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A randomized controlled study of power posing before public speaking exposure for social anxiety disorder: No evidence for augmentative effects

Michelle L. Davis[a],*, Santiago Papini[a], David Rosenfield[b], Karin Roelofs[c], Sarah Kolb[d], Mark B. Powers[a,e], Jasper A.J. Smits[a]

[a] Department of Psychology and Institute for Mental Health Research, The University of Texas at Austin, United States
[b] Department of Psychology, Southern Methodist University, United States
[c] Donders Institute for Brain Cognition and Behaviour and Behavioural Science Institute, Radboud University, Netherlands
[d] Department of Psychology, University of Würzburg, Germany
[e] Baylor University Medical Center, United States

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ABSTRACT

This manuscript details a randomized controlled study designed to test the efficacy of power posing (i.e., briefly holding postures associated with dominance and power) as an augmentative strategy for exposure therapy for social anxiety disorder (SAD). Seventy-three individuals diagnosed with SAD were assigned to one of three conditions: power posing, submissive posing, or rest (no posing) prior to participating in an exposure therapy session. Participants were assessed for between-group differences in pre- and post-manipulation salivary hormone levels, within-session subjective experiences of fear, and pre- and 1-week post-treatment SAD severity outcome measures. Though the intervention resulted in decreased SAD symptom severity one week later, analyses revealed no significant between-group differences on any tested variables. Accordingly, this study provides no evidence to suggest that power posing impacts hormone levels or exposure therapy outcomes.

1. Introduction

Testosterone, a steroid androgen hormone, has been shown to be an important regulator of social motivational behavior, and particularly approach behavior. Several studies have now demonstrated that testosterone administration increases social approach motivation (Bos, van Honk, Ramsey, Stein, & Hermans, 2013; Enter, Spinhoven, & Roelofs, 2014; Hermans, Putman, Baas, Koppeschaar, & Van Honk, 2006; Hermans, Ramsey, & Van Honk, 2008; Radke et al., 2015; Terburg, Aarts, & Van Honk, 2012; van Honk et al., 2001; Van Honk, Peper, & Schutter, 2005; Van Honk & Schutter, 2007). Given these observations, testosterone levels emerge as a potentially important target for clinical interventions that rely on approach behavior. Exposure therapy, an established treatment for social anxiety disorder (SAD; Hofmann & Smits, 2008), involves systematically and repeatedly approaching feared social cues (i.e., stimuli perceived as threatening) to re-establish a sense of safety around these cues (i.e., fear extinction; Hofmann, 2008; Otto, Smits, & Reese, 2004; Powers, Smits, Leyro, & Otto, 2007). Though efficacious for SAD (Hofmann & Smits, 2008), there is much room for improvement, and thus targeting testosterone levels may hold clinical value. Indeed, recent basic research shows that testosterone administration can facilitate approach toward

angry (i.e., perceived socially threatening) faces (Enter, Spinhoven, & Roelofs, 2016), and reduce gaze avoidance (Enter, Terburg, Harrewijn, Spinhoven, & Roelofs, 2016) among persons with SAD. Accordingly, increasing testosterone levels prior to exposure therapy may lead to enhanced fear extinction and thus better outcomes. In addition to testing the effects of direct testosterone administration, it is also important to develop and test non-pharmacological augmentation strategies that are preferable to patients and easily implemented into an exposure session, and thus easier to disseminate (McHugh, Whitton, Peckham, Welge, & Otto, 2013).

Results from one study indicate that it may be possible to manipulate testosterone via changes in posture (Carney, Cuddy, & Yap, 2010). In this study, men and women were asked to hold either poses associated with dominance and high power (e.g., expansive, open postures; or power poses) or poses associated with submission and low power (e.g., contractive, closed postures; or submissive poses) for two minutes. Participants in the power posing condition evidenced increases in testosterone levels, decreases in cortisol levels, and increases in subconscious feelings of power and risk taking. Though a recent study (Ranehill et al., 2015) – published after the current study was initiated – successfully replicated the findings regarding power posing leading to increased subjective feelings of power, they found no impact of postural

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manipulation on hormone levels. However, it is important to note that the Ranehill study protocol deviated from the Carney study in important ways (e.g., participants were given the rationale for the postures rather than using deception; see review by Carney et al. (2015)). Additionally, a recent review notes a history of the embodied effects of expansive postures on feelings of dominance and power (Carney, Cuddy, & Yap, 2015), including increased feelings of power, action orientation, and risk taking, as well as decreased threat perception and fear. Accordingly, there is overlap between the psychological and anxiolytic effects of power posing and the effects of testosterone administration, and the Carney et al. (2010) study lends preliminary evidence that power posing may cause increases in endogenous testosterone levels.

