The effect of progressive muscle relaxation on daily cortisol secretion

Chellew, K., Evans, P., Fornes-Vives, J., Pérez, G. and Garcia-Banda, G.

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The effect of Progressive Muscle Relaxation on Daily Cortisol Secretion

Abbreviated Progressive Muscle Relaxation (APMR) is a shortened version of Jacobson's (1939) original technique, designed to induce feelings of deep relaxation by systematically tensing and relaxing 16 muscle groups and by learning to focus on and discriminate between the resulting sensations of tension and relaxation (Bernstein & Borkovec 1973). There is empirical evidence of APMR's efficacy in reducing negative states of anxiety and perceived stress (Emery et al. 2008; Rausch, Gramling & Averbach 2006), and increasing positive feeling of relaxation (Pawlow & Jones 2002) as well as producing clinically significant improvement of tension headache in a randomized placebo controlled clinical trial (Blanchard et al. 1990).

The question addressed here is not therefore about its efficacy relative to non-intervention in reducing *psychological* stress. Rather, we ask whether a fully expected reduction in psychological stress will be matched by equivalent *physiological* stress-reduction. In assessing APMR's capacity to manage stress defined as a more holistic psychosomatic construct, this study has the potential to make an important contribution to research in this area.

The end product of the Hypothalamic-Pituitary-Adrenal (HPA) axis, cortisol is a useful but complex biomarker, owing to the need to consider its dynamic diurnal cycle. Cortisol typically rises dramatically to very high levels in the first hour post-awakening, the so-called "Cortisol Awakening Response" (CAR), decreasing thereafter (Clow et al. 2004). There is growing evidence that the dynamic changes in this brief CAR period may particularly reflect cognitive functioning to meet daily demands and challenges (Evans et al. 2012; Evans et al. 2011; Fries et al. 2009; Wetherell, Lovell & Smith 2014). However, the CAR period is extremely volatile and presents formidable challenges in terms of its reliable measurement due to the need to ensure highly accurate timing of the saliva samples from which cortisol concentrations are derived. Very large changes in cortisol values take place over very small

time intervals, which need in turn to be expressed relative to individual awakening times rather than clock-time (Smyth et al. 2013a). In regard to using cortisol as a biomarker of chronic stress, use of sample values from the brief volatile CAR period following awakening may actually be best avoided (Smyth et al. 2013b; Garcia-Banda et al. 2014). This is likely to be good practice since traditionally chronically higher levels of stress over time have tended with reasonable consistency to be linked to higher average levels of *total* daily cortisol secretion, which is clearly determined overwhelmingly by secretion rate outside the very brief CAR period (Smyth et al. 2013b).

Cortisol measures have been used before in stress intervention studies. Most typically, such studies (e.g. Dolbier & Rush 2012; Pawlow & Jones 2002; Pawlow & Jones 2005) have chosen to examine the acute effects of APMR on 'spot' cortisol levels assessed immediately before and after brief APMR sessions, sometimes even a single session. While such findings have generally been positive and supportive of APMR efficacy, there is a need to examine changes in cortisol measures chosen to provide stable and comparable estimates of average cortisol secretion prevailing over a meaningful period of time before and after intervention using multiple cortisol sampling over time. This would more clearly address the efficacy of APMR as a useful therapeutic intervention to reduce physiological stress. At least one study provides some supportive evidence of APMR efficacy in this respect (Krajewski et al. 2011) but drawing firm conclusions is very significantly limited by its total sample of only 7 individuals. Our study is designed to provide much firmer evidence of change which may endure beyond intervention, and where intervention itself extends beyond a single short session of APMR.

Accordingly, this study examines response to APMR in a sample of first-year university students, who were recruited as part of a wider program of research on stress and cortisol. Going to university is reportedly a stressful time for at least 50% of students (Regehr, Glancy

& Pitts 2013). Heavy academic demands and the need to forge novel social networks can for some be huge stressors (Dolbier & Rush 2012), creating worrying rates anxiety and depression. Therefore, apart from convenience, there are reasons that may make this a suitable population for this investigation. We hypothesized that one week of intensive APMR delivered by a trained professional would be effective in reducing both psychological and physiological stress. Using methodological techniques to ensure accurate timing of cortisol samples, we have sought in this study to provide best estimates of total cortisol secretion based on multiple sampling over two days, in weeks before and after APMR intervention. For all such estimates we have carefully excluded values within the brief post-awakening period of dramatic rise.

