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Psychophysiological Cardiovascular Functioning in Hostile Defensive Women

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1. Introduction

Cardiovascular diseases are the leading causes of death and disability in the world, in both developed and developing countries, and also in both sexes. In fact one third of annual deaths worldwide are due to cardiovascular problems, according to the WHO (World Health Organization) estimated 17.3 million people died from CVDs in 2008, over 80% of CVD deaths take place in low- and middle-income countries, and by 2030, almost 23.6 million people will die from CVDs (in http://www.who.int/cardiovascular_diseases). Therefore, is a serious problem, and not only in industrialized countries, indeed, is an epidemic that not only continues but it is precisely in the developing countries, where it currently is increasing dramatically. On the other hand, prevalence and mortality from these diseases among *women* has increased in an exaggerated way. This for several reasons: first, as mentioned, in women the death rate from CVDs has increased significantly, equaling or exceeding that of the male population, so we think it is of great importance to focus on studies considering this sector only a few risk factors have also increased; second, the sample with which we had consisted mostly of women, given the characteristics of it (psychology undergraduates), which were removed the few men who participated in the study. Thus, from these data and indications, and since most studies have focused on people of both sexes or only male, we considered appropriate to carry out research with a sample of only women.

Moreover the etiology of CVD is multidimensional, that is, factors involving genetic, physiological, chemical, nutritional, environmental and psychosocial, and moreover, is not fully known. It is known that a number of cardiovascular risk factors that may contribute to the development, progression or maintenance of CVD, called "classic risk factors”. Among the most important are: age, sex, cholesterol, hypertension, smoking, physical inactivity and obesity. However, surprisingly, these factors fail to explain more than 50% of the variance in predicting cardiovascular risk, whether considered independently or when considered together (Chesney, 1996; Gump & Matthews, 1999). Nevertheless a large proportion of CVDs are preventable, but they continue to increase mainly because preventive measures are inadequate.

Therefore, research about this topic has been long and extensive, especially directed to seek and discover other factors, beyond the "classics", are also contributing to the development

CVD (Brydon et al., 2010; Chida & Steptoe, 2009; Everson-Rose & Lewis, 2005; Jorgensen & Kolodziej, 2007; Vella & Friedman, 2009). We refer to *psychosocial factors*. Thus, the relationship between different psychosocial variables and the risk of CVD has been studied extensively since the mid past century, mainly for these two reasons.

2. Psychosocial risk factors and cardiovascular disease

In the search and exploration for other risk factors that may explain the etiology of CVD, psychosocial factors have gained importance to such an extent that research has been able to explain the mechanisms of action of these variables on CVD. The results obtained in various research studies have confirmed the relationship between psychosocial factors and the atheromatous plaque, which constitutes the basic injury occurring in CVD (Kaplan, et al, 1983; Kaplan, et al, 1987; Manuck, et al, 1983; Manuck, Kaplan & Matthews, 1986; Manuck, et al, 1989; Jennings, et al, 2004; Vale, 2005). The mechanisms involved in its formation, which are mechanical and chemical factors are seriously affected by psychosocial processes, and especially by the stress response. In these processes, emotions cause a faster heart rate and higher blood pressure, leading to increased blood flow and turbulence. In addition, there is a mobilization of lipids which exceeds the body's metabolic requirements, and which facilitates aggregation to artery walls and heart tissue. This relationship between psychosocial factors and CVD has received the generic name of "Hypothesis of the cardiovascular reactivity", and has been supported by various prospective studies (Keys & Taylor, 1971; Schiffer, et al, 1976; Manuck, et al, 1992; Steptoe, et al, 2000).

To date, research has shown that individuals who tend to display strong responses and reactivity are at increased risk of CVD (Manuck et al., 1992; Palmero et al., 2006; Treiber, et al, 2003; Matthews, et al, 2006). The argument that defends this refers to the stereotype response: if the cardiovascular reactivity is a characteristic of an individual and is physiological stable and consistent, then the same response patterns will be seen every time the individual is faced with a situation of stress. Evidently with certain limitations, laboratory situations can be regarded as a procedure that provides information on an individual's physiological functioning in real life (Allen, et al, 1987; Allen & Matthews, 1997; Palmero, et al, 2002; Moseley & Linden, 2006; Palmero, et al, 2007). Thus individuals, whose pattern of cardiovascular functioning is characterized by the expression of exaggerated responses, are those who, with time, are likely to experience some cardiovascular dysfunction (Everson, et al, 1996; Markovitz, et al, 1998; Strike, et al, 2003). Given this relationship between excessive cardiovascular response and CVD, research efforts have focused on finding any variable causing an increase in such responses as this will mean an increase in the likelihood of suffering from one of these diseases.

From the study on the classic Type A behavior pattern through the anger-hostility complex and hostility were conducted, in recent years, research has focused on the *defensive hostility* (high hostility and high defensiveness) as a risk factor in CVD (Guerrero & Palmero, 2010; Helmers & Krantz, 1996; Jamner, et al, 1991; Larson & Langer, 1997; Shapiro, et al, 1995; Palmero, et al, 2007; Vella & Friedman, 2007). The trait of defensive hostility reflects an approach–avoid conflict between the desire for social approval and distrust of those who can provide such support, and currently can be considered as one of the psychosocial factors with more weight and empirical support in its relationship with CVD.

