Supporting Information

Results

SPS induced acute and sustained PTSD-like alterations in state-dependent qEEG power spectra in the parietal cortex.

SPS significantly disrupted the normal qEEG power spectra in the parietal cortex in a manner similar to that observed in the frontal cortex with a few notable differences. On Day 0, SPS significantly altered qEEG power spectra in the parietal cortex during light phase wake (black line, Figure S1A) (frequency [F100,900 = 12.19, P < 0.0001], treatment [F1,9 = 37.86, P < 0.0001] interaction [F100,900 = 12.19, P < 0.0001]) and dark phase wake (black line, Figure S1D) (frequency [F100,900 = 6.7, P < 0.0001], interaction [F100,900 = 6.7, P < 0.0001]) but did not cause reduced high gamma power as in the frontal cortex. During light phase NREM sleep, SPS caused a short-term rebound in delta power (black line, Figure S1B) (frequency [F100,900 =7.69, P < 0.0001], treatment [F1,9 = 8.99, P = 0.0150], interaction [F100,900 = 7.69, P < 0.0001]), but had no effect during dark phase NREM sleep (black line, Figure S1E). Unlike in the frontal cortex, SPS caused a significant increase in alpha and low beta power during light phase REM sleep, (black line, Figure S1C) (frequency [F100,800 = 3.61, P < 0.0001], interaction [F100,800 = 3.61, P < 0.0001]), and dark phase REM sleep(black line, Figure S1F) (frequency [F100,900 = 8.09, P < 0.0001], interaction [F100,900 = 8.09, P < 0.0001]), but had little or no effect on theta.

Similar to the frontal cortex, SPS-induced qEEG changes were largely sustained in the parietal cortex during light phase wake (colored lines, Figure S1A) (frequency [F100,900 = 4.0, P < 0.0001], treatment [F3,27 = 9.17, P = 0.0002], interaction [F300,2700 = 5.59, P < 0.0001])

and dark phase wake (colored lines, Figure S1D) (frequency [F100,900 = 4.48, P < 0.0001], interaction [F300,2700 = 2.52, P < 0.0001]); light phase NREM, (colored lines, Figure S1B) (frequency [F100,900 = 5.65, P < 0.0001], treatment [F3,27 = 4.65, P = 0.0095], interaction [F300,2700 = 3.88, P < 0.0001]); and light phase REM (colored lines, Figure S1C) (frequency [F100,900 = 1.47, P = 0.0028], interaction [F300,2700 = 1.89, P < 0.0001]), and dark phase REM sleep (colored lines, Figure S1F) (interaction [F300,2700 = 2.13, P < 0.0001]).

SHAM treatment had no sustained effect on sleep-wake architecture.

See Table S1 for statistical analysis.

SHAM treatment had minor effects on state-dependent qEEG power spectra in the frontal and parietal cortices.

In contrast to the robust and sustained effects of SPS, SHAM treatment had only minor effects on qEEG relative spectral power in the frontal cortex on Day 0 during light phase wake (Figure S2A) (frequency [F100,900 = 4.95, P < 0.0001], treatment [F1,9 = 5.8, P = 0.04], interaction [F100,900 = 4.95, P < 0.0001]), dark phase wake (Figure S2D) (frequency [F100,900 = 2.57, P < 0.0001], interaction [F100,900 = 2.57, P < 0.0001]), light phase NREM (Figure S2B) (frequency [F100,900 = 8.41, P < 0.0001], treatment [F1,9 = 8.14, P = 0.0190], interaction [F100,900 = 8.41, P < 0.0001]), dark phase NREM (Figure S2E) (frequency [F100,900 = 2.28, P < 0.0001]), interaction [F100,900 = 2.28, P < 0.0001]), light phase REM (Figure S2C) (frequency [F100,900 = 1.37, P = 0.0134], interaction [F100,900 = 1.37, P = 0.0134]), and dark phase REM sleep (Figure S2F) (frequency [F100,900 = 1.36, P = 0.0139], interaction [F100,900 = 1.36, P = 0.0139]).

