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Microanalysis of the relationship between goal self-efficacy and cardiovascular reactivity. A test for the moderating role of incentive value and the mediating role of anxiety

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Abstract

Eighty-three subjects were asked to solve a series of 15 mathematical problems after having been randomly assigned to an easy (5 problems) or to a difficult (9 problems) performance challenge. In addition to this, in order to create a sense of threat, subjects were told that a loud noise would be contingent upon failing to attain the performance challenge. Goal self-efficacy was computed from calculating the difference between self-efficacy level and the performance challenge. Intrinsic incentive value was derived from a six-item scale measuring the intensity of negative affects expected by the subject to be contingent upon a hypothetical failure to attain the goal. Systolic and diastolic blood pressure, and heart rate were collected during both the task performance and a prior rest phase. Anxiety-state was measured during the task performance as well. Statistical analyses revealed that interaction of goal self-efficacy and intrinsic incentive value partially predicted systolic blood pressure and heart rate reactivity. A main effect of self-efficacy was found on diastolic blood pressure. Overall, data best fitted to non-linear, quadratic functions in which either very low or very high self-efficacy, jointly with high incentive value, led to an enhanced cardiovascular reactivity. These findings are partially consistent with those previously obtained, and point to a complex model in which self-efficacy exerts specific effects on each cardiovascular parameter, usually in a non-monotonical way. Again, incentive value appeared as a moderator, but anxiety-state did not appear as a mediator of the relationship between goal self-efficacy and cardiovascular reactivity.

Keywords: Self-efficacy; Incentive value; Perceived control; Anxiety; Blood pressure; Heart rate

1. Introduction

Self-efficacy is a specific perceived control construct emerged from Social Cognitive Theory (Bandura, 1977), and refers, in Skinner's terms (Skinner, 1996), to competence expectancies, i.e. to the control that a person expects to exert over the generation and performance of his or her own behavior. Self-efficacy has been postulated as a cognitive mechanism that establishes reciprocal influences with emotional and motivational processes at behavior, experience, and physiologic levels (Bandura, 1997). With regard to the last, Bandura (1977) suggested that (1) a great sense of capacity (self-efficacy) inversely correlates with peripheral physiological reactivity (understood as an unspecific arousal), and (2) this could be the biological pathway by which self-efficacy affects illness vulnerability (Bandura, 1992, 1997; O'Leary, 1990, 1992; Haidt and Rodin, 1999). This theoretical approach to cognitive control of peripheral physiological reactivity has promoted a large amount of experimental research (Bandura et al., 1982, 1985, 1987; Barrios, 1983; Biran and Wilson, 1981; Feltz, 1982; Feltz and Mugno, 1983; Gerin et al., 1995, 1996; Sanz and Villamarín, 1997, 2001; Sanz et al., 2006; Wiedenfeld et al., 1990; Wright and Dill, 1993) that has not yielded, until today, a consistent pattern of findings.

Three major issues arise when dealing with the role of self-efficacy on physiological reactivity. The first issue refers to the conditions (or restrictions) under which self-efficacy judgements participate in physiological regulation. The second issue

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deals with the direction in which self-efficacy affects physiological reactivity. The third issue poses the way in which self-efficacy exerts its peripheral effects: either moderating an unspecific arousal, or yielding specific patterns of physiological reactivity.

1.1. Conditions under which self-efficacy regulates physiological reactivity

Early studies in the 1980s depicted a miscellaneous landscape about the relationship between self-efficacy and physiological reactivity. The studies carried out by Bandura (Bandura et al., 1982, 1985, 1987; Wiedenfeld et al., 1990) seemed to demonstrate a linkage between the efficacy beliefs change and the activity on several biological systems (cardiovascular, hormonal–HPA axis-, pain and immunity mechanisms). Contrarily, other researchers (Barrios, 1983; Biran and Wilson, 1981; Feltz, 1982; Feltz and Mugno, 1983) reported either a lack of relationship, or a linkage in the opposite direction (in terms of causality and/or slope). These authors suggested that self-efficacy does not play any role on physiological regulation and/or it merely acts as an epiphenomenon, as was pointed out by Kirsch (1985). In fact, there were important methodological differences between previous studies. Those carried out by Bandura and colleagues were usually done in clinical patients, so in the context of a highly relevant therapeutic intervention for the well-being of the subjects, whereas the other body of research constituted a heterogeneous group of procedural settings (subjects, scenarios, tasks, etc.) that purportedly implied a lower personal commitment. Moreover, Social Cognitive Theory (SCT) postulates that self-efficacy exerts its motivational and emotional effects interacting with outcome expectancies (Bandura, 1982), which is a postulate that has not still been proven with regard to biological regulation. Thus, it seemed to be reasonable to infer that the contradictory results about the effects of efficacy beliefs on physiological reactivity could be overcome by taking into account outcome expectancies as a factor that exerts moderator effects. Outcome expectancy is a construct that has been derived from Bandura's theoretical framework (Bandura, 1982), and refers to the anticipated consequences of behavior. So it is a specific cognition about reinforcement contingencies. Amongst the dimensions that can be differentiated within outcome expectancies (Bandura, 1997; Dawson et al., 2001), in our previous studies we analyzed the role of incentive value as a moderator of the self-efficacy/physiological reactivity relationship. Overall, the results obtained in these studies clearly suggested that the effect of self-efficacy on physiological reactivity in coping situations only (or specially) appears when the perceived consequences derived from a behavior are important to the person (i.e., when incentive value is high). This moderator effect of incentive value has been found on heart rate, systolic BP, skin temperature, skin conductance, and respiratory rate, but not on diastolic BP.