One strand of the wider program of research mentioned above involved exploration of detailed cortisol profiles in relation to personality and in particular Neuroticism as a possible stress vulnerability trait (Garcia-Banda et al. 2011; Garcia-Banda et al. 2014). While we had no firm hypothesis in this study in regard to the possible modulation of any intervention effects by Neuroticism, the availability of these data allowed us to include Neuroticism along with sex, age and smoking status in subsidiary analyses to demonstrate if necessary that any obtained stress reduction effects were independent of these individual difference variables.

Method

Participants

First year students from the University of Balearic Islands were recruited annually into this APMR intervention study over a period of four years from a larger sample of students who had already provided psychometric data for research purposes. Over the course of the four years, six groups attended a week's course of APMR training. Overall 101 student volunteers provided complete data, including saliva samples and attendance at a one-week course of APMR relaxation training. Sixty-six were female. Mean age was 21.18 years (SD = 5.141). For regression analysis purposes, age was best operationalized as a dichotomous variable with

a large younger group (n=67) aged 18-20 years, and a smaller tail (N=34) of students older than 20 (age range 21-42 years). In total, 63 were high on Neuroticism and 38 were low (see Table 1).

[Table 1 near here]

Intervention

APMR sessions were conducted by an expert and university trainer (third author) in this technique who remained blind to collected data until the completion of this study. The APMR was performed following strictly the standard procedures set forth by Bernstein and Borkovec (1973). APMR training consists of five 45 minute session of tensing and releasing 16 muscle groups (dominant and non-dominant hand and forearm, dominant and non-dominant biceps, forehead, upper cheeks and nose, lower cheeks and jaws, neck and throat, chest, shoulders and upper back, abdominal or stomach region, dominant and non-dominant thigh, calf and foot) designed to produce both cognitive and physiological relaxation. Instructions encouraged participants to focus on sensations associated with release of muscle tension and feelings of comfort. They were advised not to tense muscle groups that felt strained or that aggravated pain.

Measures

Neuroticism. At the time of initial recruitment into the wider program of research of which this study forms a part, the NEO-FFI (Costa & McCrae 1999) was used to evaluate neuroticism using 12-item subscale (score range = 0 to 48). Participants responded on a 5-point Likert scale from 0 (*totally disagree*) to 4 (*totally agree*). The internal consistency value for our sample was .83. Participants were pre-selected as being high (> 85th percentile) or low (< 15th percentile) on the NEO-FFI Neuroticism scale (Costa & McCrae 1999) and Neuroticism was thus analyzed as a dichotomous variable.

Survey of Recent Life Experiences (SRLE). The SRLE was developed by Kohn and McDonald (1992) and covers the following areas: mundane annoyances, domestic responsibilities, work, romance, friends, family, other social relationships, finances, environment, time pressure, competitive standing (in terms of abilities, attractiveness, etc.), and future security. Participants indicated the extent of their recent life experiences over the past month on the following 4-point scale: 1 = not at all part of my life; 2 = only slightly part of my life; 3 = distinctly part of my life; and 4 = very much part of my life. Total SRLE score is computed by adding all the values given (1 to 4) to each question (range: 41-164). The internal consistency value for our sample was .93 (original value .90; Khon & McDonald 1992).

Cortisol. Salivary cortisol measures were collected with a cotton swab chewed for one minute, stored in a capped plastic vial ("Salivette" Sarstdet Inc.). These samples were centrifuged at 3000g for 3 minutes, and then the filtrates were stored frozen at -80ºC until analysis. Before analysis, the samples were thawed, mixed, centrifuged and analyzed without pre-treatment. To reduce error, all samples of each participant were analyzed in one assay. Salivary cortisol was measured using a modification of the Bayer ADVIA Centaur cortisol assay, a competitive direct chemiluminescence's immunoassay that uses a rabbit polyclonal antibody. Endogenous cortisol contained in the samples competes with a cortisol labeled with acridinium-ester for the binding sites of the anti-cortisol rabbit polyclonal antibody-coated paramagnetic particles. The intra- and inter-assay coefficients of variation were less than 10% for 0.30 μg/dL of cortisol.