Michael Nedelcovych

On Days 1, 2, and 7, SHAM treatment modestly altered power spectra during light phase wake (Figure S2A) (frequency [F100,800 = 3.34, P < 0.0001], interaction [F300,2400 = 1.51, P < 0.0001]), dark phase wake (Figure S2D) (frequency [F100,800 = 2.73, P < 0.0001], interaction [F300,2400 = 1.85, P < 0.0001]), light phase NREM (Figure S2B) (interaction [F300,2400 = 1.48, P < 0.0001]), dark phase NREM (Figure S2E) (frequency [F100,800 = 3.81, P < 0.0001], interaction [F300,2400 = 1.36, P = 0.0001]), light phase REM (Figure S2C) (frequency [F100,800 = 2.00, P < 0.0001], interaction [F300,2400 = 2.42, P = 0.0001]), and dark phase REM sleep (Figure S2F) (frequency [F100,800 = 3.48, P < 0.0001], interaction [F300,2400 = 1.98, P = 0.0001]).

In the parietal cortex on Day 0, SHAM treatment modestly altered power spectra during light phase wake (Figure S3A) (frequency [F100,900 = 11.79, P < 0.0001], treatment [F1,9 = 16.38, P = 0.0029], interaction [F100,900 = 11.79, P < 0.0001]), dark phase wake (Figure S3D) (frequency [F100,900 = 6.28, P < 0.0001], treatment [F1,9 = 10.33, P = 0.0106], interaction [F100,900 = 6.28, P < 0.0001]), light phase NREM (Figure S3B) (frequency [F100,900 = 4.74, P < 0.0001], treatment [F1,9 = 14.54, P = 0.0041], interaction [F100,900 = 4.74, P < 0.0001], treatment [F1,9 = 14.54, P = 0.0041], interaction [F100,900 = 4.74, P < 0.0001], interaction [F100,900 = 2.14, P < 0.0001], interaction [F100,900 = 2.14, P < 0.0001], interaction [F100,900 = 1.38, P = 0.0107], treatment [F1,9 = 5.19, P = 0.0488], interaction [F100,900 = 1.38, P = 0.0107]).

On Days 1, 2, and 7, SHAM treatment modestly altered power spectra in the parietal cortex during light phase wake (Figure S3A) (frequency [F100,800 = 3.42, P < 0.0001], interaction [F300,2400 = 1.50, P < 0.0001]), dark phase wake (Figure S3D) (frequency [F100,800 = 4.20, P < 0.0001], interaction [F300,2400 = 1.66, P < 0.0001]), light phase NREM

3

(Figure S3B) (interaction [*F*300,2400 = 1.51, P < 0.0001]), and dark phase REM sleep (Figure S3F) (frequency [*F*100,800 = 1.45, P = 0.0044], interaction [*F*300,2400 = 1.30, P = 0.0010]).

SHAM treatment had no prolonged effect on SWA

Relative to BL, SHAM increased SWA on Day 0 (time [F2,54 = 32.03, P < 0.0001], interaction [F2,54 = 5.68, P = 0.0058], treatment [F1,54 = 42.23, P < 0.0001]) consistent with the rebound effects of sleep deprivation (*32*), but produced no prolonged effect on SWA.

SHAM treatment had minor effects on body temperature

In contrast to SPS, SHAM treatment had only minor effects on body temperature (Figure S5) during wake (Day 0: hour [F8,162 = 4.07, P = 0.0002], treatment [F1,162 = 23.24, P < 0.0001], interaction [F8,162 = 4.07, P = 0.0002]; Days 1,2,7: hour [F11,418 = 4.2, P < 0.0001], treatment [F3,418 = 4.35, P = 0.005]), NREM (Day 0: hour [F8,162 = 3.42, P = 0.0012], treatment [F1,162 = 15.79, P = 0.0001], interaction [F8,162 = 3.42, P = 0.0012]; Days 1,2,7: hour [F11,420 = 4.00, P < 0.0001], treatment [F3,420 = 4.13, P = 0.0066]), and REM sleep (Days 1,2,7: hour [F11,385 = 2.37, P = 0.0077]).

