeted tissue manipulation is purported to facilitate regional lymphatic drainage, reduce the levels of proinflammatory mediators in the interstitium,<sup>17</sup> and potentially increase the activation threshold of sympathetic neurons.<sup>18</sup> Because PTSD is associated with both elevated levels of proinflammatory cytokines and SNS hyperactivity, we hypothesized that an FCS protocol designed to reduce inflammation in the neuronal sheaths of all pre- and postganglionic sympathetic pathways will improve PTSD symptoms.

## METHODS

All procedures were approved by the Solutions Institutional Review Board (IRB) (IRB Registration #: IORG0007116 Federal wide Assurance #: IRB00008523). All subjects signed IRB-approved written informed consent, completed a baseline screening to determine eligibility, and were randomly assigned to FCS treatment group or waitlist control group. See CONSORT flow chart (Figure 1).

### Subjects

The study targeted first responders and veterans with a history of trauma exposure and PTSD symptoms. Interested subjects were referred by their primary care physicians, physical therapists or massage therapists or responded to fliers placed in the community. Subjects did not receive monetary compensation for their time and were not charged for the FCS treatment.

### Inclusion and Exclusion Criteria

Inclusion criteria were (1) Ability to provide informed consent, (2) First responders or veterans who experienced one or more potentially traumatic events (PTE) identified by the Life Events Checklist for DSM-5 (LEC-5),<sup>19</sup> (3) Volunteers who had a Clinician Administered PTSD Scale for DSM-5 past month version Clinician-Administered PTSD Scale for DSM-5 (CAPS-5)<sup>20</sup> score  $\geq 11$  (corresponds to  $\geq$  mild PTSD symptoms); (Frank

Weathers personal communication 2023), (4) willingness to receive 3 FCS treatments, and (5) willingness to maintain stable doses of pharmacotherapy or psychotherapy for PTSD and/or depression before and during the study.

Exclusion criteria were as follows: (1) Subjects who had a CAPS-5 score  $\leq 10$ , (2) Subjects who had a history of receiving FCS treatment in the past, and (3) Subjects with a history of sexual assault were excluded given the manual nature of the FCS intervention.

### Procedures

Subjects who provided informed consent were screened using LEC-5 to assess exposure to PTEs in a respondents' lifetime. A trained, doctorate level psychologist administered the CAPS-5 for all subjects who met criteria for a qualifying PTE. Subjects with a baseline (T0) CAPS-5 score  $\geq 11$  were randomized to either the treatment group or waitlist control group using the website [www.randomizer.org](http://www.randomizer.org). For the waitlist control group, the CAPS-5 and other psychometric measures were scheduled to be repeated at 1 month (T1), 3 months (T2), and 6 months (T3) after the baseline (T0) assessments. After baseline psychometric assessments at T0, the FCS group received three 1.5-hour FCS treatments followed by repeat assessments at 1 month (T1), 3 months (T2), and 6 months (T3) after the last FCS treatment. All subjects also completed self-reported questionnaires to report changes in depression, anxiety, somatic symptoms, life satisfaction, and subjective report of overall improvement at the same time points as the CAPS-5 interviews. The psychologist was blind to group assignment of the subjects. Changes to participants' pharmacotherapy or psychotherapy were noted in the study visits.

