



Just Keep Breathing: Predicting Biofeedback-Training Stress Reduction Through Proactive and Reactive Aggression

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Abstract

Proactive aggression is coercive, deliberate and related to low arousal. Reactive aggression is fear-induced, impulsive, responsive behaviour due to processing and encoding stimuli as more arousing than normal. With relaxation treatment components possessing large effect sizes in anger and aggression treatment, it is important whether the distinction between proactive and reactive aggression with corresponding distinctions in arousal during stressful situations changes the efficacy of relaxation training. Thus, this study ascertained using 22 students to what extent the degree of proactive and reactive aggression – measured with the Reactive Proactive Questionnaire – of an individual predict the efficacy of a 4-session biofeedback virtual reality training in reducing heightened arousal – measured by skin conductance, heartbeat per minute and the Physical Arousal Questionnaire – during a stressful event – being the Trier Social Stress Task – including interoceptive awareness as possible mediator. Results indicates those with higher reactive aggression are worse in reducing physiological arousal using a relaxation training, and there is a disparity with interoceptive awareness, self-regulation reducing psychological arousal, and body listening reducing physiological arousal. It shows those with reactive aggression either need more guidance to implement the relaxation, or other training-techniques in reducing arousal might have more effect, for which further research is required.

Keywords: Proactive, reactive, aggression, biofeedback, relaxation

Just Keep Breathing

Predicting Biofeedback-Training Stress Reduction Through Proactive and Reactive Aggression

Aggression is a disruptive emotion holding many definitions. In this study, it is defined as “behaviors intended to hurt, harm or injure another person” (Murray-Close, Ostrov, Nelson, Crick, & Coccaro, 2010, p. 393). While once deemed an essential survival aid (Fechtner, 2012), nowadays it correlates to poorer peer relationships, worse school motivation, fewer shared activities, use of violent strong-arm tactics, higher initiation of fighting and proneness to more serious delinquency (Raine et al., 2006). At its worst, it can develop into Oppositional Defiant Disorder, with a prevalence of 1-3% under adolescents, or Conduct Disorder, with a prevalence of 3-9%, of which 1-2% are aggressive types (Centre, 2005). What makes aggression difficult to counter, as according to the catharsis hypothesis, is that anger expression reduces anger arousal and aggressive engagement which, problematically, implies expressing aggression causes positive reinforcement, facilitating future aggressions (Tyson, 1998). Therefore, a high-efficacy treatment and prevention which is optimally engaging, interactive and adaptive for high adherence (Charles & Black, 2004; Granic, Lobel, & Engels, 2014; van Rooij, Lobel, Harris, Smit, & Granic, 2016), is imperative.

Aggression has two distinctive categories originating from social information processing model deviations. Reactive aggression links to the hostile attribution bias, ascertaining social cues as more threatening while interpreting and encoding them (Mayberry & Espelage, 2007). Proactive aggression, in turn, correlates to a higher confidence in the ability to aggress for goal obtainment (Mayberry & Espelage, 2007).

Proactive aggression, also called instrumental aggression (Tyson, 1998), is coercive and deliberate (Connor, Steingard, Anderson, & Melloni, 2003) with little autonomic arousal and

stimulation seeking tendencies (Raine et al., 2006). Derived from the Social Learning Theory (Bandura, 1978; Connor et al., 2003), proactive aggression is learned behaviour maintained by the environment and positive reinforcement (Mayberry & Espelage, 2007).

Reactive aggression is “a fear-induced, irritable, and hostile affect-laden defensive response to provocation and involves a lack of inhibitory functions, reduced self-control, and increased impulsivity” (Raine et al., 2006, p. 3). According to Berkowitz’ revised Dollard’s frustration-aggression hypothesis (Connor et al., 2003), reactive aggression is caused by aversive events, called thwartings, that evoke negative affect, eliciting frustrations, which produce aggressive inclinations (Berkowitz, 1989).

Despite relatively little attention paid to anger treatment effects (Glancy & Saini, 2005), a meta-research overview by Glancy and Saini (2005) on participants who received anger or aggression therapy, indicates treatments with a relaxation component possess the largest effect sizes for efficacy. These relaxation treatments, however, do not distinct between proactive and reactive aggression. This distinction could be important, because proactive aggression theoretically links with lack in emotional depth and low emotional and physiological arousal (Murray-Close et al., 2010; Wagner & Abaied, 2016). Reactive aggression, however, is linked to heightened sensitivity to possibly threatening stimuli – hypervigilance – next to worse self-regulation capabilities, particularly under environmental stress (Wagner & Abaied, 2016). Thus, heightened reactive aggression should cause poor coping and higher arousal in arousing situations, while heightened proactive aggression should produce a lower arousal increase during similar conditions.

Stress physiologically causes aggression due to heightened arousal through the HPA axis and hormonal activity (Bertsch, Böhnke, Kruk, Richter, & Naumann, 2011; Dickerson & Kemeny, 2004; Fechtner, 2012), causing a fight (aggression) or flight (anxiety) response (Fechtner, 2012). Hence, relaxation trainings are conducted to dampen arousal while stressful

situations occur and as consequence reduce anxiety (flight) and aggression (fight) responses. Therefore, theoretically, a relaxation training would be effective against reactive aggression, where stress is heightened and stress coping mechanisms are less effective compared to those who are less aggressive. For proactive aggression, however, where arousal is already lower during stress, not much improvement due to relaxation technique effects can be expected in arousing situations. Hence, this study is conducted to examine the possible existence of this distinction in treatment results between aggression groups. Significant findings improve aggression treatment efficacy by improving the sensitivity of the training to a more focused target group.

A recent relaxation technique more commonly used for anxiety treatment, is biofeedback (van Rooij et al., 2016). Biofeedback is when unperceivable physiological processes are translated to perceivable feedback signals the participant can then try to alter through behavioural strategies (Lehrer, 2007; Tamminga, 2017; Tyson, 1998; van Rooij et al., 2016; Wagner & Abaied, 2016). Over time, alterations should endure even without translation (Schwartz & Andrasik, 2017). This research examines the distinction between proactive and reactive aggression on arousal levels before and after a relaxation-technique-based biofeedback training during a stress-elicitation task. More specifically, in this study is ascertained to what extent the degree of proactive and reactive aggression – measured with the Reactive Proactive Questionnaire (RPQ; Mackay, 2004) – of an individual predict the efficacy of a 4-session biofeedback virtual reality training (DEEP; Harris & Smit, 2014) in reducing heightened arousal – measured by skin conductance, heartbeat per minute and the Physical Arousal Questionnaire (PAQ; Kallen, 2002) – during a stressful event – being the Trier Social Stress Task (TSST; Kirschbaum, 1993). Higher reactive aggression should then predict lower arousal post-training, while proactive aggression should insignificantly predict arousal change.