Several studies of CVD patients demonstrate that subjects with high scores in defensive hostility show higher rates of ischemia during a mental stress situation, greater damage by infusion and a longer duration of ischemia during daily activities (Helmers, et al, 1995). As well, a field study conducted with paramedics showed a higher cardiac response by people with a high hostility defensive when they deal with stress situations (Jamner, et al, 1991). These results, usually obtained from real situations, appear to be supported by laboratory studies (Jorgensen, et al, 1995; Shapiro, et al, 1995; Helmers & Krantz, 1996; Larson & Langer, 1997; Palmero et al., 2002; Palmero et al., 2007), which indicates the existence of a subgroup of people who are characterized by high "Defensive Hostility" (DH), as well as a greater cardiovascular response. In general, DH individuals show greater cardiovascular response during the task phase that other groups can be formed when combining hostility and defensiveness variables (Larson & Langer, 1997). And, unlike this group, find another subgroup which is characterized by low hostility and low defensiveness and show a lower cardiovascular response -low risk group-.

However as these studies show, various inconsistencies were also found, which originated, at least in part, from the different tasks used to measure the cardiovascular variables. These results suggest the relevance of broadening the research spectrum whose aim it is to strengthen the association between psychological variables and cardiovascular response from the understanding that the exaggerated response would be the link between these and CVD. In other words, it seems appropriate to establish whether defensive hostility can be seen as the toxic component in relation to CVD.

Therefore, the *general objective* pursued with this study refers to the delimitation of the effects of defensive hostility on cardiovascular response in women, in a real stress situation. Specifically, exploring the relationship between defensive hostility, which is a better predictor than hostility alone, and cardiovascular responses (HR, SBP and DBP) in this tonic dimension. That is, considering all three phases of the experiment (adaptation: A, task: T and recovery: R) to establish the significance of the functional psychophysiological profiles in the four experimental groups (DH: high hostility-high defensiveness, HH: high hostility-low defensiveness, Def: low hostility-high defensiveness, LH: low hostility-low defensiveness).

From these premises and the proposed main objective has been carried out this research with the ultimate aim to contribute both to the development of the theoretical basis on psychosocial variables that can be considered as cardiovascular risk factors and its subsequent application in clinical practice as well as contribute to methodological development in the field of psychophysiological research.

The *general hypothesis* suggests that individuals with high scores in defensive hostility display the highest values with the psychophysiological variables. Specifically, we expect that these individuals show the greatest average values recorded in the variables (HR, SBP and DBP), and in all the phases of the experiment (A, T and R), compared to those shown by the individuals of the other three groups. In addition, DH group will be characterized by less adaptive psychophysiological profile.

3. Empirical study

3.1 Study design

In this section we show data from a recent study published by the authors (Guerrero and Palmero, 2010). One hundred and thirty female students from a *Universitat Jaume I* participated in this research. The mean age of the participants was 20.34 years (*SD*=2.06). The criteria to form the groups were the scores obtained with both the Ho Inventory (Cook & Medley, 1954) and the Social Desirability Questionnaire (Crowne & Marlowe, 1960). We used the median as the cut-off point to classify participants as "high" or "low" in each variable, thus the sample was composed as follows: 30 Defensive Hostility (DH), 40 High Hostility (HH), 42 Defensive (Def), and 18 Low Hostility (LH).

3.2 Instrumentation

Cardiovascular responses were measured with the registration system Biopac MP150 with NIBP module 100A, both were connected to a personal computer to monitor and store all the responses. Specifically, this registration system was used to measure the physiological responses: heart rate, systolic blood pressure and diastolic blood pressure. Also, this system recorded these cardiovascular parameters continuously and noninvasively.

Hostility was measured with the Hostility Inventory of Cook and Medley (1954), specifically the Composite Hostility Score (Chost) consisting in three subscales: cynicism, hostile feelings and aggressive responses. In previous studies, this information led to a scale being provided with a greater ability to predict the response and cardiovascular reactivity in comparison with the Ho scale provided as a whole (Barefoot et al., 1989; Christensen & Smith, 1993; Guerrero, 2008).

Defensiveness was measured with the Spanish version (Ávila & Tomé, 1989) of the Social Desirability Questionnaire of Crowne and Marlowe (1960). It consists of 33 items of choice alternatives (true or false) that reflect socially desirable behaviors and cognitions.

Also, reports and self-reports were also used to collect some data on behavioral habits related to health issues, and various personal and socio-demographic data.

3.3 Study procedure

A real academic exam was used as a situation of stress. More specifically, we used an exam of the degree of Psychology; this situation represents a real mental stress task for students.

Data were collected individually in one session. Following informed consent, each subject completed a questionnaire of demographics, previous medical history and noted any medication. Then, they went into the experimental cabin where they were asked to sit comfortably in an armchair and a sensor was connected to their non dominant wrist. From this time onward, both instructions and exam questions were submitted through the projector.

Following this registration session, it was necessary to remove the sensors and to go to another room to complete the corresponding scales. Finally, they were thanked for their collaboration and left.