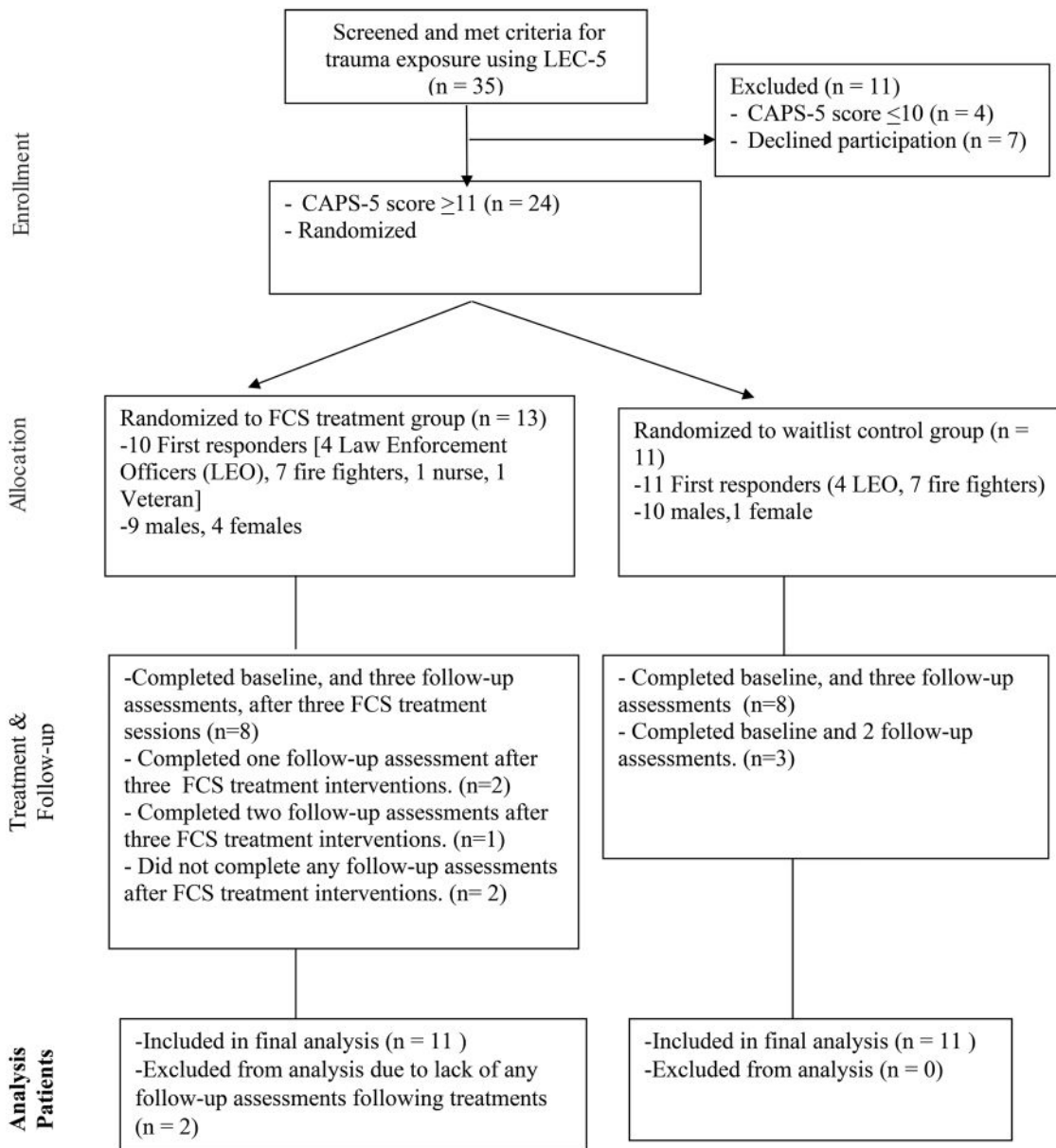
### Primary Outcome Measure

The primary outcome measure in this study was a change in PTSD severity measured by a change in CAPS-5 score. To aid clinical interpretation, response rates ( $\geq 10$  point decrease from baseline CAPS-5 score)<sup>21</sup> were also compared between the FCS treatment and the waitlist control group.

### Secondary Outcome Measures

All subjects completed the following self-reported psychometric measures at T0, T1, T2, and T3:

1. Beck Depression Inventory (BDI)<sup>22</sup> and the Patient Health Questionnaire-9 (PHQ-9)<sup>23</sup> to measure changes in the severity of depressive symptoms.
2. Patient Health Questionnaire-15 (PHQ-15)<sup>24</sup> to assess somatic symptom severity.
3. Generalized Anxiety Disorder-7 (GAD-7)<sup>25</sup> to assess changes in anxiety.
4. Satisfaction with Life Scale (SWLS)<sup>26</sup> to measure subjective happiness.
5. Pain Self-Efficacy Questionnaire (PSEQ)<sup>27</sup> to assess confidence in ability to do tasks and activities despite pain.



Abbreviations: Fascial Counterstrain (FCS)

Figure 1. Consort chart. Abbreviation: FCS, fascial counterstrain.

6. The Patient Global Impression of Change (PGIC) was administered only in the FCS treatment group at T1, T2, and T3 to measure subjective overall improvement after treatment.<sup>28</sup>

**Fascial Counterstrain Protocol**

The Fascial Counterstrain treatment group received three 90-minute FCS treatments after their baseline measures (T0). The Fascial Counterstrain Protocol used in this study targets the SNS and was provided by 1 of 2 certified practitioners (a physical therapist or a naturopathic physician). The waitlist control group were offered 3 FCS treatments after completion

of the study. The 2 FCS practitioners were also blinded to subjects’ assignment to either the FCS treatment or waitlist control group. Fascial Counterstrain treatments start with an assessment phase which involves identification of diagnostic, cutaneous tender points (TPs). Tender points are defined as tense, tender, edematous, cutaneous masses approximately 1 cm in diameter<sup>29</sup> considered to be a result of convergent spinal, nociceptive pathways that convey neuronal signals generated by deep tissue inflammation.<sup>17,30</sup> Surface TPs identified in the SNS FCS protocol were named according to the corresponding deep tissue inflammatory structures that generate the surface TP, including inflammation surrounding pre and postganglionic sympathetic neurons and ganglia.

Following the identification of TPs, the second phase involves treatment of the underlying areas of inflammation to facilitate regional lymphatic drainage, resulting in a decrease in the concentration of interstitial inflammatory mediators.<sup>17</sup> Fascial Counterstrain treatments involve manually shortening of the involved tissues to create a deep tissue “vacuum.” Each treatment position or “release” is then held for 30-45 seconds, to maximize the drainage of inflammatory mediators into the larger, regional lymphatic vessels. A successful FCS treatment is confirmed by a substantial improvement in post-treatment deep tissue mobility in the area of primary inflammation in addition to substantial, and lasting reduction in surface TP sensitivity.

### Structures Targeted by the Fascial Counterstrain Protocol

The Fascial Counterstrain protocol targets the entire SNS which is a 2-neuron, efferent pathway that includes both the pre and post ganglionic sympathetic neurons and ganglia. Preganglionic sympathetic neurons originate in the intermediolateral column of the spinal cord and extend from the first thoracic to the second lumbar spinal cord segments. These preganglionic neurons synapse onto postganglionic neurons located in the paravertebral ganglia (sympathetic chain ganglion) or the prevertebral ganglia (collateral ganglion). The postganglionic efferent neurons release norepinephrine in target organs, stimulating adrenergic receptors which control each organ’s individual “fight or flight” response. One exception to the 2-neuron sympathetic pathway design, is the preganglionic neurons that directly innervate the adrenal medulla and result in a rapid release of epinephrine and norepinephrine into the bloodstream when activated. In addition, the SNS has a bidirectional relationship with the brainstem, limbic system, and prefrontal cortex through a complex neural and hormonal network which helps the body maintain homeostasis and mount a rapid response to real and perceived threats.<sup>31</sup>

