SPECIAL ISSUE ON CENTRAL SENSITIZATION

The central sensitization inventory: A user's manual

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Correspondence Randy Neblett, PRIDE, Dallas, TX, USA. Email: randyneblett@pridedallas.com The Central Sensitization (CSI) Inventory was introduced in 2012. It was initially intended as a screener to help identify when presenting symptoms may be related to central sensitization or indicate the presence of a central sensitivity syndrome. It has now been translated and validated in a number of European, Asian, and South American languages. This article provides an overview of CSI rationale, development, recommended uses, and research results, including evidence of validity and reliability, in clinical and non-clinical subject samples.

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1 | CENTRAL SENSITIZATION

Central Sensitization (CS) refers to an amplification of neural signaling within the central nervous system, resulting in pain hypersensitivity (Woolf, 2011). Symptoms of CS include allodynia, hyperalgesia, expansion of the receptive field beyond the area of peripheral nerve supply, and prolonged pain after a stimulus has been removed (Latremoliere & Woolf, 2009). A number of CS-related biological mechanisms have been identified, including dysregulation of ascending and descending tracks in the central nervous system (Ren & Dubner, 2002; Yunus, 2007; Heinricher et al., 2009; van Wijk & Veldhuijzen, 2010; Kindler, Bennett, & Jones, 2011); over-activation of glial cells, resulting in the release of pro-inflammatory cytokines (Ji, Berta, & Nedergaard, 2013; Loggia et al., 2015; Nijs et al., 2017); dysfunction of the stress system, including the hypothalamic-pituitary-adrenal axis (Van Houdenhove & Luyten, 2009); decreased production of pain-inhibiting neurotransmitters, and increased production of pain-augmenting neurotransmitters, including excess production of brain-derived neurotropic factor (BDNF) (Caumo et al., 2016; Deitos et al., 2015; Nijs, Meeus et al., 2015; Phillips & Clauw, 2011; Trang, Beggs, & Salter, 2011).

Central Sensitization has been most often identified with fibromyalgia, and associated chronic widespread pain and sensitivity, with no observable pathology or nociceptive etiology. However, CS has also been identified in subsets of patients with clear evidence of tissue trauma, pathology, and/or nociceptive components, including multiple sclerosis (Fernández-de-las-Peñas et al., 2015), osteoarthritis (Akinci et al., 2016; Lluch, Torres, Nijs, &

Van Oosterwijck, 2014), rheumatoid arthritis (Meeus et al., 2012), and post-surgical breast cancer (Fernández-Lao et al., 2011). Interestingly CS has also been tied to various other conditions in which pain is not a primary symptom, including post-traumatic stress disorder, multiple chemical sensitivity, restless leg syndrome (Yunus, 2007, 2015) over-active bladder (Reynolds, Dmochowski, Wein, & Bruehl, 2016), and chronic hives (Torresani, Bellafiore, & De Panfilis, 2009), suggesting that CS may not only be associated with pain hypersensitivity but can also involve hypersensitivity to other stimuli, including lights, sounds, fragrances, skin irritants, bodily sensations, and stress-evoking life events (Nijs, Goubert, & Ickmans, 2016; Yunus, 2007, 2015).

Many studies have demonstrated that CS can be induced in human volunteers by activating nociceptors in a variety of ways, including electrical stimulation, capsaicin injections, mustard oil injections, acid, heat burn, UV burn, and hypertonic saline (Woolf, 2011). Subjective evidence for central-related pain hypersensitivity has been demonstrated by differences in self-reported pain severity ratings to heat, cold, electrical, and pressure stimuli between subjects with and without pain disorders (Yunus, 2007). Objective measures of CS, including brain imaging (Robinson, Craggs, Price, Perlstein, & Staud, 2011; Walitt, Ceko, Gracely, & Gracely, 2016), cortical excitability parameters assessed by transcranial magnetic stimulation (TMS), and levels of brain-derived neurotrophic factor (BDNF) (Caumo et al., 2016; Deitos et al., 2015) have demonstrated biological differences between control subjects and those with CS-related pain disorders.