1.1. Aims

The current manuscript details a proof-of principle study examining power posing as an augmentative strategy for exposure therapy for SAD. We tested whether power posing (compared to submissive posing or rest) would (1) increase testosterone; (2) result in superior exposure therapy outcomes (i.e., decreased symptom severity and fear responding during a public speech); and (3) whether testosterone changes predicted future symptom reduction among individuals engaging in power posing. Due to the aforementioned research indicating potential decrements in cortisol (Carney et al., 2010) and/or anxiolytic effects of power posing (Riskind & Gotay, 1982; Welker, Oberleitner, Cain, & Carré, 2013), we also tested whether power posing (compared to submissive posing or rest) would (4) decrease cortisol; and (5) result in increased self-reported fear within the exposure session.

2. Method

2.1. Participants

Participants (aged 18–70) were recruited from advertisements at the University of Texas and in the Austin community (see Table 1). Participants (N = 73) were diagnosed with SAD as their primary psychiatric diagnosis (i.e., the most important source of current distress) and endorsed fear of public speaking as a primary concern. Exclusion criteria included current use of testosterone enhancing products or corticosteroid medications, a lifetime history of bipolar or psychotic disorders, a history of substance or alcohol use disorders in the past six months, significant suicidal ideation, current utilization of psychotherapy for SAD, and prior non-response to exposure therapy. Participants using psychotropic medication could participate in the study if they had been on a stable dose of medication for three weeks prior to the treatment session. Participants were not paid for their participation, though students were offered course credit. All participants completed in the informed consent process prior to beginning the study procedures. This study was registered on clinicaltrials.gov (NCT02482805).

Table 1
Participant Characteristics by Condition (N = 73).

Variable	Power Posing (n = 26)		Submissive Posing (n = 27)		Rest (n = 20)	
	Mean	SD	Mean	SD	Mean	SD
Age	26.88	6.99	25.26	8.99	24.60	5.83
LSAS-performance	37.96	14.39	36.19	10.92	37.85	10.16
	n	%	n	%	n	%
White	14	53.8	14	51.9	11	55.0
Female	18	69.2	18	66.7	16	80.0
College graduate	11	42.3	16	59.3	9	45.0

2.2. Procedures

2.2.1. Eligibility screening

Participants first completed an online prescreen, which was examined for clear exclusion criteria (e.g., no social anxiety symptoms, current exposure therapy treatment, etc.). Participants who appeared eligible were invited to participate in a phone interview for diagnostic screening, using the Mini International Neuropsychiatric Interview (M.I.N.I.; Sheehan et al., 1998) to evaluate the presence of psychiatric inclusion and exclusion criteria. Eligible participants were invited to participate in the treatment session. See Fig. 1 for consort diagram.

2.2.2. Randomization

Participants were randomized to participate in power posing, submissive posing, or rest (no posture manipulation) using a randomization sheet developed by an independent investigator. Randomization was blocked by subject pool (i.e., community participants versus University students, and low SAD severity versus high SAD severity) to control for potential differences in compensation and baseline severity levels. The cut-off for high SAD severity was a score of 70 or higher on the pretreatment Liebowitz Social Anxiety Scale (LSAS) total score. Randomization information was placed in envelopes that were not opened by the therapist until immediately prior to the posture manipulation (i.e., the therapist was blind to treatment condition throughout the rationale and exposure planning components of the treatment session).

2.2.3. Treatment session

Rodebaugh, Levinson, and Lenze (2013) described a standardized test (i.e., clinical assay) for examining augmentative strategies (e.g., pharmacotherapy) for exposure therapy for SAD in an efficient, feasible manner. This protocol involves a standardized exposure therapy session in which participants plan a public speaking exposure expected to elicit a peak fear rating (using the Subjective Units of Distress Scale or SUDS, described below) of 75. The intent of this approach is to standardize the experience of anxiety (using predicted SUDS), rather than standardizing elements of the procedures, in order to provide a "clinical assay" that can be employed to test augmentation strategies and mechanisms of change. In addition to the study by Rodebaugh et al. (2013), there are several examples of fruitful research projects utilizing similar "clinical assays" to test augmentative strategies, many of which have formed the basis for subsequent treatment development research (Powers, Smits, & Telch, 2004; Ressler et al., 2004; Sloan & Telch, 2002; Smits et al., 2013; Telch et al., 2014; Wolitzky & Telch, 2009). We conducted a similar (though not identical) protocol to the Rodebaugh study, allowing participants to vary the following flexible elements in their exposure: topic of speech, utilization of confederate audience members, availability of notes, time for preparation, and reaction of experimenter. Therapists were three graduate-student level therapists trained and supervised by the senior author.