1.2. Direction of the effects

This second issue is far from a clear picture, since in some studies a negative-slope relationship between self-efficacy and physiological reactivity was reported, but in others, a positive trend was outlined. Moreover, sometimes the relationship seemed to be fitted to a linear function, but in other studies, a non-monotonic shape was found (Bandura et al., 1985). In our previous studies, self-efficacy appeared to be either positively or negatively related to heart rate and systolic blood pressure depending upon the experimental setting (reward vs. avoidance paradigms, respectively). These apparently contradictory results also appear at a theoretical level from both Social Cognitive Theory and Theory of Intensity Motivation. On the one hand, Social Cognitive Theory proposes that anxiety, in coping with aversive consequences, is partially regulated by self-efficacy: the lower the self-efficacy to perform a behavior that let to avoid negative outcomes, the greater the anxiety, especially if such outcomes are appraised as highly aversive (Bandura, 1982, 1986; Villamarín, 1994). As was pointed above, several studies carried out in clinical contexts showed that an increase in self-efficacy was accompanied by a decrease in both self-reported anxiety and physiological reactivity (Bandura et al., 1982, 1985). But, on the other hand, in experimental studies on achievement motivation (Bandura and Cervone, 1983, 1986), also rooted in Social Cognitive Theory, it was observed that a high self-efficacy level led to an enhanced effort to perform the task. Moreover, theory of intensity of motivation (Brehm and Self, 1989) postulates that cardiovascular reactivity increases directly with effort spent on the task, until a limit (potential motivation) that is, in part, a function of incentive value and probability to attain it. As a whole, the empirical results obtained from this theoretical framework (Wright and Dill, 1993; Wright and Dismukes, 1995; Wright and Gregorich, 1989; Wright et al., 1990, 1992, 1994; Eubanks et al., 1992) are in line with this prediction. Nevertheless, these apparently contradictory postulates and results are not necessarily incompatible, but rather, as Gerin et al. (1992, 1996) have stressed, this fact appoints to a couple of independent factors affecting to cardiovascular reactivity in active coping: the sense of control and the effort to control. The first (sense of control) has been suggested to establish an inverted relationship with cardiovascular reactivity, but the second (effort to control) would establish a direct relationship with it. Empirical evidence (Gerin et al., 1995, 1996) seems to give support to this dual effect of self-efficacy on cardiovascular response in active coping paradigms.

1.3. Effects on unspecific arousal vs. specific psychophysiological patterns

As it was said above, Bandura suggested a diffuse, unspecific effect of efficacy beliefs on arousal; in earliest studies, this assumption led to measure a few number of biological parameters (thus making difficult to find specific patterns of psychophysiological reactivity). Contrarily, when several indicators were jointly taken into account (Sanz, 1998), a more complex landscape has emerged. Concretely, the effect of self-efficacy on a specific psychophysiological variable seemed to be different from that exerted on the other variables in terms of magnitude of the effect, direction (slope), and shape (linear vs. non-linear) of the predictive model, and the role of other

moderator variables (e.g. incentive value). For example, systolic BP seems to depend upon the interaction between self-efficacy and incentive value, but diastolic BP would only depend upon self-efficacy main effect. As Sanz (1998) has stressed, although a low self-efficacy combined with a high incentive value for the task clearly leads to an overall increase on peripheral physiological reactivity (when all the psychophysiological parameters are taken into account as a whole), each "cognitive pattern" (self-efficacy × incentive value) seems to activate different psychophysiological patterns, purportedly congruent with coping style to be displayed by the subject (simplifying, in terms of *fight* or *flight*). This finding is convergent with results obtained from other theoretical approaches of coping and perceived control concerning psychophysiological regulation (Obrist, 1975; Obrist et al., 1978; Manuck et al., 1978; Kasprowitz et al., 1990), and is congruent with the diversity of underlying neural regulation mechanisms for biological functions (Cacioppo, 1994; Guyton and Hall, 2000; Waldstein et al., 1997).

Taking into account these arguments, we arranged the present study, which was designed attending to these methodological and theoretical conditions:

1. It might enable the analysis of the functional relationship between self-efficacy and peripheral physiological reactivity. In order to do this, the methodological approach must be essentially quantitative.
2. It might embrace all the full-range of self-efficacy, in order to detect the two hypothetical effects (sense of control – linked to threat appraisal – and effort to control — linked to commit with task performance and effort) present at different magnitude alongside its range.
3. A common experimental manipulation might embrace the entire sample so that it generates a twofold contingency of both threat and challenge.
4. A large enough number of psychophysiological variables might be measured in order to assess variable-specific regulation, and each one must be considered as independent concerning its linkage to specific expectancies (self-efficacy and incentive value).

Moreover, several innovations or changes were done in relation to our past studies:

1. *Goal self-efficacy (GSE)*. In his most recent analysis of self-efficacy construct, Bandura (1997) proposes that, in order to define and assess self-efficacy in achievement tasks, it is necessary to establish accurately the task difficulty. In this regard, self-efficacy is always a prediction to attain a specific goal, and depends on both the difficulty of the goal and the perceived ability of the person. Some studies from other theoretical frameworks have analyzed the interaction between perceived ability and task difficulty on cardiovascular reactivity (Wright and Dill, 1993; Wright and Dismukes, 1995; Wright et al., 1994), but in our previous works, we examined the effects of self-efficacy on physiological reactivity without taking jointly into account these two aspects. Therefore, in present work, we intended to analyze the effects of positive and negative discrepancies between perceived ability and task difficulty (namely, the goal self-efficacy) on cardiovascular reactivity.
2. *Intrinsic incentive value.* In previous research on the effects of specific perceived control on physiological reactivity, only extrinsic incentives have been usually considered. These are normally defined as external consequences, such as money, social praises, electric shocks, or loud noises, which are contingent upon an arbitrary attainment or failure criterion for a task. Nevertheless, as many authors have stressed (Bandura, 1986; Csikszentmihalyi, 1998; Deci and Ryan, 1985), the incentives cannot only be extrinsic, but also intrinsic. Amongst these, the most important are perhaps the affective states derived from success or failure in achievement tasks (Bandura, 1986). Consequently, in this work we intended to study the effect of intrinsic incentives on cardiovascular reactivity. Concretely, intrinsic incentive value has been here defined as the intensity of negative affective state that subjects anticipate they will experience in case of a hypothetical failure to attain the performance goal.