The Fascial Counterstrain treatment protocol used in this study specifically targeted the sympathetic neural pathways, originating from the first thoracic to the second lumbar spinal segments, including the direct preganglionic branches to the adrenal medulla. The Fascial Counterstrain treatments also addressed the postganglionic sympathetic pathways that innervate target organs from the first cervical spinal segments down to the sacrococcygeal junction. For a complete list of the FCS TPs and treatments included in this study, please see FCS treatment manuals.<sup>32,33</sup>

### Statistical Analysis

There was considerable variability in the timing of assessments for both the FCS treatment group and the waitlist control group. For instance, the interval from baseline (T0) to the final follow-up (T3) assessment ranged from 5.7 to 11.2 months in the FCS treatment group and from 5.9 to 7.6 months in the waitlist control group. In addition, among participants in the FCS group, the intervals between the first, second, and third

treatment sessions varied considerably. This variability made it necessary to distinguish the effects of the FCS intervention from changes that might occur spontaneously over time, as indicated by the changes in outcome measures observed in the waitlist control group. We used a piecewise linear mixed-effects regression model to isolate and evaluate the effects of FCS treatment on primary and secondary outcomes, while accounting for variations in the timing of assessments and FCS treatments across participants.

The model is based on the following core assumptions:

1. Pre-treatment trend: Before FCS treatment, both the FCS and control groups are assumed to follow a shared linear trend in primary and secondary outcome scores. The pre-treatment trend in the waitlist control group estimated using data from baseline (T0) to the final follow-up (T3) is applied to the pre-treatment period from baseline (T0) until the time of the first FCS treatment for the FCS treatment group.
2. Post-treatment trend: Following the last FCS treatment, patients in the FCS treatment group are assumed to follow a potentially different linear trajectory, which may or may not match the natural progression observed in the waitlist control group.
3. Fascial Counterstrain Treatment effect (discontinuity): The model identifies the treatment effect by measuring the average change in primary and secondary outcomes immediately before and after the 3 FCS treatments. To isolate this effect, we collapsed the days between the first and last FCS treatment into a single time point effectively removing the duration of treatment from the timeline. The corresponding potential discontinuity in the individual score trajectories, and the size of this discontinuity, represents the primary treatment effect.

In the Fascial Counterstrain treatment group, symptom scores were modeled as 2 distinct linear phases: a pre-treatment trend and a post-treatment trend, separated by a discontinuity representing the treatment effect. To standardize the analysis, each patient’s timeline was realigned so that the date of their first treatment was set as time zero. For the waitlist control group, changes in symptoms were modeled as a single continuous linear trend, with timelines aligned to the first follow-up after baseline (T1), which was set to time zero.

This modeling strategy allows for robust estimation of the impact of FCS treatment while controlling for individual differences in follow-up intervals.

The regression formula for the mean outcome measure as function of time was:

$$\text{Outcome measure}(t) = \beta_0 + \beta_1 \cdot t + \beta_2 \cdot I_{\text{group}} + \beta_3 \cdot I_{\text{FCS}} + \beta_4 \cdot I_{\text{FCS}} \cdot t$$

Here,  $\beta_i$ 's are regression coefficients,  $t$  is time,  $I_{\text{group}}$  is a group identifier (control vs. treatment), and  $I_{\text{FCS}}$  is a treatment indicator (treatment was not received vs. treatment was received).  $\beta_1$  is the pre-treatment rate (change in the score per month),

and  $\beta_4$  is the change in the pre-treatment rate after patients receive their treatment. The sum of  $\beta_1$  and  $\beta_4$  is the post-treatment rate.  $\beta_3$  is the discontinuity in the average score which refers to the main effect of treatment. The overall regression model includes a random intercept for each patient, and an additional random intercept for each treated patient at time-points after the treatment was received. This additional random intercept accounts for the different number of days removed from individual timelines and for the different individual responses to treatment. This model is applied to both primary and all the secondary outcomes, and includes all non-missing data for both waitlist controls and FCS treatment groups.

According to the model, the main effect of treatment was the change in primary and secondary outcomes in the FCS group immediately before and after the course of the 3 FCS treatments. To ensure that the changes in outcomes between the groups were compared over the same time frame, the model calculated the difference between the average change in outcomes immediately before and after 3 FCS sessions in the FCS treatment group ( $\beta_3$ ) and compared it to the average change in the waitlist control group, scaled to the 19.5-day period  $\beta_1^* = 0.64 \times \beta_1$  (19.5 days is 64% of an average month). This approach allows for direct statistical comparison of changes in outcomes over equivalent durations in FCS treatment group and waitlist controls.