Prior to the session, participants completed questionnaires assessing demographic and SAD severity measures. At the onset of the session, participants first watched a video describing the cognitive-behavioral model of SAD and the rationale for exposure therapy. Therapists then familiarized the participant with the SUDS scale and guided participants in designing a 5-min speech exposure with specific behavioral goals designed to decrease avoidance. The participants then participated in the posturing manipulation protocol (see below). Following the posturing manipulation, they began their speeches after a brief wait period. Participants provided fear ratings at the start and end of each speech (recalling the highest level of fear they experienced over the course of the speech), and delivered the same 5-min speech (with the same behavioral goals) three times. After the exposures, participants processed the exercise with the therapist.
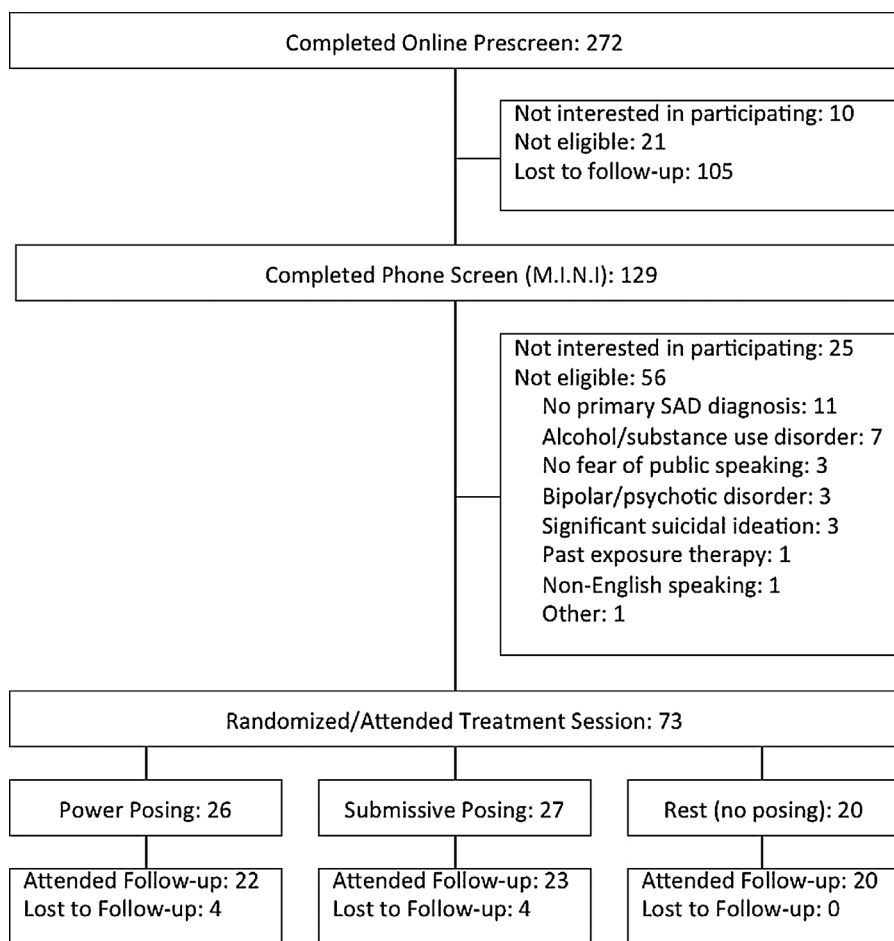
Fig. 1. Consort diagram.