In sum, we intended to study the functional relationship between, on the one hand, goal self-efficacy and intrinsic incentive value and, on the other hand, cardiovascular reactivity and anxiety in a laboratory procedure based on the performance of a task within a double contingency of threat and challenge. Three hypotheses were tested:

1. Goal self-efficacy exerts its effects on cardiovascular reactivity more acutely when the consequences of the failure are highly aversive (i.e. when the intrinsic incentive value is high), with the exception for diastolic blood pressure, which is only linked to goal self-efficacy.
2. Goal self-efficacy relates to cardiovascular reactivity in a non-monotonically, positive quadratic shape when both effects exerted (sense of control and effort to control) are concurrently present. In particular, cardiovascular reactivity arises when goal self-efficacy is very high or very low. In other words, the relationship between goal self-efficacy and each of cardiovascular variables fits a quadratic (U-shaped) function better than a linear function.
3. Anxiety-state, as the affective outcome of low sense of control, is a mediator of the relationship between goal self-efficacy and cardiovascular reactivity.

2. Materials and methods

2.1. Subjects

The sample consisted of eighty-three healthy volunteers, of which 66 were women and 17 were men, with an average age of 20.5 years. They were chosen from a group of students in their first course of psychology. After recruitment, volunteers were telephoned in order to set a date and time for the experimental session, as well as for receiving instructions on their behavior

prior to the session. These instructions specified: (a) the need for them to avoid consuming psychoactive substances (tobacco, coffee, cola drinks, tea, alcohol or marijuana) as much as possible during 4 h prior to the experimental session, and (b) a recommendation to choose a session time that would not be preceded by (or was previous to) stressful events (an exam, a visit to the doctor…), or by intense physical exercise.

2.2. Apparatus

A PC connected to two screens via an S-VGA splitter was used. One of them (19″) was the one with which the subject interacted from the experimental room and through which he or she was provided with instructions to carry out the experimental task. The other computer screen allowed the subject's behavior to be observed in real time from the control room by the experimenter. In addition, a set of microphones and loud speakers was arranged, which allowed for communication, if needed, between the experimenter and the subject. Finally, a Welch Allyn oscillometric electrosphygmomanometer was used to measure blood pressure (BP) and heart rate. According to the manufacturer, the device's margin of error for blood pressure measurement is ±3 mm Hg. Reliability of this device was previously tested against a SE-1000 oscillometric electrosphygmomanometer (Sein Ltd.); measuring in a counterbalanced order on the two arms at rest; results indicated a correlation of $r = 0.96$. The cuff was placed on the subjects' non-dominant arm and used in accordance with the recommendations by the Association for the Advancement of Medical Instrumentation (1992) for automatic measurement of blood pressure.

2.3. Questionnaires

SPSS Data Entry® 3.0 was used to create a computerized questionnaire designed to assess the subjects' compliance with the pre-experimental instructions and other control variables recorded in order to screen the sample or proceed to statistical control: the consumption of psychoactive substances, antihypertensive and hormonal treatments, cardiovascular pathologies, and stressful events. Likewise, other personal (such as age and gender) and procedural variables (such as laboratory temperature, time, and experimenter) were also recorded. This software also contained a computerized version of State-Trait Anxiety Inventory (STAI) by Spielberger et al. (1970), adapted into Spanish (Seisdedos, 1994). Both state and trait anxiety questionnaires appeared at different stages of the experimental session.

In addition, within the computer program guiding the entire experimental session, which was designed using PowerPoint (Microsoft Office 2000®) and macros from Visual Basic, various questions were included in order to evaluate self-efficacy level and intrinsic incentive value. According to the last recommendations by Bandura (1997), self-efficacy level was evaluated through a one-item measure: "*How many problems do you think you will be able to solve of the 15 you must do?*" Afterwards, the subjects responded to the item "*What do you think is the probability that you will achieve the goal we have set for you?*" (goal self-efficacy validity index) on an 11-point scale; this item was arranged in order to assess the convergent validity of goal self-efficacy measure (that is a computed index). In addition, a six-item questionnaire was included to assess intrinsic incentive value. Subjects were told to think about a hypothetical failure to reach the challenge performance, and then they had to respond on a 5 level scale (from "*not at all*" to "*very much*") about their anticipated feelings: frustration, dissatisfaction, sadness, anger, shame, and blame. Finally, an item was arranged to assess noise aversivity (i.e. extrinsic incentive value) on a 5-point scale.

2.4. Procedure

When the subject arrived at the laboratory, he or she was greeted by an experimenter ($n = 3$, one female, two males) assigned randomly to sessions without regard of subjects' gender. After making the subject comfortable in the experiment chair, the experimenter gave to the subject an informed consent form to read and sign. Then, the blood pressure cuff was placed on the subject's non-dominant arm following the guidelines set forth by Shapiro et al. (1996) and a check was made to ensure that the electro-sphygmomanometer was functioning properly through a pre-experimental blood pressure reading with a two-fold purpose: to get the subject used to the sensation of the cuff inflating, and to obtain a pre-experimental record to be employed if needed as a covariate in the inferential statistical analyses. During this stage, the proper functioning of the subject–experimenter intercom system was also checked. Then the experimental procedure itself began that, from then on, was totally computerized. The subject began the session filling in a control checklist of questions (consumption of psychoactive substances, pre-experimental stress, etc.) and the anxiety-trait questionnaire.

Immediately thereafter, a 5-min baseline took place, during which the subject saw neutral images (landscapes) on the computer screen while listening to classical music through the headphones. The subject's blood pressure and heart rate were taken in minutes two and four.

