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Heart Rate vs Stress Indicator for Short Term Mental Stress

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Authors' contributions

This work was carried out in collaboration between all authors. All authors read and approved the final manuscript.

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ABSTRACT

Heart rate variation (HR) being identified as depending on subjects' stress state when submitted to short term mental stress, this study aimed at analyzing whether or not it could be possible to find a mathematical relationship between the average heart rate variation and the intensity *S* of a stress indicator in case of short term mental stress, whatever the stress indicator is. The method consisted in working the hypothesis by gathering data providing HR and ratio of frequency power of HRV (Heart Rate Variability) for different level of stress, HRV being considered as a stress indicator and presenting the advantage of being widely used in studies, therefore providing numerous data in the literature. From this data, a mathematical model was designed and then assessed by testing its reliability when applied to HR variation versus different types of stress indicators (EMG, GSR, Work Load, questionnaires such as STAI-S, ALES). The correlation obtained between the model and the data provided by the literature (24 points from 8 studies

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gathering 272 subjects) gave r=.95 (p<.0001) which allowed us to validate the model. Limits of the model were identified and discussed.

Keywords: Heart rate; mental stress; stress factor; stress indicator.

1. INTRODUCTION

Many research domains have identified and studied mental stress as a crucial factor of influence or consequence: for example mental stress may affect performance [1] or social interaction [2-4], may cause or be a consequence of pathologies [5-11]; it is also studied when combined with tiredness [12] or related to sleepiness [13,14]. Different kinds of mental stress were identified, mainly chronic stress [15,16] and short term stress (or acute stress) [16-19].

Two ways have been developed for stress state assessment: subjective (through questionnaire; e.g. [20-25] or objective through physiological measurements; e.g. [16,18,26-30]. The former provides an "image" of the stress perceived by the subjects afterwards (subjective state) and the latter gives access to an instant state which is the subjects' physiological response to stress (physiological state). Both subjective and physiological states are subjects' responses to one or several stress factors (or stressors). We can therefore conclude that the subjects' stress response doubles in intensity from one context to another if the questionnaire score shows an increase from 20 to 40 or when the subjects' heart rate (HR) increases from 60 to 120bpm. In parallel, it is not always easy to rate how a stressor might double in intensity. For example, if we imagine a subject working in a noisy environment and find out that this subject is stressed due to the noise (see for example the analysis of job environment for nurses in [8] or for naval personals in [25]), what must be done to increase the intensity of the stressor? Must we increase the intensity of the noise (measured in dB), or must we increase the average frequency of the noise (measured in Hz) or both? The answer to this question is particularly difficult and is not available in the scientific literature. However, it is trivial to assume that one context is more stressful than another: a noisy environment with 100 dB is probably more stressful than an environment with 50 dB.

Nevertheless, in spite of many studies available about the effect of stress, none addresses the continuous variation of subjects' responses with quantified stress increase. Studies often compare two conditions of stress through stress indicators such as subjective or objective measurements.

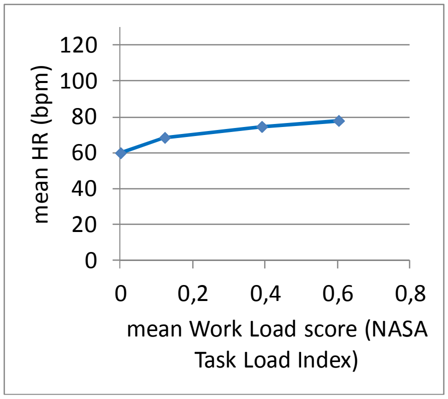
Yet, considering studies available in the literature, we found that healthy subjects' response in terms of average heart rate could present a typical shape and varied with increasing level of stress, stress being objectified by an indicator such as the difficulty of the context [31-33] or an indicator such as the workload [34] or the assessment of perceived stress [35]; the shape of the curve between the average heart rate HR and the stressor indicator S could be seen as $HR \propto S^a$ where a was a positive number smaller than 1 (see examples Fig. 1) in the case of healthy subjects under short term mental stress.

As the conditions of stress were different from one study to another, this finding let us think that, in case of healthy adult subjects submitted to short term mental stress, there could be a generalized relationship between the average heart rate considered as a physiological response to the stress, and this relationship being independent from the kind of stressor but depending on its intensity (Fig. 2).

