Yoga Asana Sessions Increase Brain GABA Levels: A Pilot Study

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ABSTRACT

Objectives: The aim of this study was to compare changes in brain γ-aminobutyric acid (GABA) levels associated with an acute yoga session versus a reading session. It was hypothesized that an individual yoga session would be associated with an increase in brain GABA levels.

Design: This is a parallel-groups design.

Settings/location: Screenings, scan acquisitions, and interventions took place at medical school-affiliated centers.

Subjects: The sample comprised 8 yoga practitioners and 11 comparison subjects.

Interventions: Yoga practitioners completed a 60-minute yoga session and comparison subjects completed a 60-minute reading session.

Outcome measures: GABA-to-creatinine ratios were measured in a 2-cm axial slab using magnetic resonance spectroscopic imaging immediately prior to and immediately after interventions.

Results: There was a 27% increase in GABA levels in the yoga practitioner group after the yoga session (0.20 mmol/kg) but no change in the comparison subject group after the reading session (0.001 mmol/kg) (t = −2.99, df = 7.87, p = 0.018).

Conclusions: These findings demonstrate that in experienced yoga practitioners, brain GABA levels increase after a session of yoga. This suggests that the practice of yoga should be explored as a treatment for disorders with low GABA levels such as depression and anxiety disorders. Future studies should compare yoga to other forms of exercise to help determine whether yoga or exercise alone can alter GABA levels.

INTRODUCTION

The practice of yoga includes postures (asanas), breathing methods (pranayama), chanting, and meditation (dhyana). As the use of these techniques increases, it is important that possible mechanisms underlying the effects of these practices be elucidated. Yoga has shown promise in improving symptoms associated with depression, anxiety disorders, and epilepsy. These disorders are associated with low γ-aminobutyric acid (GABA) states and are effectively treated with pharmacologic agents that increase the activity of the GABA system. We hypothesize that the ability of yoga to decrease symptoms in the same disorders is in part mediated through the GABA system and that such
changes can be measured using magnetic resonance spectroscopy (MRS).

LOW GABA LEVELS ARE FOUND IN MOOD AND ANXIETY DISORDERS

GABA dysfunction is a factor in mood disorders. In rats, multiple efficacious treatments for affective disorders (e.g., valproate, lithium, tricyclic antidepressants, and electroconvulsive therapy [ECT]) have been shown to increase brain GABA levels. Cerebrospinal fluid studies have demonstrated low GABA levels in depressed subjects compared to controls. MRS measurements of occipital GABA levels demonstrate that patients with major depression have decreased GABA levels compared to controls. When treated with serotonin selective reuptake inhibitors (SSRIs) or ECT, these same patients showed clinical improvement and increased GABA levels. Low GABA activity has also been observed in anxiety disorders.

YOGA AS A TREATMENT FOR MOOD AND ANXIETY DISORDERS

Yoga has been used to reduce symptoms associated with depression and anxiety disorders. Two reviews of the literature, reporting studies in which yoga was used to treat depression and anxiety, identified five randomized controlled trials of depression, of which four reported significant reduction in symptoms, and six randomized studies of anxiety, of which all reported a significant reduction in symptoms.

GABA LEVELS, SEIZURES, AND YOGA

Increasing brain GABA levels is one mechanism through which seizure frequency is decreased. MRS has documented increases in brain GABA levels associated with the administration of antiepileptic agents. Additionally, the practice of yoga has shown promise as an adjunctive treatment for epilepsy. We speculate that the antiepileptic effect of yoga, like the anti-epileptic effects of many seizure medications, is mediated through the GABA system.

Although research in this area would benefit from more carefully controlled studies, the available studies suggest that yoga may be used to decrease symptoms associated with low brain GABA states, such as depression, anxiety disorders, and epilepsy. We hypothesize that one mechanism through which yoga decreases the symptoms associated with these conditions is an increase in brain GABA levels. MRS is a valuable tool in this line of inquiry, because it allows changes in brain neurochemistry, in the form of GABA levels, to be correlated with a behavioral intervention, such as the practice of yoga. Understanding the relationship between behavior and neurochemistry would be an important step in understanding how the practice of yoga exerts its beneficial effects.

