Psychopathy and Heart Rate Variability: A New Physiological Marker for the Adaptive Features of Boldness

Pilar Segarra, Rosario Poy, Victoria Branchadell, Pablo Ribes-Guardiola, & Javier Moltó

Affective Neuroscience Lab, Department of Basic and Clinical Psychology, and Psychobiology. Universitat Jaume I, Castelló, Spain

Author's note: Data and code for this study are available upon reasonable request from the corresponding authors.

Funding: This work was supported by the Ministerio de Ciencia, Innovación y Universidades (Spain; Grant PID2019-104522GB-I00; Grant FPU18/01613 to VB).

Declarations of interest: none

Correspondence concerning this article should be addressed to:

Pilar Segarra, Department of Basic and Clinical Psychology, and Psychobiology, Universitat Jaume I Avenida Sos Baynat s/n 12071, Castelló, Spain. E-mail: segarra@uji.es

Javier Moltó, Department of Basic and Clinical Psychology, and Psychobiology, Universitat Jaume I Avenida Sos Baynat s/n 12071, Castelló, Spain. E-mail: molto@uji.es

Abstract

The boldness disposition of the triarchic model of psychopathy is theorized to entail, aside from maladaptive proclivities (narcissism, fearless risk-taking), some adaptive features (e.g., immunity to stressful events, high self-esteem, and emotional resilience) that seem to predispose high boldness individuals to an effective emotional regulation in response to environmental demands. The high frequency band of heart rate variability —an index of parasympathetic cardiac vagal activity— is a well-validated physiological index of emotional self-regulation and mental health resilience. The aim of this study was to examine the unique predictive contributions of triarchic dispositions of boldness, meanness, and disinhibition on resting vagally-mediated heart rate variability (vmHRV) in a sample of 241 undergraduates (60 men) assessed via the Triarchic Psychopathy Measure (TriPM; Patrick, 2010). A multiple regression analysis was conducted on vmHRV in which TriPM Boldness, Meanness, and Disinhibition scores were entered as predictors, along with gender, age, body mass index, mean resting heart rate, and respiratory activity. Results showed that only TriPM Boldness —but not Meanness or Disinhibition— scores significantly predicted vmHRV (positively), thus evidencing that adequate emotional self-regulation is one of the adaptive features encompassed by the boldness disposition. These findings encourage further use of vmHRV as a physiological marker of boldness and contribute to shedding light on the nomological network surrounding the construct of boldness in psychopathy.

Nowadays, the triarchic model (Patrick et al., 2009) is one of the most influential conceptualizations of psychopathy because, among other reasons, it links symptomatic features of psychopathy to neurobiological systems and processes. Specifically, the triarchic model conceptualizes psychopathy in terms of three distinct but interrelated biobehavioral dispositions or psychological constructs related to normal-range personality traits that have direct neural and behavioral referents (Patrick & Drislane, 2015).

The disinhibition disposition entails a phenotypic style characterized by impulsiveness, lack of behavioral restraint, and difficulties in affective control. Disinhibition is viewed as a liability factor for externalizing psychopathology and corresponds to the neurobehavioral dimension of inhibitory control that putatively reflects frontal-brain-based differences in the capacity for behavioral restraint. The meanness disposition encompasses a constellation of phenotypic features linked to a lack of ability to understand other~~'s~~ people's feelings and welfare that are manifested behaviorally by strategic exploitation of others, callousness, lack of close attachments and predatory aggression, among others. Meanness is thought to reflect a biologically based predatory orientation involving deficient empathic sensitivity and weak affiliation/attachment capacity that is expressed in antagonistic externalizing psychopathology. The boldness disposition follows Cleckley's (1941/1976) concept of psychopathy as a personality disorder that entails an outward appearance of psychological (adaptive) normality that masks a severe pathological maladjustment. With this in mind, boldness is theorized to represent the paradox underlying psychopathy, thus comprising a phenotypic style characterized by maladaptive proclivities (e.g., narcissism, fearless risk-taking, and failures to learn from punishment experiences) in conjunction with certain adaptive features (e.g., low levels of anxiety or fear, immunity to stress, a socially potent interpersonal style). Boldness is hypothesized to correspond to the neurobehavioral dimension of threat sensitivity, reflecting individual differences in the reactivity of the brain's