The recording session consisted in three phases: adaptation (A), task (T) and recovery (R), with duration of 10, 20 and 10 minutes, respectively.

a. *Adaptation phase* (10 min): there was no stimulus. The purpose of this period was for participants to become familiar with the environment. The psychophysiological variables were recorded in their tonic dimension to establish baseline levels with the aim of obtaining the participants' usual levels under rest conditions.
b. *Task phase* (20 min): the 20 stimuli that formed the experimental task were presented: an objective test of 20 questions with four alternative answers. The stimuli were separated by a one-minute period, and the duration of this phase was therefore 20 minutes. This phase was considered an overall stressful period, and its variables were recorded in their phasic dimension.
c. *Recovery phase* (10 min): no stimulus was presented. The variables were considered in their tonic dimension to see how these variables recovered their usual levels after the stress situation. These data allow us to ascertain how long the organism needs to achieve its usual values following a situation of stress. This is extremely relevant when considering the consequences caused by the stress situation from a neuroendocrinological viewpoint because the greater the time needed by the organism to recover its baseline levels, the greater the exposure to the effects of substances released by the organism because of this situation of stress (catecolamnines and cortisol).

3.4 Statistical analysis

The first approaches were the descriptive analysis and correlations, and an analysis of variance (ANOVA) was carried out for a more detailed analysis of the results. Then, according to the main objective, that of analyzing the relationship between defensive hostility and the cardiovascular responses to study the functional significance of each group's profiles during all three phases, namely in their tonic dimension, an ANOVA was carried out whose design was 4 groups (DH, HH, LH and Def) x 3 phases (adaptation, task and recovery) with repeated measures for the phase variable. Subsequently, a univariate analysis of variance has been conducted for each phase to obtain a more accurate description of the potential differences encountered should such differences be given from the post hoc Tukey test with which the groups involved in them will be determined.

3.5 Study results

Data will be presented in the following figures, which reflect the psychophysiological profiles obtained from analysis of data for each physiological variable separately.

Although, before that, we like to refer to the profiles described by Kelsey (1993) on which we relied. There are different ways to respond physiologically to stressful events, which depends on external factors (situational) and internal factors (personality variables). In this regard, and from the classic proposal Kelsey, we noted three response patterns, which reflect corresponding profiles associated with different forms of reaction to stressful events: *habituation, sensitization* and support or *constant*.

- Habituation.

When you perceive a situation as potentially threatening or novel, occurs an increase in cardiovascular reactivity. After an exposure time in such a situation, and after that initial increase, there is a phenomenon of habituation, during which we see a progressive decrease of the initial values. This phenomenon is considered essential in the process of adaptation and regulation of humans and lower animals, demonstrating the ability to self-regulatory organism, that is, it can be activate to deal with a potentially dangerous or threatening situation and, in turn, is able to return to baseline (BL) once the situation has gone or is under control.

- Sensitization.

In this case, the individual responds to an agent or stressor stimulus with a high cardiovascular reactivity (the phenomenon of sensitization), similar to the previous pattern but without habituation occur. Instead, it produces a progressive increase in reactivity over the situation. It shows a organism's inability to return to baseline, which is highly detrimental to the heart muscle and overall health.

- Constant.

In this third pattern occurs an initial increase, similar to that of the other two patterns, but no habituation or sensitization occurs. This increase remains constant throughout the stress. Thus, this pattern is also maladaptive; since there is no preparation behavior is essential in the adaptation to the environment (Cannon, 1932) or has the ability to return to baseline levels.

From these profiles is observed that only the first is adaptive, decreasing when the individual cardiovascular reactivity to stressful events faced long periods of time. On the one hand, the initial increase in activation allows for better coping with the situation and the subsequent decline after a period of time, it is necessary to avoid damaging the body and maintain homeostasis (Palmero, Breva, et al., 1994; Palmero, Espinosa et al., 1995).

Concerning *heart rate* in Figure 1 shows the obtained profiles by different groups.