The second subject of this study concerns possibly mediating effect of interoceptive awareness. Interoceptive awareness is “conscious perception of the body [which] influences all afferent information which arises within the body” (Craig, 2004; Domschke, Stevens, Pfleiderer, & Gerlach, 2010; Werner, Kerschreiter, Kindermann, & Duschek, 2013, p. 39). It rests on the neurally confirmed (Craig, 2004; Critchley, Wiens, Rotshtein, Öhman, & Dolan, 2004) somatic marker theory of Damasio, who theorised somatic (bioregulatory process) markers influence emotion, decision-making, attention, working memory formation and retrieval (Domschke et al., 2010), through distinct interoceptive pathways (Craig, 2004), both consciously and unconsciously (Bechara & Damasio, 2005). Interoceptive awareness could interact with biofeedback and aggression through various ways. Firstly, as biofeedback exists out of behaviourally responding to body signals, it could interact with training efficacy by inter-individually differing body perception accuracies. Secondly, higher interoceptive awareness could be part of higher general awareness, including hypervigilance – characteristic of reactive aggression but not proactive aggression.

While little to no research has been conducted on interoceptive awareness regarding aggression, several anxiety studies have found interesting results. Individuals with higher interoceptive awareness report emotions with higher intensity (Werner et al., 2013) and trait anxiety (Domschke et al., 2010; Spada, Nikčević, Moneta, & Wells, 2008), characteristic of reactive aggression. Werner and colleagues, however, found participants with high interoceptive sensitivity had, despite insignificant physiological arousal differences, less reported state and habitual public-speaking anxiety during public speaking (Werner, Duschek, Mattern, & Schandry, 2009). This indicates higher interoceptive awareness causes lower arousal during stressful situations. In this research is posited, with interoceptive awareness – measured with the Multidimensional Assessment of Interoceptive Awareness Questionnaire (MAIA; Mehling, 2012) – as mediator between the two aggression types and the aggression

reduction, that due to hypervigilance, reactive aggression predicts higher interoceptive awareness while proactive aggression does not. Furthermore, it is expected higher interoceptive awareness predicts higher arousal reduction.

Methods

Participants

676 Dutch Radboud University students ($M_{\text{age}} = 20.32$ years; $SD_{\text{age}} = 2.36$; range: 17-30; gender: 133 males, 544 females) recruited via SONA (Sona Systems Ltd., 2018) for half a participant point. Upon scoring moderate to high on the shortened version of the Depression Anxiety Stress Scales (anxiety ≥ 4 , stress ≥ 8) (Henry & Crawford, 2005), a small selection was invited to the lab sessions, being rewarded either 3.5 study points or 40 euros. The final lab sample, excluding incomplete physiological data and drop-outs – most due to VR-sickness (Fernandes & Feiner, 2016) and scheduling conflicts – included 22 Dutch Radboud University students ($M_{\text{age}} = 21.32$ years; $SD_{\text{age}} = 2.32$; range: 18 – 25; gender: 3 males, 19 females).

Procedure

For the experiment, participants filled in an online screening which included the DASS-21, RPQ and MAIA. Upon passing the screening, they were invited to the lab sessions 1 and 2 via e-mail, for the first week. During the lab sessions, participants filled in a consent form, and were thereafter attached to the Biopac 160M (Biopac Systems Inc., 2016a), by putting the ECG electrodes on their left ankle and both wrists, and putting the skin conductance electrodes with conductance gel on the volar surfaces of the distal phalanges of the non-dominant index and middle finger. During session 1, participants completed a baseline measurement of one minute, performed the TSST of two and a half minutes, filled in the PAQ regarding the stress task, and finally played the first session of DEEP for 10 minutes. Afterwards they were detached and session 3 and 4 were planned in for the week afterwards. Session 2 and 3 were similar to session 1, except no TSST took place, nor planning for further sessions. During session 4, the DEEP training was conducted prior to the stress task, which was done last. A schematic overview is given in figure 1, and instruments are further detailed below.

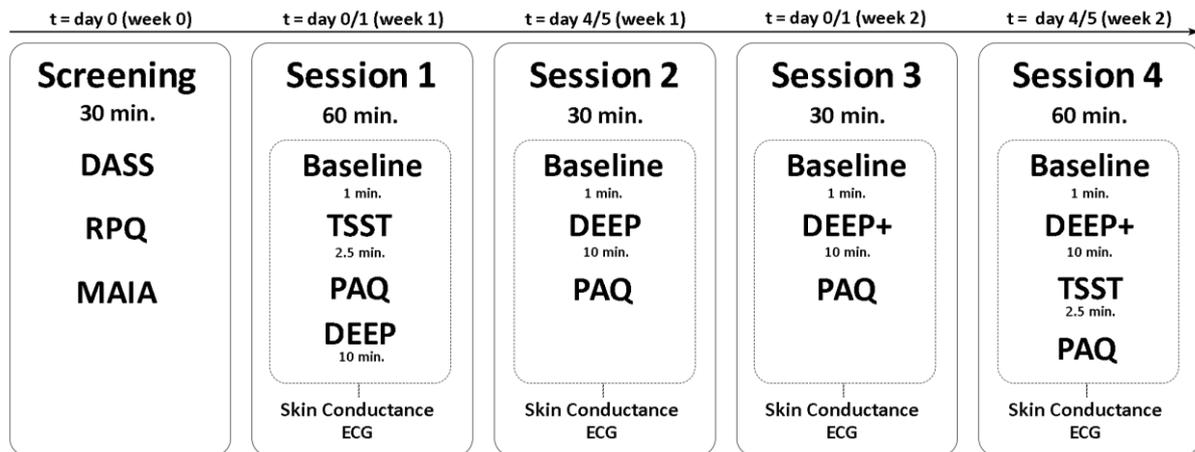


Figure 1. General Procedure.

Instruments

Depression Anxiety Stress Scales (DASS-21). The DASS-21 is a validated 21-item 4-point questionnaire used to measure depression (e.g. “Ik voelde me somber en zwaarmoedig”) anxiety (e.g. “Ik was angstig zonder enige reden”) and stress (e.g. “Ik vond het moeilijk me te ontspannen”) in the individual based on the previous week (Henry & Crawford, 2005). The questionnaire has sufficient internal consistencies according to literature ($\alpha_{\text{depression}} = .91$, $\alpha_{\text{Anxiety}} = .84$, $\alpha_{\text{stress}} = .90$)(Brown, Chorpita, Korotitsch, & Barlow, 1997). In this study, the alpha’s were similarly sufficient ($\alpha_{\text{depression}} = .88$, $\alpha_{\text{Anxiety}} = .81$, $\alpha_{\text{stress}} = .87$).

Reactive Proactive Aggression Questionnaire (RPQ). The RPQ is a validated instrument to measure the two factor-aggression model with, according to literature, an internal consistency in adolescents of .86 for proactive aggression, .84 in reactive aggression and .90 for total aggression (Raine et al., 2006). In this study, the Dutch validated version was used, which contains 23 items that can be answered with never, sometimes and often. 12 items measured proactive aggression (e.g. “Hoe vaak heb je gevochten om te laten zien wie de baas was?”) and 11 reactive aggression (e.g. “Hoe vaak heb je woede uitbarstingen gehad?”); (Cima, Raine, Meesters, & Popma, 2013). The internal consistencies in this study were .78 for reactive aggression, and .90 for proactive aggression.

Multidimensional Assessment of Interoceptive Awareness Questionnaire (MAIA).

The MAIA (Mehling et al., 2012) is a 32-item Likert-Scale questionnaire measuring different aspects of interoceptive awareness with different scales, their internal consistency according to their source and in this study outlined in table 1 below.