defensive motivational system —based on the amygdala and affiliated structures. Several studies support this by showing diminished startle potentiation during exposure to threat stimuli (e.g., Esteller et al., 2016), reduced cortical late positive potential (LPP) to negative versus neutral pictures during passive viewing (Ellis et al., 2017), and deficient CS+/CS- electrodermal (López et al., 2013) and electrocortical differentiation (Paiva et al., 2020) in fear conditioning in high fearless dominance/boldness individuals. In addition, boldness has shown consistent negative associations with self-report measures of fearfulness/anxiety and internalizing symptomatology (Latzman et al., 2020; Poy et al., 2014). It is worth nothing that boldness has been related to maladaptive response perseveration in the face of increasing punishment contingencies (Ribes-Guardiola et al., 2020), but also to an enhanced task switching performance under threat conditions (Yancey et al., 2019), suggesting that resistance to the impact of danger or punishment cues might involve adverse outcomes or an adaptive resilient style depending on the situation. Boldness has recently been shown to predict greater emotional well-being during COVID-19 outbreak (Sica et al., 2021) and reduced frequency of protective behaviors (Paiva et al., 2021), supporting the idea that boldness' protection from emotional distress may in turn lead to behaviors that increase the risk of getting the disease. From this standpoint, it seems likely that their low threat sensitivity might be somehow responsible for the fact that high boldness individuals show both good emotional regulation (resilient functioning) and behavioral dysregulation (reckless and unrestrained responses) under stressful or threatening situations. Although the neurophysiological correlates of threat processing deficits linked to triarchic boldness (e.g., reduced fear-potentiated startle, deficient fear conditioning, diminished LPP to aversive stimuli) are relatively well-studied, empirical evidence about physiological correlates for the potentially adaptive features of boldness (as adequate emotional regulation) is still lacking.

Resting heart rate variability (HRV) —the variation in the time interval between consecutive heartbeats in milliseconds— is currently widely considered as a well-validated physiological index of emotional regulation capacity (see Appelhans & Luecken, 2006, and Balzarotti et al., 2017, for reviews). The high frequency band of HRV (HF-HRV) —which reflects the parasympathetic influence (via the vagus nerve) on the sinoatrial node of the heart (Berntson et al., 1997)— has been specifically proposed to be a transdiagnostic biomarker of self-regulation (the ability to regulate behavioral, emotional, and cognitive processes) and mental health resilience (e.g., Beauchaine & Thayer, 2015; Thayer et al., 2012).

In this line, the neurovisceral integration model (Thayer & Lane, 2000, 2009) emphasizes the interplay between this vagally-mediated cardiac activity and the brain structures related to emotional processing-and suggests that several prefrontal cortical areas modulate cardiovascular activity via the inhibition (in safe contexts) and disinhibition (under threat conditions) of the amygdala (see Thayer & Lane, 2000, for a review of the three routes by which the amygdala would lead to cardiac vagal control). Given that HF-HRV is positively related to amygdala-medial prefrontal cortex functional connectivity and shows similar associations with criterion measures of the nomological network of boldness (i.e., positive correlations with stress immunity, emotional resilience, subjective well-being, and negative relationships with internalizing psychopathologies and self-report measures of fearfulness; see Thayer et al., 2012), we hypothesized that HF-HRV and triarchic boldness would be positively related. The only study that has examined the relationship between vagally-mediated heart rate variability and psychopathy features supports this hypothesis to some extent. Thus, in a sample of male prisoners, Hansen et al. (2007) found that the interpersonal facet of psychopathy —which partially captures the measurement domain of boldness (Venables et al., 2014)— explained most of the HF-HRV. However, the predictive contribution of boldness features to HF-HRV is yet to be directly tested.