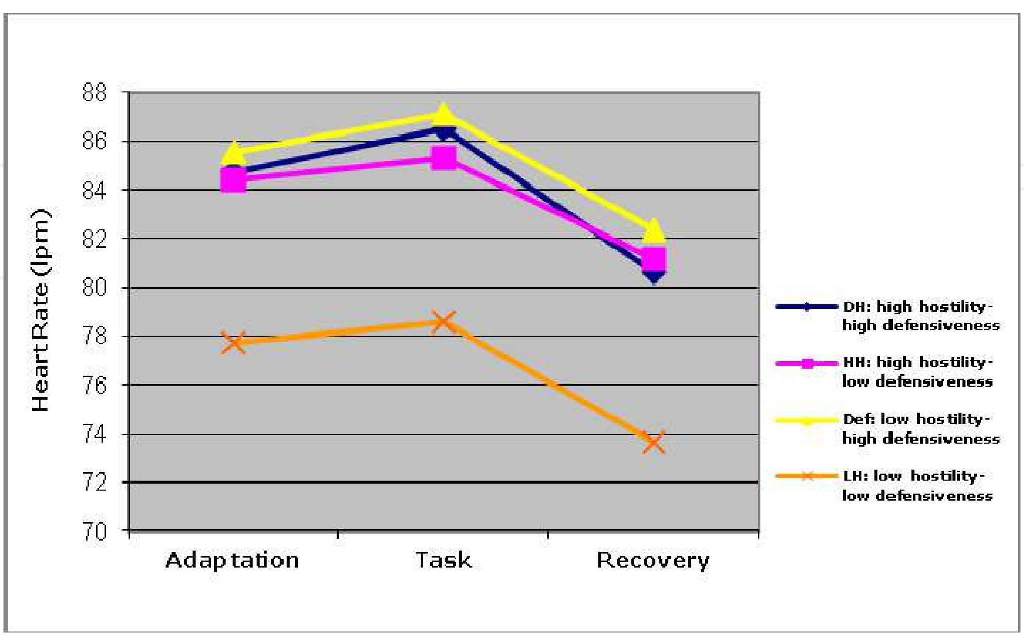


Fig. 1. Heart rate and defensive hostility during three phases.

As for the functional significance of the various profiles, as seen in Figure 1, all groups show the habituation trend, so profiles are adaptive. Although, LH group shows the more adaptive pattern, as it presents a greater and faster recovery to their baseline levels.

Concerning to *systolic blood pressure* as seen in Figure 2, all groups show the habituation trend. Again, LH group presents a more adaptive pattern, with lower values in the recovery phase.

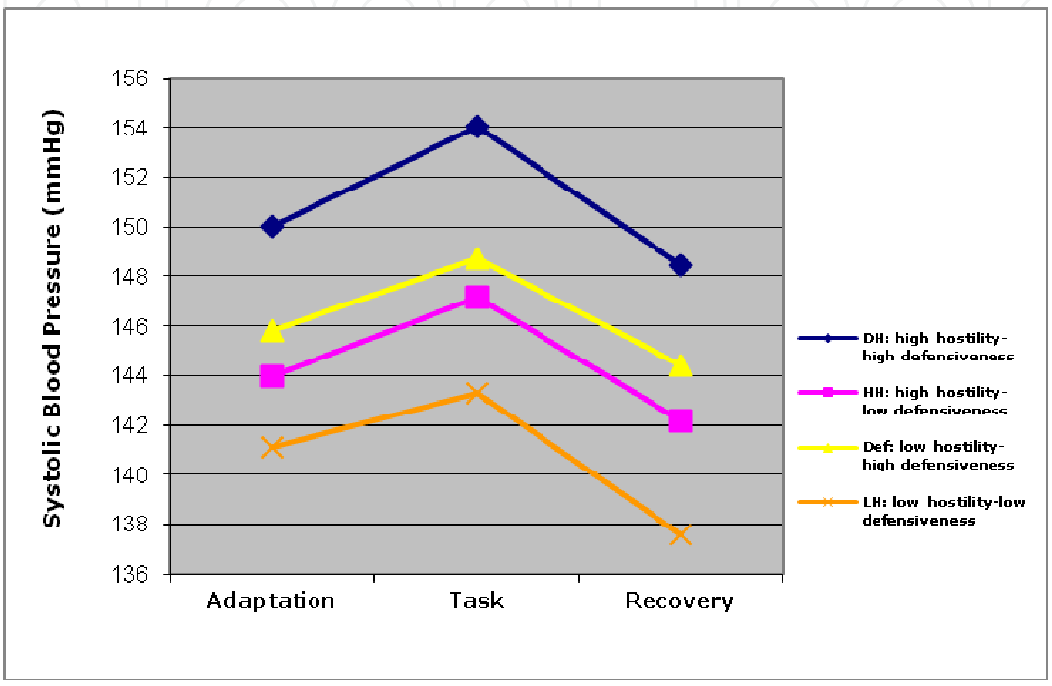


Fig. 2. Systolic blood pressure and defensive hostility during three phases.

Here we also find that DH is the group that obtained the greatest values in systolic pressure in all three phases.

An additional interesting fact is the effect of variable defensiveness, although the difference was not statistically significant, we see that the two groups with high defensiveness, DH and Def, presented the higher SBP values in all three phases (A, T and R)

Concerning *diastolic blood* pressure as seen in Figure 3, all groups show the habituation trend, and again LH group presents a more adaptive pattern.

Again, we see that DH is the group that shows the highest values in all three phases. In this case also notes the effect of variable defensiveness, although neither are statistically significant differences. Two groups with high defensiveness, DH and Def, presented the higher DBP values, but only in adaptation and task phases.

Figure 2 and 3 demonstrate clear differences between SBP and DBP between the psychophysiological patters of extreme groups: DH presents higher values than LH group. And the other two groups are in an intermediate position, with values very similar among them and throughout the three phases.