## RESULTS

Baseline characteristics for the FCS treatment and waitlist control groups of subjects are presented in **Table 1**. Although the mean CAPS-5 score at baseline was 9 points higher in the FCS group compared to the waitlist control group, the difference

**Table 1.** Sample Characteristics and Outcome Measures Evaluated at the Baseline

Baseline characteristic	Control group	Treatment group	All subjects
Sample size, n	11	11	22
Gender, n (%)			
Female	1 (9%)	3 (27%)	4 (18%)
Male	10 (91%)	8 (73%)	18 (82%)
Age, mean (SD)	47.1 (7.7)	44.8 (11.5)	45.9 (9.7)
Outcome measure, mean (SD)			
CAPS-5	28.2 (9.8)	37.2 (17.4)	33.7 (14.5)
BDI	14.8 (4.4)	18.8 (11.9)	16.7 (8.8)
PHQ-9	6.9 (3.9)	10.0 (7.0)	8.4 (5.7)
PHQ-15	8.3 (4.6)	9.7 (4.9)	8.9 (4.7)
GAD-7	4.6 (3.3)	9.2 (6.5)	6.8 (5.5)
SWLS	14.7 (6.6)	11.2 (6.4)	13.1 (6.6)
PSEQ	47.2 (8.4)	45.9 (8.7)	46.6 (8.4)

Abbreviations: CAPS-5, Clinician-Administered PTSD Scale for DSM-5; BDI, Beck Depression Inventory; GAD-7, Generalized Anxiety Disorder-7; PSEQ, Pain Self-Efficacy Questionnaire; PHQ-15, Patient Health Questionnaire-15; PHQ-9, Patient Health Questionnaire-9; SWLS, Satisfaction with Life Scale.

was not statistically significant ( $P = .15$ , unequal variance  $t$ -test).

### Primary Outcome Measure: CAPS-5

Preliminary inspection of the CAPS-5 data (**Figure 2**) confirmed that the FCS treatment group had a substantial decrease in PTSD severity 1 month after treatment (T1) compared to the waitlist controls. The decrease in CAPS-5 score was sustained at least for 6 months after FCS treatment.

The results from the piece-wise linear mixed-effects regression model including significance levels for within and between group comparisons for primary and secondary outcome measures are listed in **Table 2**. **Figure 3A and B** depicts the changes in CAPS-5 scores during repeated assessments at the individual subject level for the waitlist control group and FCS treatment group respectively. **Figure 3B** illustrates changes in CAPS-5 scores in the FCS treatment group before and after FCS treatment, as determined by the model.

Three FCS treatments administered over an average of 19.5 days [95% CI (15.2-23.8)] resulted in a significantly greater reduction in CAPS-5 scores in the FCS treatment group compared to the waitlist control group [18.7-point decrease, 95% CI (8.7-28.7) vs. 1.3-point decrease, 95% CI (0.7-1.9); main effect for FCS treatment:  $P < .001$ ; FCS group vs control group comparison:  $P = .0017$ ], (**Table 2** and **Figure 3**). The slopes of pre- and post-treatment splines in the FCS group were not significantly different and correspond to approximately 2.0-point decrease in CAPS-5 score per month (**Figure 3B**).

Subjects were also compared using the 5-level categorical classification of PTSD, a scale which ranges from asymptomatic to extreme PTSD. There were 20 patients (10 controls, 10 treated) who could be evaluated by the change in severity category between baseline and the 1-month assessment. In both groups, no patient moved to a worse category. Among the controls, 2 patients improved by at least 1 category, and 8 stayed in the same category. In the FCS treatment group, all 10 patients improved by at least 1 category and no patients remained in the same category (20% improvement in controls versus the 100% improvement in FCS treatment group; Fisher's Exact test,  $P = .002$ ). Clinically meaningful response or improvement in PTSD symptoms defined as a 10-point decrease in CAPS-5 severity, was seen in 80% of the FCS treatment group compared to a 20% response rate in the waitlist group (Fisher's Exact test,  $P = .057$ ).

### Secondary Outcome Measures

Compared to waitlist controls, the FCS treatment group had statistically significant decreases in the severity of self-reported depressive symptoms, anxiety symptoms, somatic symptoms, and a statistically significant increase in subjective report of satisfaction with life before and after the 3 FCS treatments (**Table 2**). As with the primary outcome measure (CAPS-5), all secondary outcome measures showed significantly larger declines in the FCS treatment group compared to the waitlist control group before and after FCS treatment. The statistical

**Table 2.** Regression Analysis for the Primary and the Secondary Outcomes

Outcome measure	Pre-treatment score rate (CI) $\beta_1$	Post-treatment score rate (CI) $\beta_1 + \beta_4$	Change in score rate (CI) $\beta_4$	Main treatment effect (CI) $\beta_3$	Main treatment effect P-value ( $\beta_3$ )	Group comparison P-value ( $\beta_3 - \beta_1^*$ )
CAPS-5	-2.0*** (-3.0, -1.0)	-2.1** (-3.4, -0.8)	-0.1 (-1.7, 1.5)	-18.7*** (-28.7, -8.7)	<.001	.0017
BDI	0.06 (-0.6, 0.7)	-0.6 (-1.9, 0.5)	-0.7 (-1.9, 0.5)	-9.5** (-15.6, -3.4)	.0039	.0041
PHQ-9	0.3 (-0.2, 0.8)	-0.2 (-1.0, 0.6)	-0.5 (-1.5, 0.4)	-6.6** (-10.8, -2.3)	.0038	.0032
PHQ-15	-0.02 (-0.4, 0.3)	-0.2 (-0.7, 0.3)	-0.2 (-0.8, 0.5)	-3.6* (-6.6, -0.7)	.019	.020
GAD-7	0.01 (-0.3, 0.3)	-0.2 (-0.6, 0.3)	-0.2 (-0.7, 0.3)	-5.3* (-9.4, -1.3)	.013	.014
SWLS	0.07 (-0.4, 0.6)	0.3 (-0.5, 1.0)	0.2 (-0.7, 1.1)	5.0* (0.6, 9.4)	.028	.031
PSEQ	-0.7* (-1.3, -0.05)	-0.1 (-1.1, 0.9)	0.5 (-0.6, 1.7)	6.3 (-2.0, 14.7)	.13	.11