The main goal of this study was to explore the association between resting vagally-mediated heart rate variability (vmHRV) and boldness above and beyond the meanness and disinhibition dispositions of the triarchic model of psychopathy, in order to examine the usefulness of vmHRV as a positive, physiological correlate of boldness. On the basis of the close link between vagally-mediated cardiac activity and emotional regulation ability via amygdala inhibition/disinhibition (see Thayer et al., 2009), and the fact that certain features of the boldness disposition are theoretically related to emotional adjustment such as immunity to stressful events or emotional resilience (see Patrick et al., 2009), it was expected that high vmHRV would be exclusively associated with higher self-reported boldness scores.

Method

Participants

Study participants were 245 volunteer undergraduate psychology students between the ages of 18 and 25 years. No participant reported diagnosis of mental disorder or pharmacological treatment that could alter cardiac activity at the time of testing. Four male undergraduates were excluded from the analysis after participation due to alcohol use 24 h prior to the ECG recording. The final sample comprised a total of 241 Caucasian participants (181 women, 60 men), with a mean age of 20.01 years (SD = 1.95). All participants signed a written informed consent form and received academic credit for their participation.

Measures

Triarchic Psychopathy Measure (TriPM; Patrick, 2010). The TriPM is a 58-item self-report inventory specifically designed to measure the three phenotypic domains proposed in the triarchic model of psychopathy (Patrick et al., 2009). The items were answered using a 4-point Likert scale (3 = *true*, 2 = *somewhat true*, 1 = *somewhat false*, 0 = *false*). The Spanish translation of the TriPM had previously shown good criterial validity (Esteller et al., 2016;

Poy et al., 2014). Internal consistencies (alpha coefficients) for Boldness, Meanness, and Disinhibition scores in the current sample were .78, .81, and .79, respectively.

Heart Rate Variability (HRV). The electrocardiogram (ECG) was recorded using 8 mm In Vivo Metric Ag/AgCl surface electrodes (Standard Lead II placement) at a 1000 Hz sampling rate using a Coulbourn V75-04 High Gain Isolated Bioamplifier (with high and low cutoffs set at 40 Hz and 8 Hz, respectively). Electrodes were placed on the right wrist and the left ankle; the ground electrode was placed on the right ankle. Analog ECG signals provided by VPM software (Cook, 2002) were transferred to Kubios HRV analysis Package 2.2 (Tarvainen et al., 2014). Artifacts detected within the R-to-R series were removed by applying an artifact correction level that differentiated abnormal inter-beat-intervals (IBIs), in milliseconds, from median IBIs using a piecewise cubic spline interpolation method. Following established guidelines (Laborde et al., 2017), the R-to-R intervals were subjected to an autoregressive power spectrum density method (AR model order = 16) to obtain absolute powers of high frequency (HF; 0.15-0.4 Hz) band, an index of vagal cardiac activity mediated by the parasympathetic system (Berntson et al., 1997). An ECG-derived respiration (EDR) measure was also computed using the Kubios' algorithm to estimate sinus respiratory arrhythmia from peak HF-HRV values.

Procedure

During the first semester, participants filled out the TriPM at sessions with a maximum of 45 subjects. ECG recording was conducted individually in an isolated, dimly lit room during the second semester. All participants were asked not to smoke, engage in vigorous physical activity, or drink caffeine 2 h before the experiment

After arriving at the laboratory, participants were weighed, measured, and informed about the experimental session protocol, which had been approved by the ethics committee of the university and was carried out in accordance with the Declaration of Helsinki. The

electrodes were then attached to the participants and, after a period of acclimatization, the 5-min baseline-resting ECG recording started. As recommended by guidelines reports (e.g., Laborde et al., 2017; Quintana et al., 2016), participants were seated in a comfortable armchair (with knees at a 90º angle, both feet resting on the footrest and hands on the armrests) and were asked to breathe spontaneously and to remain with their eyes open throughout the recording time. After a short break, students who voluntarily kept on participating in the experimental session underwent a passive picture viewing task that included measurement of startle reflex responses (results reported in Esteller et al., 2016). Participants then completed a brief survey asking for their age, alcohol consumption in the 24 h prior to the experiment, and time elapsed since the last tea or coffee consumption.