The aim of the present article was to test/determine the hypothesis of pattern $HR \propto S^a$ ($0<a<1$) where S is a stress indicator linked to the stress intensity. Such a correlation was already assumed by Levy et al. [36: 1237] but did not give rise to a model.

When considering the available data in the literature, no measurements were reported for series of stressful contexts that might give information about the variation of HR versus stress intensity which was not surprising according to what is reported above.

Yet many studies gave the ratio of power frequencies for low vs high values of heart rate variability (ratio LF/HF of HRV) as a relevant and confident parameter to differentiate levels of stress state. This is obtained by analyzing fluctuations in beat-to-beat periods (HRV), then applying spectral analysis of HRV highlighting



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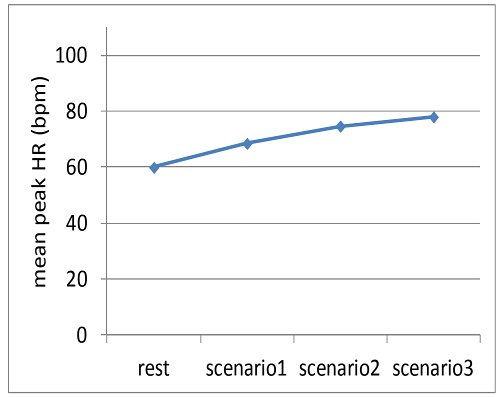

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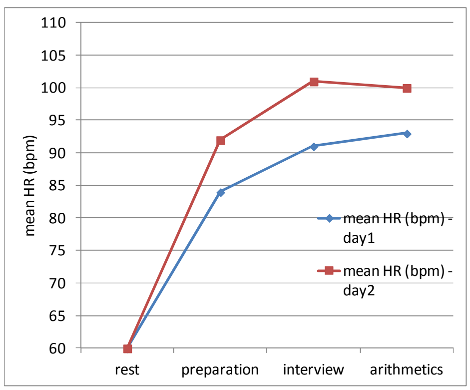

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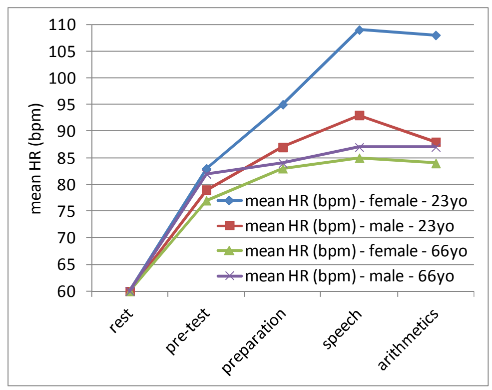

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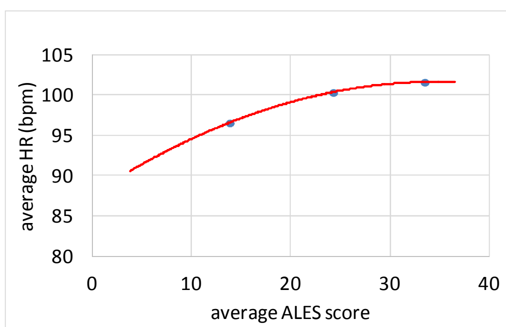

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Fig. 1. a) Cinaz et al. [34] for *N*=7 healthy adult subjects characterized stress through subjective assessment of the workload b) Spierer et al. [31] submitted *N*=3 healthy adult subjects to graduate levels of stress through different scenarii c) Petrowski et al. [32] submitted *N*=25 healthy adult subjects to graduate levels of stress through different situations d) Kudielka et al. [33] did the same for *N*=60 healthy adult subjects e) Berton et al. [35] assessed perception of stress using the Appraisal of Life Event Scale for *N*=20 healthy adult subjects involved in medical training simulation

several frequency bands among which [37] a low-frequency component reflecting sympathetic activity (usually referred to as LF: 0.04-0.15Hz) and a high-frequency component reflecting parasympathetic activity (usually referred to as HF; 0.15-0.4Hz). The ratio of LF to HF power illustrates the autonomic parasympathetic and sympathetic balance and is used as an indicator of stress [38-40]. Despite the fact that studies usually presented only two experimental conditions for comparative analysis of one stress state to another, by compiling data of different studies, it was here found possible to cover a large range of different stress states. We thus introduced a hypothesis of representativeness assuming that, in first approximation, the ratio LF/HF was directly representative of the stress intensity.