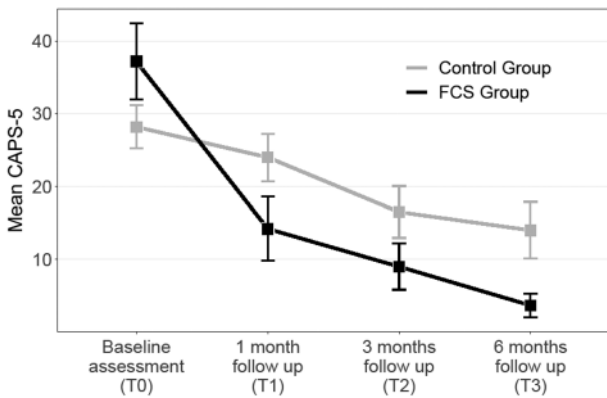
Score rates are defined as changes in scores per month. The 95% CI are provided in parenthesis. The *P*-value listed in column 6 corresponds to the main effect of treatment ( $\beta_3$  parameter in the model). The *P*-value listed in column 7 reflects the significance of the difference in treatment effects between the fascial counterstrain group and the waitlist control group, ( $\beta_3 - \beta_1^*$  in the model, where  $\beta_1^*$  is pre-treatment change score per 19.5 days).

Abbreviations: CAPS-5, Clinician-Administered PTSD Scale for DSM-5; BDI, Beck Depression Inventory; GAD-7, Generalized Anxiety Disorder-7; PSEQ, Pain Self-Efficacy Questionnaire; PHQ-15, Patient Health Questionnaire-15; PHQ-9, Patient Health Questionnaire-9; SWLS, Satisfaction with Life Scale.

\**P* < .05.

\*\**P* < .01.

\*\*\**P* < .001.



**Figure 2.** Mean (+SE) Clinician-Administered PTSD Scale for DSM-5 (CAPS-5) score in the fascial counterstrain group and waitlist controls. Bars represent standard errors. The fascial counterstrain treatment group received 3 fascial counterstrain treatments between the baseline assessment (T0) and 1-month follow-up (T1). Because assessments did not occur at identical time intervals across participants, outcome data were extrapolated to match standardized time points for visualization. The statistical model in Table 2 and Figure 3 accounted for the temporal variability in assessments.

significance of within and between-group differences is reported in the last 2 columns of Table 2. Finally, although the PSEQ score in the FCS treatment group showed a 6.3-point increase in confidence in ability to do tasks and activities despite pain, this change was not significantly different from the waitlist control group.

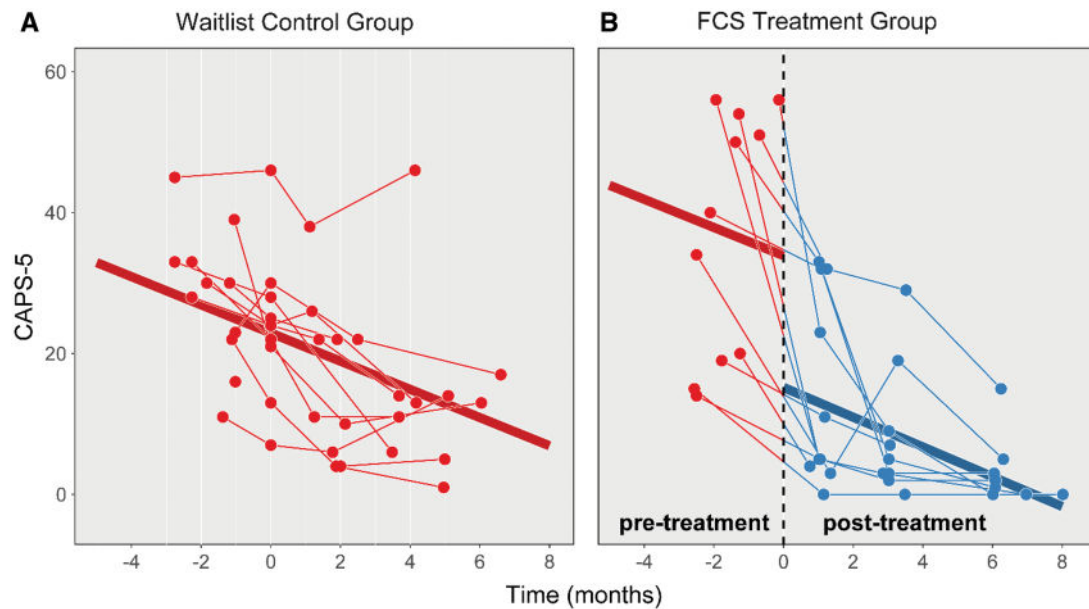
Patient Global Impression of Change (PGIC) was assessed in the FCS treatment group at T1, T2, and T3 time points. PGIC scores at T2 were significantly higher than T1 and PGIC scores at T3 were significantly higher than T2, confirming greater subjective overall improvement in the FCS group after FCS treatment (+3.9 points (*P* < .001) and +3.2 points (*P* < .001) respectively, unequal variance *t*-test). There were no reported adverse events in either group.

## DISCUSSION

This proof-of-concept study is the first to confirm that treatment with 3 sessions of FCS resulted in a statistically significant and rapid decrease in clinician-rated PTSD symptom severity, self-reported depression, self-reported anxiety levels, and somatic symptoms. In addition, the FCS treatment group reported significant increases in self-reported satisfaction with life and subjective overall improvement compared to waitlist controls. PTSD clinical response rates in the FCS group were substantially higher compared to waitlist controls. Because the CAPS-5 scores in the FCS treatment group continued to decrease for up to 6 months (T3) following the last FCS treatment, it is likely that the rapid improvements in PTSD symptoms were sustained over this time period.