Statistical analysis

Prior to all analyses, HF-HRV power values in m^2 were transformed into natural logarithms (lnHF) to fit to the assumptions of the linear analysis. A three-stage regression analysis was conducted to test our main hypothesis about the unique predictive contribution of boldness disposition to vmHRV. Step 1 included gender (0 = men, 1 = women), age (in years), body mass index (BMI; Kg/m^2), mean resting heart rate (HR; in beats per minute), and ECG derived respiration (EDR) as predictors, given their demonstrated influence on inter-individual differences in HRV (e.g., Laborde et al., 2017; Quintana et al., 2016). TriPM Meanness and Disinhibition scores were entered at Step 2, and TriPM Boldness scores were added at Step 3 to test for the predictive contribution of this disposition alone to vmHRV after controlling for its overlap with the physiological variables and the other triarchic dispositions.

Results

Descriptive statistics and bivariate correlations between variables of interest are shown in Table 1 (left). Regression coefficients predicting lnHF at Step 3 of the hierarchical regression

model are shown in Table 1 (right)[1]. As expected, HR (β = -0.47) and EDR (β = -.0.19) were significant predictors of vmHRV at Step 1, $F(5, 235) = 14.94$, $p < .001$, $R^2 = .241$. Neither TriPM Meanness nor TriPM Disinhibition scores contributed to the prediction of lnHF at Step 2 ($\Delta R^2 = .006$, $p = .41$), in which HR (β = -0.47), EDR (β = -0.18), and Gender (β = 0.14) were significant predictors, $F(7, 233) = 10.92$, $p < .001$, $R^2 = .247$. TriPM Boldness scores significantly increased the explained variance at Step 3 ($\Delta R^2 = .014$, $p = .036$), overall model $F(8, 232) = 10.26$, $p < .001$, $R^2 = .261$. Thus, consistent with our hypothesis, inter-individual differences in boldness accounted for physiological differences in vmHRV above and beyond the triarchic dispositions of meanness and disinhibition, even after adjusting for the contribution of known relevant covariates.

Discussion

This was the first study to focus explicitly on the relationship between cardiac vagal activity —measured via resting vagally-mediated heart rate variability (vmHRV)— and the behavioral dispositions of boldness, meanness, and disinhibition of the triarchic model of psychopathy (Patrick et al., 2009). Our results demonstrated, as hypothesized, that vmHRV was positively related to TriPM Boldness scores. This finding is consistent with numerous studies showing that both HF-HRV and boldness can be adequate indicators —coming from different assessment domains (physiological and self-report, respectively)— of an adaptive and healthy psychological functioning. Thus, it is important to highlight that heart rate variability has traditionally been considered as a protective factor against internalizing symptomatology (e.g., Beauchaine &Thayer, 2015), just as the boldness disposition of the

[1] The sensitivity power analyses conducted in G*Power (see Faul et al., 2007) revealed that the linear multiple regression (fixed model, R^2 deviation from zero) with 241 participants and eight predictors would be sensitive to an effect size of $f^2 = 0.06$ (critical $F = 1.98$).

triarchic model has recently been demonstrated to be (e.g., Latzman et al., 2020). Furthermore, these two measures are linked with psychologically well-adjusted personality variables such as high emotional resilience, good executive functioning, high-self-esteem, and subjective well-being —for example, see Sleep et al. (2019) for correlations of boldness, and Holzman & Bridget (2017) and Thayer et al. (2012) for relationships of heart rate variability with aforementioned variables.

Although the relevance of the triarchic boldness disposition for psychopathy has recently been the subject of intense scientific debates (for a review, see Lilienfeld et al., 2012; Miller & Lyman, 2012)—, the fact that this disposition is assumed to reflect individual differences in the reactivity of the brain's defensive motivational system to threat signals makes it especially relevant to the field of psychopathy. Boldness is the only triarchic disposition consistently related to low neuroticism and high extraversion (e.g., Miller et al., 2016; Poy et al., 2014), personality traits theoretically associated with a weak behavioral inhibition system (BIS; Gray, 1987, and Gray & McNaughton, 2000), responsible for inhibiting or regulating approach behavior that might lead to adverse outcomes in response to threats of punishment. Indeed, research is beginning to learn about the specific role of boldness in some well-documented threat processing psychopathic deficits including maladaptive response perseveration in the face of punishment (Ribes-Guardiola et al., 2020), diminished startle responses to threat pictures (Esteller et al., 2016), and reduced amplitudes of late positive potentials to aversive signals (Ellis et al., 2017; Paiva et al., 2020).