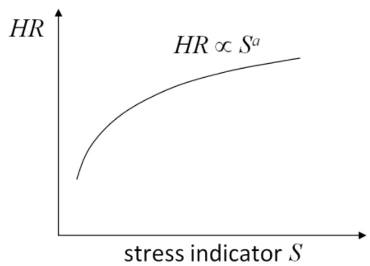

Fig. 2. Hypothesis regarding a model for HR variation with stress indicator for healthy adult subjects submitted to short term mental stress

Therefore the research question changed in: "Is it possible to find a mathematical relationship between the average heart rate variation and the intensity *S* of a stress indicator in case of short term mental stress, whatever the stress indicator is?" considering the indicator *S* as the ratio of power frequencies for low vs high values of heart rate variability (ratio LF/HF of HRV).

The present article aims at proposing an answer to this question by seeking the relationship and then verifying its consistency with various stress indicators within the restricted framework mentioned in the research question: subjects are healthy adults and stress is of mental and short term type. These two considerations are thus at the outset limits of the expected model.

2. MATERIALS AND METHODS

2.1 Design

The method consisted in working the hypothesis of pattern by gathering data providing HR and ratio of frequency power of HRV for different conditions of stress. The resulting correlation was then used to formulate a mathematical expression of the hypothesis of pattern shaping the model. The model was then assessed by testing its reliability when applied to HR variation versus different types of stress indicators. In case of success, this would validate the hypothesis of representativeness; if not, this would lead to rejecting both hypothesis (that of pattern and that of representativeness) and thus the model.

2.2 Working the Hypothesis of Pattern for the Model

The hypothesis of pattern was that a relationship of type $HR \propto S^a$ ($0<a<1$) might be found in specific conditions:

- With healthy subjects not to get any bias due to the distortion induced by any biological or mental disease,
- With adult subjects as it was shown that children could react differently than adults in certain conditions of stress [33],
- In case of mental stress only (not combined with physical stress obviously influencing the heart rate),
- In case of short term stress only (as opposed to chronic stress which relates to a different process).

These criteria were necessary to ensure the homogeneity of data as done by others (see for example [3]).

This first phase consisted in gathering articles of the literature providing several conditions of short term mental stress for samples of subjects with quantified data regarding the average heart rate and a stress indicator.

The choice of stress indicator was led by three criteria: i) being a relevant indicator of stress and thus being widely accepted by the scientific community, ii) being widely used in order provide data covering a large range of values through several studies, iii) providing data in accordance with the aforementioned specifications. This resulted in choosing the power ratio of heart rate variability as stress indicator (ratio LF/HF of HRV) as suggested in §1.

Then a power trendline was calculated in accordance with the assumption in §1 through a least square method with a correlation coefficient r_H calculated in a logarithm space and the associated significance p_H. We decided to reject the correlation if r_H <.8 or p_H >.001.

Although the period for the literature review was not limited, the aforementioned criteria and, after analysis of the papers available, the choice of

HRV as the relevant stress indicator bounded this period de facto.

2.3 Shaping the Model

Two hypotheses were combined. A first hypothesis of pattern consisted in assuming a general power variation of HR with a stress indicator *S*. A second hypothesis of representativeness assumed the ratio LF/HF to represent *S*, the equation of the power trendline obtained in the previous step being used to fix the power coefficient *a* of the expected model of type: $HR = kS^a + c$ with 0<*a*<1. Then, mathematical developments helped us to formulate an expression for the coefficient *k* of the model. For this aim, we used the properties of power functions when values tending to zero, and we combined the result with the fact that, when tending to zero, the power model could be approximated by a linear function as shown by some of the collected data. This helped us to provide an expression of the expected model and its adjustment key.

2.4 Subjects

Assessment of the model was done by considering studies in the literature providing each at least 3 pairs of values (*HR*; *S*) where *HR* was the value of the average heart rate for a sample of subjects submitted to a given stress condition characterized by *S*, a quantified stress indicator. This stress indicator was chosen as various as possible from one study to another. Hence *S* could be the score obtained with workload assessment or through stress questionnaires, or could be measurements of stress of muscles or skin conductivity for instance. For each study, the model was adjusted according to the protocol defined in the previous step. Finally, the set of calculated HR, HR_{model}, was compared to the measured HR provided by the studies, HR_{meas}. The model was rejected if the resulting correlation provided a correlation coefficient r_m <.8 or a related significance p_m >.001. Rejecting the model would show that one of the hypothesis (pattern or representativeness) was false.