Table 1

MAIA Scales, Subscales, Items, Source and Study Internal Consistencies

Scale	Subscale(s)	Items	Literature α	Study α
Awareness of body sensations	Noticing	4	.69	.67
Emotional reaction and attentional response to sensations	Not-distracting	3	.69	.54
	Not-worrying	3	.69	.12
Capacity to regulate attention	Attention regulation	7	.87	.87
Awareness of the connection between body sensations and emotional awareness	Emotional awareness	5	.82	.83
	Self-regulation	4	.83	.80
	Body listening	3	.82	.79
Trusting body sensations	Trusting	3	.79	.89

The MAIA subscales used in this study, being self-regulation (e.g. “Wanneer ik mij overmand voel, kan ik een rustige plek in mezelf vinden.”) and body listening (e.g. “Ik luister naar signalen van mijn lichaam om meer over mijn emotionele toestand te vernemen.”) as they concern interoceptive awareness regarding to emotions without emotional awareness itself, both have alphas indicating sufficient internal consistency ($\alpha_{\text{self-regulation}} = .80$; $\alpha_{\text{body-listening}} = .79$). The MAIA has been able to detect significant changes in self-reported interoceptive awareness through training (Bornemann, Herbert, Mehling, & Singer, 2015).

PAQ The PAQ (Kallen, 2002) is a 7-item self-report questionnaire developed for assessment of the perceived state of physiological arousal. Each item consists of a 9-point scale (0–8) for indication of arousal the participant experiences (e.g. “Kun je je hart voelen kloppen?”). The scales have according to Kallen (2002) an internal consistency that ranges from .64 for baseline measurements to .81 for stress measurements. In this study, the internal consistency ranged from $\alpha = .65$ -.79 for baseline measurements to $\alpha = .81$ -.84 for stress

measurements. To get a self-report arousal reduction score, four PAQ measurements were derived from this study and averaged, after which the following equations were applied:

- (1) Session 1 PAQ difference = Session 1 PAQ post-TSST - Session 1 PAQ post-baseline
- (2) Session 4 PAQ difference = Session 4 PAQ post-TSST - Session 4 PAQ post-baseline
- (3) EDA Reduction = Session 1 PAQ difference - Session 4 PAQ difference

The end results is one PAQ Reduction score, signifying the degree of reduction in arousal increase due to stress compared to the participant his or her calm state of being, comparing two stressful events before and after the 4-session relaxation training.

Trier Social Stress Task (TSST). The TSST (Kirschbaum, Pirke, & Hellhammer, 1993) is a validated instrument used for eliciting social-evaluative stress in an individual (Kudielka, Hellhammer, Kirschbaum, Harmon-Jones, & Winkielman, 2007). As adaptation from the original, loosely following the research of Hollenstein and colleagues (2012), in this research the participants were first asked to stand and give – as soon as instructed – a two minute presentation on a subject of their own preference. They were allowed to switch from subject if needed. They were also told the speech will be recorded by the second researcher in another room, while the first researcher stayed in the room with the participant to check whether no errors occurred on the physiological recordings. The second researcher not in the room informed the participant when to start and stop via the speaker, and the participant was asked to ignore the first researcher that is in the room. Both the instructions and the speech itself were recorded physiologically and subsequent questionnaires regarding the task

pertained both stages. For the second TSST, which was unannounced, participants had to choose another topic.

DEEP. DEEP (Harris & Smit, 2014) is a game based on the combination of Virtual Reality Exposure Therapy (VRET) and biofeedback, like Intrepid (Repetto et al., 2009) and Mindlight (Wijnhoven, Creemers, Engels, & Granic, 2015), which are effective in treatment of anxiety-related disorders such as phobias (Botella, Fernández-Álvarez, Guillén, García-Palacios, & Baños, 2017), PTSD (Seitz, Poyrazli, Harrisson, Flickinger, & Turkson, 2014), and social anxiety (Anderson et al., 2013). In DEEP, Players move through an underwater environment quickest through optimal diaphragmatic breathing, which in a real-world setting reduces anxiety (Chen, Huang, Chien, & Cheng, 2016). Player diaphragm expansion, recorded by a stretch sensor, is indicated with a growing and shrinking circle (see figure 2a and 2b). Subpar breathing (below 50% of full capacity) makes players slow down and eventually sink to the sea-bottom. During DEEP+ sessions, DEEP changes after 2.5 minutes from peaceful to exposure environment for 5 minutes, additionally training biofeedback application for stressful situations whilst a stressful situation occurs (see figure 2c). A research conducted by van Rooij and colleagues (2016) showed 7-minute DEEP-players had significantly reduced state anxiety comparing pre- and post-session measurements ($p = .046$, 2016).



Figure 2. DEEP gameplay depiction with minimal diaphragmatic expansion (a), maximal diaphragmatic expansion (b) and during a DEEP+ session.

Skin Conductance (SCL). Skin conductance, or electrodermal activity (EDA), was measured using Biopac (Biopac Systems Inc., 2016a, 2016b), with a gain of $5\mu\text{S}/\text{V}$ (10HZ, DC, DC). Arousal of perceived acute stress or challenge (Wagner & Abaied, 2016) causes sympathetic activation (Dawson, Schell, & Filion, 2007) producing sweat gland activity (Dawson et al., 2007), heightening the conductivity of the skin. Heightened skin conductance reactivity, in turn, has been positively linked to (reactive) aggression and negatively with psychopathy, correlated to proactive aggression (Lorber, 2004; Raine et al., 2006). For this study, as per usual for chronic stimuli, skin conductance reactivity (SCL in μS)(Dawson et al., 2007) of participants was used as arousal indicator. Four measurement windows were gathered per participant, being EDA during the session 1 baseline, the session 1 stress task, the session 4 baseline and session 4 stress task, which were each averaged to derive one value per window. To calculate pre-to post training arousal reduction, equation 4-6, similar to equation 1-3, were used which are outlined below.

$$(4) \quad \text{Session 1 EDA difference} = \text{Session 1 EDA TSST} - \text{Session 1 EDA baseline}$$

$$(5) \quad \text{Session 4 EDA difference} = \text{Session 4 EDA TSST} - \text{Session 4 EDA baseline}$$

$$(6) \quad \text{EDA Reduction} = \text{Session 1 EDA difference} - \text{Session 4 EDA difference}$$

Thus for EDA reduction, a positive value signifies a lower increase of arousal has taken place between the session 4 baseline and stress task, than the increase between the session 1 baseline and stress-task.