The development of CS, and associated symptoms, is often related to trauma. CS is most often associated with physical trauma/injury (McBeth, Harkness, Silman, & MacFarlane, 2003; Myrtveit, Skogen, Wenzel, & Mykletun, 2012; Wenzel, Mykletun, & Nilsen, 2009), but can it also result from other types of trauma, such as certain infections (e.g., Hepatitis C, Epstein Barr, Lime Disease) and emotional trauma, including childhood abuse (Häuser, Kosseva, Üceyler, Klose, & Sommer, 2011; Kindler, Jones, Perrin, & Bennett, 2010; Phillips & Clauw, 2011; Wilson, 2010; Yunus, 2008). Spinal injuries to the low back (Sanzarello et al., 2016; Bid, Soni, & Rathod, 2016) and neck (Van Oosterwijck, Nijs, Meeus, & Paul, 2013) appear to be especially susceptible to developing CS symptoms, including chronic widespread pain (Kindler et al., 2010; McBeth et al., 2003; Myrtveit et al., 2012; Wenzel et al., 2009) and fibromyalgia (Buskila & Mader, 2011; Buskila, Neumann, Vaisberg, Alkalay, & Wolfe, 1997; Waylonis & Perkins, 1994).

After CS develops, little or no nociceptive stimulus is necessary to perpetuate and sustain a state of hyperalgesia or allodynia. Painful sensations can occur in the absence of either peripheral pathology or noxious stimuli (Latremoliere & Woolf, 2009). Because no apparent cause for pain can be identified (pain is no longer nociceptive in nature) physicians and other health care providers may have a tendency to interpret these patients as neurotics, malingerers, or somatizers (Yunus, 2012, 2015).

2 | HISTORICAL BACKGROUND OF THE CSI

The development of the Central Sensitization Inventory (CSI) was initially inspired by articles from Muhammad Yunus (Yunus, 2000, 2007) and Lindsay Kinder (Kindler et al., 2011). A very compelling argument was made that many pain-related syndromes, previously viewed as separate disorders, and often termed as "functional" or "medically unexplained," have a common etiology of CS. Yunus introduced the term Central Sensitivity Syndrome (CSS) to describe these disorders (Yunus, 2000). Proposed members of the CSS family include fibromyalgia, irritable bowel syndrome, temporomandibular joint disorder, migraine and tension headache, myofacial pain syndrome, and some chronic pelvic pain disorders (e.g., interstitial cystitis, primary dysmenorrhea) (Kindler et al., 2011; Phillips & Clauw, 2011; Yunus, 2007, 2015). Other types of disorders, of which pain is not a primary component, have been added to the CSS family, including post-traumatic stress disorder, multiple chemical sensitivity, and restless leg syndrome (Phillips & Clauw, 2011; Yunus, 2007). Although these conditions may appear unrelated, they all share a common theme of hypersensitivity to stimuli.

When investigating the supporting literature, one finds a striking over-lap in diagnoses among the CSS family, especially regarding fibromyalgia (Phillips & Clauw, 2011; Woolf, 2011; Yunus, 2007). For instance, in a review of previous studies, Yunus (2012) reported that 13% to 52% of TMD patients, 10% to 40% of headache patients, and 20% to 65% of IBS patients also met criteria for fibromyalgia. A relatively large amount of co-morbid symptomology is also evident among various CSSs, including insomnia, feeling "unrefreshed" after sleeping, difficulty concentrating, bowel and bladder problems, and fatigue (Yunus, 2007, 2015). It is also clear that psychiatric disorders, emotional symptoms (including anxiety and depression), and trauma (including childhood abuse) are often associated with CSSs (Arnold et al., 2006; Henningsen, Zimmermann, & Sattel, 2003; Phillips & Clauw, 2011; Spiegel et al., 2015). It was within this framework that the CSI was developed.