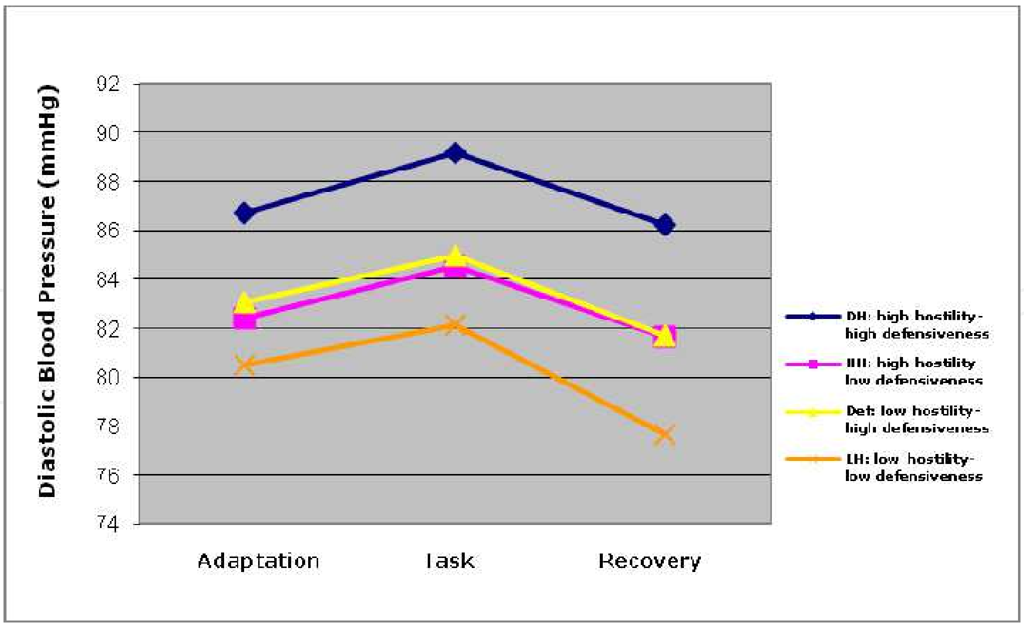


Fig. 3. Diastolic blood pressure and defensive hostility during three phases.

4. Conclusion

In general, the DH group presented higher values for the cardiovascular variables during the three phases, as well as a slow recovery. Thus, we believe that *defensive hostility* has proved to be a more appropriate criterion than hostility alone when determining the possible risk of cardiovascular dysfunction.

So, consistent with our main objective, and in relation to establishing the functional significance of the general profiles in the four groups through considering the three experiment phases, our results are in the expected direction. Specifically, the DH group obtains the highest values in the three phases and variables, particularly in the blood pressure index. Additionally, extending the findings available to therefore include the recovery phase, we have found that this group of individuals takes longer to recover after a stress situation, that is, they score the highest values encountered during the recovery phase.

About the *cardiovascular parameters*, the three most commonly used in these studies were HR, SBP and DBP (Swain & Suls, 1996), to combine the criteria and to enhance the comparison between different results from various research studies. All the parameters have been captured, recorded, stored and analyzed in a highly reliable, non invasive and continuous manner, which is especially relevant in the case of blood pressure. They are easily accessible and provide us with highly reliable information on cardiac and vascular functioning over different time periods, that is, to reflect the changing psychophysiology profile of individuals at all times.

Among these parameters, we note blood pressure reflects the greatest differences between the two extreme groups (DH and LH): introducing a greater response, activation and recovery for the DH group. On the other hand, HR reveals a clear difference in the lowest values submitted by the low-risk group (LH) compared to a more homogeneous response from the other three groups in all the phases. So, we suggest that the cardiovascular functioning of hostile defensive individuals in such situations is best reflected through blood pressure, specifically through diastolic pressure, an index which seems to appear truer for the recovery phase, as reflected by the significant differences found between the two extreme groups.

More specifically, showing each cardiovascular physiological variable separately, the following was observed.

Regarding the *HR*, although there is no effect of interaction between hostility and defensiveness, since the group HD presents values very similar to those groups HH and Def, HR shows how the low-risk group, LH , presents the lowest values along the three phases compared to the other groups.

We must also emphasize for the three groups with higher levels in HR, which is the group Def -although differences are not statistically significant- which presents the highest levels in all three phases, reflecting a major effect of the variable defensiveness, specifically in the adaptation and task phases where groups with high defensiveness (DH and Def) show the highest levels.

Our data also specifies that it is in the recovery phase where there are statistically significant differences between the Def group and the LH group.

Regarding the *SBP*, beside show the effect of interaction between hostility and defensiveness, a fact that reflects the important values of the DH group, the main effect of the defensiveness variable also exist in three phases, which means that subjects with high scores in defensiveness obtain higher values than subjects with low scores in defensiveness.

In addition, our data indicate that statistically significant differences that exist between DH and LH groups are specifically at the task and the recovery phases, in turn, indicates that there are no differences between groups with respect to their baseline levels.

The fact that the DH group presents higher scores for these values, leads us to suggest that defensive hostility rather than hostility alone better predicts the cardiovascular function in situations of stress.

Regarding the *DBP*, it seems that the real meaning of this variable can be seen only with the consideration of defensive hostility, such as denoting the effects of interaction in all three phases. Like the previous case, our data indicate that statistically significant differences also exist between DH and Def groups, but in this case appear in the recovery phase. There was also a main effect of the defensiveness variable, but is almost imperceptible.

Regarding the *functionality of the profiles* all four groups in three variables were adaptive. But in blood pressure case, it seems interesting to note that while the four groups that tend to indicate profiles of habituation, the fact that the individuals' defensive hostile obtains these statistically significant values during the recovery phase means that this group of individuals takes significantly longer to return to the baseline values of situations without stress. Thus, the profile of hostile defensive subjects would be less adaptive, because in this group the recovery is more slow and gradual. The profiles of the three remaining groups are adaptive, with a faster recovery.