Together with the aforementioned markers, the findings of the present study suggest that vmHRV could also be incorporated into psychopathy research as a potential new physiological indicator of boldness and, in addition, it could be extremely useful to explore the role of threat sensitivity and parasympathetic cardiac activation in certain phenotypic outcomes of psychopathy. The vmHRV indexes the capacity of the prefrontal cortex to

modulate subcortical circuits of fear responses, and the association between vmHRV and individual differences in boldness could also contribute to shedding light on the top-down regulatory control processes of negative emotions that have recently been hypothesized to underlie this disposition of psychopathy (see Yancey et al., 2019). This potential etiological hypothesis would be consistent both with evidence of impaired prefrontal-amygdala connectivity in psychopathy (e.g., Motzkin et al., 2011), and with proposed links between the prefrontal cortex and the septo-hippocampal system (Gray & McNaughton, 2000), the core neurobiological substrate of the BIS. At this point, two key aspects of the triarchic model of psychopathy need to be emphasized (see Patrick, 2018): (1) this model does not assume that the dispositions of boldness, meanness, and disinhibition should correspond directly with neurobiological systems of threat reactivity, affiliative capacity, and inhibitory control, respectively, but rather proposes that these dispositions, as dimensions of variation in biobehavioral functioning across individuals, can be operationalized using indicators from different measurement domains and serve to establish bridges between clinical problems and neurobiological processes, and (2) from this standpoint, psychopathological symptoms reflect the interplay of biobehavioral systems with environmental influences over time and developmental stages. Consequently, the first step in understanding the etiology of psychopathy would be to identify multiple correlates of these systems and to explore how individual differences in their functioning are related to distinct configurations of psychopathic expressions. The relevance of the finding of the present study lies in the identification of resting heart rate variability as a marker of boldness and, therefore, stands as the first step toward identifying variables from the physiological and behavioral response domains that correlate with the biobehavioral system of threat reactivity.

However, these results should be considered in light of some limitations. On the one hand, the use of a sample of undergraduates with a small age range makes it difficult to

generalize our results to other populations and age ranges. In addition, the use of a sample with unequal gender ratio may influence our results, although participants' gender was included as a variable in the analyses. Future research in gender-balanced samples of different types (community, clinical, criminal) and ages (child, adolescent, adult) is needed to investigate these issues. On the other hand, the use of self-report instruments to assess dimensions in undergraduates may narrow the range of scores on a particular triarchic disposition of psychopathy. Moreover, it would be desirable to incorporate different operationalizations of the triarchic dispositions other than the TriPM scales. Future work should also try to operationalize constructs of psychopathy using indicators from multiple domains of measurement (physiological, behavioral, self-report), in order to establish a psychoneurometric quantification of biobehavioral dispositions.

Despite these limitations, the present study provides evidence supporting vmHRV as a potential physiological correlate of the boldness disposition that could help advance our understanding of the different etiological processes and pathways underlying psychopathic dispositions as specified in the triarchic model. Once the patterns of associations (convergent and discriminant) of vmHRV with other reliable multiple indicators have been established, researchers should be able to determine whether this indicator contributes to the emergence, expression, and temporal course of a given psychopathic phenotype. This research strategy will not only provide a comprehensive picture of the correlates associated with (maladaptive and adaptive) features of the boldness disposition, but it will undoubtedly also have a major impact on the understanding of psychopathy in neurophysiological terms, thus contributing to the National Institute of Mental Health's Research Domain Criteria (NIMH RDoC; Insel et al., 2010) framework, which promotes a multidomain, biobehavioral approach to examine the nature of mental health and clinical psychopathologies.