ECG (BPM). Contrary to skin conductance, not only sympathetic but also parasympathetic activity influences heart rate (Dawson et al., 2007), which is linked with stress through sympathetic nervous system activation (Appelhans & Luecken, 2006; Wagner & Abaied, 2016). While breathing in air temporarily deactivates parasympathetic influences,

giving as consequence a heart rate increase, breathing out restores the influence, causing a heart rate decrease (Appelhans & Luecken, 2006). Therefore, learned controlled breathing, in which breathing out takes longer (7s) than breathing in (3s), should make for less heart rate increases in stressful situations, thus a lower heart rate variability compared to the (non-stressful) baseline of an individual. Low resting heartrate has been correlated with anti-social behaviours (Lorber, 2004; Ortiz & Raine, 2004) and (proactive) aggression (Lorber, 2004). High heart rate reactivity has been associated with (reactive) aggression. (Lorber, 2004). For this study, ECG has been measured using Biopac (Biopac Systems Inc., 2016a, 2016c), using a gain of 2000 (Norm, LP, 1.0HZ). Afterwards, four measurement windows were once again gathered per participant, being heart rate during the session 1 baseline, the session 1 stress task, the session 4 baseline and session 4 stress task. Due to much noise in the data, the ECG was put through a passband of 5-15Hz (Pan & Tompkins, 1985) with a Blackman -61dB window and subsequently centred using a digital FIR filter, after which the positive peaks were used to detect heart rate with a baseline window width of 100 milliseconds, noise rejection of 5%, and windowing units of 40 to 120 beats per minute. Afterwards, heartbeats below 40 were removed, due to the human heartbeat not going lower while awake in a resting state. Only then the values were averaged to create one mean for each window. To calculate pre-to post training arousal reduction, equations 7-9 were used, outlined below.

$$(7) \quad \text{Session 1 BPM difference} = \text{Session 1 BPM TSST} - \text{Session 1 BPM baseline}$$

$$(8) \quad \text{Session 4 BPM difference} = \text{Session 4 BPM TSST} - \text{Session 4 BPM baseline}$$

$$(9) \quad \text{BPM Reduction} = \text{Session 1 BPM difference} - \text{Session 4 BPM difference}$$

Thus for heart-rate reduction in beats per minute, a positive value signifies a lower arousal increase has taken place between the session 4 baseline and stress task, than the increase between the session 1 baseline and stress-task.

Analysis. All data for this study was prepared in different software for different goals. Physiological data was prepared in Biopac (2016a), while questionnaire key calculations and reliability analyses were conducted using SPSS (v25). Finally, the preliminary and main analyses, including assumptions, were conducted using R (R Core Team, 2018). Two preliminary analyses were conducted. The first entailed paired t-test analyses to inspect the instrumental validity of the adapted version of the TSST, regarding whether a significant change of arousal occurred between baseline and stress conditions. Secondly, a regression analysis was conducted for reactive and proactive aggression on the first session arousal difference to inspect whether the RPQ scales significantly predict arousal increase due to stress. The first main analysis concerns testing aggression predictability on arousal reduction, using regression models. Separate analyses for the different types of aggression (reactive, proactive) and arousal (EDA, BPM, PAQ) were chosen as it was not the goal to inspect shared variance, but to inspect separate predictabilities. The second main analysis regards mediation of interoceptive awareness, for which several structural equation models (SEMs) were constructed, which can be found in figure 3. The directionality is theoretically underpinned due to interoceptive awareness and aggression both being proven predictors of arousal (Tyson, 1998; Werner et al., 2009; Werner et al., 2013). Moreover, proactive and reactive aggression are both traits, while interoceptive awareness is situationally applicable. In these models, the covariance between proactive and reactive aggression was included as well, as both aggression types come from the same questionnaire and both measure distinctions of the construct aggression. With the inability to test model validity, explained later in the result section, the model with covariance included is both most theoretically and rationally valid.

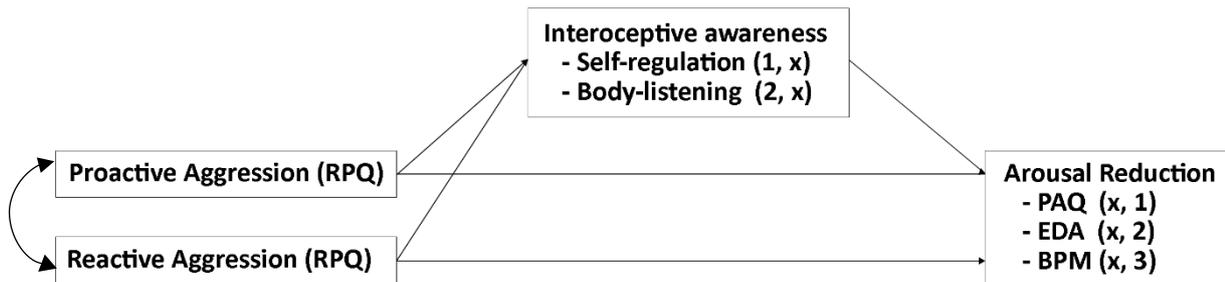


Figure 3. Mediation model (research question 2). The six created models are using: Self-regulation and PAQ (1,1), Self-regulation and EDA (1,2), Self-regulation and BPM (1,3), Body-listening and PAQ (2,1), Body-listening and EDA (2,2) and Body listening and BPM

For the analyses in R (R Core Team, 2018), several packages were used. After translating the data set into R using the package foreign (R Core Team, 2017), for descriptive data, the package Pastecs was used (Grosjean, Ibanez, & Etienne, 2018) with the package Lattice for box and density plots (Sarkar, 2008) and the package Car so inspect the homogeneity of groups (Fox, Friendly, & Weisberg, 2013). Furthermore, the package SemPlot (Epskamp & Stuber, 2017) was used to create the structural equation models, which uses the package Lavaan (Rosseel, 2012). All parameter estimates have been bootstrapped, to be better warranted against the low sample size and slight non-normality of the data. Graphical representations of the data included in the results was made by the package Ggplot 2 (Wickham, 2016), using Ggpubr for paired data-based boxplots (Kassambara, 2017).

Results

Before the preliminary analyses, several modifications to the variables were made. Upon assumption-based analyses, proactive and reactive scales were found to be positively skewed and kurtosed. Therefore, both were transformed using a mild correction, reactive aggression transformed with a square root, and proactive aggression being transformed with a natural logarithm (ln). As consequence, both scales acquired normal distribution. There were no outliers in the data more than 3 standard deviations from the mean. The unstandardized descriptive statistics of all variables for the main analyses are outlined in table 2. For the main analyses, all variables were standardized.

Table 2

Descriptive statistics of all used (unstandardized) variables for the main analyses.

Variables	<i>M (SE_{mean})</i>	<i>SD</i>	<i>Skew</i>	<i>Kurtosis</i>
Proactive aggression(transformed)	0.57 (0.16)	.74	0.79	-.78
Reactive aggression (transformed)	2.52 (0.16)	0.78	0.42	0.28
MAIA body listening	3.48 (0.15)	0.70	-0.31	-0.90
MAIA self-regulation	2.60 (0.21)	1.00	-0.02	-1.00
BPM reduction	3.25 (2.28)	10.71	-0.12	-0.025
PAQ reduction	3.55 (1.77)	8.29	.88	.011
EDA reduction	-0.70 (0.48)	2.24	.22	.39

Notes. $N = 22$.