Specifically, in our view, while DBP continues the pattern of other variables during the task, and displays an interaction effect between hostility and defensiveness, it is the most important variable to detect the cardiovascular functioning of hostile defensive individuals in the recovery phase. Once again, these arguments lead us to suggest that it is desirable to consider this variable, along with the inclusion of the recovery phase.

In short, along with the results corresponding to the tonic dimension of the cardiovascular variables being studied, it appears that *defensive hostility* is more appropriate than hostility alone to understand cardiovascular functioning in stressful situations. So, defensive hostility identifies a dimension of personality that, ultimately, would be a better predictor of the cardiovascular response in particular and of cardiovascular disease in general. It would be more fitting than hostility alone to explain and understand cardiovascular functioning in stressful situations.

The DH group, which shows that those individuals with high scores in hostility and high scores in defensiveness, are those who reflect the highest values in activation and cardiovascular response, but only when faced with the demands of a challenging task. For this reason, and as we have pointed out, it seems appropriate to use a real situation of stress as an experimental task because this specific environment is the best scenario to see the psychophysiological response style that characterizes the individuals being studied. In addition, if the situation of stress is sufficiently long, there is also the possibility of locating the adjustment mechanism of individuals to the sustained demands of this stressful activity. Those hostile individuals seeking to perform an action that is not disagreeable to others display the greatest difficulty in controlling their hostile experiences. The result of this inability to properly monitor hostility experiences produces a sustained increase in the activation of the sympathetic system which, in turn, gives rise to significant increases in the cardiovascular variables studied: HR, SBP and DBP. One particular sensitivity of the SBP is to capture this sympathetic activation which suggests its suitability differential in such studies.

Thus, these findings show the relationship between defensive hostility and cardiovascular functioning in situations of stress by the various cardiovascular register indexes (HR, SBP and DBP) and by considering the various parameters analyzed, namely response, activation and cardiovascular recovery, have been demonstrated. As mentioned above, we believe that defensive hostility has proved to be a more appropriate criterion than hostility alone when determining the possible risk of cardiovascular dysfunction.

Regarding the three *experimental phases*, we can state the following. About the *recovery phase*, we think that its inclusion in the experimental research laboratory is especially important since it provides vital information on restoring physiological parameters. This phase is a basic and essential element in the detection of the possible risks of future dysfunctions (Guerrero & Palmero, 2006). As seen, the recovery phase profile in the DH group has shown a slower recovery, especially in terms of blood pressure, and more specifically in DBP. Thus, the inclusion of the recovery phase in this type of experimental laboratory, it constitutes a basic and essential element in the detection of possible risks of future dysfunctions. From a neuroendocrinological viewpoint, it is important to consider the consequences caused by the situation since the more time the organism needs to recover the baseline levels, the greater the exposure to the effects of the substances released (catecholamines and cortisol).

About the *task phase*, by considering the *duration* parameter, which is scarcely taken into account in such research, we believe it appropriate to propose an experimental task that is long enough to establish a genuine cardiovascular functioning, that is more likely to be correct, by appreciating how the adjustment to a stressful situation is produced, or not. The fact that short or moderately long tasks have been systematically used may have masked the dysfunctional connotations of the response profiles in the different groups.

Regarding the *tasks* used as a stressful situation in the laboratory, the importance of creating and using a type of task that involves a real stress situation must be highlighted, that is, one as close as possible to everyday situations. Thus, one can understand some of the inconsistencies found which, in turn, provide the ecological laboratory experiments in this field with more validity. In this respect, we believe that the task used in this research, a real exam, is a task with connotations of personal and social threats, which also requires investing considerable effort to be able to deal with it actively and successfully, and this has been reflected by the significant differences obtained in the three experiment phases.

Concluding, in our modest opinion, with this and other research we have conducted we provide more empirical support about the great relevance within the theoretical framework on DH as a possible psychosocial cardiovascular risk factor also in women. However, as a future research direction, probable variability among females as compared to males necessitates concentrated research in this area, and we recognize the need for separate data analysis for males. Also, the study should be replicate with other samples to see if there are similar results and generalize these obtained data.

5. References

Allen, M.T. & Matthews, K.A. (1997). Hemodynamic responses to laboratory stressors in children and adolescents: The influences of age, race, and gender. *Psychophysiology*, *34*, 329-339.

Allen, M.T., Sherwood, A., Obrist, P.A., Crowell, M.D., & Grange, L.A. (1987). Stability of cardiovascular reactivity to laboratory stressors: A 2 ½ year follow-up. *Journal of Psychosomatic Research*, *31*, 639-645.

Brydon L., Strike P.C., Bhattacharyya M.R., Whitehead D.L., McEwan J., Zachary I., and Steptoe A. (2010). Hostility and physiological responses to laboratory stress in acute coronary syndrome patients. *J Psychosom Res*, 68 (2), 109-116.

Cannon, W.B. (1932). *The wisdom of the body*. New York: Norton.

Chida Y. and Steptoe A. (2009). The Association of Anger and Hostility with Future Coronary Heart Disease. A Meta-Analytic Review of Prospective Evidence. *J Am Coll Cardiol*, 53 (11), 936-946.