The extent of decline in CAPS-5 score with FCS treatments in this study is similar to the decrease reported after 6 weeks of sertraline,<sup>34</sup> after 24 weeks of a combination of prolonged exposure and sertraline<sup>35</sup> or after 8 weeks of mantram repetition or present centered therapy.<sup>36</sup> In contrast to several months required for most evidence-based psychotherapies and pharmacotherapies to improve PTSD symptoms, the FCS associated decrease in PTSD symptom severity occurred over the course of only 20 days. Furthermore, unlike the rapid decrease in PTSD symptom severity that lasted less than a month following ketamine infusion,<sup>35,37</sup> the reduced PTSD symptom severity with FCS was sustained for several months after the completion of treatment. In addition, FCS treatment was not associated with any of the adverse effects reported with trauma focused therapy or pharmacotherapies, to include nausea, headaches, dry mouth, blurry vision, dizziness, and dissociation.

Although the exact mechanism by which FCS treatment decreases activity of the SNS with resultant decrease in PTSD symptoms is currently unknown, preclinical research findings support 3 plausible explanations. First, as stated previously, elevated levels of peripheral and central pro-inflammatory



**Figure 3.** Change in posttraumatic stress disorder symptoms severity for individual subjects in the waitlist control group and the fascial counterstrain treatment group. Figure 3(A). Clinician-Administered PTSD Scale for DSM-5 (CAPS-5) scores and timing of assessments relative to T1 (set to 0) for individual patients in the waitlist control group (dots). Linear trend from the model (solid line) indicates a CAPS-5 score decrease of  $\sim 2$ /month. Figure 3(B) CAPS-5 scores and timing of assessments relative to treatment (broken line) in the fascial counterstrain treatment group. Pre-treatment assessments and post treatment assessments are located on the negative and positive side of the time axis, respectively. Linear trend estimated from the control group was applied to the pretreatment data of the fascial counterstrain group (solid line, negative time). The post-treatment trend (solid line, positive time) was determined for and fitted to fascial counterstrain patients only. Note discontinuous CAPS-5 score drop of  $\sim 19$  in the fascial counterstrain group immediately after treatment (within fascial counterstrain treatment group:  $P < .001$ ; fascial counterstrain treatment group vs. control group:  $P = .0017$ ; see also Table 2).

cytokines have been shown to lower the activation threshold of sympathetic neurons.<sup>18</sup> Because FCS has been hypothesized to decrease interstitial inflammation,<sup>17</sup> the targeted FCS protocol can decrease inflammation in neuronal sheaths of the SNS with resultant decrease in both SNS hyperactivity and PTSD symptoms. Second, proinflammatory biomarkers activate chemically sensitive peripheral nociceptors, resulting in stimulation of somatosympathetic reflexes<sup>38</sup> and increased SNS activity. Therefore, FCS-related reductions in the levels of pro-inflammatory cytokines can potentially reduce nociceptive activity, resulting in lower sympathetic drive and decreased PTSD symptom severity. Finally, elevated sympathetic drive can cause vasoconstriction of the neck and spinal vasculature which can directly impact blood flow to the brain stem, including the vagal nuclei.<sup>39</sup> Therefore, reducing sympathetic activity with FCS may help restore normal vascular perfusion to the medulla, enhancing vagal tone and leading to a global reduction in sympathetic nervous system activation.

### Limitations

There was substantial individual variability in the duration between the 3 treatments and the timing of follow-up assessments in the FCS group and the waitlist control group. However, the statistical model used in the study was constructed specifically to disentangle the effects of FCS from the gradual decrease in CAPS-5 score with time, which was observed in both groups. The inclusion of a waitlist control, without an active comparison group, is an additional limitation. Lastly, the

waitlist control subjects were not reassessed after they received 3 FCS treatments at the end of the study participation.

This proof-of-concept study is the first to demonstrate that three 1.5-hour sessions of FCS manual therapy targeting the SNS resulted in a rapid and statistically significant reduction in clinician-rated PTSD symptom severity, as well as patient-reported depressive, anxiety, and somatic symptoms. Large, randomized studies in PTSD should compare the effectiveness of FCS manual therapy to trauma-focused therapy or evidence-based pharmacotherapy to evaluate its potential as a rapid, cost-effective, and scalable public health intervention.

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### CONFLICT OF INTEREST STATEMENT

Holly Christy is employed by and owns Element 7 Wellness, a medical clinic that offers Fascial Counterstrain (FCS) treatment. Brian Tuckey is the developer of the FCS techniques and is employed by and owns Tuckey and

Associates, a physical therapy clinic that offers FCS treatment. Holly Christy and Brian Tuckey are paid instructors for The Jones Institute and the Counterstrain Academy, which provides training and education in FCS. Nirnaya Miljajac and Nayak Polissar received financial compensation for conducting the statistical analysis. Meena Vythilingam does not have any commercial or financial relationships that could be construed as a potential conflict of interest. The findings and conclusions contained in this article are solely those of the authors and do not represent the official policy or position of the U.S. Department of Health and Human Services.

### DATA AVAILABILITY

The data that support the findings of this study are available upon request from the corresponding author.

### INSTITUTIONAL REVIEW BOARD

Solutions Institutional Review Board. Federalwide Assurance (FWA) #: IRB00008523.