Adherence Electronic Monitoring. Timing cortisol adherence was measured by Medication Event Monitoring System (MEMS) Track Caps device (AARDEX, Ltd., Zug, Switzerland). Participants took an absorbent cotton swab at each assigned sampling time from a plastic bottle with a microchip lid that recorded the time of each opening. After collecting a

saliva sample, participants stored the swab in a pre-labeled plastic tube (Salivette, Sarstedt, Barcelona, Spain) and manually registered the time (e.g., 8:30 am) on the protocol-required form everytime they took a saliva sample. The registered time by hand was then compared with the time measured by the MEMS caps, to guarantee that the "subjective" information provided by participants was accurate. An AARDEX interface and software were used to transfer time collection from the MEMS to PC (Broderick et al. 2004). In addition, participants programmed their mobiles to beep at the established times in order to further enhance compliance. Discrepancy between MEMS and protocol-required timing of saliva samples could thus be used to check the sensitivity of any hypothesized effects to degree of timing errors.

Procedure

First year UIB students gave informed consent in their classes. Those who wanted to participate in the study provided their e-mail address and mobile phone number. In order to evaluate the effect of APMR on perceived stress participants completed the SRLE scale the week before and after the training. Cortisol secretion was assessed by collecting five measures of cortisol across the day (awakening, 45 min, 2.5h, 8h and 12h), on two days (Tuesday and Thursday), one-week before (pre) and one-week after (post) the intervention. To provide a degree of control for the possibility that any cortisol reduction over the course of weeks in the main study may have reflected simple habituation over time rather than the intervention, we included an additional baseline cortisol measure taken two-weeks prior to the pre-intervention in half of the six cohort groups. Additionally, participants used the MEMS Caps to register each time they took a saliva sample. Moreover, students filled the information protocol registering the exact time of each sample, including wake-time ("as soon as you open your eyes and before getting up"), eating times, caffeine intake, medication taken, or if they had siesta, or they did sport, etc. (see Adam & Kumari 2009). Participants were not given reimbursement

for their participation, although at the end of the study they received detailed information about their personality and cortisol profiles.

Treatment of Data and Statistical Analysis

Outcome measures were examined for normality of distribution. Extreme outlying scores (+/- three-standard deviation) were winsorized and in the case of cortisol were root transformed to reduce skewness statistics to approximately twice the standard error or less.

A Total Cortisol Secretion (TCS) measure was calculated as area under the curve (AUC) of cortisol measures collected at 0.75h, 2.5h, 8h, and 12h after awakening on Tuesday and Thursday for each time period (pre- and post-intervention), using the standard trapezoid formula (Pruessner et al. 2003).

Outcome effects were examined using mixed regression modeling (MRM), an approach deemed most appropriate for multilevel designs incorporating repeated measures over time with fixed and random parameters (Blackwell, Mendes de Leon & Miller 2006). This approach has been used in particular to model dynamic aspects of the diurnal cortisol cycle (Smyth et al. 2013a), and in this case enabled us to examine over two time periods (pre and post) the effect of APMR on SRLE and TCS. Similar two-level models were constructed for both dependent variables. In each case we assumed random intercepts and random slopes at the first level (Model A), which were modelled as outcomes at level 2 (Model B) when between-persons covariates were introduced. The goal in Model B was to determine which person-level characteristics might modulate differences at the within-person level.

Model A represents solely within-person effects and included the fixed covariates of pre- and post- intervention weeks, wake-time, and, for TCS only, sampling day within weeks (Tuesday vs. Thursday). The dichotomous variable of sampling day was effect coded such that zero represented cortisol secretion effects for other variables averaged across both sampling

days. For similar reasons and following convention (Blackwell et al. 2006), wake-time was participant-centred such that scores represented the purely within-participant effect of changes in wake-time across occasions, with each score computed as a person's raw wake-time minus their own mean wake-time over all four study days. Thus again a value of zero in the model equations would represent conditions in which wake-time was assumed to be average for each participant. Finally the key covariate of the intervention (pre vs. post) was dummy coded (0/1) such that 0 (and therefore the intercept in the model) represented SRLE or TCS pre-intervention.