3. RESULTS

3.1 Working the Hypothesis of Pattern for the Model

The choice of stress indicator that respected three criteria listed in §2.2 led to select the power ratio of heart rate variability as indicator. This quantity was a more widely used indicator and permitted to collect a large amount of data respecting the conditions specified for the data in §2.1.

Fig. 3 summarizes the data collected from the literature. Ten studies were used, involving 202 subjects and providing *N*=32 points. Among these studies, only those regarding healthy subjects were considered, implying sometimes to select only data of the control group of the study.

The trendline obtained confirmed a relationship between HR and the stress indicator of power type. The correlation obtained gave $r_H(N=32)=.88$ ($p_H<.0001$) which allowed us to accept it as a starting formulation of the model sought. The correlation given by the HR variation vs the power ration LF/HF of the HR variability helped us to suggest the expected model could be written as:

$$HR = kS^a + c \tag{1}$$

where:

- *a* = .2
- *k* is a coefficient to be calculated, depending on S
- *c* is a constant depending on *S*

3.2 Shaping the Model

We then considered two values of the heart rate, HR_1 and HR_2, which we expressed according to (1):

$$HR_1 = kS_1^a + c \tag{2}$$

$$HR_2 = kS_2^a + c \tag{3}$$

(2) – (3) gave:

$$\delta HR = k\{S_2^a - S_1^a\} = k\left\{(S_1 + \delta S)^a - S_1^a\right\} \tag{4}$$

$$= kS_1^a\left\{\left(1 + {\delta S}/{S_1}\right)^a - 1\right\} \tag{5}$$

where the symbol δ designated the infinitesimal difference of a quantity.

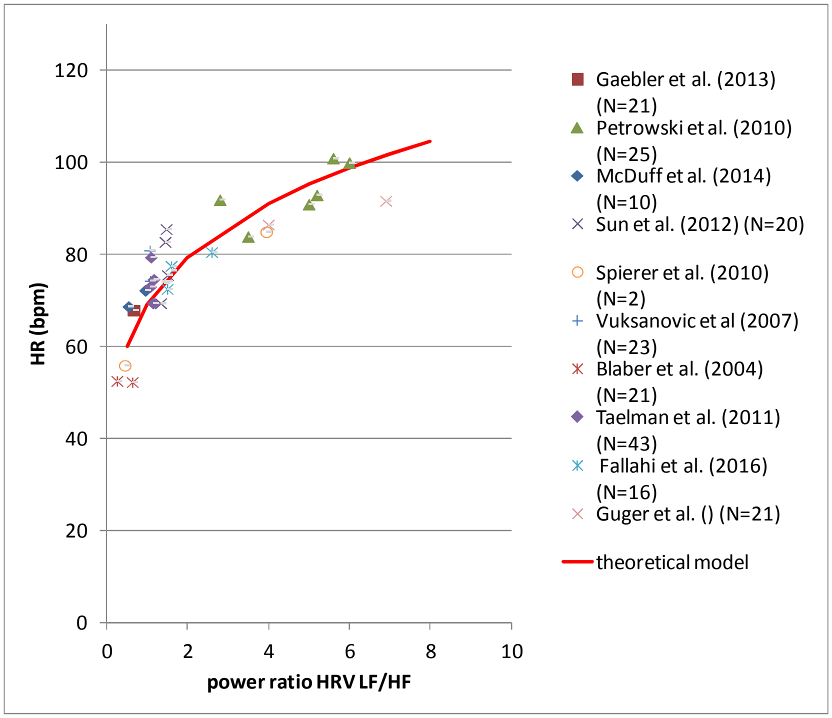

Fig. 3. Power trendline between HR and the HRV power ration LF/HF

Applying the Taylor expansion of the function $(1 \ + x)^m$ at the first order when x tends to 0:

$$(1 \ + x)^m = 1 + mx \tag{6}$$

Equation (5) became:

$$\delta HR \ = kS_1^a \left\{ \left(1 + a \, {}^{\delta S}/_{S_1} \right) \ - 1 \right\}$$

$$\delta HR \ = k \, a \, \delta S \, S_1^{(a-1)} \tag{7}$$

Besides, for S_i small, we considered that HR_i varied as a linear function of S_i:

$$HR_i = \alpha S_i \ + \beta \tag{8}$$

giving for HR_1 and HR_2:

$$HR_1 = \alpha S_1 \ + \beta \tag{9}$$

$$HR_2 = \alpha S_2 \ + \beta \tag{10}$$

(10) – (9) gave:

$$\delta HR \ = \alpha(S_2 \ - S_1 \ ) = \alpha \, \delta S \tag{11}$$

Making (7) and (11) equal, we obtained:

$$k \, a \, \delta S \, S_1^{(a-1)} = \alpha \, \delta S \tag{12}$$

giving the expression for k:

$$k \ = {}^{\alpha}/_{a \ S_1^{(a-1)}} \tag{13}$$

where:

- α is the slope of (8)
- a = .2 is the coefficient obtained through the correlation and used in (1)
- S_1 to be adjusted regarding the range of experimental data covered by the linear function (8). Further tests with experimental data led to consider that S_1 was to be adjusted to 15% of the range of experimental data covered by the linear function (8).

3.3 Assessing the Model

Eight studies were found in the literature respecting the criteria described in § 2.3 and providing 24 points spread into 8 samples of subjects (one per study) and gathering altogether 272 healthy adult subjects; when studies considered two cohorts of subjects, one made up of healthy subjects and the other made up of subjects with pathologies or psychological problems, only the healthy cohort was considered. Fig. 4 shows the measured values of HR provided by the studies vs the calculated values of HR applying the model obtained in §3.2. For each study, the legend on Fig. 4 shows information regarding the article from which was obtained the data, the number of subjects involved in the study and the stress indicator the researchers used to characterize the stress state of the subjects.

As seen on Fig. 4, the model shows a good consistency whilst being applying to six different stress indicators over eight different studies. These indicators were:

- The Work Load for Cinaz et al. [34] and Nahlinder [41], self-assessed by subjects through the NASA Task Load Index (TLX) [42],
- The stress of muscles measured through EMG for Fallali et al. [40] and for Lundberg et al. [43],
- The skin resistivity measured through GSR for Sun et al. [44],
- The stress state self-assessed through a multiquestionnaire developed by the authors for Lundberg et al. [43],
- The stress state self-assessed through the STAI-S of Spielberger [21] for Berger et al. [4] (the authors referred to [45] for this test),
- The Appraisal Life Event Scale for Berton et al. [35].

The correlation obtained on Fig. 4 for values of HR provided by the literature vs the calculated values of HR gave r_m (N =24)=.95 (p_m<.0001) which allowed us to validate the model and the hypothesis of representativeness.

4. DISCUSSION

It must first be noticed that it was rather difficult to gather data from the literature respecting all criteria listed in §2.

It is remarkable that, in this attempt to elaborate a model for heart rate variation for subjects under stress, this variation actually follows a power trendline which is generalized through a unique power coefficient *a* found here equal to .2 (validation of the hypothesis of pattern).

Yet this model has limits which reduce its domain of application. The first limits are given by the conditions for which it was set up; they are listed in section 2. Some of the limits were confirmed during the validation phase of the model: actually, the model applies for healthy adults; when subjects with disease are taken into account (for example mental pathologies like social anxiety disorder [3], panic disorder [32] or pathological disease like diabetes [46]), the model does not apply: data shows another trend to that of the curve. The model does not also apply when the