### INSTITUTIONAL ANIMAL CARE AND USE COMMITTEE

Not applicable.

### INSTITUTIONAL CLEARANCE

Not applicable.

### INDIVIDUAL AUTHOR CONTRIBUTION STATEMENT

Holly Christy (Conceptualization, Funding acquisition, Methodology, Investigation, Project administration, Writing—review & editing), Brian Tuckey (Methodology, Writing—original draft, Writing—review & editing), Nirnaya Miljajac (Formal analyses, Writing—original draft, Writing—review & editing), Nayak Polissar (Formal analyses, Writing—original draft, Writing—review & editing), and Meena Vythilingam (Methodology, Writing—original draft, Writing—review & editing, Supervision)

### REFERENCES

1. Obuobi-Donkor G, Oluwasina F, Nkire N, Agyapong VI. A Scoping Review on the Prevalence and Determinants of Post-Traumatic Stress Disorder among Military Personnel and Firefighters: Implications for Public Policy and Practice. *IJERPH*. 2022;19:1565. <https://doi.org/10.3390/ijerph19031565>
2. Hoge CW, Castro CA, Messer SC, McGurk D, Cotting DI, Koffman RL. Combat duty in Iraq and Afghanistan, mental health problems, and barriers to care. *N Engl J Med*. 2004;351:13–22. <https://doi.org/10.1056/NEJMoa040603>
3. Gross GM, Pietrzak RH, Hoff RA, Katz IR, Harpaz-Rotem I. Risk for PTSD symptom worsening during new PTSD treatment episode in a nationally representative sample of treatment-seeking U.S. veterans with subthreshold PTSD. *J Psychiatr Res*. 2022;151:304–10. <https://doi.org/10.1016/j.jpsychires.2022.04.040>
4. Steenkamp MM, Litz BT, Hoge CW, Marmar CR. Psychotherapy for military-related PTSD: a review of randomized clinical trials. *JAMA*. 2015;314:489–500. <https://doi.org/10.1001/jama.2015.8370>
5. Davis LL, Schein J, Cloutier M, et al. The economic burden of posttraumatic stress disorder in the United States from a societal perspective. *J Clin Psychiatry*. 2022;83:e2. <https://doi.org/10.4088/jcp.21m14116>
6. Schneider M, Schwerdtfeger A. Autonomic dysfunction in posttraumatic stress disorder indexed by heart rate variability: a meta-analysis. *Psychol Med*. 2020;50:1937–48. <https://doi.org/10.1017/S003329172000207X>
7. Speer K, Semple S, Naumovski N, D'Cunha N, McKune A. HPA axis function and diurnal cortisol in post-traumatic stress disorder: a systematic review. *Neurobiol Stress*. 2019;11:100180. <https://doi.org/10.1016/j.ynstr.2019.100180>
8. Fonkoue IT, Marvar PJ, Norrholm S, et al. Symptom severity impacts sympathetic dysregulation and inflammation in post-traumatic stress disorder (PTSD). *Brain Behav Immun*. 2020;83:260–9. <https://doi.org/10.1016/j.bbi.2019.10.021>
9. Dyball D, Evans S, Boos CJ, Stevelink SAM, Fear NT. The association between PTSD and cardiovascular disease and its risk factors in male veterans of the Iraq/Afghanistan conflicts: a systematic review. *Int Rev Psychiatry (Abingdon, England)*. 2019;31:34–48. <https://doi.org/10.1080/09540261.2019.1580686>
10. Karimov-Zwienenberg M, Symphor W, Peraud W, Décamps G. Childhood trauma, PTSD/CPTSD and chronic pain: a systematic review. *PLoS One*. 2024;19:e0309332. <https://doi.org/10.1371/journal.pone.0309332>
11. Bookwalter DB, Roenfeldt KA, LeardMann CA, Kong SY, Riddle MS, Rull RP. Posttraumatic stress disorder and risk of selected autoimmune diseases among US Military personnel. *BMC Psychiatry*. 2020;20:23. <https://doi.org/10.1186/s12888-020-2432-9>
12. Courtois C, Sonis J, Brown L, et al. Summary of the clinical practice guideline for the treatment of posttraumatic stress disorder (PTSD) in adults. *Am Psychol*. 2019;74:596–607. <https://doi.org/10.1037/amp0000473>
13. Bisson J, Van Gelderen M, Roberts N, Lewis C. Non-pharmacological and non-psychological approaches to the treatment of PTSD: results of a systematic review and meta-analyses. *Eur J Psychotraumatol*. 2020;11:1795361. <https://doi.org/10.1080/20008198.2020.1795361>
14. Kirkpatrick K, Khan MH, Deng Y, Shah KB. A review of stellate ganglion block as an adjunctive treatment modality. *Cureus*. 2023;15:e35174. <https://doi.org/10.7759/cureus.35174>
15. Prasad S, Jain N, Umar T, et al. Sympathetic nerve blocks for posttraumatic stress disorder: an evidentiary review for future clinical trials. *Front Psychiatry*. 2023;14:1309986. <https://doi.org/10.3389/fpsy.2023.1309986>
16. Fritz K, Novotny K, Carr CL Jr. *Physiology, Counterstrain/FPR*. StatPearls Publishing; 2020.
17. Tuckey B, Srbely J, Rigney G, Vythilingam M, Shah J. Impaired lymphatic drainage and interstitial inflammatory stasis in chronic musculoskeletal and idiopathic pain syndromes: exploring a novel mechanism. *Front Pain Res*. 2021;2:691740. <https://doi.org/10.3389/fpain.2021.691740>
18. Kenney MJ, Ganta CK. Autonomic nervous system and immune system interactions. In: *Comprehensive Physiology*. Wiley; 2014:1177–200. <https://doi.org/10.1002/cphy.c130051>
19. Gray MJ, Litz BT, Hsu JL, Lombardo TW. Psychometric properties of the Life Events Checklist. *Assessment*. 2004;11:330–41. <https://doi.org/10.1177/1073191104269954>
20. Weathers FW, Bovin MJ, Lee DJ, et al. The Clinician-Administered PTSD Scale for DSM-5 (CAPS-5): development and initial psychometric evaluation in military veterans. *Psychol Assess*. 2018;30:383–95. <https://doi.org/10.1037/pas0000486>
21. Schnurr PP, Friedman MJ, Engel CC, et al. Cognitive behavioral therapy for posttraumatic stress disorder in women: a randomized controlled trial. *JAMA*. 2007;297:820–30. <https://doi.org/10.1001/jama.297.8.820>
22. Beck BDI-I, Steer AT, Brown RA. (1996). *Beck Depression Inventory-II (BDI-II)*. APA PsychTests.
23. Kroenke K, Spitzer RL, Williams JB. The PHQ-9: validity of a brief depression severity measure. *J Gen Intern Med*. 2001;16:606–13. <https://doi.org/10.1046/j.1525-1497.2001.016009606.x>
24. Kroenke K, Spitzer RL, Williams JBW. The PHQ-15: validity of a new measure for evaluating the severity of somatic symptoms. *Psychosom Med*. 2002;64:258–66. <https://doi.org/10.1097/00006842-200203000-00008>
25. Spitzer RL, Kroenke K, Williams JBW, Löwe B. A brief measure for assessing generalized anxiety disorder: the GAD-7. *Arch Intern Med*. 2006;166:1092–7. <https://doi.org/10.1001/archinte.166.10.1092>