This study examined brain GABA levels in two groups: established yoga practitioners (YP) and comparison subjects (CS), to test the hypothesis that brain GABA levels increase in YP after a 60-minute yoga session relative to CS after a 60-minute reading period. To our knowledge, this is the first study to measure the effect of yoga on brain GABA levels.

MATERIALS AND METHODS

Study design

Subjects were recruited through advertisements. Informed consents, approved by the Boston Medical Center and McLean Hospital Institutional Review Boards, were obtained for each subject. All subjects were evaluated using the Structured Clinical Interview for DSM-IV Axis I Disorders: Patient Edition (SCID) and the Addiction Severity Index (ASI). All Axis I diagnoses were established using SCID criteria. Variables related to alcohol and nicotine use were obtained from the ASI. A yoga history, medical history, and physical examination were performed to determine eligibility. All subjects participated in a baseline magnetic resonance spectroscopic imaging (MRSI) scan that took approximately 60 minutes. Subjects then participated in a 60-minute intervention, consisting of the practice of yoga (YP) or reading (CS). The intervention was followed by a second MRSI scan.

Inclusion/exclusion criteria

Subjects were 18–45-year-old men and women with no past or current history of psychiatric illness, alcohol or substance abuse or dependence. Women were required to be using an acceptable method of birth control and have a negative urine pregnancy test prior to scanning. YP needed to report practicing yoga at least 2 days per week for at least 4 months, whereas CS needed to have no previous history of yoga practice. Individuals with contraindication to magnetic resonance evaluation (e.g., pregnancy, claustrophobia, a cardiac pacemaker, or ferrous implant) or medical histories that could have influenced scan results (e.g., head injury with loss of consciousness greater than 15 minutes, neurological illness, or serious medical illness) were excluded. No psychoactive prescription or nonprescription medications were allowed.

Interventions

The following procedures were used to facilitate a consistent yoga experience across subjects. YP were instructed to modify their usual practice to a 60-minute time period that focused on the yoga postures (asanas). Brief quiet pe-
riods at the beginning and end of the 60-minute intervention were allowed, but at least 55 minutes of the hour were spent doing the asanas and associated breathing exercises (pranayama). Pranayama or meditation not associated with an asana or the quiet periods described above were not allowed. All yoga sessions were observed by research staff with yoga training. The practice guidelines resulted in the observation of a practice containing very similar sequences of well-known asanas (e.g., sun salutation, standing poses, sitting poses, twists, supine and prone poses, inverted poses, balancing poses, and backbends). The study design stressed the practice of asanas because they are observable, as opposed to the internal state of meditation. The reading material used by CS was screened by research staff to exclude self-help or religious topics. Reading materials consisted of periodicals in the waiting area and books of popular fiction brought by the subjects.

**Menstrual stage as a covariate**

GABA levels change over the menstrual cycle with a decline during the luteal phase. Increased GABA levels associated with the follicular phase have been reported using MRS. Because menstrual stage could affect brain GABA levels, menstrual and contraceptive histories were obtained. Cycling female subjects were classified as being in the follicular stage if they were in the 14 days immediately after the first day of the last period or the luteal stage if they were in the 14 days immediately after the first day of the last period on the day of the imaging session. Women using hormonal forms of birth control were classified in a separate category because they were not cycling normally.

**Image acquisition and analysis**

**Equipment.** All data were collected on a 4 Tesla (T) Varian, UnityINOVA, whole-body MR system running VNMR 1.1b (Varian Inc., Palo Alto, CA), using a volumetric TEM design (Bioengineering Inc., Minneapolis, MN) RF head coil operating at 170.3 MHz for proton imaging.