2.2.4. Posturing manipulation protocol

Individuals were randomized to one of three posturing conditions completed prior to the speech exposures. In order to prevent attribution effects, participants were not informed of the true purpose of these poses. Instead, we followed the procedures of the Carney et al. (2010) study, providing the participants with the rationale that they were being put into certain bodily positions to test physiological responses (via the heart rate and skin conductance monitors) as a function of sensor placement relative to their heart. After planning their speech exposure, participants put on heart rate and skin conductance monitors (i.e., watches and finger bands). Participants in the power posing condition were asked to hold two 1-min, open, expansive postures associated with high power. Participants in the submissive posing condition were asked to hold two 1-min, closed, contractive postures associated with low power. These postures were identical to those used in the Carney et al. (2010) study. Participants in the rest (no posture) condition were asked to sit and wait for 2 min, with the rationale that their physiological responses were being tested (but without the sensor placement rationale). Hormone samples were assessed immediately prior to the posturing manipulation protocol, then 17 min later (see full description of hormone sampling below).

2.2.5. Post-treatment session

At one week post-treatment, individuals returned to the laboratory to complete questionnaires assessing for changes in SAD severity, as well as to participate in a behavioral assessment task (BAT). They delivered the same 5-min speech they planned and delivered during the treatment session (to the same audience and with the same speech element manipulations and behavioral goals) as the same indices were measured as in the exposure session (e.g., SUDS). After the BAT, the

therapist discussed with the participant how they could continue to apply home-practice strategies. Participants were then debriefed about the posturing deception and those who wished to receive additional exposure-based treatment were provided with CBT treatment referrals in the Austin community.

2.3. Measures

2.3.1. Salivary hormone samples

Participants provided a passive drool saliva sample (1) immediately prior to the behavioral posturing manipulation, then (2) 15 min later (e.g., approximately 17 min after the first sample), immediately prior to the speech exposures. This is in line with the Carney et al. (2010) study, wherein a change in hormone levels was evident 17 min after power posing. Saliva samples were collected using the passive drool protocol (in 2 ml vials). The samples were stored at −20 °C until radio immune assay was performed by Dr. Clemens Kirschbaum's laboratory in Dresden, Germany, which yielded both testosterone and cortisol values (for descriptions of specific methodology used by this laboratory, see: Miller, Plessow, Kirschbaum, & Stalder, 2013; Reardon, Herzhoff, & Tackett, 2016).

2.3.2. Liebowitz social anxiety scale (LSAS)

The LSAS is a 24-item scale which yields separate scores for fear and avoidance in both social and performance situations. It is a commonly used outcome measure in SAD treatment studies and has very good psychometric properties (Heimberg et al., 1999; Safren et al., 1999). As participants in this study endorsed fear of public speaking as their primary concern (and because the exposure consisted of public speaking exposure), we utilized the "social performance" subscale of

the LSAS as a primary outcome measure. This subscale utilizes fear and avoidance ratings for the 13 items of the LSAS assessing performance situations (e.g., "giving a report to a group", "acting, performing, or giving a talk in front of an audience"). The range of the LSAS performance total subscale is 0–78. The LSAS was completed prior to the treatment session and one week later.

2.3.3. Subjective units of distress scale (SUDs)

Participants provided fear ratings at the beginning and end of each speech (i.e., initial, end, and retrospective peak SUDS) using the SUDS scale (Wolpe & Lazarus, 1966), which ranges from 0 to 100. We used the peak rating within the exposure session (i.e., the highest rating of the three speeches), as well as the difference between this peak rating and the end (final) rating after the third speech, as process measures to examine for between-group differences in within-session fear activation and fear extinction. We opted to use the peak SUDS rating across the three exposures within the exposure session (rather than an average peak SUDS) given the instructions in the study protocol (i.e., designing an exposure to elicit a peak SUDS rating of 75 across the three exposures). We also looked for between-group differences in peak fear during the BAT as a primary outcome measure.

2.4. Analyses

An a priori power analysis (power = 0.8, α = 0.05) was conducted to determine the sample size necessary to detect hormonal effects given the effect sizes in the Carney et al. (2010) study (i.e., $f = 0.36$ for testosterone and $f = 0.47$ for cortisol findings). This analysis indicated that we needed a total sample size of at least 63 to detect the smallest of these effects ($f = 0.36$). Accordingly, our sample provided us an adequate (though not definite, given power of 0.8) chance of detecting similarly sized effects to those seen in the Carney study.