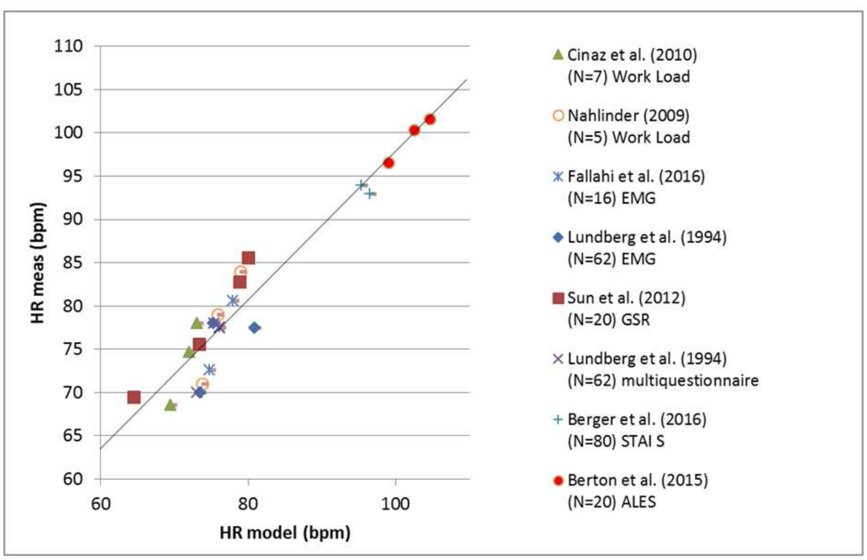

Fig. 4. Measured values of HR provided by the literature vs the calculated values of HR applying the model (r_m=.95; p_m<.0001)

stress process is disturbed: for example, Goodie et al. [47] presented subjects under stressful conditions, some of the subjects having a lower level of stress due to an instant feedback of their stress state; data related to these latter subjects with feedback did not match the model. Then come the limits due to the range over which the model was tested: It does not cover values of HR exceeding 101 bpm. Yet, we might assume that the model follows an asymptotic trend for higher values.

Another limit appeared during the assessment phase. It concerned the post-stress relaxation phase: Data related to this phase did not match the model [14,32,33,40,41,47]. Therefore, the present model is clearly exclusively devoted to the short term mental stress experienced without feedback of stress state for the subjects.

5. CONCLUSION

Our study validated a model describing short mental stress of healthy adult subjects through the expression of heart rate versus a stress indicator of the following form:

$$HR \ = kS^{a} + c \tag{14}$$

with:

$$k \ = {}^{\alpha}\!/_{a \ S_1^{(a-1)}} \tag{15}$$

where:

- *α* is the slope of the linear function linking *HR* and S_1 in the neighborhood of 0,
- *a* = .2
- S_1 adjusted to 15% of the range of experimental data covered by the linear function.

In practice, short term mental stress situations may be encountered in daily or professional life for example when experiencing a difficult interpersonal interaction, taking an exam, performing an unusual and/or difficult work activity (from the mental standpoint), dealing with an unexpected and surprising situation, being trained on simulator to manage a critical situation.

The model has shown a good reliability for short term mental stress of healthy adult subjects (p_m<.0001) when the situation did not involve subjects in physical efforts.

Further experiments are needed to investigate the limits of the model. It was said at the beginning of §4 that the model did not apply for subjects with diseases. However we may assume that a modified model (with another value of the power *a* for example) could fit data of patients concerned by a given disease or a family of diseases. Similarly, we might assume the same for patients experiencing two consecutive episodes of short term mental stress: in this study we noticed that our model only fitted data for the first episode of stress; we may assume that a modified model could describe the second one. Data available in the literature is not numerous enough to permit the elaboration of these models. This thus offers the perspective of several series of experiments to explore these assumptions. If modified forms of this model for HR response under mental stress could describe these particular cases, this might contribute to improving their understanding.

CONSENT

The present study is based on data from experiments published in the scientific literature for which the respective authors managed the subjects' informed consent when necessary.

ETHICAL APPROVAL

The present study is based on data from experiments published in the scientific literature for which the respective authors obtained ethical approval when necessary.

COMPETING INTERESTS

The authors have declared that no competing interests exist.

REFERENCES

1. Hayes JP, LaBar KS, Petty CM, McCarthy G, Morey RA. Alterations in the neural circuitry for emotion and attention associated with posttraumatic stress symptomatology. Psychiatry Research: Neuroimaging. 2009;172(1):7-15.
2. DeLongis A, Folkman S, Lazarus RS. The impact of daily stress on health and mood: Psychological and social resources as mediators. Journal of Personality and Social Psychology. 1988;54(3):486-495.
3. Gaebler M, Daniels JK, Lamke JP, Fydrich T, Walter H. Heart rate variability and its neural correlates during emotional face processing in social anxiety disorder. Biological Psychology. 2013;94(2):319-330.