**Proton-MRSI.** Using sagittal images, the bottom of the 2-cm-thick MRsI slab was aligned with the anterior commissural–posterior commissural line and then rotated 20 degrees. Oblique T1-weighted images were then acquired, allowing visualization of the cortex and deep gray structures for voxel placement. The two-dimensional J-resolved magnetic resonance spectroscopic imaging (2D-JMRSI) acquisition sequence used a slice-selective spin-echo MRSI scheme modified to incrementally acquire spectra at each phase-encode step with increasing echo-time to sample the J-coupling of the coupled metabolites. This acquisition collected 24 individual echo time (TE)-stepped spectra for each of the 96 circular, sparsely-sampled k-space points, with the TE ranging from 30 milliseconds to 490 milliseconds in 20 millisecond increments. Acquisition parameters were as follows: repetition time (TR) = 1.25 seconds, sampling matrix = 14 × 14 (circular-sparse), spectral bandwidth = 2 kHz, complex time-points = 1024, field of view (FOV) = 24 × 24 cm, slab thickness = 2 cm, number of averages = 1, nominal voxel volume = 4.5 cc (effective voxel size ~8 cc), total scan duration = 48 minutes.

**Proton-MRI processing/analysis.** The raw k-space data were read into a zero-padded 16 × 16 matrix, for all 24 TE. The TE-series (24 k-space 2D-JMRSI data sets) were zero-filled out to 64 k-space 2D-JMRSI data sets in TE and digitally apodized with a 0.5-Hz Gaussian filter prior to Fourier transforming in the TE dimension. Each J-resolved, 2D-JMRSI k-space set was then digitally filtered in kx and ky with a Hanning partial k-space filter to produce J- and spatially resolved spectra. For each subject, an 8 × 7 2D-JMRSI matrix was shifted in the x and y dimensions in order to position the rectangular matrix of voxels inside the skull. Voxels anterior to the ventricles were omitted because of artifacts from the sinus cavity in the frontal regions. In addition, voxels falling outside the brain were omitted, ensuring that only voxels fully inside the brain were included. Molar estimates of global brain GABA levels were calculated for each subject by correcting the TE-averaged creatine peak area for T1 and T2-weighting, using values of 1.72 seconds, and 0.273 seconds, respectively, and acquisition parameters (TR/TE min = 1.25 seconds/30 milliseconds). The raw GABA peak areas were then multiplied by a scaling correction factor, accounting for the differing number and intensity of resonances in the 2D GABA spectrum at J = 7.5 Hz, as well as the estimated 12% macromolecule contribution from the single-voxel data as determined in a prior study. Each corrected GABA/Cr ratio was then multiplied by 8.34 mmol/L, using the average of the gray/white-matter creatine levels (9.11 mmol/L/7.58 mmol/L) reported by Dager et al., to derive molar estimates of global GABA concentration in the brain. All spectra were fitted using the LC Model spectral analysis tool (Stephen Provencher, © 1992–2003). Statistical analyses. Data were examined for distribution normality and homogeneity of variance between groups. In bivariate analyses, categorical data were analyzed using Fisher’s exact tests. Continuous and ordinal data were analyzed using student (two-sample) t tests. Analyses of covariance (ANCOVA) were used to control for covariates of a priori theoretical interest (i.e., baseline GABA level, gender, and menstrual stage). Statistical significance required a two-tailed p value <0.05. Analyses used SPSS Version 13.0 (SPSS, Inc., Chicago, IL).

**RESULTS**

**Subject acquisition**

Twenty-nine (29) subjects came to the screening interview (14 YP/15 CS), of which 22 met entrance criteria (10 YP/12 forms of birth control were classified in a separate category because they were not cycling normally.
CS). Two (2) YP were not scanned because of illness. Twenty (20) subjects participated in the scanning session (8 YP/12 CS). Nineteen (19) subjects (8 YP/11 CS) had complete data sets; spectral data on 1 CS was of poor quality and could not be used. Subjects were healthy and did not take prescription medications, except birth control pills (YP = 3, CS = 3) and antibiotics for acne (YP = 1), in the month prior to the screening interview. Thirty days prior to enrollment, over-the-counter medications were limited to nonsteroidal anti-inflammatory medications and acetaminophen.