References

Appelhans, B. M., & Luecken, L. J. (2006). Heart Rate Variability as an Index of Regulated Emotional Responding. *Review of General Psychology, 10*(3), 229–240. https://doi.org/10.1037/1089-2680.10.3.229

Balzarotti, S., Biassoni, F., Colombo, B., & Ciceri, M. R. (2017). Cardiac vagal control as a marker of emotion regulation in healthy adults: A review. *Biological Psychology, 130*, 54-66. https://doi.org/10.1016/j.biopsycho.2017.10.008

Beauchaine, T., & Thayer, J. (2015). Heart rate variability as a transdiagnostic biomarker of psychopathology. *International Journal of Psychophysiology, 98*(2), 338–350. https://doi.org/10.1016/j.ijpsycho.2015.08.004

Berntson, G. G., Bigger, J. T., Eckberg, D. L., Grossman, P., Kaufmann, P. G., Malik, M., Nagaraja, H. N., Porges, S.W., Saul, J. P., Stone, P. H., & van der Molen, M.W. (1997). Heart rate variability: Origins, methods, and interpretive caveats. *Psychophysiology, 34*(6), 623–648. https://doi.org/10.1111/j.1469-8986.1997.tb02140.x

Cleckley, H. (1976). *The mask of sanity*. St. Louis, MO: Mosby (Original work published 1941).

Cook, E. W., III (2002). *VPM reference manual*. Birmingham, AL: Author.

Ellis, J. D., Schroder, H. S., Patrick, C. J., & Moser, J. S. (2017). Emotional reactivity and regulation in individuals with psychopathic traits: Evidence for a disconnect between neurophysiology and self-report. *Psychophysiology, 54(10)*, 1574-1585. https://doi.org/10.1111/psyp.12903

Esteller, À., Poy, R., & Moltó, J. (2016). Deficient aversive-potentiated startle and the triarchic model of psychopathy: The role of boldness. *Biological Psychology*, *117*, 131-140. https://doi.org/10.1016/j.biopsycho.2016.03.012

Faul, F., Erdfelder, E., Lang, A. G., & Buchner, A. (2007). G* Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods*, *39*(2), 175-191. https://doi.org/10.3758/BF03193146

Gray, J. A. (1987). *The psychology of fear and stress*. Cambridge University Press.

Gray, J. A., & McNaughton, N. (2000). *The Neuropsychology of anxiety: an enquiry into the functions of the septo-hippocampal system (2nd ed.)*. Oxford University Press.

Hansen, A. L., Johnsen, B. H., Thornton, D., Waage, L., & Thayer, J. F. (2007). Facets of psychopathy, heart rate variability and cognitive function. *Journal of Personality Disorders*, *21*(5), 568-582. https://doi.org/10.1521/pedi.2007.21.5.568

Holzman, J., & Bridgett, D. (2017). Heart rate variability indices as bio-markers of top-down self-regulatory mechanisms: A meta-analytic review. *Neuroscience and Biobehavioral Reviews*, *74*, 233-255. https://doi.org/10.1016/j.neubiorev.2016.12.032

Insel, T., Cuthbert, B., Garvey, M., Heinssen, R., Pine, D. S., Quinn, K., Sanislow, C., & Wang, P. (2010). Research domain criteria (RDoC): toward a new classification framework for research on mental disorders. *The American Journal of Psychiatry,*

Laborde, S., Mosley, E., & Thayer, J. (2017). Heart rate variability and cardiac vagal tone in psychophysiological research – Recommendations for experiment planning, data analysis, and data reporting. *Frontiers in Psychology*, *8*, 213. https://doi.org/10.3389/fpsyg.2017.00213

Latzman, R. D., Palumbo, I. M., Krueger, R. F., Drislane, L. E., & Patrick, C. J. (2020). Modeling relations between triarchic biobehavioral traits and DSM internalizing disorder dimensions. *Assessment, 27*(6), 1100-1115. https://doi.org/10.1177/1073191119876022

Lilienfeld, S. O., Patrick, C. J., Benning, S. D., Berg, J., Sellbom, M., & Edens, J. F. (2012). The role of fearless dominance in psychopathy: Confusions, controversies, and

clarifications. *Personality Disorders: Theory, Research, and Treatment, 3(3)*, 327-340. https://doi.org/10.1037/a0026987