Preliminary analyses. Paired t-tests for each session and each arousal measurement were conducted between the stress and baseline arousal means, having met the assumptions of no outliers – except for EDA_{stress} in session 4 ($SD_z = 3.13$), which was kept in due to having a small sample and little difference in results with the outlier taken out – and dependence of observations. For heartbeat per minute, with normally distributed data for session 1 ($W = .95$, $p = .35$) and 4 ($W = .95$, $p = .37$), there was a significant arousal increase between the baseline

($M= 77.13, SD= 13.49$) and stress ($M= 95.03, SD= 11.79$) condition means in session 1 ($t(21)= 8.98, p < .001$) and baseline ($M= 79.18, SD= 12.62$) and stress ($M= 93.83, SD= 12.89$) condition means in session 4 ($t(21)= 8.50, p < .001$). For skin conductance in micro Siemens, with non-normally distributed data for session 1 ($W= .87, p = .0082$) – therefore using a Wilcoxon signed rank t-test – but normally distributed data for session 4 ($W= .95, p = .28$), there was a significant arousal increase between the baseline ($M= 6.35, SD= 3.01$) and stress ($M= 8.41, SD= 3.55$) condition means in session 1 ($V(21)= 253, p < .001$) and baseline ($M= 5.32, SD= 3.62$) and stress ($M= 8.08, SD= 4.88$) condition means in session 4 ($t(21)= 6.68, p < .001$). For the 5-item PAQ scale, with normally distributed data for session 1 ($W= .92, p = .062$) and 4 ($W= 97, p = .62$), there was a significant arousal increase between the baseline ($M= 18.32, SD= 8.05$) and stress ($M= 26.45, SD= 11.13$) condition means in session 1 ($t(21)= 4.20, p < .01$) and baseline ($M= 10.18, SD= 7.22$) and stress ($M= 14.77, SD= 10.51$) condition means in session 4 ($t(21)= 2.57, p = .018$). All significant data is depicted in figure 4 below.

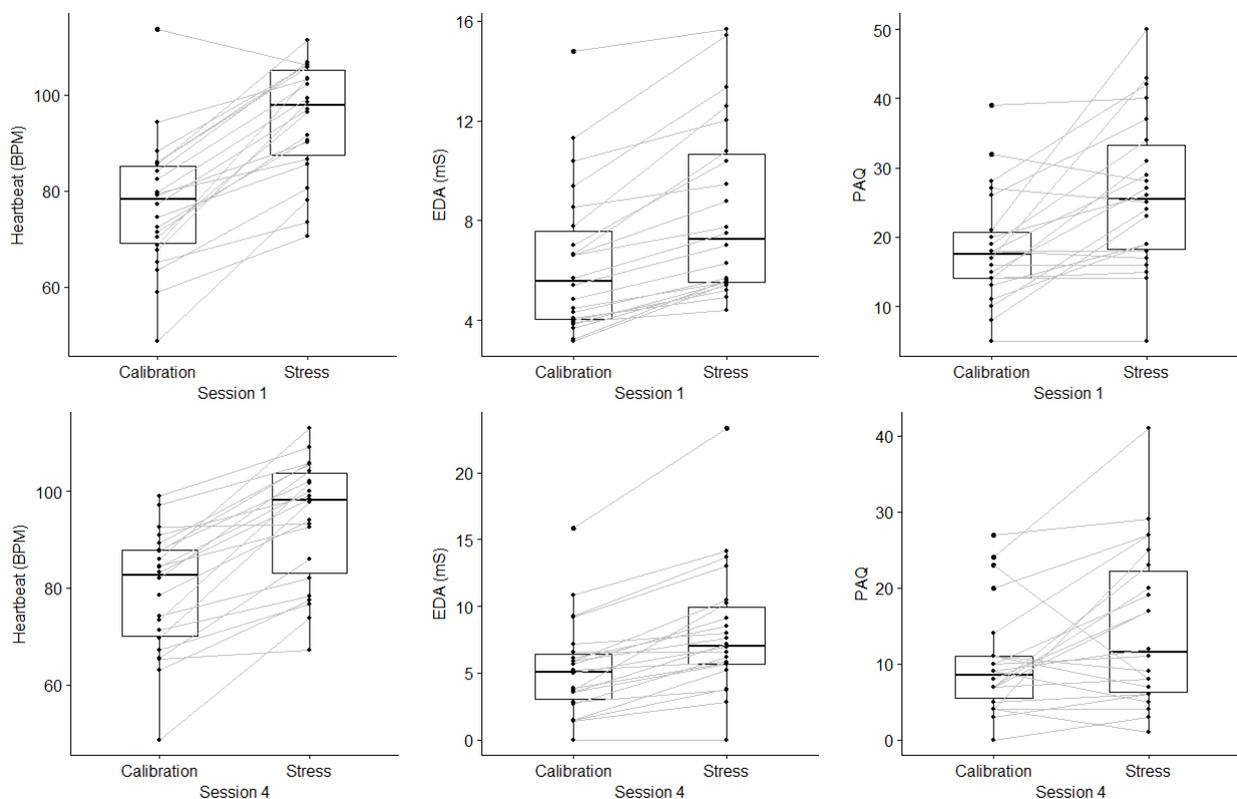


Figure 4. Significant paired t-test differences between calibrations and stress conditions in boxplots. Lines between the plots show data from the same participant.

To examine the predictability of proactive and reactive aggression on participant arousal increase during the stressful event compared to the respective baseline measurement in the first session, several separate regression analyses were conducted. In these regression models, either proactive or reactive aggression was used as independent variable predicting arousal difference (see equations 1, 4 and 7) of session 1, either by heartbeat (BPM), skin conductance (EDA) or through the Physiological Arousal questionnaire (PAQ). Assumptions of no outliers and normally distributed data – using the transformed standardized reactive and proactive scale variables – were considered sufficiently met. Regarding proactive aggression predicting heartbeat increase during stress, the data was sufficiently linear with weak heteroscedasticity. Both the regression model ($F(1, 20) = .148, p = .70$) with an R^2 of .007 and the regression coefficient ($b = 1.09, SE = 2.83, t(20) = .38, p = .71$) were insignificant. For reactive aggression predicting a heartbeat increase during stress, both linearity and homoscedasticity were deemed met. Both the regression model ($F(1, 20) = .344, p = .56$) with an R^2 of -.003, and the regression coefficient ($b = -1.56, SE = 2.66, t(20) = -.59, p = .56$), however, were once again insignificant. Regarding proactive aggression predicting an increase of electrodermal activity during stress, the assumptions linearity and homoscedasticity were met, but both the regression model ($F(1, 20) = .0000060, p = .99$) with an R^2 of -.05 and the regression coefficient ($b = .0029, SE = .38, t(20) = .008, p = .99$) held insignificant results. For reactive aggression predicting a skin conductance increase during stress, both linearity and homoscedasticity seemed met. The regression model ($F(1, 20) = .81, p = .38$) with an R^2 of .039 and the regression coefficient ($b = -18.08, SE = 20.09, t(20) = -.90, p = .38$) were again insignificant. Regarding proactive aggression predicting reported physiological arousal increase during stress, linearity and homoscedasticity both seemed not met, with a regression model ($F(1, 20) = .00016, p = .97$), R^2 of 7.92×10^{-5} and regression coefficient ($b = .11, SE = 2.76, t(20) = .040, p = .96$) that were insignificant. For reactive

aggression predicting reported physiological arousal increase during stress, both linearity seemed met and homoscedasticity seemed sufficient. The regression model ($F(1, 20) = 5.841$, $p = .025$) with an R^2 of .22 and the regression coefficient ($b = -5.54$, $SE = 2.30$, $t(20) = -2.42$, $p = .025$) were both significant, showing a mild negative relationship ($r = -.47$) outlined in the graph below. This means participants with higher reactive aggression had a smaller arousal increase from their respective baseline during the stress-task in the first session, as measured with the physiological arousal questionnaire. The significant standardized regression equation: $PAQ \text{ increase (S1)} = -.51\beta_{\text{reactive aggression}}$, is graphically represented in figure 5 below.