Cook, W.W. & Medley, D.M. (1954). Proposed hostility and pharisaic-virtue scales for the MMPI. *Journal of Applied Psychology*, *38*, 414-418.

Crowne, D. P. & Marlowe, D. (1960). A new scale of social desirability independent of psychopathology. *Journal of Consulting Psychology, 24* (4), 349-354.

Chesney, M.A. (1996). New behavioral risk factors for coronary heart disease: Implications for intervention. In K. Orth-Gomer y N. Schneiderman (eds.): *Behavioral Medicine Approaches to Cardiovascular Disease Prevention* (pp. 169-182). Mahwah, NJ: Erlbaum.

Everson, S.A., Kaplan, G.A., Goldberg, D.E., & Salonen, J.T. (1996). Anticipatory blood pressure response to exercise predicts future high blood pressure in middle-aged men. *Hypertension*, *27*, 1059-1064.

Everson-Rose, S.A.; Lewis, T.T. (2005). Psychosocial factors and cardiovascular diseases. *Annual Review of Public Health, 26*, 469-500.

Guerrero, C., and Palmero, F. (2006). Percepción de control y respuestas cardiovasculares. I*nternational Journal of Cliniccal and Health Psychology, 6* (1), 145-168.

Guerrero, C. (2008). Metodología en psicofisiología cardiovascular: procedimiento alternativo para la medición de la reactividad y su relación con la hostilidad defensiva. Tesis doctoral publicada. Universitat Jaume I, Castellón. En http://www.tesisenxarxa.net/TDX- 0206108-131614/index.html.

Guerrero, C., and Palmero, F. (2010). Impact of defensive hostility in cardiovascular disease. *Behavioral Medicine*, *36* (3), 77-84.

Gump, B.B. &Matthews, K.A. (1999). Do background stressors influence reactivity to and recovery from acute stressors? *Journal of Applied Social Psychology*, *29*, 469-494.

Helmers, K. F., Krantz, D. S., Merz, C. N. B., Klein, J., Kop, W. J., Gottdiener, J.S., & Rozanzki, A. (1995). Defensive hostility: Relationship to multiple markers of cardiac ischemia in patients with coronary disease. *Health Psychology, 14,* 202-209.

Helmers, K.F. & Krantz, D.S. (1996). Defensive hostility, gender and cardiovascular levels and responses to stress. *Annals of Behavioral Medicine, 18*, 246-254.

Jamner, L.D., Shapiro, D. Goldstein, I.B., & Hug, R. (1991). Ambulatory blood pressure and heart rate in paramedics: Effects of cynical hostility and defensiveness. *Psychosomatic Medicine*, *51*, 285-289.

Jennings, J.R., Kamarck, T.W., Everson-Rose, S.A., Kaplan, G.A., Manuck, S.B., & Salonen, J.T. (2004). Exaggerated blood pressure responses during mental stress are prospectively related to enhanced carotid atherosclerosis in middle-aged Finnish men. *Circulation, 110*: 2198–2203.

Jorgensen, R.S.; Abdul-Karim, K.; Kahan, T.A., & Frankowsi, J.J. (1995). Defensiveness, cynical hostility and cardiovascular reactivity: A moderator analysis. *Psychotherapy and Psychosomatics*, *64(3-4)*, 156-161.

Jorgensen, R.S., and Kolodziej, M.E. (2007). Suppressed anger, evaluative threat, and cardiovascular reactivity: a tripartite profile approach. *International Journal of Psychophysiology, 66*(2), 102-8.

Kaplan, J.R., Manuck, S.B., Adams, M.R., Weingand, K.W., & Clarkson, T. B. (1987). Inhibition of coronary atherosclerosis by propanolol in behaviorally predisposed monkeys fed an atherogenic diet. *Circulation, 76,* 1364-1372.

Kaplan, J.R., Manuck, S.B., Clarkson, T.B., Lusso, F.M., Taub, D.M., & Miller, E.W. (1983). Social stress and atherosclerosis in normocholesterolemic monkeys. *Science, 220,* 733-735.

Keys, A. & Taylor, H.L. (1971). Mortality and coronary heart disease among men studied for 23 years. *Archives of Internal Medicine, 128,* 201-214.

Larson, MR. & Langer, AW. (1997). Defensive hostility and anger expression: relationship to additional heart rate reactivity during active coping. *Psychophysiology, 34*, 177-184.

Manuck, S.B., Kaplan, J.R., & Clarkson, T.B. (1983). Behaviorally induced heart rate reactivity and atherosclerosis in cynomolgus monkeys. *Psychosomatic Medicine, 45,* 95-108.

Manuck, S.B., Kaplan, J.R., & Matthews, K.A. (1986). Behavioral antecedents of coronary heart disease and atherosclerosis. *Arteriosclerosis, 7,* 485-491.

Manuck, S.B., Kaplan, J.R., Adams, M.R., & Clarkson, T.B. (1989). Behavioral elicited heart rate reactivity and atherosclerosis in female cynomolgus monkeys. *Psychosomatic Medicine, 51,* 306-318.

Manuck, S.B., Olsson, G., Hjemdahl, P., & Rehnqvist, N. (1992). Does cardiovascular reactivity to mental stress have prognostic value in postinfarction patients? A pilot study. *Psychosomatic Medicine, 54,* 102-108.