3 | DEVELOPMENT AND GROWTH OF THE CSI

The CSI was originally designed as an instrument for screening patient symptomology, to help identify if symptoms may be related to CS or may indicate the presence of a CSS (Mayer et al., 2012). It was recognized by our group that patient symptom presentations are often complex and distressing. When no clear pathology can be identified to explain symptoms, physicians and other health care workers may be inclined to either (1) dismiss the symptoms as a sign of mental illness/stress/somatization or (2) order expensive and invasive assessment procedures (e.g., imaging, colonoscopy, etc.) in an attempt to find a medical cause and eventual invasive treatments (e.g., surgery) in attempt to alleviate the symptoms. However, when symptoms are related to CS, or represent a CSS, the primary target for treatment should be the central nervous system, not the periphery (Latremoliere & Woolf, 2009). For patients with CS-related disorders, medical interventions targeted in the periphery are often unnecessary, unhelpful, and potentially harmful. The CSI was an attempt to provide a relatively quick means of identifying when symptoms may be associated with CS/CSS so that additional diagnostic evaluation can be performed, to assess for CSS, and appropriate treatment can be initiated.

The items were developed based on an extensive literature search of overlapping somatic and emotional health-related symptom dimensions that have been found in previous studies to be associated with CS/CSS. As a result, the CSI contains a very heterogeneous list of 25 items. In Appendix 1 Part A, one is asked how often he/ she experiences each symptom ("never, rarely, sometimes, often, or always"). Individual items are scored from "0" (never) to "4" (always), resulting in a total score range for all 25 items from "0" to "100." It was recognized that it may be important to know if subjects were aware of previous CSS or related diagnoses, so a second section was developed. Appendix 2 Part B asked if one has been previously diagnosed with seven common CSS diagnoses (tension headaches/migraines, fibromyalgia, irritable bowel syndrome, restless leg syndrome, temporomandibular joint disorder, chronic fatigue syndrome, and multiple chemical sensitivities) and three CS-related diagnoses (depression, anxiety/panic attacks, and neck injury). CSI B is for information only and is not scored.

At the time of this writing, the CSI had been translated, validated, and published in Dutch (Kregel et al., 2016), French (Pitance et al., 2016), Spanish (Cuesta-Vargas, Roldan-Jimenez, Neblett, & Gatchel, 2016), Gujarati (Bid, Soni, Rathod, & Ramalingam, 2016), Brazilian Portuguese (Caumo et al., 2016), Serbian (Knezevic et al., 2017), and Japanese (Tanaka et al., 2017). The author of the present article has also been in communication with other groups who are in the process of translating and validating the CSI in Italian, Turkish, German, Korean, Nepali, Russian, and Tatar (personal communications). The English version of the CSI can be found in the appendix. Multiplelanguage versions of the CSI, with supporting references, can be found at www.pridedallas.com/questionnaires.

4 | PSYCHOMETRIC RELIABILITY OF THE CSI

The CSI has been found to be psychometrically sound in all published studies to the present. The original English version of the CSI demonstrated good test-retest reliability and internal consistency (Pearson's r = .82; Cronbach's

 α = .88, respectively) (Mayer et al., 2012). Similar results have been found in the other published translated versions (Bid, Soni, Rathod, & Ramalingam, 2016; Caumo et al., 2017; Cuesta-Vargas et al., 2016; Knezevic et al., 2017; Kregel et al., 2016; Pitance et al., 2016; Tanaka et al., 2017), with test-retest from .85 to .97 and Cronbach's alpha from 0.88 to 0.91.