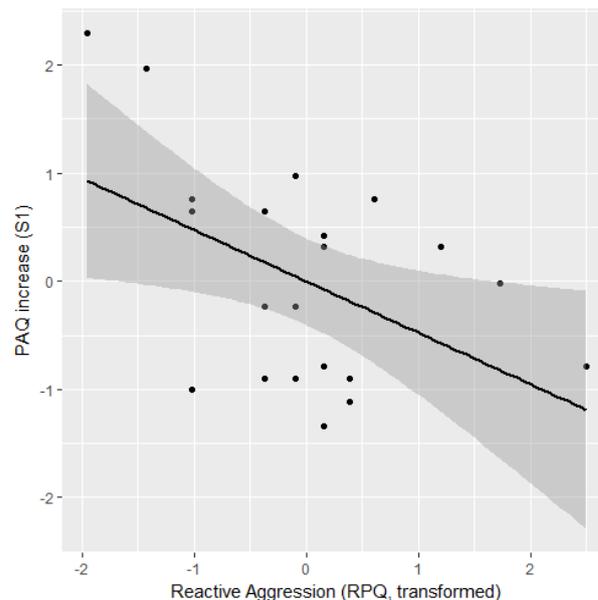


Figure 5. Linear graph showing the regression of psychological arousal increase of the participant from their respective baseline during the stress-task, predicted by the reactive aggression of the participant, using the standardized regression line.

Main regression analyses. With the main variables described in table 2 sufficiently meeting the requirements of no outliers and a normalized univariate distribution, the assumptions of homoscedasticity and linearity were inspected for each regression model, after which the analyses themselves were conducted. These entailed either proactive or reactive aggression being an independent variable predicting a reduction of arousal increase during a

stressful situation comparing pre- and post-training sessions, using the three arousal reduction means created with equation 1 through 9 as dependent variables. These were heartbeat per minute, skin conductance and reported physiological arousal.

For proactive aggression predicting BPM reduction, both assumptions of linearity and homoscedasticity seemed met. The regression model is insignificant ($F(1, 20) = .7, p = .41$), with an R^2 of .034, proactive aggression being an insignificant positive predictor ($\beta = .018, SE = 2.20, t(20) = -.84, p = .41$). Regarding reactive aggression predicting BPM reduction, the assumption of homoscedasticity seems met, but the assumption of linearity violated. The regression model is insignificant ($F(1, 20) = .13, p = .73$), with reactive aggression being an insignificant negative predictor ($\beta = -.079, SE = .23, t(20) = -.36, p = .73$).

For proactive aggression predicting EDA reduction comparing pre- to post- training arousal during a stressful event, the assumption of linearity was deemed met, while the assumption of homoscedasticity was not. The regression model is insignificant ($F(1, 20) = 2.75, p = .11$), with an R^2 of .12, proactive aggression being an insignificant negative predictor ($\beta = -.35, SE = .21, t(20) = -1.66, p = .11$). Regarding reactive aggression predicting EDA reduction comparing pre- to post- training arousal during a stressful event, the assumption of homoscedasticity and linearity were met. The regression model is significant ($F(1, 20) = 7.624, p = .012$), with reactive aggression being a significant negative predictor ($\beta = -.53, SE = .19, t(20) = -2.76, p = .012$). This means participants with higher reactive aggression experienced a smaller arousal reduction from their respective baseline comparing pre- and post-training stressful events, as measured with skin conductance. This significant result is graphically represented in figure 6 below, with the according standardized regression equation ($EDA \text{ reduction} = -.53\beta_{\text{reactive aggression}}$).

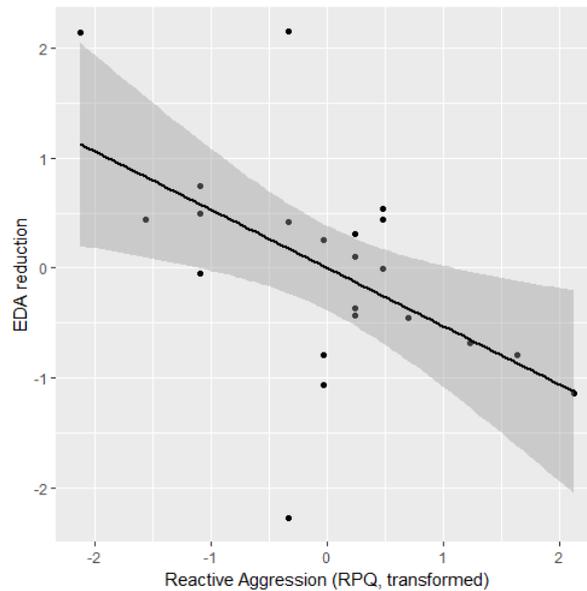


Figure 6. Linear graph showing the regression of the skin conductance reduction of arousal increase of the participants during stress compared to pre-training, predicted by the reactive aggression of the participant.

For proactive aggression, predicting PAQ reduction, the assumption of linearity and homoscedasticity was not met. The regression model is insignificant ($F(1, 20) = .040, p = .84$), with an R^2 of .0020, proactive aggression being an insignificant negative predictor ($\beta = -.048, SE = .22, t(20) = -.2, p = .84$). Regarding reactive aggression predicting PAQ reduction, the assumption of homoscedasticity and linearity both did not seem met. The regression model is insignificant ($F(1, 20) = 3.04, p = .097$), with reactive aggression being an insignificant negative predictor ($\beta = -.36, SE = .21, t(20) = -1.74, p = .097$).

Main mediation models. A path analysis model was constructed examining associations between reactive and proactive aggression, interoceptive awareness and the aforementioned arousal reductions, all being exogenous constructs. Due to two different scales of interoceptive awareness and three different ways in which arousal reduction was measured, 6 mediation models were analysed in this study. In these models, as shown in figure 3, both reactive and proactive aggression indicate interoceptive awareness and arousal reduction, while interoceptive awareness additionally indicates arousal reduction, creating five direct paths, and 2 indirect paths. These SEM models being saturated as consequence, no model fit

analyses could be conducted and it has to be assumed this is the correct model based on theory. Therefore, the model was constructed to match both theory and the study design, which includes a covariance between proactive and reactive aggression, found significant in all models ($\beta = .590, SE = .239, p = .014$).

For model 1, shown in figure 7, taking standardized heartbeats per minute and using standardized interoceptive awareness of body listening as mediator, only interoceptive awareness by listening to the body was a significant positive predictor on BPM reduction ($\beta = .535, SE = .179, p < .01$). Both the indirect effect of proactive aggression ($\beta = .020, SE = .143, p = .88$) and reactive aggression ($\beta = -.101, SE = .147, p = .49$) on BPM reduction via interoceptive awareness by listening to the body were insignificant.