Three separate AN(C)OVAs were performed with treatment group (0, 1, 2) as a predictor variable to determine whether individuals assigned to power posing (relative to submissive posing or rest) evidenced the following changes in dependent variables: (Aim 1) increased post-manipulation testosterone levels (covarying for pre-manipulation testosterone levels), (Aim 2) decreased peak SUDS at BAT (covarying for pre-treatment SAD severity using the LSAS-performance score), and decreased post-treatment LSAS-performance (covarying for pre-treatment LSAS-performance). We also tested whether testosterone changes from pre- to post-manipulation were predictive of primary outcome variables (Aim 3). Both hormone variables (i.e., changes in both testosterone and cortisol from pre- to post-manipulation) were added to the models as predictors simultaneously, as they could have opposite effects. Finally, to test whether power posing elicited anxiolytic effects, two additional AN(C)OVAs were performed with treatment group as a predictor variable to determine whether individuals who participated in power posing evidenced (Aim 4) decreased post-manipulation cortisol levels (covarying for pre-manipulation cortisol levels), and (Aim 5) lower peak SUDS within the exposure session. In all analyses, demographic (sex, age, race, cohabitation status, education) and pre-treatment severity (LSAS-performance) variables were added to the models as potential predictors, as well as therapist (dummy coded for the 3 therapists) and number of confederates during exposures. In the hormone analyses, all variables from an endocrine questionnaire (assessing the influence of factors like food intake, exercise, and sleep on hormone levels) were additionally added to the model as potential predictors. Given that no study outcomes changed as a function of their inclusion/exclusion, all non-significant control variables were dropped from the models prior to reporting.

For each of the analyses above we also calculated Bayes Factors (BFs), which have been recommended as a metric for quantifying the support of one hypothesis over another (e.g. Dienes, 2014; Wagenmakers, 2007; Wagenmakers, Morey, & Lee, 2016). BFs are particularly useful when traditional approaches yield a non-significant p-

value, which may be indicative of either inconclusive findings (i.e., alternative and null hypotheses are equally supported) or evidence in favor of the null hypothesis (e.g., treatments are equivalent on the outcome measure). For example, a BF of 10 suggests the alternative hypothesis is 10 times more probable than the null hypothesis, a BF of 1 suggests neither hypothesis is more probable (i.e., hypotheses are equally supported by the data), and a BF of 0.10 (i.e., 1/10) suggests the null hypothesis is 10 times more probable than the alternative—strong evidence that treatments are equivalent. Though BFs can be interpreted as continuous indicators, some conventional cutoffs have been recommended: 1. strong (BF > 10) or moderate (3 < BF < 10) evidence in favor of the alternative hypothesis; 2. inconclusive results (0.33 ≤ BF ≤ 3); or 3. strong (0 < BF < 0.10) or moderate (0.11 < BF < 0.32) evidence in favor of the null (Lee & Wagenmakers, 2014). For ease of interpretation, these guidelines (i.e., *strongly/moderately favors alternative, inconclusive, strongly/moderately favors null*) are reported in italics after BFs in the results section. In the current analyses, the strength of the evidence in favor of (or against) a treatment effect was evaluated by comparing the full model to the model without the treatment variable (including nuisance covariates when applicable). All ANOVA and Bayesian analyses were conducted in JASP (Version 0.8.1.1; JASP Team, 2017). Bayesian analyses used JAPS's default Cauchy priors (r scale): fixed effects = 0.5, random effects = 1, and covariates = 0.354 (Rouder, Morey, Speckman, & Province, 2012).

3. Results

The 73 individuals included in the analyses were on average 26 years old ($SD = 7.48$), female (71.2%), white (53.4%), and college-educated (49.3%). The average baseline LSAS (performance subscale) score was 37.27 ($SD = 11.96$). From treatment session to 1-week follow-up there were significant reductions in both pre-post LSAS-performance scores (mean change = −4.41; $SD = 8.04$) and peak SUDS (mean change = −21.42; $SD = 18.53$). There were no significant differences in these baseline characteristics between groups (see Table 1). In hormone analyses, six testosterone data points and seven cortisol data points were identified as outliers (i.e., standardized residuals were > 3 standard deviations outside the mean for the condition). Analyses were performed with and without these data points and primary outcomes were unaffected by their exclusion, therefore, the testosterone and cortisol analyses presented include all data. Of the 73 participants enrolled in the study, only 69 completed the post-treatment LSAS, and only 66 participants completed the post-treatment BAT. The consort diagram is presented in Fig. 1.