5 | FACTOR STRUCTURE OF THE CSI

Factor analyses from different published translated versions of the CSI have produced some similar but conflicting results. The original English CSI (Mayer et al., 2012) determined a 4-factor structure: 1. Physical Symptoms, 2. Emotional Distress, 3. Headache/Jaw Symptoms, and 4. Urological Symptoms. Three items that did not load on any of the factors. A confirmatory factor analysis (CFA) of the French version confirmed the same 4-factor structure, with the same three items that did not load on any of the factors (Pitance et al., 2016). A CFA of the Dutch version resulted in a similar 4-factor structure, but the item pool for each factor was somewhat different than the English version, and five items did not load on any of the factors (Kregel et al., 2016). CFA of the Brazilian Portuguese version also determined a 4-factor solution, but with a somewhat different item pool and 1 item that did not load on any of the factors (Caumo et al., 2017). CFA of the Serbian version confirmed the 4-factor solution, with all items retained, and determined the presence of a single second-order general factor (Knezevic et al., 2017). Exploratory analysis of the Japanese version determined a 5-factor model (Tanaka et al., 2017). A principal component analysis with Maximum Likelihood Extraction (MLE) determined a 1-factor solution in the Spanish version (Cuesta-Vargas et al., 2016). To help resolve these factor structure discrepancies, a large multi-country study was performed with a coalition of research groups from The Netherlands, Spain, France, Italy, Serbia, Brazil, and the United States (Cuesta-Vargas et al., 2018). CSI data from 1,987 subjects were pooled into a single database for analysis. A bi-factor solution was determined (Rodriguez, Reise, & Haviland, 2016). The results confirmed the same four orthogonal factors found in previous studies but found that the reliability of the four factors was too low to be recommended for subscales. CSI items in each of the four factors were: Physical Symptoms (1, 2, 5, 6, 8, 9, 12, 14, 17, 18, 22), Emotional Distress (3, 13, 15, 16, 23, 24), Headache/Jaw Symptoms (4, 7, 10, 19, 20), and Urological Symptoms (11, 21, 25). One general factor, representing "CS-Related Symptoms," showed substantial reliability [i.e., Cronbach α = 0.92; Omega ω = 0.95; and omega hierarchical ω -h = 0.89]. The results of this study suggest that only total CSI scores should be used and reported. However, for individual clinical purposes, a review of individual item scores may be beneficial for better understanding the patient's symptom presentation.

6 | INTERPRETING CSI SCORES

A score of 40 or higher has been recommended as a reasonable cutoff to alert health care professionals that a patient's symptom presentation may indicate the presence of CS/CSS. This 40-point cut-off was determined based on its ability to discriminate between CSS and non-patient subjects (Neblett et al., 2013): sensitivity (81%) was good in correctly identifying CSS patients, and specificity (75%) was adequate in correctly identifying non-patient comparison subjects. A separate study with the French CSI found even better sensitivity (95%) and specificity (90%) with distinguishing between fibromyalgia subjects and acute ankle pain and control subjects. This 40-point cut-off score has been recommended as one component of an algorithm for helping to identify CS-related pain (vs. neuropathic and nociceptive pain) in generalized chronic pain (Nijs, Malfliet, Ickmans, Baert, & Meeus, 2014; Nijs, Torres-Cueco et al., 2014) and low back pain subjects (Nijs, Apeldoorn et al., 2015), and for classifying of temporomandibular joint disorder subtypes (Monaco, Cattaneo, Marci, Pietropaoli, & Ortu, 2017). Most recently, five severity levels have been developed to help aid in the clinical interpretation of the CSI (subclinical = 0–29; mild = 30–39; moderate = 40–49; severe = 50–59; and extreme = 60–100) (Neblett, Hartzell, Mayer, Cohen, & Gatchel, 2017). The 40-point cut-off has shown promise in some studies with other clinical populations (Bennett, Walsh, Thompson, & Krishnaney, 2017; Kim, Yoon, Yoon, Yoo, & Ahn, 2015). Some groups, however, have found different cutoff scores to more accurately identify significant levels of CS-related symptomology for specific populations, such as migraine subjects (Aguila et al., 2016). Furthermore validation of severity level scores in different subject populations are needed to verify their clinical usefulness. Preliminary analyses of the five severity levels, however, has been positive. In three separate studies, different chronic pain populations were placed in severity groups based on CSI scores. Mean scores for each severity group were significantly different than all the other severity groups, demonstrated good score discrimination (Knesvic et al. 2017; Neblett, Hartzell et al., 2017). Perhaps these severity cutoffs can be a useful guide for clinicians and researchers in the clinical interpretation of CSI scores for other clinical populations. It is possible, however that alternative severity level cutoffs may be found to be more useful in different language versions of the CSI or with different subject populations (van Wilgen et al., 2018).