Demographics and yoga practice

There were no differences between the two cohorts in age, gender, education, ethnicity, marital status, body mass index, overall alcohol consumption or consumption to the point of intoxication in the last 30 days, nicotine use in the last 30 days, and nicotine use in the last year (Table 1). All subjects were white with the exception of one Asian in the comparison group. All anatomical magnetic resonance imaging scans were without brain abnormalities. Details of individual yoga practices are reviewed in Table 2. There was no history of yoga practice in the CS.

Analysis of baseline GABA levels

At baseline, the mean GABA levels were 0.75 ± 0.18 mmol/kg for the YP and 0.94 ± 0.20 mmol/kg for the CS (t = 2.11, df = 17, p = 0.050). When menstrual stage was treated as a covariate, the differences in the GABA baseline values between the YP and CS groups remained significant (F[1, 16] = 11.83, p = 0.003); the effect of menstrual stage was also significant (F[1, 16] = 7.66, p = 0.014). A post-hoc t test revealed that subjects in the follicular stage (N = 2) were a greater number of males in the control group, an ANCOVA contrasting the groups on GABA change values of the YP and CS groups remained significant (F[1, 16] = 6.51, df = 1, p = 0.021); baseline GABA levels did not exhibit an independent effect (F[1, 16] = 1.60, p = 0.224). Because there were no differences between the two cohorts in age, gender, education, ethnicity, marital status, body mass index, overall alcohol consumption or consumption to the point of intoxication in the last 30 days, nicotine use in the last 30 days, and nicotine use in the last year (Table 1), all of whom were in the CS group, exhibited significantly greater baseline GABA levels (1.05 ± 0.05 mmol/kg) than subjects in the luteal phase (N = 3) (0.75 ± 0.11 mmol/kg), all of whom were in the YP group (t = 3.40, df = 3, p = 0.04) (Table 3).

Analysis of changes in GABA levels

After the 60-minute interventions, the mean GABA levels were 0.95 ± 0.17 mmol/kg for the YP group and 0.94 ± 0.21 mmol/kg for the CS group. Using a two-sample t test, there was a significant difference between the GABA change values (from pre- to postintervention) of 0.20 ± 0.18 mmol/kg (range = 0.49) for the YP group and −0.001 ± 0.05 (range = 0.18) for the CS group (t = −2.99, df = 7.87, p = 0.018). Further analysis using an ANCOVA controlling for menstrual stage confirmed a significant difference in GABA change values (F[1, 16] = 10.19, p = 0.006), whereas menstrual stage was not found to exhibit an independent effect (F[1, 16] = 0.130, p = 0.72). In addition, a second ANCOVA controlling for baseline GABA levels found a significant difference between the GABA change values of the YP and CS groups (F[1, 16] = 10.36, df = 1, p = 0.005); the effect of gender was nonsignificant (F[1, 16] = 0.171, p = 0.68). Spectral data demonstrate an increase in the GABA peak after the yoga asana session in a single YP (Fig. 1). Individual GABA changes for all subjects are shown in Figure 2.

DISCUSSION

This study demonstrated that a 60-minute yoga asana session in established YP is acutely associated with a 27% increase in GABA levels. When differences in GABA change scores between groups were analyzed controlling for base-

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Table 1. Demographic Characteristics of Yoga Practitioners (YP) (N = 8) and Comparison Subjects (CS) (N = 11)