In Model B, the following level 2 (between-persons) fixed covariates were included: sex, age category, smoking status, neuroticism group, and allocation (or not) to an additional baseline (pre-intervention) assessment of outcome measures. All these dichotomous variables were effect coded such that -1 represented the category values of female, younger age, non-smoker, low neuroticism, and absence of additional baseline assessment, and +1 represented binary opposites. A preliminary full Model B was run including all covariates to examine their statistically independent effects on baseline (intercept coefficient) levels of dependent variables, and all two-way interactions involving intervention (pre-post slope coefficient). The latter test within the model for possible modulation of within-person intervention effects by between-persons covariates. The final Model B presented here involved backward elimination of covariates with no significant effects on intercept or intervention slope coefficients.

Results

Full details of all analyses including coefficients for estimating all Model A and final Model B effects for both stress measures are given in Table 2.

Effect of APMR on SRLE

The results of Model A indicate that the intercept (denoting SRLE baseline) before APMR training was 72.03. The slope coefficient (-6.90) for the intervention effect was statistically significant (F = 39.88; df = 1, 89.71; $p < .001$) and is an estimate of the reduction in SRLE measured stress in the week following the intervention, with wake-time held at its zero (mean) value. Wake-time was not associated with overall SRLE scores nor with changes in SRLE post-intervention. The final model B yielded similar intercept (69.21) and intervention slope (-6.84) values, the latter remaining highly significant (F = 39.98; df = 1, 89.76; $p < .001$) and, expressed in percentage terms, the intervention was followed by an approximately 10% reduction in SRLE measured stress, equivalent to a Cohen 'd' effect size of 0.38 (see Figure 1). Model B yielded a significant (F = 47.44; df = 1, 92.60; $p < .001$) main effect coefficient of 10.74, for neuroticism. This coefficient estimates that high neuroticism participants tended to report 10.74 (approximately 16%) more SRLE stress units than the study average, and low neuroticism participants equivalently less. The Neuroticism x Intervention interaction was not significant, so there was no suggestion that Neuroticism modulated the main finding of a reduction in SRLE stress following the intervention. Equally there were no other significant main effects on overall level of SRLE stress reporting and no evidence of modulation of the significant intervention effect, with all other terms being excluded from the final model in the process of process of backward elimination (see Table 2).

[Table 2 near here]

Effect of APMR on Cortisol Secretion

The results of Model A using MRM analysis indicate that average total cortisol secretion (TCS) before APMR training (intercept) was 1.055 root units (equivalent to 5.48 μg/dl). The estimate of slope coefficient for intervention was -.003 root units (-0.45 μg/dl), (F = 6.49; df = 1, 123.45; $p < .012$). In this case slope was equal to differences between pre and post intervention means, with sampling day and wake-time held at zero (mean) values. Thus

APMR was followed by a significant decrease in cortisol secretion a week after the training. Later wake-time was significantly associated (F = 13.91; df = 1, 222.12; $p < .001$) with lower cortisol regardless of time point (pre- or post-intervention) with a significantly negative slope coefficient of -.004 in the equation based on root units. This would translate into a reduction of 0.56 μg/dl for every hour that participants might wake up later than their own typical average time. The intervention*wake-time interaction was not significant indicating that the efficacy of the intervention in reducing cortisol was not associated with any pre-post changes in wake-time. No significant effects were evident involving day of week.

Model B yielded closely similar estimates of intercept and intervention slope to Model A indicating a similar degree of significant (F = 6.94; df = 1, 133.78; $p < .009$) cortisol reduction of approximately 8% following the intervention (see Figure 1), and equivalent to a Cohen 'd' effect size of 0.30. More detailed descriptive examination of total AUC cortisol secretion over the course of the day (excluding secretion before 4 min post-awakening) showed that pre-post diminutions in mean secretion rates were consistent across the three post-45 min sampling periods (i.e., 45 min – 2.5h, 2.5 – 8h, and 8h – 12h). Consecutive mean secretion rates (ugs/dl/h) were 1.05, 0.49, and 0.29 in the pre-condition, and 0.98, 0.43, 0.27 in the post-condition. In both conditions, the same normal diurnal decline in secretion is apparent. None of the level 2 covariates interacted significantly with the intervention covariate, suggesting that the degree of cortisol reduction did not depend on gender, age, neuroticism, smoking status or whether participants' baseline cortisol was assessed once or twice before the intervention. Regardless of pre or post occasions, males had overall higher levels of cortisol than females (F = 4.93; df = 1, 92.07; $p < .029$), younger students (<21 years) had higher levels of cortisol than older ones (F = 9.14; df = 1, 91.44; $p < .003$), and participants whose baseline cortisol was assessed twice before the APMR intervention had significantly lower levels of cortisol than

participants who had a single week pre-intervention baseline ($F = 44.18$; $df = 1, 91.32$; $p < .001$).