7 | VALIDITY OF THE CSI

Despite its name, the CSI does not measure CS. No self-report instrument can do that. But there is growing evidence that CSI scores are associated with CS-related symptoms and diagnoses. A recent systematic review identified 14 CSI studies, which were determined to have good-to-excellent quality of evidence (Scerbo et al., 2017). The authors concluded that the CSI generates reliable and valid data to quantify the severity of CS-related symptoms.

7.1 | Evidence of convergent validity

Strong correlations have been found among total CSI scores and other validated self-report measures of pain intensity, depressive symptoms, anxiety symptoms, sleep disturbance, pain catastrophizing, the ability to concentrate, and perceived disability/pain interference/quality of life, all of which have been associated with CS/CSS (Caumo et al., 2017; Huysmans et al., 2018; Knezevic et al., 2017; Kregel et al., 2017; Neblett et al., 2013, 2015; Neblett, Hartzell et al., 2017; Neblett, Hartzell, Williams et al., 2017; Tanaka et al., 2017).

Central sensitization inventory scores have been shown to be associated with CS-related pain patterns. Higher CSI scores were associated with wider body area distribution of self-reported knee pain, and lower pain thresholds, in a group of osteoarthritis patients who were scheduled to undergo primary total knee arthroplasty (Lluch Girbés et al., 2016); increased widespread pain sensitivity in a group of shoulder patients who were undergoing quantitative sensory testing (QST) (Coronado, Mackie, Simon, & George, 2014); longer pain duration, higher pain intensity, and more widespread pain pattern in a general chronic pain population (van Wilgen et al., 2018); and higher pain intensity and increased pain behavior (as assessed with a 1-min stair-climbing test) in a group of subjects with chronic nonspecific low back pain (Huysmans et al., 2018).

Additional evidence has been demonstrated by associations among CSI scores and known risk factors for CS. Higher CSI scores were associated with longer duration of pain (Knezevic et al., 2017; van Wilgen et al., 2018), childhood abuse history and a major depressive disorder diagnoses (Neblett et al., 2015) in general chronic pain populations and longer length of pre-admission disability, more pre-admission surgeries, greater number of injured body parts, childhood abuse history, and major depressive disorder diagnoses in a group of chronic spinal disorder patients entering functional restoration treatment (Neblett, Hartzell, Williams et al., 2017).

Finally, positive correlations have been found among CSI scores and objective biological markers of CS. CSI scores were associated with brain gamma aminobutyric acid (GABA) levels in migraine subjects (Aguila et al., 2016). Furthermore a Receive Operator Curve (ROC) analysis indicated that subjects with CSI scores >22.5 were nearly five times more likely to have migraine than those with lower scores. Another study found that CSI scores

were associated with higher levels of serum brain-derived neurotrophic factor (BDNF) in chronic pain patient samples (especially those with fibromyalgia) compared to pain-free control subjects (Caumo et al., 2017). The same study also found that higher CSI scores were associated with lower pain-pressure thresholds and dysfunction of the descending pain-modulatory system in the fibromyalgia patients, as measured with a cold-pressor conditioned pain modulation test. A separate study with chronic spinal pain subjects found a significant, but weak correlation between CSI scores and pain pressure thresholds, but no significant correlation with a conditioned pain modulation test (Kregel et al., 2017).

7.2 | Evidence of discriminant validity

Total CSI scores have been shown to discriminate between subjects with general chronic musculoskeletal pain and pain-free controls (Kregel et al., 2016); between subjects with fibromyalgia and both acute ankle sprain and pain-free controls (Pitance et al., 2016); among patients with fibromyalgia/chronic widespread pain regional chronic low back pain, and non-patient university students and faculty (Mayer et al., 2012); between subjects with interstitial cystitis, overactive bladder, and healthy controls (McKernan, Cohn, Bruehl, Dmochowski, & Reynolds, 2017); and between subjects with, and without, a diagnosed CSS (Caumo et al., 2017; Knezevic et al., 2017; Neblett et al., 2013; Neblett, Hartzell et al., 2017).