4. Berger J, Heinrichs M, von Dawans B, Way BM, Chen FS. Cortisol modulates men's affiliative responses to acute social stress. Psychoneuroendocrinology. 2016; 63:1-9.
5. Al-Baldawi R. Migration-related stress and psychosomatic consequences. International Congress Series, Elsevier. 2002;1241:271-278.
6. Segerstrom SC, Miller GE. Psychological stress and the human immune system: A meta-analytic study of 30 years of inquiry. Psychological Bulletin. 2004;130(4):601.
7. Nakao M. Work-related stress and psychosomatic medicine. Bio Psycho Social Medicine. 2010;4(1):1-8.
8. Kane PP. Stress causing psychosomatic illness among nurses. Indian Journal of Occupational and Environmental Medicine. 2009;13(1):28-32.
9. Madhura S, Subramanya P, Balaram P. Job satisfaction, job stress and psychosomatic health problems in software professionals in India. Indian Journal of Occupational and Environmental Medicine. 2014;18(3):153.
10. Combs MA, Canu WH, Broman-Fulks JJ, Rocheleau CA, Nieman DC. Perceived stress and ADHD symptoms in adults. Journal of Attention Disorders. 2015;19(5): 425-434.
11. Dossmann R. Stress assessment in psychiatry: Interview vs questionnaire. Stress Self-assessment & Questionnaires - Choice, Application, Limits. 2015;51-55.
12. Costin A, Costin N, Cohen P, Eisenach C, Marchlinski F. Effect of exercise on heart-rate response to mental stress in teenagers. European Journal of Preventive Cardiology. 2013;20(4):593-596.
13. Woodward SH, Arsenault NJ, Voelker K, Nguyen T, Lynch J, Skultety K, Sheikh JI. Autonomic activation during sleep in posttraumatic stress disorder and panic: A mattress actigraphic study. Biological Psychiatry. 2009;66(1):41-46.
14. Cho HS, Kim YW, Park HW, Lee KH, Jeong BG, Kang YS, Park KS. The relationship between depressive symptoms among female workers and job stress and sleep quality. Annals of Occupational and Environmental Medicine. 2013;25(1):paper#12.
15. Maslova LN, Bulygina VV, Markel AL. Chronic stress during prepubertal development: Immediate and long -lasting effects on arterial blood pressure and anxiety-related behavior. Psychoneuroendocrinology. 2002;27:549-561.
16. Schubert C, Lambertz M, Nelesen RA, Bardwell W, Choi JB, Dimsdale JE. Effects of stress on heart rate complexity - A comparison between short-term and chronic stress. Biological Psychology. 2009;80:325-332.
17. Davezies PH. Stress et pouvoir d'agir: Données biologiques. Santé & Travail. 2008;64.

   Available:http://www.alternatives-economiques.fr/page.php?controller=article&action=htmlimpression&id_article=39496&id_parution=791
18. Montano N, Porta A, Cogliati CH, Costantino G, Tobaldini E, Rabello Casali K, Iellamo F. Heart rate variability explored in the frequency domain: A tool to investigate the link between heart and behavior. Neuroscience and Biobehavioral Reviews. 2009;33:71-80.
19. Keitel A, Ringle M, Schwartges I, Weik U, Picker O, Stockhorst U, Deinzer R. Endocrine and psychological stress responses in a simulated emergency situation. Psychoneuroendocrinology. 2011; 36:98-108.
20. Cohen S, Kamarck T, Mermelstein R. A global measure of perceived stress. Journal of Health and Social Behavior. 1983;24:385-396.
21. Spielberger CD. Manual for the state-trait anxiety inventory. Consulting Psychologist Press Inc., Palo Alto, California; 1983.
22. Karasek RA, Theorell R. Healthy work, stress, productivity and the reconstruction of working life, the free press. Basic books: New York; 1990.
23. Jehel L, Paterniti S, Brunet A, Louville P, Guelfi J. L'intensité de la détresse péritraumatique prédit la survenue des symptômes post-traumatiques parmi des victimes d'agressions. L'Encéphale. 2006; 1(32):953-956.
24. Kilminster S, Bridger RS. Modified work and well-being questionnaire for cohort study of stress in the naval service: Psychometric validation. Unpublished MoD Report; 2007.
25. Bridger RS, Brasher K, Kilminster SAD. Job stressors in naval personnel serving

on ships and in personnel serving ashore over a twelve month period. Applied Ergonomics. 2011;42:710-718.

26. Steptoe A. Stress, social support and cardiovascular activity over the working day. International Journal of Psychophysiology. 2000;37:299-308.