López, R., Poy, R., Patrick, C. J., & Moltó, J. (2013). Deficient fear conditioning and self-reported psychopathy: The role of fearless dominance. *Psychophysiology, 50*(2), 210-218. https://doi.org/10.1111/j.1469-8986.2012.01493.x

Miller, J. D., Lamkin, J., Maples-Keller, J. L., & Lynam, D. R. (2016). Viewing the triarchic model of psychopathy through general personality and expert-based lenses. *Personality Disorders: Theory, Research, and Treatment, 7(3),* 247-258. http://dx.doi.org/10.1037/per0000155

Miller, J. D., & Lynam, D. R. (2012). An examination of the Psychopathic Personality Inventory's nomological network: A meta-analytic review. *Personality Disorders: Theory,Research, and Treatment, 3(3)*, 305-326. https://doi.org/10.1037/a0024567

Motzkin, J. C., Newman, J. P., Kiehl, K. A., & Koenigs, M. (2011). Reduced prefrontal connectivity in psychopathy. *Journal of Neuroscience*, *31*(48), 17348-17357. https://doi.org/10.1523/JNEUROSCI.4215-11.2011

Paiva, T. O., Almeida, P. R., Coelho, R. C., Pasion, R., Barbosa, F., Ferreira-Santos, F., Bastos-Leite, A. J., & Marques-Teixeira, J. (2020). The neurophysiological correlates of the triarchic model of psychopathy: An approach to the basic mechanisms of threat conditioning and inhibitory control. *Psychophysiology, 57*(8), e13567. https://doi.org/10.1111/psyp.13567

Paiva, T. O., Cruz-Martins, N., Pasion, R., Almeida, P. R., & Barbosa, F. (2021). Boldness personality traits are associated with reduced risk perceptions and adoption of protective behaviors during the first COVID-19 outbreak. *Frontiers in Psychology, 12*, 974. https://doi.org/10.3389/fpsyg.2021.633555

Patrick, C. J. (2010). Triarchic Psychopathy Measure (TriPM). Retrieved from https://www.phenxtoolkit.org/protocols/view/121601?origin=search

Patrick, C. J. (2018). Psychopathy as masked pathology. In C. J. Patrick (Ed.), *Handbook of Psychopathy* (2nd ed., pp. 3-21). The Guilford Press

Patrick, C. J., & Drislane, L. E. (2015). Triarchic model of psychopathy: Origins, operationalizations, and observed linkages with personality and general psychopathology. *Journal of Personality, 83*(6), 627-643. https://doi.org/10.1111/jopy.12119

Patrick, C. J., Fowles, D. C., & Krueger, R. F. (2009). Triarchic conceptualization of psychopathy: Developmental origins of disinhibition, boldness, and meanness. *Development and Psychopathology, 21*(3), 913–938. https://doi.org/10.1017/S0954579409000492

Poy, R., Segarra, P., Esteller, A., López, R., & Moltó, J. (2014). FFM description of the triarchic conceptualization of psychopathy in men and women. *Psychological Assessment, 26*(1), 69-76. https://doi.org/10.1037/a0034642.

Quintana, D. S., Alvares, G. A., & Heathers, J. A. (2016). Guidelines for reporting articles on psychiatry and heart rate variability (GRAPH): recommendations to advance research communication. *Translational Psychiatry, 6*, e803. https://doi.org/10.1038/tp.2016.73

Ribes-Guardiola, P., Poy, R., Segarra, P., Branchadell, V., & Moltó, J. (2020). Response perseveration and the triarchic model of psychopathy in an undergraduate sample. *Personality Disorders: Theory, Research, and Treatment, 11(1)*, 54–62. https://doi.org/10.1037/per0000371.