[Figure 1 near here]

Discussion

These results confirm findings from controlled studies that Abbreviated Progressive Muscle Relaxation (APMR) can significantly reduce cortisol secretion, suggesting that this could be representative of reduced physiological responding to stress ~~psychological stress~~ (Emery et al. 2008; Rausch, Gramling & Averbach 2006). The SRLE was used as a hassles measure (Kohn & McDonald 1992) suitable for determining accurately how much stress participants have experienced over a short period of time, in this case, one-week.

However the principle aim of this study was to test the hypothesis that improvement in the self-report based outcome would be matched by improvement in a stress-relevant biomarker, viz. cortisol. The cortisol measure (TCS) was carefully chosen and constructed to ensure its fitness for purpose. Careful attention was paid to timing accuracy of saliva sampling, exclusion of samples in the immediate post-awakening period, and adequate multiple sampling during the course of each of four proximal days, two days in each period. In such circumstances, construction of a TCS measure of the kind employed in this study can provide a stable estimation of a participant's prevailing cortisol level over at least the period around the sampling days. Particular emphasis is drawn to the fact that in averaging over more than one day, the TCS measure used in this study attenuates the influence of within-day acute cortisol responses to situational demands and excludes entirely the influence of within-participant fluctuations in the most volatile period of the diurnal cycle which follows awakening.

Using a robust physiological outcome measure based on accurately timed cortisol sampling over multiple days, we were able to confirm our hypothesis that a one-week course of APMR decreased significantly students' ~~levels of~~ cortisol ~~physiological stress~~ one-week after the intervention. In terms of control features of the design, approximately half the participants provided stress outcome measures in an additional baseline trial one week before the pre-intervention week common to all participants. If the cortisol reduction between pre- and post- intervention sampling found for all participants were due to a simple temporal effect reflecting perhaps "habituation" of a temporary cortisol reactivity to the novelty and challenge of the saliva collection protocol itself, then the efficacy of the intervention, assessed in terms of cortisol, might appear to be significantly less in the sub-group whose post-intervention represented their third, as opposed to second, exposure to the saliva collection protocol. In essence, if simple repetition of the cortisol sampling protocol rather than intervention were the only factor promoting cortisol reduction over time, then greater cortisol reduction should be apparent following intervention in those participants exposed to only one pre-intervention base-line assessment. This would be manifested by a significant interaction between presence of additional base-line and pre versus post intervention period. Results showed no significant influence of an additional baseline exposure to saliva collection on the efficacy of the intervention. The cohort groups which were selected for additional baseline cortisol assessment did have significantly lower cortisol overall (i.e., both pre and post intervention). However, simple absolute differences between cohorts, where the cohorts have been recruited and the cortisol assayed over a significant course of time, as in this study design, are not unexpected. As is the case when differing average values pertain for equivalent studies from different laboratories, such differences in absolute cortisol values cannot readily be interpreted.

Another variable which was examined and statistically controlled in this study was wake-time. Later wake-time was significantly associated with lower cortisol secretion. People who

woke up later had lower cortisol secretion compared with people who woke up earlier. Wake-time effects on cortisol secretion levels have been reported before in the psychophysiology literature (Edwards et al. 2001; Kudielka & Kirschbaum 2003; Okun et al. 2010). However the crucial finding, in terms of the focus of this paper, was that the intervention effect was independent of wake-time. In other words there was no evidence that the efficacy of the APMR in reducing TCS was mediated by any changes in wake-time between pre- and post-intervention. Equally, no modulating role was found for neuroticism and APMR was equally effective in reducing stress measures in both high and low neuroticism groups. Sex, age, and smoking status were not related to psychological stress. However, sex and age effects were apparent for cortisol measures. Male and younger people presented higher levels of TCS, which is in line with the results found by Seeman et al. (2001). However none of these variables in any way modulated the pattern of cortisol ~~stress~~ reduction apparent over the trial period for all participants.