3.1. Aims 1–3: does power posing result in testosterone increases that facilitate SAD symptom reduction?

3.1.1. Does power posing (prior to the session) lead to higher testosterone levels 20 min later?

Covarying for pre-manipulation testosterone levels, $F(1,69) = 293.21$, $p < 0.001$, partial $\eta^2 = 0.81$, there were no significant differences in post-manipulation testosterone levels by condition, $F(2,69) = 0.62$, $p = 0.54$, partial $\eta^2 = 0.003$, $BF = 0.20$ *(moderately favors null)*. Table 2 provides the post-manipulation testosterone and cortisol means by condition, adjusted for pre-manipulation hormone levels.

3.1.2. Does power posing facilitate the effects of exposure therapy on social anxiety symptom reduction?

Covarying for pre-treatment LSAS-performance scores, $F(1,65) = 100.18$, $p < 0.001$, partial $\eta^2 = 0.60$, there were no significant differences in (1 week) post-treatment LSAS-performance scores by condition, $F(2,65) = 0.39$, $p = 0.68$, partial $\eta^2 = 0.01$, $BF = 0.17$ *(moderately favors null)*. Similarly, controlling for sex, $F(1,62) = 7.86$,

Table 2
Estimated Marginal Means of Outcome Measures.

	Testosterone[a]		Cortisol[b]		LSAS-performance[c]		Peak SUDS (exposure)[d]		Peak SUDS (BAT)[d]	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Power Pose (n = 26)	50.24	6.97	4.27	0.46	33.54	1.65	74.14	2.66	56.40	4.04
Submissive Pose (n = 27)	61.12	6.95	3.71	0.44	33.33	1.55	75.86	2.59	56.32	4.04
Rest (n = 20)	57.40	8.01	3.97	0.53	31.60	1.76	74.12	3.11	44.37	4.47

[a] Pre-manipulation testosterone covariate evaluated at 57.77.
[b] Pre-manipulation cortisol covariate evaluated at 4.09.
[c] Adjusted for pre-treatment LSAS-performance score covariate evaluated at 37.30.
[d] Controlling for sex.

$p = 0.01$, partial $\eta2 = 0.10$, there were no significant differences in peak SUDS at the post-treatment BAT by condition, $F(2,62) = 2.82$, $p = 0.07$, partial $\eta2 = 0.08$, BF = 0.95 *(inconclusive)*. Estimated marginal means of outcome measures are presented in Table 2.

3.1.3. Do testosterone changes from pre- to post-manipulation predict social anxiety symptom reduction?

Controlling for pre-treatment LSAS, $F(1,65) = 106.61$, $p < 0.001$, partial $\eta2 = 0.60$, neither testosterone change, $F(1,65) = 1.72$, $p = 0.19$, partial $\eta2 = 0.01$, BF = 0.15 *(moderately favors null)*, nor cortisol change, $F(1,65) = 3.78$, $p = 0.06$, partial $\eta2 = 0.02$, BF = 0.39 *(inconclusive)*, were predictive of post-treatment LSAS scores. Controlling for pre-treatment LSAS, $F(1,62) = 4.76$, $p = 0.03$, partial $\eta2 = 0.07$, neither testosterone change, $F(1,62) < 0.001$, $p = 0.98$, partial $\eta2 < 0.001$, BF = 0.38 *(inconclusive)*, nor cortisol change, $F(1,55) = 2.33$, $p = 0.13$, partial $\eta2 = 0.03$, BF = 1.01 *(inconclusive)*, were predictive of peak SUDS at BAT.

3.2. Aims 4 & 5: does power posing affect fear during the session?

3.2.1. Does power posing decrease cortisol levels 20 min later?

Covarying for pre-manipulation cortisol levels, $F(1,69) = 81.03$, $p < 0.001$, partial $\eta2 = 0.54$, there were no significant between-condition differences in post-manipulation cortisol levels, $F(2,69) = 0.34$, $p = 0.68$, partial $\eta2 = 0.01$, BF = 0.16 *(moderately favors null)*.