After the baseline, the subjects saw on the computer's screen a description of the task to be carried out, and six training trials were done. The purpose of these trials was for the subject to become familiar with the task so that he or she could make a sufficiently precise judgment on his or her perceived capacity to carry it out successfully (self-efficacy level). After this, the subjects were informed about the task, which consisted of a series of 15 problems with a difficulty level similar to that of the trial items. Immediately afterward, the subjects' self-efficacy level was evaluated. In order to reject the possibility that the effect of goal self-efficacy and incentive value effects on cardiovascular reactivity and anxiety were moderated by goal level (i.e., they were different depending upon performance challenge), two goal (difficulty) levels were arranged in a between-groups experimental procedure. Half of the subjects (high difficulty group) were ask to solve correctly at least 9 of the 15 problems, and the other half (low difficulty group) were ask to solve correctly at least 5 problems. After this, self-efficacy level and goal self-efficacy validity index were assessed. Immediately

thereafter, the subject was aware of the coping-avoidance contingency. He or she, without previous warning, heard through the headphones a loud noise (105 dB; white noise; 5 s duration), and was informed that if his or her performance did not reach the required level, he or she would be exposed to the same sound at the end of the task, but this time for a duration of 30 s. Thus, the subjects were placed in a condition of active coping in which they could avoid an aversive stimulus contingent upon failure to reach a certain performance level in the task. Afterwards, subjects responded to the intrinsic incentive value scale.

The task then started, and it was divided into two blocks of trials (of eight and seven problems each, respectively). Measurement of blood pressure and heart rate began 50 s after the beginning of each of the two test sets. After the end of the first block of trials, subjects completed the on-screen STAI-state questionnaire.

2.5. Task

The task consisted of a series of 15 arithmetic problems; a problem and four response choices (the correct solution among them) appeared on the computer screen. The problems were made up of a combination of two or three arithmetical operations (such as "*two times two times five*"). The subject had a 7-s time limit for responding (by using the mouse to click on the correct response) before the problem disappeared from the screen. During each test, a continuous noise distracter was used to simulate the sound of a timer. The interval between problems was 10 s, during which preparatory instructions for the following problem appeared. This is the classical task that we have used to test the relationship between perceived control and physiological reactivity (Sanz, 1998; Sanz and Villamarín, 2001; Sanz et al., in press), because of (a) it implies a great amount of mental workload, (b) it consequently induces a great mobilization and expenditure of energy resources (peripheral physiological reactivity), (c) it is very easy to build performance indicators, (d) it is very sensible to affective interference, (e) in literature about the topic are usually found similar tasks, and (f) there is a great individual variability in either performance and peripheral physiological reactivity aroused by task in the context of our experimental paradigm. The length of each problem was calibrated in prior tests such that the probability of solving each problem would be 50% for the sample set as a whole. Thus, from the outset, this would lead one to expect that the subjects with high goal (a challenge of 9 problems correct out of 15) would estimate that their probability of reaching the goal would be fewer than 50%. Contrarily, the subjects with low goal (a challenge of 5 problems out of 15) would estimate their probability of reaching the goal would be over 50% overall.

2.6. Data preparation

BP and heart rate raw data were transformed before being statistically processed. Delta (Δ) scores were derived as the difference between the values in the first and second sets of the task and the final reading in baseline. Therefore, two reactivity values were obtained for systolic and diastolic blood pressure, and for heart rate: initial Δ score (corresponding to first set of problems) and final Δ score (second set of problems); the reliability of this mathematical transformation was tested and supported by Llabre et al. (1991).

It is worth noting that heart rate raw data used to calculate Δ scores were obtained by dividing the pulse waves counted into time expended by the electro sphygmomanometer to detect BP; this calculation was automatically done by this device, avoiding the overestimation problem (Reyes del Paso and Vila, 1998). This strategy to measure heart rate makes it possible to match measurement intervals for BP and heart rate, but does not permit the use of correction strategies for deviant interbeat intervals (Porges and Byrne, 1992). This reliability problem leads to the assumption that the relationship between heart rate and other variables would be larger than those found here.

On the other hand, goal self-efficacy was computed by subtracting the performance challenge proposed to a subject from his or her self-efficacy (SE) level. For example, if SE level was 9 points (subject perceiving him/herself as capable to solve 9 problems of 15) and performance challenge was 5 problems of 15, then resulting goal self-efficacy (GSE) was 4 points. Descriptive analysis showed that this variable presented the expected good properties in our sample: mean = −0.30, median = 0, and a broad and homogeneous dispersion: rank = 17 (from −8 to 9, being full rank = 19), sd = 3.6, kurtosis = −0.07 and skewness = 0.10.

In order to include goal self-efficacy as a factor in exploratory ANOVAs or ANCOVAs, subjects were classified into 4 groups: very low goal SE, when goal self-efficacy was −4 points or lower (n = 18); low goal SE when goal self-efficacy was −3 to −1 points (n = 17); high goal SE when goal self-efficacy was 0 to 3 points (n = 31); and very high goal SE when goal self-efficacy was 4 points or higher (n = 14).