Sica, C., Perkins, E. R., Latzman, R. D., Caudek, C., Colpizzi, I., Bottesi, G., Caruso, M., Giulini, P., Cerea, S., & Patrick, C. J. (2021). Psychopathy and COVID-19: Triarchic model traits as predictors of disease-risk perceptions and emotional well-being during a

global pandemic. *Personality and Individual Differences*, 176, 110770. https://doi.org/10.1016/j.paid.2021.110770

Sleep, C. E., Weiss, B., Lynam, D. R., & Miller, J. D. (2019). An examination of the Triarchic Model of psychopathy's nomological network: A meta-analytic review. *Clinical Psychology Review, 71,* 1-26. https://doi.org/10.1016/j.cpr.2019.04.005

Tarvainen, M. P., Niskanen, J. P., Lipponen, J. A., Ranta-Aho, P. O., & Karjalainen, P. A. (2014). Kubios HRV–heart rate variability analysis software. *Computer Methods and Programs in Biomedicine*, *113*(1), 210–220. https://doi.org/10.1016/j.cmpb.2013.07.024

Thayer, J. F., Åhs, F., Fredrikson, M., Sollers III, J. J., & Wager, T. D. (2012). A meta-analysis of heart rate variability and neuroimaging studies: implications for heart rate variability as a marker of stress and health. *Neuroscience & Biobehavioral Reviews*, *36*(2), 747-756. https://doi.org/10.1016/j.neubiorev.2011.11.009

Thayer, J. F., & Lane, R. D. (2000). A model of neurovisceral integration in emotion regulation and dysregulation. *Journal of Affective Disorders*, *61*(3), 201-216. https://doi.org/10.1016/S0165-0327(00)00338-4

Thayer, J. F., & Lane, R. D. (2009). Claude Bernard and the heart–brain connection: Further elaboration of a model of neurovisceral integration. *Neuroscience & Biobehavioral Reviews, 33*(2), 81-88. https://doi.org/10.1016/j.neubiorev.2008.08.004

Venables, N., Hall, J., & Patrick, C. (2014). Differentiating psychopathy from antisocial personality disorder: A triarchic model perspective. *Psychological Medicine, 44*(5), 1005-1013. doi:10.1017/S003329171300161X

Yancey, J. R., Bowyer, C. B., Foell, J., Boot, W. R., & Patrick, C. J. (2019). Boldness moderates the effects of external threat on performance within a task-switching paradigm. *Journal of Experimental Psychology: Human Perception and Performance, 45*(6), 758-770. https://doi.org/10.1037/xhp0000631

Table 1. Descriptive Statistics and Pearson's zero-orden Correlations for Study Variables (left side), and Regression Coefficients Predicting Vagally-Mediated Resting Heart Rate Variability (lnHF) at Step 3 of the Hierarchical Regression Model (right side)

Variable	M	SD	1	2	3	4	5	6	7	Predictors	B	SE	*β*	t	*p*	95% CI [LL, UL]
										Intercept	8.648	0.823		10.510	**.000**	
1.ln (HF)	6.61	0.87	___							Gender	0.340	0.143	**.169**	2.377	**.018**	[0.029, 0.308]
2.Age	20.01	1.95	. 09	___						Age	0.003	0.026	.007	0.116	.907	[-0.108, 0.122]
3. BMI (kg/m^2)	22.88	3.92	.02	.05	___					BMI	0.024	0.013	.107	1.795	.074	[-0.010, 0.225]
4.EDR	0.25	0.05	**-.22**	**-.17**	**.14**	___				EDR	-3.145	1.114	**-.168**	-2.825	**.005**	[-0.286, -0.051]
5.HR (bpm)	78.02	12.23	**-.45**	-.13	.04	.10	___			HR	-0.033	0.004	**-.468**	-7.519	**.000**	[-0.591, -0.346]
6.Boldness	27.83	8.13	**.18**	.09	**-.13**	**-.18**	**-.13**	___		Boldness	0.014	0.006	**.127**	2.114	**.036**	[0.009, 0.246]
7.Meanness	11.52	7.01	.12	.02	.02	**-.19**	**-.13**	**.22**	___	Meanness	0.008	0.009	.067	0.907	.366	[-0.078, 0.211]
8.Disinhibition	17.66	7.98	**.13**	-.04	.03	-.11	**-.18**	.08	**.51**	Disinhibition	0.001	0.007	.012	0.182	.856	[-0.119, 0.143]

Significant associations are highlighted in bold (for values greater than |.16|, $p < .01$).
CI = confidence interval for β; LL = lower limit, UL= upper limit