Michael Nedelcovych

Figure S1. SPS induced acute and sustained alterations in qEEG power spectra in the parietal cortex. In the light phase (top panels), SPS caused (A) a transient increase in high gamma; (B) an acute rebound, but a persistent subsequent reduction in delta power during NREM sleep; and (C) an acute increase in alpha, and a prolonged decrease in delta power during REM sleep. In the dark phase (bottom panels), SPS caused (D) a transient increase in theta, alpha, and low gamma with a sustained increase in beta during wake; (E) no change during NREM sleep; and (F) an acute increase in alpha during REM sleep. Day 0 only includes values from remaining hours of the light phase immediately after SPS treatment. Data are depicted as mean + SEM (n = 9-10). Background shades delineate power bands delta (δ), theta (θ), alpha (α), beta (β), low and high gamma (γ). Comparison between treatment and BL performed by repeated measures two-way ANOVA. Colored lines below data points correspond to each day and indicate P < 0.01 in Bonferroni *post hoc* test.

Figure S2. SHAM treatment had minor effects on qEEG power spectra in the frontal cortex. In the light phase (top panels), SHAM treatment had only minor effects during light phase (A) wake, (B) NREM, and (C) REM sleep, and during dark phase (D) wake, (E) NREM, and (F) REM sleep. Day 0 only includes values from remaining hours of the light phase immediately after SHAM treatment. Data are depicted as mean + SEM (n = 9-10). Background shades delineate power bands delta (δ), theta (θ), alpha (α), beta (β), low and high gamma (γ). Comparison between treatment and BL performed by repeated measures two-way ANOVA. Colored lines below data points correspond to each day and indicate P < 0.01 in Bonferroni *post hoc* test.

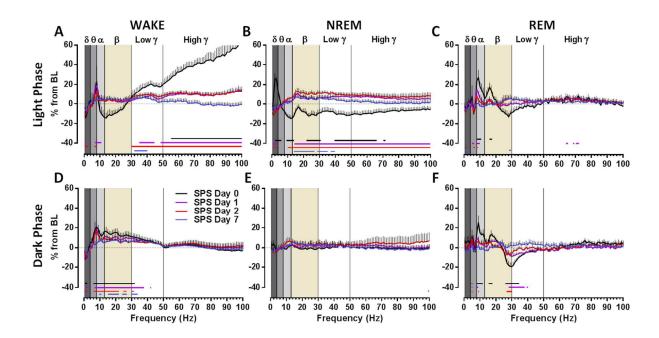
Figure S3. SHAM treatment had minor effects on qEEG power spectra in the parietal cortex. In the light phase (top panels), SHAM treatment had only minor effects during light phase (A)

wake, (B) NREM, and (C) REM sleep, and during dark phase (D) wake, (E) NREM, and (F) REM sleep. Day 0 only includes values from remaining hours of the light phase immediately after SHAM treatment. Data are depicted as mean + SEM (n = 9-10). Background shades delineate power bands delta (δ), theta (θ), alpha (α), beta (β), low and high gamma (γ). Comparison between treatment and BL performed by repeated measures two-way ANOVA. Colored lines below data points correspond to each day and indicate P < 0.01 in Bonferroni *post hoc* test.

Figure S4. SHAM treatment had no prolonged effect on SWA. During the light (rodent quiescent) phase, SHAM treatment caused an initial rebound in slow wave activity (SWA), but subsequently had no effect. Data are depicted as mean - SEM (n = 9-10). $^{P} < 0.01$, $^{P} < 0.001$, Day 0 vs. BL in Bonferroni *post hoc* test.

Figure S5. SHAM treatment had minor effects on body temperature. SHAM treatment moderately reduced temperature relative to BL at various time points after treatment. Black bars indicate dark phases. Comparison between treatment and BL performed by two-way ANOVA. * P < 0.05 in Bonferroni *post hoc* test.