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>YP</th>
<th>CS</th>
<th>t/df</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>25.75 (5.15)</td>
<td>26.55 (7.62)</td>
<td>0.25</td>
<td>0.80</td>
</tr>
<tr>
<td>Gender (male)</td>
<td>1/8 (13%)</td>
<td>6/11 (55%)</td>
<td>Exact</td>
<td>0.15</td>
</tr>
<tr>
<td>Education (years)</td>
<td>16.56 (1.59)</td>
<td>15.82 (2.23)</td>
<td>−0.80</td>
<td>0.43</td>
</tr>
<tr>
<td>Ethnicity (white)</td>
<td>8/8 (100%)</td>
<td>10/11 (91%)</td>
<td>Exact</td>
<td>1.00</td>
</tr>
<tr>
<td>Alcohol consumptiona</td>
<td>2.75 (2.43)</td>
<td>3.82 (2.82)</td>
<td>0.86</td>
<td>0.40</td>
</tr>
<tr>
<td>Alcohol intoxicationb</td>
<td>0.25 (0.46)</td>
<td>0.91 (1.58)</td>
<td>1.31</td>
<td>0.21</td>
</tr>
<tr>
<td>Nicotine usea</td>
<td>0.00 (0.00)</td>
<td>0.00 (0.00)</td>
<td>Exact</td>
<td>1.00</td>
</tr>
<tr>
<td>Nicotine useb</td>
<td>3/8 (38%)</td>
<td>1/11 (9)</td>
<td>Exact</td>
<td>0.26</td>
</tr>
<tr>
<td>Marital status (never married)</td>
<td>7/8</td>
<td>10/11</td>
<td>Exact</td>
<td>0.86</td>
</tr>
<tr>
<td>Body–mass index</td>
<td>24.03 (3.56)</td>
<td>25.48 (5.44)</td>
<td>0.66</td>
<td>0.49</td>
</tr>
</tbody>
</table>

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aIn past 30 days, using the Addiction Severity Index.

bIn last year.

cUnequal variance.

dMarital status: YP: 7 never married, 1 married/CS: 10 never married, 1 divorced.

t, t-value; df, degrees of freedom.
line differences in GABA levels, menstrual stage, and gender, the differences in GABA change scores between the two groups remained significant. The acute elevation of brain GABA levels following the practice of yoga asanas in experienced practitioners suggests that yoga asanas may be an efficacious treatment for low GABA states.

The CS group also had two MRSI scans separated by a 60-minute reading period; however, they did not demonstrate an increase in GABA levels. Because the change in GABA levels was only found in the YP group, it is unlikely that the increase in GABA levels was related to increased familiarity with the scanning environment or a 60-minute

<table>
<thead>
<tr>
<th>No.</th>
<th>Type of yoga training</th>
<th>Years</th>
<th>Current sessions/week</th>
<th>Session length</th>
<th>Minutes/week</th>
<th>Meditation</th>
</tr>
</thead>
<tbody>
<tr>
<td>YP1</td>
<td>Ashtanga</td>
<td>3</td>
<td>4&lt;sup&gt;a&lt;/sup&gt;</td>
<td>90&lt;sup&gt;a&lt;/sup&gt;</td>
<td>360</td>
<td>S</td>
</tr>
<tr>
<td>YP2</td>
<td>Ashtanga</td>
<td>2</td>
<td>5&lt;sup&gt;a&lt;/sup&gt;</td>
<td>90&lt;sup&gt;a&lt;/sup&gt;</td>
<td>270</td>
<td>S</td>
</tr>
<tr>
<td>YP3</td>
<td>Ashtanga Vinyasa</td>
<td>4</td>
<td>5&lt;sup&gt;a&lt;/sup&gt;</td>
<td>105&lt;sup&gt;a&lt;/sup&gt;</td>
<td>525</td>
<td>M</td>
</tr>
<tr>
<td>YP4</td>
<td>Ashtanga Vinyasa</td>
<td>1.5</td>
<td>3&lt;sup&gt;a&lt;/sup&gt;</td>
<td>90&lt;sup&gt;a&lt;/sup&gt;</td>
<td>270</td>
<td>S</td>
</tr>
<tr>
<td>YP5</td>
<td>Ashtanga Vinyasa Hatha</td>
<td>10</td>
<td>5&lt;sup&gt;a&lt;/sup&gt;</td>
<td>60&lt;sup&gt;a&lt;/sup&gt;</td>
<td>300</td>
<td>M &amp; S</td>
</tr>
<tr>
<td>YP6</td>
<td>Ashtanga Vinyasa Iyengar Kundalini Power</td>
<td>7</td>
<td>6&lt;sup&gt;a&lt;/sup&gt;, 3&lt;sup&gt;b&lt;/sup&gt;</td>
<td>30&lt;sup&gt;a&lt;/sup&gt;, 150&lt;sup&gt;b&lt;/sup&gt;</td>
<td>630</td>
<td>S</td>
</tr>
<tr>
<td>YP7</td>
<td>Hatha Kirpaulo Iyengar Bikram</td>
<td>2</td>
<td>3&lt;sup&gt;a&lt;/sup&gt;</td>
<td>60&lt;sup&gt;b&lt;/sup&gt;</td>
<td>180</td>
<td>S</td>
</tr>
<tr>
<td>YP8</td>
<td>Hatha</td>
<td>2</td>
<td>4&lt;sup&gt;a&lt;/sup&gt;</td>
<td>90&lt;sup&gt;a&lt;/sup&gt;</td>
<td>360</td>
<td>N</td>
</tr>
</tbody>
</table>