Furthermore, a Principal Components Analysis was done on the 6-item scale assessing intrinsic incentive value, showing a one-dimensional structure, with all items jointly explaining 67% of variance. A reliability analysis was then performed, indicating a good internal consistency for the scale (Cronbach's

Table 1
Means or percentages for control and independent variables by goal self-efficacy groups

	Age	Gender (% of women)	% stressful events	% depressor drugs	% stimulant drugs	Extrinsic incentive value	Intrinsic incentive value	Goal self-efficacy validity index	STAI-R
GSE very low (n = 17)	20.7	83.3	23.5	5.5	50.0	3.7	7.3	2.8	22.3
GSE low (n = 18)	20.1	100	41.1	0	60.5	3.5	7.4	4.3	21.7
GSE high (n = 31)	20.1	87.1	41.2	3.2	48.4	3.4	9.7	5.5	23.2
GSE very high (n = 14)	21.0	50.0	26.7	14.2	35.7	3.8	9.4	8.3	23.6

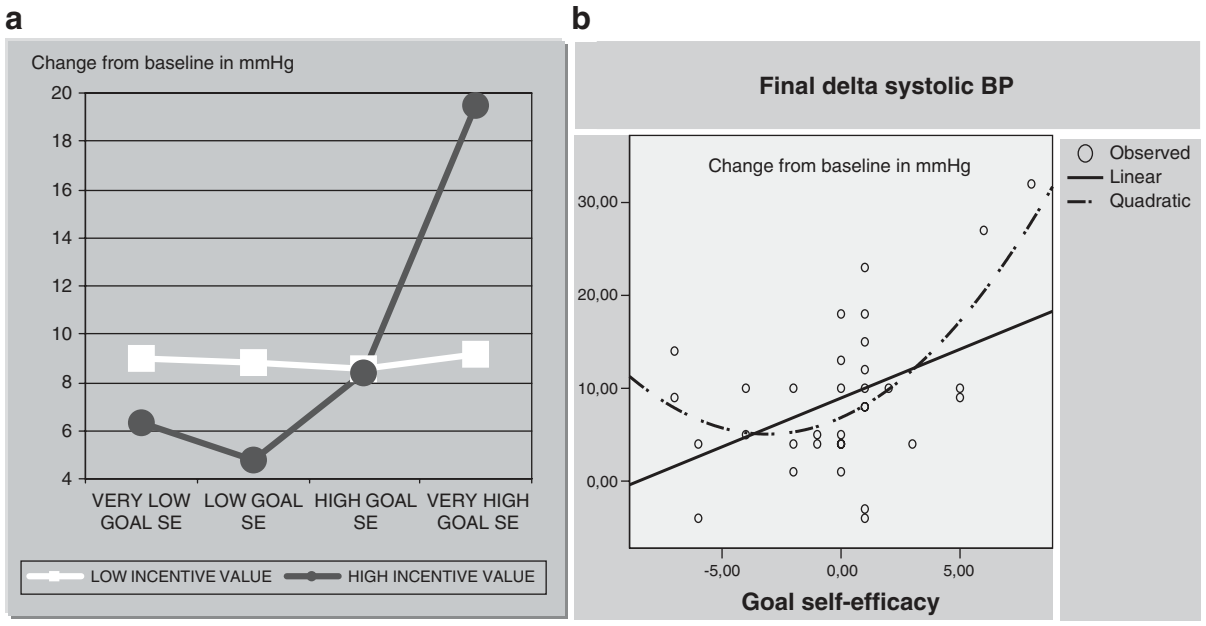

Fig. 1. (a, left) Mean final systolic BP delta scores (raw change from baseline) as a function of goal self-efficacy (categorized) and intrinsic incentive value. (b, right) Curve fitting a quadratic vs. a linear model. *X*-axis: goal self-efficacy; *Y*-axis: final systolic BP delta scores; only high intrinsic incentive value subjects.

$\alpha = 0.89$). Consequently, an index of intrinsic incentive value was derived by adding scores of all the items. In order to use this variable as a factor in exploratory ANOVAs or ANCOVAs, it was dichotomized (high-$n = 35$ – vs. low-$n = 45$ – intrinsic incentive value) using the sample median.

As can be seen, the categorical transformation of both goal self-efficacy and intrinsic incentive value yielded a non-homogenous distribution of subjects among groups, which could reduce the robustness of the ANOVAs done with these data; concretely, some cells had only six subjects. Despite this, it did not suppose a major limitation to interpret the results, since (1) ANOVAs only were planned to identify interactions between independents variables, and the conclusions are derived from the analyses in which original, quantitative data were used — linear and non-linear regressions; and (2) the statistical criterion used to interpret the results was $p = 0.025$ (not $p = 0.05$, a less conservative but more usual criterion).

Finally, direct scores of anxiety-state were computed to be used in statistical analyses. To avoid sample reduction, an algorithm using SPSS was used for missing values correction on data from three subjects.

2.7. Statistical design and analysis

One subject was rejected for statistical analyses due to antihypertensive treatment. Two subjects were also rejected due to significant data loss. Therefore, final sample was made up of 80 subjects. Moreover, data collection partially failed in some of the subjects, although this was not considered a reason for excluding them from the statistical analyses. This does explain the slight variation in the degrees of freedom among the different analyses that appear below.

Dependent variables showed a mild mean correlation ($r = 0.34$; $p < 0.01$), ranging from $r = -0.03$ (n.s.) to $r = 0.60$ ($p < 0.001$). Despite this, univariate inferential analyses were planned and performed on each dependent variable. The reasons are either theoretical or methodological (as was pointed out above):

1. The assumption that perceived control exerts specific regulation on different physiological parameters.
2. The nesting among the Δ scores derived from first and second trials' blocks records (initial and final reactivity, respectively) for each cardiovascular variable.

The analysis strategy consisted of three steps. The core of the analysis (steps 2 and 3) was done on original quantitative variables, in congruence with the nature of the design.

In order to test hypothesis 1 (interaction between goal self-efficacy and intrinsic incentive value), first step was done using non-continuous, transformed (categorized) variables. An ANCOVA was done on each physiological index and on anxiety-state in order to reject the possibility that task difficulty interacted with goal self-efficacy, intrinsic incentive value, or its interaction. As none of the analyses done yielded any interactive effect in which task difficulty was implicated, dependent variables again underwent ANOVAs using a 4 × 2 (GSE × intrinsic incentive value) factorial model.[1]

The second step, based upon the original quantitative independent variables, was planned to test whether data would better fit a quadratic than a linear model, as was postulated in

[1] The correlation matrix indicated that some control variables were correlated with psychophysiological reactivity indexes and/or with anxiety-state, concretely: gender, experimenter, pre-experimental basal levels of systolic BP, diastolic BP and heart rate, caffeine consumption and anxiety-trait. Nevertheless, their effects on dependent variables did not reach statistical significance in the context of full models of ANCOVA.