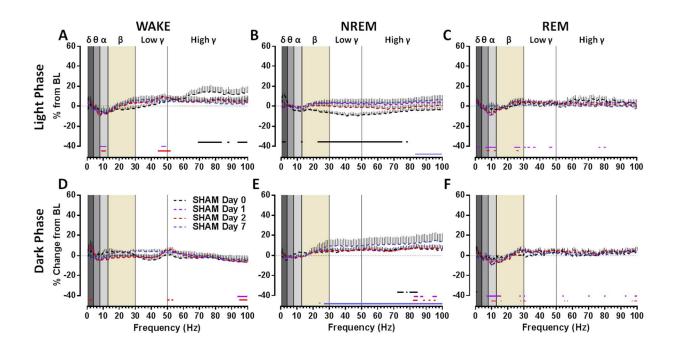




	Light Phase					
	SHAM BL	SHAM Day 1	SHAM Day 2	SHAM Day 7	F	Р
WAKE (min/hr)	16.8 ± 0.7	16.5 ± 0.7	16.3 ± 0.6	14.8 ± 0.8	2.59	.125
NREM (min/hr)	35.6 ± 0.8	36.2 ± 0.6	36.7 ± 0.6	37.5 ± 0.8	1.76	.181
REM (min/hr)	7.5 ± 0.3	7.4 ± 0.3	7.0 ± 0.3	7.6 ± 0.3	1.06	.385
WAKE bouts/hr	12.8 ± 0.7	12.9 ± 0.7	12.8 ± 1.0	11.6 ± 1.1	1.51	.238
NREM bouts/hr	13.5 ± 0.8	13.4 ± 0.6	13.2 ± 0.9	12.3 ± 0.9	1.39	.270
REM bouts/hr	5.3 ± 0.3	5.0 ± 0.3	5.2 ± 0.4	5.5 ± 0.3	.914	.449
WAKE bout (min)	1.3 ± 0.1	1.3 ± 0.1	1.3 ± 0.1	1.3 ± 0.1	.350	.790
NREM bout (min)	2.8 ± 0.2	2.8 ± 0.2	2.9 ± 0.2	3.2 ± 0.3	2.60	.076
REM bout (min)	1.5 ± 0.1	1.5 ± 0.1	1.4 ± 0.1	1.4 ± 0.1	.603	.620
			Dark Phase			
	SHAM BL	SHAM Day 1	SHAM Day 2	SHAM Day 7	F	Р
WAKE (min/hr)	39.8 ± 0.9	37.5 ± 1.7	37.9 ± 1.4	37.7 ± 0.7	2.22	.111
NREM (min/hr)	17.6 ± 0.9	19.5 ± 1.5	19.2 ± 1.3	19.69 ± 0.7	2.42	.091
REM (min/hr)	2.6 ± 0.2	3.0 ± 0.3	2.9 ± 0.3	2.6 ± 0.2	1.29	.300
WAKE bouts/hr	9.4 ± 0.4	11.2 ± 0.6	11.1 ± 0.5	10.8 ± 0.8	2.31	.102
NREM bouts/hr	9.3 ± 0.4	11.2 ± 0.6	11.1 ± 0.5	10.9 ± 0.8	2.41	.092
REM bouts/hr	2.6 ± 0.3	2.8 ± 0.3	2.8 ± 0.3	2.8 ± 0.2	.208	.890
WAKE bout (min)	4.4 ± 0.3	3.5 ± 0.3	3.6 ± 0.3	3.7 ± 0.3	2.06	.132
NREM bout (min)	1.9 ± 0.1	1.8 ± 0.1	1.7 ± 0.1	1.9 ± 0.1	.485	.696
REM bout (min)	1.0 ± 0.0	1.1 ± 0.0	1.0 ± 0.0	1.0 ± 0.1	1.14	.353

 Table S1. SHAM treatment had no persistent effect on sleep-wake architecture.







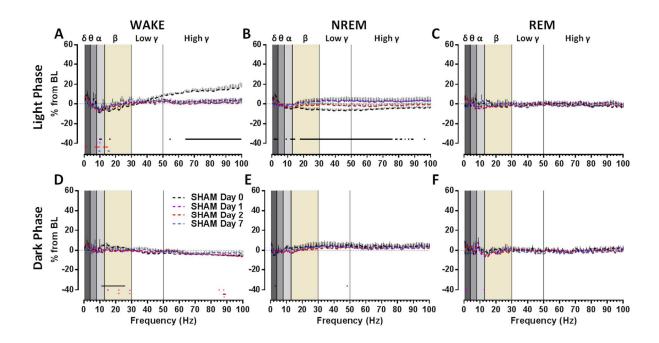


Figure S4