Self-reported CSS diagnoses on CSI part B have also been found to correlate with physician-diagnosed CSSs and total CSI scores. Several studies have shown that CSI scores rise as the number of self-reported CSS diagnoses increases (Caumo et al., 2017; Neblett et al., 2013, 2015; Neblett, Hartzell et al., 2017). In one study, the percentage of chronic spinal pain disorder patients who reported a comorbid CSS diagnosis increased in a stair-step pattern, from 11% in a subclinical CSI severity group to 56% in an extreme CSI severity group. Agreement between physician-diagnosed CSSs and self-reported CSSs, in a group of general chronic pain subjects, was relatively high with fibromyalgia, headache, irritable bowel syndrome and moderate with restless leg syndrome and temporomandibular joint disorder (Neblett et al., 2013).

7.3 | Evidence of predictive validity

Higher CSI scores (above the proposed 40-point cutoff), obtained before participating in knee arthroplasty, predicted higher dosages of post-surgical analgesics and more severe post-surgical pain intensity ratings (Kim et al., 2015). CSI scores above 40 were the strongest predictive variable, of all those studied, for persistent pain 3 months after surgery, with an odds ratio of 5.091. In a separate study of spinal fusion surgery subjects, higher pre-surgical CSI scores (above 40) predicted worse post-surgical self-reported disability, depressive symptoms, and quality of life, and a longer post-surgical hospital stay (Bennett et al., 2017). In fact, for each 10-point score increase above 40, the average length of stay increased by 6.4%.

8 | TREATMENT RESPONSIVENESS OF THE CSI

Though it was originally designed as a symptom screener, there is some recent evidence that the CSI may be an effective treatment outcome measure. CSI scores decreased following five sessions (over 2 months) of a conventional physiotherapy program and a McKenzie exercise program in two groups of chronic non-specific low back pain subjects (Bid, Soni, Yadav, & Rathod, 2017). Results were significantly better in the McKenzie program. CSI scores, and other associated self-reported symptoms (pain intensity, pain-related anxiety, sleep disturbance, perceived disability depressive symptoms, and somatization symptoms) improved in a group of chronic spinal disorder patients who completed a functional restoration treatment program (Neblett, Hartzell, Williams et al., 2017). An as-yet unpublished report found similar decreases in CSI scores, and other associated symptoms, in a group

general chronic pain subjects who completed a separate functional restoration program (Jimenez, Aboussssouan, Mandell, & Huffman, 2017).

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9 | CLINICAL USE OF THE CSI

As stated previously, the CSI was originally intended as a screener. It can be included in an initial patient evaluation, along with a review of symptoms, medical evaluation, medical history, mental health evaluation, and other relevant self-report questionnaires. It may be especially useful for patients who present with pain, or other physical symptoms, of unknown origin.

Some clinical indications of CS include pain levels that are disproportionate to the extent of injury, a neuroanotomically illogical pain pattern, and hypersensitivity of senses unrelated to the musculoskeletal system (Nijs et al., 2016). So, low CSI scores (along with no clinical evidence of CS) suggest that additional medical tests may be warranted to evaluate the cause of the symptoms. High CSI scores (along with clinical evidence of CS) suggest that the patient should be first evaluated for a CSS and/or CS involvement before additional medical tests are ordered. Detailed guidelines are available (with CSI scores as one component) for identifying when pain is likely related to CS (Nijs, Torres-Cueco et al., 2014; Nijs et al., 2016). Also, established criteria for diagnosing individual CSSs, such as fibromyalgia (Wolfe & Häuser, 2011; Wolfe et al., 2016), are readily available.

Treatment of CSSs and/or CS-related pain often requires a different approach than for nociceptive pain (Latremoliere & Woolf, 2009). A number of studies have demonstrated that tricyclic compounds, serotonin norepinephrine re-uptake inhibitors (such as duloxetine and tramadol), and alpha-2-delta ligands (such as pregabalin and gabapentin) are efficacious for CS-related pain disorders and a variety of other related CSSs (Phillips & Clauw, 2011). In addition to medication, a biopsychosocial combination of difference desensitizing strategies (e.g., pain neuroscience education, exercise therapy, cognitive behavioral therapy, biofeedback, and relaxation training) has been recommended (Adams & Turk, 2015; Kerns, Sellinger, & Goodin, 2011; Nijs, Malfliet et al., 2014). In fact, an interdisciplinary treatment approach may be the best option for treating CS-related symptoms of chronic pain (Gatchel & Okifuji, 2006; Nijs, Apeldoorn et al., 2015).