Markovitz, J.H., Raczynski, J.M., Wallace, D., Chettur, V., & Chesney, M.A. (1998). Cardiovascular reactivity to video game predicts subsequent blood pressure increases in young men: The CARDIA study. *Psychosomatic Medicine*, *60*, 186-191.

Matthews, K.A., Zhu, S., Tucker, D.C, & Whooley, M.A. (2006). Blood pressure reactivity to psychological stress and coronary calcification in the coronary artery risk development in young adults study. *Hypertension, 47*(3): 391-395.

Mente, A. & Helmers, K.F. (1999). Defensive hostility and cardiovascular response to stress in young men. *Personality and Individual Differences*, *27(4)*, 683-694.

Moseley, J.V. & Linden, W. (2006). Predicting blood pressure and heart rate change with cardiovascular reactivity and recovery: results from 3-year and 10-year follow up. *Psychosomatic Medicine, 68*: 833-843.

Palmero, F., Guerrero, C., Gómez, C. y Carpi, A. (2006). Certezas y controversias en el estudio de la emoción. *Revista Electrónica de Motivación y Emoción*, *9*, 23-24. *Psychosomatic Medicine, 68*: 833-843.

Palmero, F., Breva, A. & Landeta, O. (2002). Hostilidad defensiva y reactividad cardiovascular en una situación de estrés real. *Ansiedad y Estrés, 8*(2-3), 115-142.

Palmero, F., Iñiguez, C., Guerrero, C., Carpi, A., Díez, J.L. & Diago, J.L. (2007). Hostilidad, psicofisiología y salud cardiovascular. *Avances de Psicología Latinoamérica. Colombia, 25* (1), 22-43.

Schiffer, F., Hartley, L.H., Schulman, C.L., & Abelmann, W.H. (1976). The quiz electrocardiogram: A new diagnostic and research technique for evaluating the relation between emotional stress and ischemic heart disease. *American Journal of Cardiology, 37,* 41-47.

Shapiro, D., Goldstein, I.B., & Jamner, L.D. (1995). Effects of anger/hostility, defensiveness, gender, and family history of hypertension on cardiovascular reactivity. *Psychophysiology*, *32,* 425-435.

Steptoe, A., Cropley, M., & Joekes, K. (2000). Task demands and the pressures of everyday life: Associations between cardiovascular reactivity and work blood pressure and heart rate. *Health Psychology*, *19*, 46-54.

Strike, P.C., Magid, K., Brydon L., Edwards, S., McEwan, J.R., & Steptoe, A. (2003). Exaggerated platelet and hemodynamic reactivity to mental stress in men with coronary artery disease. *Psychosomatic Medicine, 66*: 492–500.

Swain, A., & Suls, J. (1996). Reproducibility of blood pressure and heart rate reactivity: A meta-analysis. *Psychophysiology, 33,* 162-174.

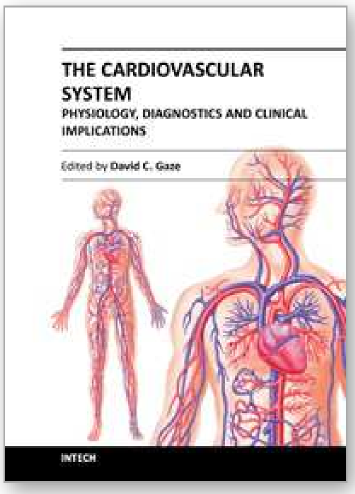
Treiber, F.A., Kamarck, T., Schneiderman, N., Sheffield, D., Kapuku, G, & Taylor, T. (2003). Cardiovascular Reactivity and Development of Preclinical and Clinical Disease States. *Psychosomatic Medicine, 65*, 46-62.

Vale, S. (2005). Psychosocial stress and cardiovascular diseases. *Postgraduate Medical Journal*, *81*, 429-435.

Vella, E.J. (2003). *Autonomic Characteristics of Defensive Hostility: Reactivity and Recovery to Active and Passive Stressors*. Published thesis. Blacksburg, Virginia.

Vella, E.J. y Friedman, B.H. (2007). Autonomic characteristics of defensive hostility: Reactivity and recovery to active and passive stressors. *International Journal of Psychophysiology, 66,* 95-101.

Vella, E.J., and Friedman, B.H. (2009). Hostility and anger-in: Cardiovascular reactivity and recovery to mental arithmetic stress. *Int J Psychophysiol*, 72, 253-259.



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The cardiovascular system includes the heart located centrally in the thorax and the vessels of the body which carry blood. The cardiovascular (or circulatory) system supplies oxygen from inspired air, via the lungs to the tissues around the body. It is also responsible for the removal of the waste product, carbon dioxide via air expired from the lungs. The cardiovascular system also transports nutrients such as electrolytes, amino acids, enzymes, hormones which are integral to cellular respiration, metabolism and immunity. This book is not meant to be an all encompassing text on cardiovascular physiology and pathology rather a selection of chapters from experts in the field who describe recent advances in basic and clinical sciences. As such, the text is divided into three main sections: Cardiovascular Physiology, Cardiovascular Diagnostics and lastly, Clinical Impact of Cardiovascular Physiology and Pathophysiology.