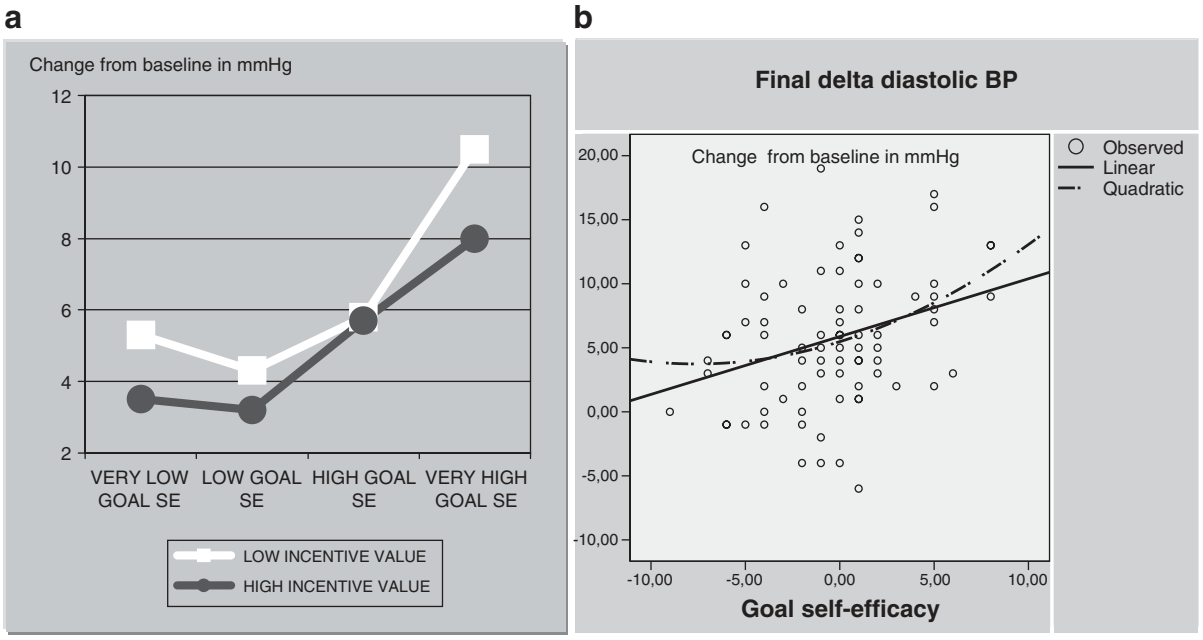

Fig. 2. (a, left) Mean final diastolic BP delta scores (raw change from baseline) as a function of goal self-efficacy and intrinsic incentive value (categorized). (b, right) Curve fitting a quadratic vs. a linear model. *X*-axis: goal self-efficacy; *Y*-axis: final diastolic BP delta scores; whole sample.

hypothesis 2. Regression curve-fit analyses of goal self-efficacy on dependent variables were carried out taking into account if their relationship was or not moderated by intrinsic incentive value, as it was obtained in the first step. Two models were tested and compared: linear model and quadratic model.

Finally, a third step was planned in order to test hypothesis 3 (the mediator role for anxiety-state); so, partial correlations between goal self-efficacy and cardiovascular indexes were done controlling for anxiety-state.

3. Results

3.1. Independent and control variables

GSE computed index presented a high, positive correlation ($r=0.75$; $p<0.005$) with the measure of GSE validity index. ANOVA of GSE validity index using dichotomized GSE as a factor yielded the same results ($F(3,76)=25.28$; $p<0.005$). Planned contrasts (Helmert) indicated that all groups had mean values of GSE validity index different from each other (very high

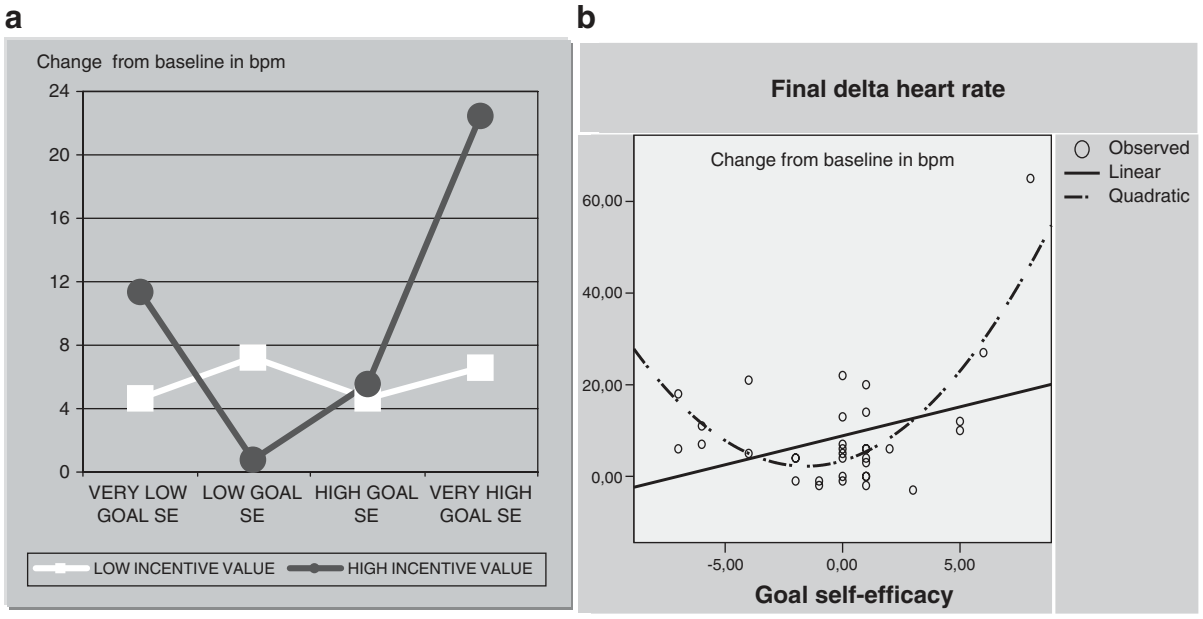

Fig. 3. (a, left) Mean final heart rate delta scores (raw change from baseline) as a function of goal self-efficacy and intrinsic incentive value (categorized). (b, right) Curve fitting a quadratic vs. a linear model. *X*-axis: goal self-efficacy; *Y*-axis: final heart rate delta scores; only high intrinsic incentive value subjects.