3.2.2. Does power posing decrease peak SUDS within the exposure session?

Controlling for sex, $F(1,69) = 10.85$, $p = 0.002$, partial $\eta2 = 0.14$, there were no significant differences between conditions in fear activation (peak SUDS[1]), $F(2,69) = 0.15$, $p = 0.86$, partial $\eta2 = 0.004$, BF = 0.14 *(moderately favors null)*. Estimated marginal means are presented in Table 2. Moreover, the average peak SUDS during the treatment session exposure session was 77.16 ($SD = 13.85$) and the average peak SUDS at BAT was 56.05 ($SD = 20.03$). A repeated measures ANOVA of phase, condition, and the phase by condition interaction showed that, whereas the overall change in SUDS was significant and with a large effect size across the complete sample, $F(1,63) = 94.11$, $p < 0.001$, partial $\eta2 = 0.58$, BF > 100 *(strongly favors alternative)*, there was no significant effect of condition, $F(2,63) = 0.90$, $p = 0.41$, partial $\eta2 = 0.03$, BF = 0.28 *(moderately favors null)*, or a phase by condition interaction, $F(2,63) = 2.34$, $p = 0.11$, partial $\eta2 = 0.02$, BF = 0.18 *(moderately favors null)*. This overall change in SUDS represents an average decrease of approximately 27% from pre- to post-treatment (see Fig. 2 for SUDS by condition).

4. Discussion

We conducted a randomized controlled study to compare the effects

of power posing, submissive posing, and rest (no posing) prior to a public speaking exposure for individuals with SAD ($N = 73$). This study was based on theory and preliminary evidence that power posing may increase testosterone levels, which may enhance the efficacy of exposure therapy for SAD by increasing approach toward perceived-threatening social stimuli. Across all hypotheses, ANCOVAs yielded non-significant results. Bayesian analyses can be helpful in removing ambiguity when traditional analyses yield non-significant p-values (i.e., do non-significant results indicate inconclusive findings, or evidence in favor of the null hypothesis?). These analyses suggested our data provided moderate evidence in favor of the null hypothesis that power posing *does not* result in increased testosterone levels, decreased cortisol levels, decreased subjective levels of fear during exposure, or superior reductions in SAD symptom severity. However, our data were inconclusive in assessing whether power posing affects changes in peak SUDS one week post-exposure, with neither the null nor the alternative hypothesis receiving greater support. Finally, analyses assessed whether testosterone or cortisol changes prior to exposure were predictive of exposure outcomes regardless of condition. Our data were inconclusive, except for providing moderate evidence that testosterone change was not related to changes in SAD symptom severity.

Possible explanations for null findings are related to the sample size, population, and study design. Though our study was powered to detect medium to large effect sizes (as found in the Carney study), it may have been underpowered to detect small effect sizes. Additionally, to our knowledge, our study is the first to apply power posing toward a clinical population (i.e., individuals diagnosed with SAD). It is possible that any hormonal and/or psychological effects of power posing may not have been evident due to the stressful nature of the public speaking exposure for this group of individuals, which may have led to hormonal changes which obscured any effects of our study manipulation. Though we designed the study based on the Carney et al. (2010) study, several features of the study differed from this trial. Individuals in our study were likely highly stressed during the power posing procedures, given that they were aware they would be participating in an upcoming public speaking exposure. We also did not utilize a filler task during the pose (whereas the Carney study had individuals in the study complete a social filler task). Additionally, the Carney study was conducted in 2008–2009, whereas our study was conducted after extensive media and University course coverage of power posing. Though we attempted to control for whether participants in the study were blind to the purpose of the study manipulation by questioning them about the deception at follow-up, it is possible that they were aware of the literature on power posing and of the true purpose of the study. Finally, it is possible that the timing/dose of power posing was not appropriate to yield hormonal changes or enhance exposure outcomes in this study, but that there could be effects of power posing that would be evident with differential hormone sample assessment times (i.e., shorter or longer time between samples) and/or a larger "dose" of power posing (e.g., longer holding of poses, a shorter period of time between power posing and exposures, or power posing within the exposure session rather than prior to it).
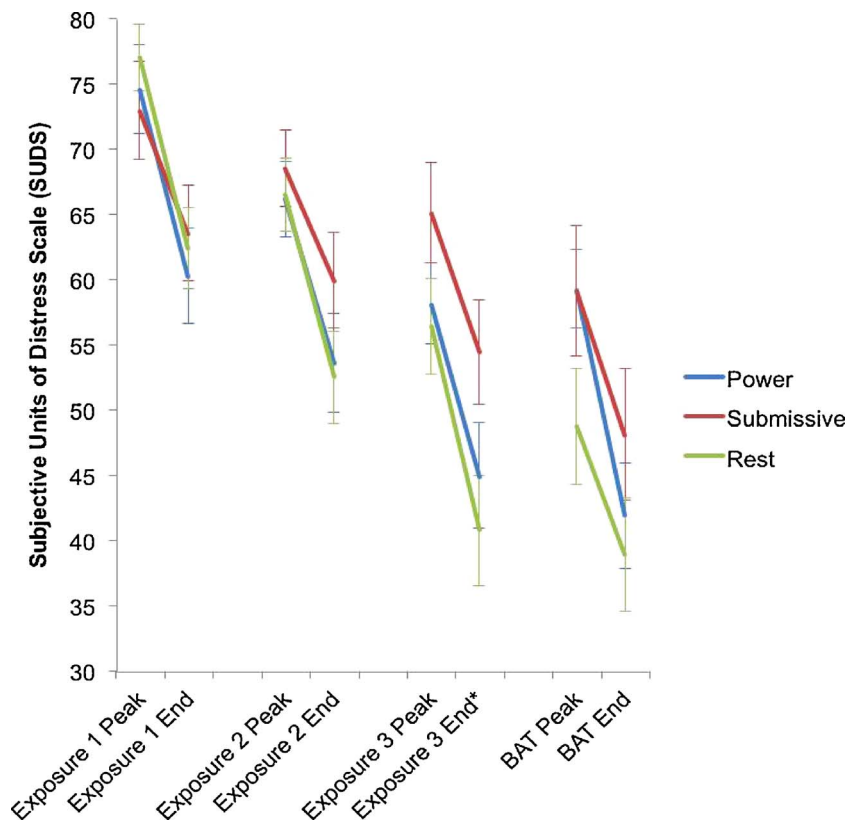
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[1] In post-hoc analyses, overall outcomes did not change when using the average peak SUDS across the three public speaking exposures (rather than the peak rating across the three).