<sup>a</sup>Yoga class.  
<sup>b</sup>Individual practice.  
S, 5–10 minutes of rest (shavaana) at end of yoga practice; M, regular meditation practice (>1 hour per week); N, no meditation practice.

The CS group also had two MRSI scans separated by a 60-minute reading period; however, they did not demonstrate an increase in GABA levels. Because the change in GABA levels was only found in the YP group, it is unlikely that the increase in GABA levels was related to increased familiarity with the scanning environment or a 60-minute reading period.

<table>
<thead>
<tr>
<th>ID</th>
<th>YP</th>
<th>GABA scan 1</th>
<th>GABA scan 2</th>
<th>GABA change</th>
<th>CS</th>
<th>GABA scan 1</th>
<th>GABA scan 2</th>
<th>GABA change</th>
</tr>
</thead>
<tbody>
<tr>
<td>YP1</td>
<td>H</td>
<td>0.95</td>
<td>0.87</td>
<td>−0.08</td>
<td>N/A</td>
<td>0.81</td>
<td>0.86</td>
<td>0.05</td>
</tr>
<tr>
<td>YP2</td>
<td>H</td>
<td>0.44</td>
<td>0.78</td>
<td>0.35</td>
<td>H</td>
<td>1.14</td>
<td>1.15</td>
<td>0.02</td>
</tr>
<tr>
<td>YP3</td>
<td>L</td>
<td>0.86</td>
<td>1.07</td>
<td>0.21</td>
<td>N/A</td>
<td>1.13</td>
<td>1.04</td>
<td>−0.10</td>
</tr>
<tr>
<td>YP4</td>
<td>H</td>
<td>0.83</td>
<td>0.82</td>
<td>−0.01</td>
<td>N/A</td>
<td>0.78</td>
<td>0.83</td>
<td>0.05</td>
</tr>
<tr>
<td>YP5</td>
<td>L</td>
<td>0.75</td>
<td>1.10</td>
<td>0.35</td>
<td>N/A</td>
<td>0.56</td>
<td>0.51</td>
<td>−0.05</td>
</tr>
<tr>
<td>YP6</td>
<td>L</td>
<td>0.64</td>
<td>0.74</td>
<td>0.10</td>
<td>N/A</td>
<td>0.77</td>
<td>0.76</td>
<td>−0.01</td>
</tr>
<tr>
<td>YP7</td>
<td>H</td>
<td>0.95</td>
<td>1.19</td>
<td>0.24</td>
<td>H</td>
<td>1.03</td>
<td>1.12</td>
<td>0.09</td>
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<tr>
<td>YP8</td>
<td>N/A</td>
<td>0.62</td>
<td>1.03</td>
<td>0.41</td>
<td>F</td>
<td>1.08</td>
<td>1.09</td>
<td>0.01</td>
</tr>
</tbody>
</table>