27. Fairclough S, Houston K. A metabolic measure of mental effort. Biological Psychology. 2004;66:177-190.

28. Müller M, Hänsel M, Fichtner A, Hardt F, Weber S, Kirschbaum C, Rüder S, Walcher F, Koch T, Eich Ch. Excellence in performance and stress reduction during two different full scale simulator training courses: A pilot study. Resuscitation. 2009; 80(8):919-924.

29. Rohleder N, Nater U. Determinants of salivary a-amylase in humans and methodological considerations. Psychoneuroendocrinology. 2009;34:469-485.

30. Bailon R, Mainardi L, Orini M, Sörnmo L, Laguna P. Analysis of heart rate variability during exercise stress testing using respiratory information. Biomedical Signal Processing and Control. 2010;5:299-310.

31. Spierer D, Griffiths E, Sterland T. Fight or flight: Measuring and understanding human stress response in tactical situations. The Tactical Edge, Summer. 2009;30-40.

32. Petrowski K, Herold U, Joraschky P, Mück-Weymann M, Siepmann M. The effects of psychosocial stress on heart rate variability in panic disorder. German Journal of Psychiatry. 2010;13(2):66-73.

33. Kudielka BM, Buske-Kirschbaum A, Hellhammer DH, Kirschbaum C. Differential heart rate reactivity and recovery after psychosocial stress (TSST) in healthy children, younger adults, and elderly adults: The impact of age and gender. International Journal of Behavioral Medicine. 2004;11(2):116-121.

34. Cinaz B, La Marca R, Arnrich B, Tröster G. Monitoring of mental workload levels. In Proceedings of IADIS eHealth Conference. 2010;189-193.

35. Berton J, Rinau E, Conte M. Appraisal of life event scale (ALES) to differentiate mental stress perceived by physicians and nurses involved in the same simulation Training Situation of Anesthesiology, Stress Self-assessment & Questionnaires - Choice, Application, Limits. 2015;39-43.

36. Levy WC, Cerqueira MD, Harp GD, Johannessen KA, Abrass IB, Schwartz RS, Stratton JR. Effect of endurance exercise training on heart rate variability at rest in healthy young and older men. The American Journal of Cardiology. 1998; 82(10):1236-1241.

37. ESC-European Society of Cardiology and the North American Society of Pacing and Electrophysiology. Heart rate variability: Standards of measurement, physiological nterpretation and clinical use. Circulation. 1996;93:1043-1065.

38. Malik M. (Task force of the European society of cardiology). Heart rate variability standards of measurement, physiological interpretation, and clinical use. Eur Heart J. 1996;17:354-381.

39. McDuff D, Gontarek S, Picard R. Remote measurement of cognitive stress via heart rate variability. In Engineering in Medicine and Biology Society (EMBC), 36th Annual International Conference of the IEEE. 2014; 2957-2960.

40. Fallahi M, Motamedzade M, Heidarimoghadam R, Soltanian AR, Miyake S. Effects of mental workload on physiological and subjective responses during traffic density monitoring: A field study. Applied Ergonomics. 2016;52:95-103.

41. Nählinder S. Flight simulator training: Assessing the potential. Department of Management and Engineering, Linköpings Universitet; 2009.

42. Hart SG, Stavenland LE. Development of NASA-TLX (Task Load Index): Results of empirical and theoretical research. Chap. 7, In Hancock PA, Meshkati N, editors, Human Mental Workload. Elsevier; 1988; 139-183.

43. Lundberg U, Kadefors R, Melin B, Palmerud G, Hassmén P, Engström M, Dohns IE. Psychophysiological stress and EMG activity of the trapezius muscle. International Journal of Behavioral Medicine. 1994;1(4):354-370.

44. Sun FT, Kuo C, Cheng HT, Buthpitiya S, Collins P, Griss M. Activity-aware mental stress detection using physiological sensors. In Zerfos P, Montanari R, Phan TH, editors. Mobile computing, applications, and services. Springer Berlin Heidelberg. 2010;211-230.

45. Laux L, Glanzmann P, Schaffner P, Spielberger CD. State-trait-angstinventar. Beltz, Weinheim, Germany; 1981.
46. Vandeput S. Heart rate variability: Linear and non linear analysis with applications in human psychology. PhD Dissertation, Leuven Un.; 2010.
47. Goodie JL, Larkin KT. Changes in hemodynamic response to mental stress with heart rate feedback training. Applied Psychophysiology and Biofeedback. 2001; 26(4):293-309.

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