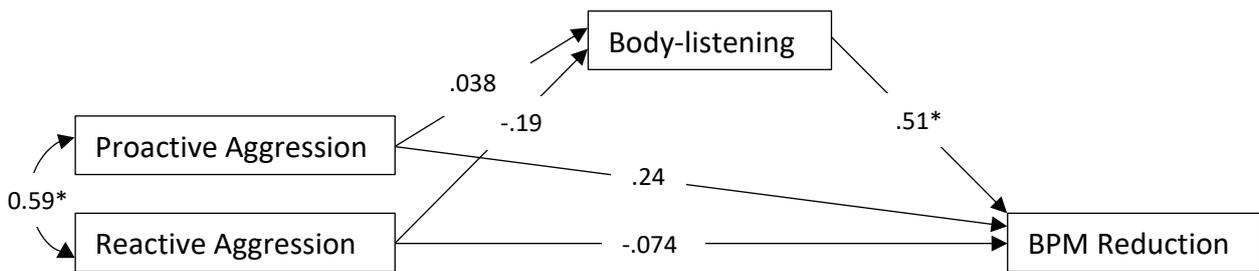


Figure 7. Mediation model of proactive and reactive aggression on heartbeat reduction, with body-listening as mediator, including all standardized regression coefficient estimates. * $p < .05$.

Regarding model 2, shown in figure 8, using interoceptive awareness by self-regulation, no significant direct or indirect regression paths were found.

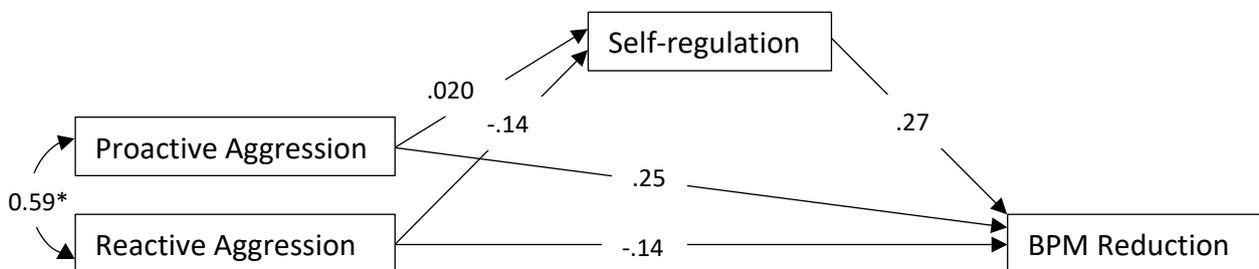


Figure 8. Mediation model of proactive and reactive aggression on heartbeat reduction, with self-regulation as mediator, including standardized regression coefficient estimates. * $p < .05$.

For model 3, as depicted in figure 9, using standardized skin conductance in micro Siemens as arousal reduction measurement and listening to the body as interoceptive awareness, only one significant regression path was found. Reactive aggression significantly predicted EDA arousal reduction ($\beta = -.530, SE = .231, p = .022$) in a negative relationship.

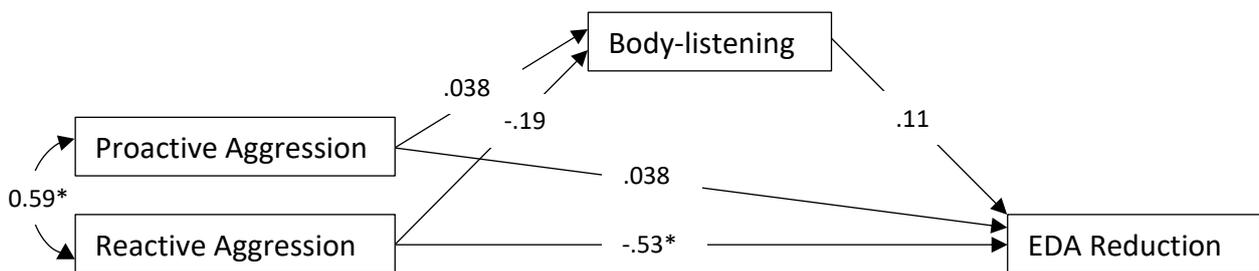


Figure 9. Mediation model of proactive and reactive aggression on skin conductance reduction, with body-listening as mediator, including all standardized regression coefficient estimates. * $p < .05$.

Model 4, as shown in figure 10, using interoceptive awareness by self-regulation, only had reactive aggression ($\beta = -.530, SE = .228, p = .020$) as a significant negative predictor of EDA arousal reduction as well.

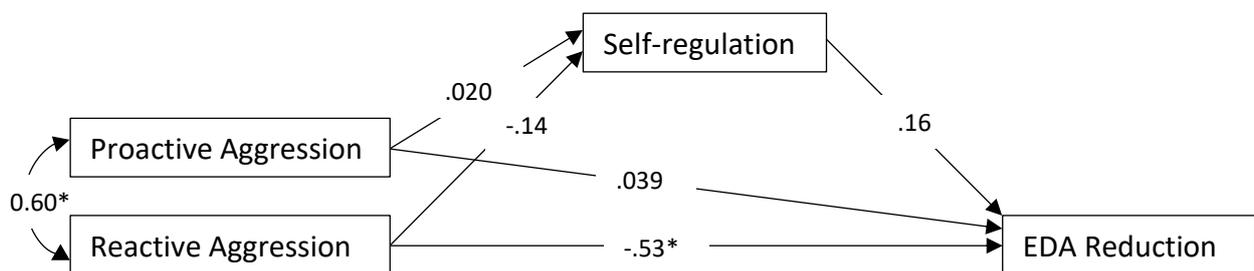


Figure 10. Mediation model of proactive and reactive aggression on skin conductance reduction, with self-regulation as mediator, including all standardized regression coefficient estimates. * $p < .05$.

Regarding model 5, shown in figure 11, concerning physiological arousal questionnaire score reduction and listening to the body as interoceptive awareness, one significant regression path was found. This was once again reactive aggression negatively predicting arousal reduction ($\beta = -.500, SE = .240, p = .037$), in this case physiological.

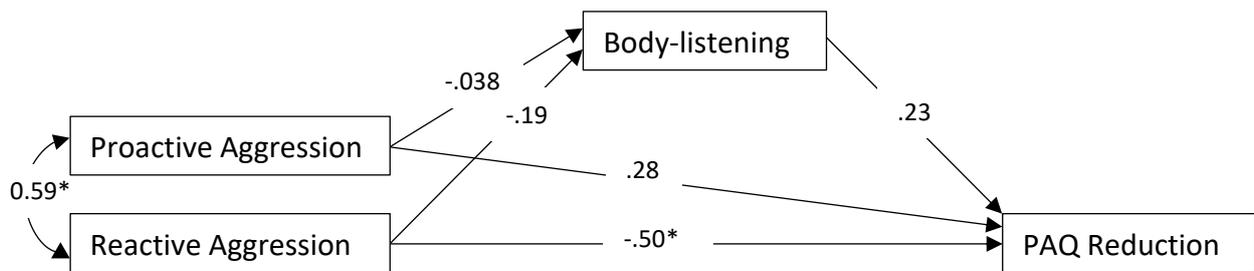


Figure 11. Mediation model of proactive and reactive aggression on self-reported physiological reduction, with body-listening as mediator, including all standardized regression coefficient estimates. $*p < .05$.