Menstrual stage: L, Luteal Stage; F, Follicular Stage; H, Hormonal Therapy; N/A, male, GABA, γ-aminobutyric acid. Scan 1 and 2 = GABA levels in mmol/kg.
period between the two scans. After the reading intervention, the mean GABA level for the CS group was 0.94 ± 0.21 mmol/kg. Multiple subjects in both groups had GABA levels greater than 1.0 mmol/kg, suggesting that the lack of change in the CS was not caused by a ceiling effect.

The finding of lower baseline GABA levels in the YP group was unexpected. The baseline difference in GABA levels between groups may be explained by the distribution of females in different stages of the menstrual cycle. The luteal phase is associated with lower GABA levels than the follicular stage.31,32 Of the subjects assigned to a menstrual stage, all subjects in the luteal stage were in the YP group, whereas all subjects in the follicular were in the CS group, a distribution that would favor higher baseline GABA lev-

FIG. 1. (A) The chemical-shift imaging grid depicts the multiple voxels within the brain that are totaled to estimate the γ-gamma aminobutyric acid (GABA) level of the whole slab. (B) J-resolved sample spectra (J = 7.5 Hz) are shown from two regions before and after yoga practice in a yoga practitioner. Spectra are displayed with LC Model fit (Stephen Provencher, © 1992–2003) and are not filtered. (C) The spectral data are removed, leaving only LC Model fit magnified 11 × to show the GABA resonance area at 2.95 ppm, which demonstrates an increase in GABA peak after yoga practice.

FIG. 2. Individual GABA levels (pre- and post-intervention) for yoga practitioners (YP) (N = 8) and comparison subjects (CS) (N = 11).
els in the CS group, as was found. Although baseline GABA values may have been affected by menstrual stage, changes in GABA levels after a yoga session seemed to be unrelated to menstrual stage. This study indicates that menstrual stage should be considered when measuring GABA levels.

One strength of the present study is the inclusion of a carefully screened population that is free of psychiatric and neurologic illness and psychoactive medications. The inclusion of YP from many different schools with varying degrees of practice can be viewed as both a strength and weakness in this study. The consistency of the yoga sessions observed allows comparison and implies that the changes in GABA levels are related to a sequence of asanas and pranayama and not limited to a specific school of yoga. Although the study was not randomized, nor was there a crossover where YP were scanned after a reading intervention, the results of this study strongly suggest that yoga can increase GABA levels. This pilot study did not control for activity levels. Other forms of exercise, such as walking, may lead to the same increases in GABA levels as observed in the YP group. Further studies are required to understand the relationship of GABA levels to standard exercises.

CONCLUSIONS

The World Health Organization reports that mental illness makes up 15% of the global burden of disease. Depression and anxiety disorders both contribute to this burden, and are attended by low GABA levels, relative to normal controls, as measured by MRS. These disorders are successfully treated with pharmacologic agents known to increase the activity of the GABA system. The significant comorbidity between depression and anxiety disorders and their successful treatment with agents that affect the GABA system support the theory that low GABA activity is involved in the pathology of these disorders and increased GABA activity is associated with symptom reduction. The literature suggests that the practice of yoga is also associated with symptom reduction in depression and anxiety disorders. This study demonstrates that brain GABA levels increase by 27% after a 1-hour yoga asana practice in experienced partakers and suggests that the practice of yoga should be explored and compared to other exercise modalities as a treatment or adjunctive treatment for disorders associated with low GABA states. The development of an inexpensive, widely available intervention, with few side effects, that is effective in alleviating the symptoms of disorders associated with low GABA states has clear public health advantages.

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