Table 2
Fitting comparisons of two regression models for statistically significant effects found on previous ANOVAs

	Linear equation fitting				Quadratic equation fitting				Best model
	df	*F*	p	Adjusted R^2	*df*	*F*	p	Adjusted R^2	
GSE (IIV=high) on final Δ systolic BP	32	8.02	0.008	0.17	31	8.87	0.001	0.32	Quadratic
GSE (IIV=high) on final Δ heart rate	32	2.13	ns	–	31	11.27	<0.001	0.38	Quadratic
GSE on final Δ diastolic BP (whole sample)	77	8.22	0.005	0.08	76	4.55	0.01	0.08	Both

Linear models correspond to the equation: $CVP_i = b_0 + (b_1 * GSE)$. Quadratic models correspond to the equation: $CVP_i = b_0 + (b_1 * GSE) + (b_2 * GSE^2)$. CVP_i = cardiovascular parameter; GSE = goal self-efficacy. IIV = intrinsic incentive value.

GSE M=8.25; high GSE M=5.51; low GSE=4.29; very low GSE=2.77). Furthermore, the intrinsic incentive value score was uncorrelated to noise aversivity ($r=0.02$, ns). In addition, Tukey's post hoc analyses showed that GSE groups did not differ in extrinsic incentive, intrinsic incentive value, and anxiety trait.

With regard to the control variables (see Table 1), GSE groups did not statistically differ in age, stressful events and drugs consumption. Gender appeared to be significantly different among groups, since very high GSE group showed a bigger percentage of men ($\chi^2=17.58$; g.l.=3; $p=0.003$).

3.2. Systolic blood pressure

On the one hand, analysis of variance performed on initial systolic BP reactivity showed neither main nor interactive effects of GSE and/or intrinsic incentive value. On the other hand, ANOVA on final systolic BP reactivity did show a main effect of GSE and an interaction of GSE × incentive value near to reach statistical significance. Simple effects analysis revealed an effect of GSE on systolic BP only when intrinsic incentive value was high ($F(3,75)=3.66$; $p=0.01$). Regression (curve-fit) analyses of GSE on final systolic BP (whole sample) were near statistical significance to fit either a quadratic model ($F(2,76)=3.54$; $p=0.04$) and a linear model ($F(1,77)=4.17$; $p=0.04$). Regression of GSE on final systolic BP when incentive value was low was not significant; contrarily, regression when incentive value was high fitted a quadratic model ($F(2,31)=8.86$; $p<0.001$; $R^2=0.36$; adjusted $R^2=0.32$) better than a linear (positive) model ($F(1,32)=8.01$; $p=0.008$; $R^2=0.20$; adjusted $R^2=0.17$) (Fig. 1) (Table 2).

3.3. Diastolic blood pressure

Analysis of variance done on initial diastolic BP reactivity showed neither main nor interactive effects of GSE or incentive value. On the other hand, ANOVA showed a statistically significant main effect of GSE on final diastolic BP ($F(3,75)=3.32$; $p=0.02$). Regression (curve-fit) analyses of GSE on final diastolic BP revealed a fit a quadratic model ($F(2,76)=4.55$; $p=0.01$; $R^2=0.11$; adjusted $R^2=0.08$), as well as to a linear model ($F(1,77)=8.22$; $p=0.005$; $R^2=0.10$; adjusted $R^2=0.08$) (Fig. 2) (Table 2).

3.4. Heart rate

ANOVA performed on initial heart rate reactivity showed neither main nor interactive effects of GSE and/or incentive value. Conversely, analysis on final heart rate reactivity revealed the main effect of GSE ($F(3,71)=3.29$; $p=0.02$) and its interaction with incentive value ($F(3,71)=3.22$; $p=0.02$) reached statistical significance; incentive value main effect was near to reach it ($F(1,71)=3.39$; $p=0.07$). Simple effects analysis indicated that the GSE effect on final heart reactivity was statistically significant only when incentive value was high. Regression (curve-fit) analysis of GSE on final heart rate in whole sample was near to fit a quadratic model ($F(2,76)=3.63$; $p=0.03$) but not a linear model. Regression of GSE on final heart rate when incentive value was low was not significant; contrarily, regression when incentive value was high fitted a quadratic model ($F(2,31)=11.26$; $p<0.0005$; $R^2=0.42$; adjusted $R^2=0.38$) but not a linear model (Fig. 3) (Table 2).

3.5. Role of anxiety-state as a mediator of self-efficacy and cardiovascular reactivity relationship

Pair-wise correlations revealed that anxiety-state direct scores only significantly correlated with initial and final systolic BP delta scores ($r=0.32$; $p=0.004$ and $r=0.25$, $p=0.02$, respectively). Correlations between anxiety-state and the other cardiovascular measures ranked from $r=0.04$ to $r=0.17$ and did not reach statistical significance.