Fig. 2. SUDS within speech exposures across time points by condition. *Notes*: Error bars are standard errors of the means. * = Significant difference in exposure 3 end SUDS between the submissive and rest conditions, $t(41) = 10.97$, $p < 0.001$. No other significant differences in means at $p \leq 0.05$.

Assuming that the above noted differences in study methodology do not prevent a comparable replication of the Carney study, our findings, along with those of the Ranehill et al. (2015) study, offer no evidence to suggest that power posing impacts hormone levels. Replication is important, however, particularly when sample sizes are relatively small, and null findings contribute to the body of literature regarding the extent of the effects (if any) on power posing. As a testament to this, a recent analysis reviewed the 33 studies identified in the Carney et al. (2015) review of power posing, and accounted for selective publishing (i.e., the failure to publish null findings, or the file-drawer effect; Rosenthal, 1979) using a *p*-curve analysis (Simmons & Simonsohn, 2017). They found that the distribution of *p*-values from the 33 published studies on power posing was indistinguishable from the expected *p*-values if the average effect size were zero, and/or if selective reporting was the sole reason for the significant effects. Thus, power posing does not appear to be a robust method of facilitating change, whether psychological, behavioral, or hormonal. Our study provides further evidence toward this end.

Finally, our hypothesis that increased testosterone during exposure therapy might lead to facilitated exposure therapy outcomes among individuals with SAD remains untested, as we were unable to successfully increase testosterone levels prior to the public speaking exposure. As highlighted by Enter et al. (2014), Enter, Spinhoven et al. (2016) and Enter, Terburg et al. (2016), this remains an important area for future research to address, perhaps by applying low doses of exogenous testosterone prior to exposure to ensure that hormone levels are sufficiently increased. Given that exposure is an effective treatment for many with SAD, it may be useful to selectively apply augmentation strategies (such as testosterone administration) to those who have previously not responded to exposure therapy (Mataix-Cols et al., 2017). Additionally, though our study yielded null findings, the single session assay utilized here (derived from Rodebaugh et al., 2013) offers a timesaving, inexpensive way to test augmentative psychopharmacological strategies, such as testosterone administration. As noted in the Results, we elicited significant variability (i.e., no floor effects) and

meaningful reductions in both pre-post LSAS-performance scores and peak SUDS elevations from treatment to BAT with only a single treatment session and 1-week follow-up. Utilizing this brief assay as a preliminary proof-of-principle test may be an important step prior to assessing effects on a full dose of exposure therapy. Indeed, assessing the null effects of power posing with this clinical assay, combined with other research regarding the effects of power posing, allows us to assume that power posing is likely not a useful strategy for enhancing exposure therapy outcomes for SAD.

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