10 | CONCLUSION

The CSI was just published in 2012 (Mayer et al., 2012). It is still in its infancy. Despite its recent development, it has received quite a bit of international attention and is now available in a number of separate languages. It is anticipated that the number of translations will continue to grow, allowing the CSI to be used and studied in a wide variety of cultures and subject samples. Its positive psychometric properties and varied and substantial evidence of reliability and validity suggest that the CSI has a promising future for clinical and research use.

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APPENDIX 1

Central sensitization inventory: Part A

NILEY

Name:

Date:

Please circle the best response to the right of each statement.

1.	I feel tired and unrefreshed when I wake from sleeping.	Never	Rarely	Sometimes	Often	Always
2.	My muscles feel stiff and achy.	Never	Rarely	Sometimes	Often	Always
3.	I have anxiety attacks.	Never	Rarely	Sometimes	Often	Always
4.	I grind or clench my teeth.	Never	Rarely	Sometimes	Often	Always
5.	I have problems with diarrhea and/or constipation.	Never	Rarely	Sometimes	Often	Always
6.	I need help in performing my daily activities.	Never	Rarely	Sometimes	Often	Always
7.	I am sensitive to bright lights.	Never	Rarely	Sometimes	Often	Always
8.	I get tired very easily when I am physically active.	Never	Rarely	Sometimes	Often	Always
9.	I feel pain all over my body.	Never	Rarely	Sometimes	Often	Always
10.	I have headaches.	Never	Rarely	Sometimes	Often	Always
11.	I feel discomfort in my bladder and/or burning when I urinate.	Never	Rarely	Sometimes	Often	Always
12.	I do not sleep well.	Never	Rarely	Sometimes	Often	Always
13.	I have difficulty concentrating.	Never	Rarely	Sometimes	Often	Always
14.	I have skin problems such as dryness, itchiness, or rashes.	Never	Rarely	Sometimes	Often	Always
15.	Stress makes my physical symptoms get worse.	Never	Rarely	Sometimes	Often	Always
16.	I feel sad or depressed.	Never	Rarely	Sometimes	Often	Always
17.	I have low energy.	Never	Rarely	Sometimes	Often	Always
18.	I have muscle tension in my neck and shoulders.	Never	Rarely	Sometimes	Often	Always
19.	I have pain in my jaw.	Never	Rarely	Sometimes	Often	Always
20.	Certain smells, such as perfumes, make me feel dizzy and nauseated.	Never	Rarely	Sometimes	Often	Always
21.	I have to urinate frequently.	Never	Rarely	Sometimes	Often	Always
22.	My legs feel uncomfortable and restless when I am trying to go to sleep at night.	Never	Rarely	Sometimes	Often	Always
23.	I have difficulty remembering things.	Never	Rarely	Sometimes	Often	Always
24.	I suffered trauma as a child.	Never	Rarely	Sometimes	Often	Always
25.	I have pain in my pelvic area.	Never	Rarely	Sometimes	Often	Always

TOTAL:

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APPENDIX 2

Central sensitization inventory: Part B

Name:

Date:

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Have you been diagnosed by a doctor with any of the following disorders?

Please check the box to the right for each diagnosis and write the year of the diagnosis.

		NO	YES	Year Diagnosed
1.	Restless Leg Syndrome			
2.	Chronic Fatigue Syndrome			
3.	Fibromyalgia			
4.	Temporomandibular Joint Disorder (TMJ)			
5.	Migraine or tension headaches			
6.	Irritable Bowel Syndrome			
7.	Multiple Chemical Sensitivities			
8.	Neck Injury (including whiplash)			
9.	Anxiety or Panic Attacks			
10.	Depression			