For model 6 however, shown in figure 12, which used self-regulation as indicator of interoceptive awareness, two significant direct regression paths were found. The first is reactive aggression negatively predicting reported physiological arousal reduction ($\beta = -.491, SE = .225, p = .029$). The second was self-regulation positively predicting arousal reduction ($\beta = .372, SE = .177, p = .036$).

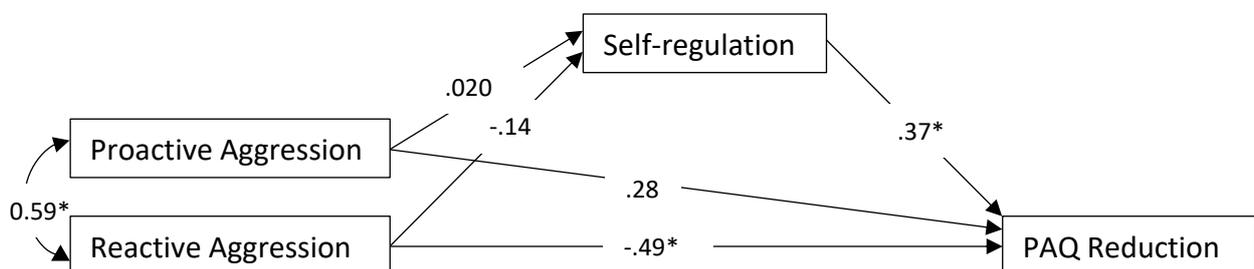


Figure 12. Mediation model of proactive and reactive aggression on self-reported physiological reduction, with self-regulation as mediator, including all standardized regression coefficient estimates. $*p < .05$.

Discussion

Putting all the results together, several conclusions can be drawn from the data. The preliminary t-test analyses show all forms of arousal significantly increased during the stress-task, both from a physiological (EDA, ECG) and psychological (PAQ) perspective. This is important, because it shows the instrument validity of the adapted stress task is strong, as supported by Hollenstein and colleagues (2012), on which this stress-task is based.

The preliminary regression analyses, however, already indicated trouble for the main analysis. The predictability that arousal increases under the stressful events which the t-tests have proven to exist, was often insignificantly predicted by the aggression types. Furthermore, the data that was significant, being reactive aggression predicting a smaller self-reported arousal increase during the first session, is against the alternative hypothesis that was taken in mind for this analysis. This is unexpected considering the studies of Mayberry and Espelage (2007), who predicted proactive aggression comes with little arousal while reactive aggression should be linked to a steeper arousal increase during stressful situations. This also seems to go against Raine and colleagues (2006), who stated reactive aggression is a fear-induced irritable effect, which socio-evaluative stress should cause (Fechtner, 2012; Kirschbaum, Pirke & Helhammer, 1993).

A design issue specific to this part of the analysis that could have caused this disparity is that participants with higher reactive aggression could already have had higher arousal when measuring the baseline, as they are put in an isolated room, are hooked to wires and are uncertain what will happen. This level of uncertainty and unpredictability regarding emotionally salient events in which furthermore a reward is promised, is known to cause anxiety and therefore higher arousal (Lowry, Johnson, Hay-Schmidt, Mikkelsen, & Shekhar, 2005). However, t-tests conducted after the main analyses for this discussion question, between participants scoring above the median on reactive aggression and below the median

on reactive aggression reveal no significant baseline differences in skin conductance, heartbeat and reported arousal during session 1. There being at least 15 minutes between participant entry into the room and the measurement of the baseline, often more, the notion of the initial arousal levels being temporally higher due to movement – e.g. cycling – can also be ruled out. Another reason for this result could be that the data is often not normally multivariately distributed, linear or heteroscedastic, which can be largely attributed to the small non-sensitive sample. As part of an ongoing research, this paper was written with a much smaller sample than necessary for regression analyses to have a reasonable standard deviation and reliability. This is an ongoing problem for the other analyses as well, especially the structural equation model, wherefore large samples are required. Furthermore, due to the sample being comprised of students, many scored low on the proactive and reactive aggression scales, amplifying the already prone non-normal distributions. One example is the mean of the RPQ scales in this study ($M_{\text{proactive}} = 1.40$, $M_{\text{reactive}} = 5.95$) being a lot lower than the means of even a normal sample from literature ($M_{\text{proactive}} = 2.79$, $M_{\text{reactive}} = 7.14$; Raine et al., 2006), let alone a sample comprised of those with actual aggression disorders. It could be that the reactive aggression scores from this sample are too low to indicate actual reactive aggression.

The main regression analyses continue the trend of the preliminary regression analyses. Reactive aggression negatively predicts skin conductance arousal reduction, meaning participants with higher reactive aggression have a higher skin conductance-based arousal increase during the second stress-task, which goes against the previously mentioned sources regarding aggression and arousal as well. One possible limitation could be the presence of much noise in the physiological data which, although careful precautions were taken, could perhaps not all have been filtered out. Another, more interesting reason, could be that those with higher reactive aggression, due to it being related to impulsivity and lower self-control

(Raine et al., 2006), do not implement the strategy that was taught to them, especially with only four sessions.

The mediation models created showed no significant mediating effects. They did, firstly, re-establish reactive aggression significantly negatively predicts training-induced reduction of arousal in stressful situations. Secondly, they indicated participants with higher interoceptive awareness in the form of listening their body had significantly reduced arousal due to stress in the form of heart beats per minute in session four compared to the first session. Those with higher interoceptive awareness in the form of self-regulation, however, showed significantly higher reduced self-reported physiological arousal due to stress in session four compared to session one. It is interesting to see self-regulation is related to behavioural arousal reduction while not physiological arousal reduction, while body listening vice versa. One reason could be that the heartbeat could be easier to listen to but seem more difficult to regulate, while behaviourally perceived arousal reduction seems much easier to change. However, further research should be conducted to further explore the reasons behind this disparity. It is important to note once again that due to having saturated models with zero degrees of freedom, no assumption could be made on whether this model significantly better than other models, therefore having a good model fit. However, as this model is based on theoretically placed exogenous variables, this is much less problematic than if, for instance, latent variables were involved.

By implementing four-session biofeedback relaxation training and relating it to different types of aggression for the first time, measuring physiological but also newly self-reported arousal, this explorative study gives new insight into the relation of reactive aggression and arousal increase in stressful situations after a relaxation training has been conducted. It counter-intuitively and counter-theoretically predicts those with higher reactive aggression are worse in reducing physiological arousal under stress by using a relaxation training. These

results could have, if reliably re-tested to counter the limitations in this research, have large consequences in the psychological field, as this would indicate those with reactive aggression either need more guidance to implement the relaxation, more sessions to make the implementations stick, or other training-techniques in reducing arousal might have more effect.

Future research regarding the limitations of this research should focus on gathering a bigger sample, and implementing this research sample that is more aggressive. A future statistical option is to apply dynamic system modelling to see whether there is a pattern of predictability instead of a predictive linear relationship between aggression and arousal. Future theoretical studies could look into further focusing on the aforementioned disparity in self-regulation and body listening and their effect on psychological and physiological arousal, or how impulsivity influences whether individuals with reactive aggression actually apply the techniques they learned to relax. After all, “a healthy mind, has an easy breath” (Gibson, 2008, p. 113).

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