Despite its strengths, this study does have some limitations. Our principal aim was to demonstrate dual impact of APMR on both psychological and physiological stress. As reported in the introduction, against non-intervention control conditions, efficacy of APMR in relation to improvement in self-reported or observed symptomology has already been established in the existing literature. Nevertheless, we should issue a caveat that our study was not intended to be a randomized controlled trial of APMR efficacy and inappropriate conclusions should not be drawn in this regard. Of interest here was the examination of intervention effects in the student population and there must be caution in generalizing from a basically healthy and young adult population to more “distressed” populations across the fuller adult age range. Such populations may be more difficult to investigate with the same degree of experimental control but they may also be deemed more in need of therapeutic intervention. Longer follow-up would

also be desirable to demonstrate if enduring gains might indicate the extent to which a "life-skill" has been acquired from this relatively short and cost-effective intervention.

Conclusions

First, this study suggests that APMR is an effective intervention to decrease both perceived stress and cortisol secretion, particularly in university students, maintaining these changes a week after the training. Efficacy effects were independent of individual differences in wake-time, neuroticism, sex, age and smoking status. Thus, given the high rates of stress-related mental health problems reported by students (Regehr, Glancy & Pitts 2013), university health services may wish to consider the benefits of making this type of intervention more widely available as a means of combating student stress and possibly helping to lower the incidence of more serious resultant anxiety and depression (Bewick et al. 2010). Relaxation training might offer real benefits to students as they seek to cope with the challenges of their degree journey. Secondly this study suggests that cortisol as a bio-measure of stress could provide a useful and informative addition to self-report outcome measures in evaluating a range of interventions in the area of stress management. However ensuring appropriate, reliable and robust cortisol measurement does involve addressing some complex issues, which are treated in more detail elsewhere (Smyth et al. 2013b).

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The authors of this study declare that they have no conflict of interest.

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Table 1

Sample characteristics in relation to baseline cortisol assessment

	Groups			
	Additional baseline		Single baseline	
Characteristics	n	%	n	%
Neuroticism				
Low-N	19	41.3%	19	34.5%
High-N	27	58.7%	36	65.5%
Age				
Younger	26	56.5%	41	74.5%
Older	20	43.5%	14	25.5%
Gender				
Female	32	69.6%	34	61.8%
Male	14	30.4%	21	38.2%
Smoking status				
Non-smoker	32	69.6%	44	80%
Smoker	14	30.4%	11	20%
Total	46	45.5%	55	54.5%

Table 2

Effects of the intervention on the two outcome variables

	Model A			*Final Model B*		
SRLE	Coeff	(SE)	*p*<	Coeff	(SE)	*p*<
Fixed effects						
Intercept	72.03	(1.91)	.001	69.21	(1.60)	.001
Intervention	-6.90	(1.12)	.001	-6.84	(1.1)	.001
Wake-time	1.22	(3.39)	.719			
Intervention*Wake-time	-2.00	(6.16)	.746			
Neuroticism				10.74	(1.52)	.001
	Variance	(SD)	*p*<	Variance	(SD)	*p*<
Random effects						
Level 1 residual	58.86	(86.00)	.001	52.71	(180.10)	.003
Intercept	289.9	(460.8)	.001	183.7	(309.30)	.001
Linear slope				10.52	(325.50)	.747
TCS	Coeff	(SE)	*p*<	Coeff	(SE)	*p*<
Fixed effects						
Intercept	1.050	(.001)	.001	1.055	(.001)	.001
Intervention	-.003	(.001)	.012	-.003	(.001)	.009
Day	-.001	(.001)	.444			
Intervention *Day	-.001	(.001)	.532			
Wake-time	-.004	(.001)	.001	-.004	(.001)	.001
Intervention*Wake-time	-.000	(.002)	.855			
Additional baseline				-.007	(.001)	.001
Gender				.003	(.001)	.029
Age				-.003	(.001)	.003
	Variance	(SD)	*p*<	Variance	(SD)	*p*<
Random effects						
Level 1 residual	.000107	(.00011)	.001	.000107	(.00011)	.001
Intercept	.000138	(.00024)	.001	.000083	(.00017)	.001
Linear slope	.000001	(.00019)	.523	.000014	(.00017)	.432

Note. SE = standard error; SD = standard deviation; TCS = total cortisol secretion.

Figure 1. Effect of abbreviated progressive muscle relaxation (APMR) intervention on psychological stress measured by the survey of recent life events (SRLE) and total cortisol secretion (TCS) following the final Model B.

* $p < .001$