26. Diener E, Emmons RA, Larsen RJ, Griffin S. The satisfaction with life scale. *J Pers Assess.* 1985;49:71–5. [https://doi.org/10.1207/s15327752jpa4901\\_13](https://doi.org/10.1207/s15327752jpa4901_13)
27. Nicholas MK, McGuire BE, Asghari A. A 2-item short form of the pain self-efficacy questionnaire: development and psychometric evaluation of PSEQ-2. *J Pain.* 2015;16:153–63. <https://doi.org/10.1016/j.jpain.2014.11.002>
28. Rampakakis E, Ste-Marie PA, Sampalis JS, Karellis A, Shir Y, Fitzcharles M-A. Real-life assessment of the validity of patient global impression of change in fibromyalgia. *RMD Open.* 2015;1:e000146. <https://doi.org/10.1136/rmdopen-2015-000146>
29. Jones L. (1995). *Strain and Counterstrain*. 2nd ed. American Academy Osteopathy.
30. Lin Q, Li D, Xu X, Zou X, Fang L. Roles of TRPV<sub>1</sub> and neuropeptidergic receptors in dorsal root reflex-mediated neurogenic inflammation induced by intradermal injection of capsaicin. *Mol Pain.* 2007;3:30. <https://doi.org/10.1186/1744-8069-3-30>, 1744-8069-3–30.
31. Wehrwein EA, Orer HS, Barman SM. Overview of the anatomy, physiology, and pharmacology of the autonomic nervous system. In: *Comprehensive Physiology*. Wiley; 2016:1239–1278.
32. Tuckey B. Fascial Counterstrain for the Nervous System 1 Upper Body and Cranium, Training Manual, 2019.
33. Tuckey B. Fascial Counterstrain for the Nervous System Part 2 Thorax, Pelvis and Lower Extremities, Training Manual, 2017.
34. Rauch SAM, Kim HM, Powell C, et al. Efficacy of Prolonged Exposure therapy, sertraline hydrochloride, and their combination among combat veterans with posttraumatic stress disorder: a randomized clinical trial. *JAMA Psychiatry.* 2019;76:117–26. <https://doi.org/10.1001/jamapsychiatry.2018.3412>
35. Fremont R, Brown O, Feder A, Murrrough J. Ketamine for treatment of posttraumatic stress disorder: state of the field. *Focus (American Psychiatric Publishing).* 2023;21:257–65. <https://doi.org/10.1176/appi.focus.20230006>
36. Bormann JE, Thorp SR, Smith E, et al. Individual treatment of posttraumatic stress disorder using mantram repetition: a randomized clinical trial. *Am J Psychiatry.* 2018;175:979–88. <https://doi.org/10.1176/appi.ajp.2018.17060611>
37. Feder A, Costi S, Rutter SB, et al. A randomized controlled trial of repeated ketamine administration for chronic posttraumatic stress disorder. *Focus (American Psychiatric Publishing).* 2023;21:296–305. <https://doi.org/10.1176/appi.focus.23021014>
38. Schmidt RF, Weller E. Reflex activity in the cervical and lumbar sympathetic trunk induced by unmyelinated somatic afferents. *Brain Res.* 1970;24:207–18. [https://doi.org/10.1016/0006-8993\(70\)90101-0](https://doi.org/10.1016/0006-8993(70)90101-0)
39. Montalbano MJ, Loukas M, Oskouian RJ, Tubbs RS. Innervation of the blood vessels of the spinal cord: a comprehensive review. *Neurosurg Rev.* 2018;41:733–5. <https://doi.org/10.1007/s10143-016-0788-6>