Moreover, in order to test hypothesis 3, partial correlations between goal self-efficacy and cardiovascular variables were planned (Table 3). Notwithstanding this, taking into account that the relationship between goal self-efficacy and cardiovascular variables did not fit the linearity assumption, predictive

Table 3
Correlations and partial correlations (controlling for anxiety-state) among cardiovascular variables and their respective predictive quadratic model based upon goal self-efficacy (GSE)

	Final Δ systolic BP (IIV=high)	Final Δ diastolic BP	Final Δ heart rate (IIV=high)
Predictive model	SBP=6.84+(1.15*GSE)+(0.19*GSE^2)	DBP=6.84+(1.15*GSE)+(0.19*GSE^2)	HR=6.84+(1.15*GSE)+(0.19*GSE^2)
Correlation predicted vs. observed data	0.603** (n=34)	0.327* (n=79)	0.651** (n=34)
Partial correlation Predicted vs. observed data (controlling for anxiety-state)	0.600** (n=34)	0.327* (n=79)	0.649** (n=34)

*$p<0.01$; **$P<0.0005$. IIV = intrinsic incentive value.

models (based upon goal self-efficacy as an independent variable) for each cardiovascular variable were previously created, employing the parameters yielded by the quadratic curve-fit analysis. Then, correlations and partial correlations (controlling for anxiety-state) between the observed data for each cardiovascular measure and its predictive model were done. Matrixes of correlations and partial correlations were very similar (Table 3), thus the control for anxiety-state did not reduce the percentage of variability of each cardiovascular parameter explained by its predictive model.

4. Discussion

Goal self-efficacy, a variable computed by subtracting difficulty (goal to be attained) from self-efficacy level, presents a high dispersion in our sample, which has allowed us to explore its physiological relevance alongside all its rank.[2] Also, the fact that goal self-efficacy is strongly correlated with goal self-efficacy validity index gives this measure a convergent validity. Finally, the lack of interactive effects between difficulty and goal self-efficacy on both cardiovascular reactivity and anxiety-state indicates that the most important element is not the challenge level (namely, the goal) per se, but rather the discrepancy (magnitude and direction) between perceived capacity and the challenge. Moreover, the lack of correlation between intrinsic incentive value and the subjective measure of noise aversivity indicates that intrinsic incentive value scores do not reflect extrinsic incentives, and therefore, it exclusively is an index of intrinsic processes.

Overall the obtained results regarding to the cardiovascular reactivity are in line with hypothesis 1, since an interaction between intrinsic incentive value and goal self-efficacy on systolic BP and heart rate has been found. As it was pointed out in previous studies, in which extrinsic incentives were assessed (Sanz and Villamarín, 1997, 2001; Sanz et al., 2006), self-efficacy seems to regulate physiological reactivity, especially when intrinsic incentive value is high, namely, when the consequences of behavior (intrinsic outcomes) are important for the individual. In contrast, diastolic BP reactivity only seems to be regulated by a direct (main) effect of goal self-efficacy, a result that had already been obtained in previous research (Sanz and Villamarín, 2001; Sanz et al., 2006).

Moreover, in congruence with hypothesis 2, we have found in curve-fit analyses that the relationship of goal self-efficacy with systolic BP and heart rate fits a U-shaped curve, in which reactivity seems to be greater when goal self-efficacy is very low or very high (but especially in this last condition, as is reflected by a mild asymmetry). In other words, this relationship seems to be adjusted to a quadratic shape, in which reactivity is greater when discrepancy between self-efficacy level and challenge increases. Contrarily, when intrinsic incentive value is low, goal self-efficacy establishes a "smooth" function with systolic BP and heart rate changes; namely, a lack of relationship has been found. The results also show that subjects who show very high goal self-efficacy underwent higher diastolic BP reactivity during the task performance. Furthermore, as the regression analyses have shown, the relationship of goal self-efficacy with diastolic BP reactivity is as close to a quadratic (U-shaped) function as to a linear function. Again, this result suggests a specific regulation for each component of cardiovascular system, but it limits the generalization of hypothesis 2 to all the measured variables.

Notwithstanding this, all these effects have been observed on final reactivity indexes only. Results obtained on initial reactivity scores showed the same trends, but they did not reach statistical significance. This can be explained considering that the active coping condition referred to consequences at the end of the task; therefore, the subjects needed to make a certain number of trials prior to evaluating the performance, the attainment of the proposed goal, and thus the avoidance of threat.

Besides, results show that anxiety-state partially depends upon an additive effect of both goal self-efficacy and intrinsic incentive value. As Bandura (1986) postulated, anxiety-state scores are greater when outcome results are important to the individual and they are viewed as poorly controllable, namely, when incentive value is high but self-efficacy is low. It is also worth noting the unexpected interaction found between both goal self-efficacy and anxiety-trait on anxiety-state: despite the progressive overall decline of anxiety-state when goal self-efficacy rises, the subjects with high or very high goal self-efficacy showed an increase in anxiety-state when they presented high anxiety-trait. Nevertheless, concerning hypothesis 3, two unexpected results have been found:

1. Anxiety-state weakly correlates with cardiovascular reactivity (only its correlation with systolic BP reach the statistical significance).
2. The partial correlations and correlations among goal self-efficacy and each of the cardiovascular variables are identical.

Consequently, these results do not confirm the mediator role of anxiety-state. This lead to formulate two alternative explanations: either (1) low sense of control is not the underlying mechanism to the increased cardiovascular reactivity found in very low goal self-efficacy subjects; or (2) low sense of control generates two parallel effects on both affective state (anxiety) and psychophysiological regulation. Further research should be done in order to elucidate this issue.

In sum, all these results depict a complex landscape that can be synthesized as follows:

1. Goal self-efficacy interacts with intrinsic incentive value to regulate cardiovascular reactivity, in such a way that goal self-efficacy affects cardiovascular reactivity especially when incentive value is high, i.e. when the consequences of behavior are important to the person. Despite this, diastolic BP is regulated in a different way.
2. Goal self-efficacy seems to regulate cardiovascular reactivity in a non-monotonic, quadratic way when all the rank of variability in self-efficacy is taken into account, with an exception for diastolic BP.

[2] In order to reduce the risk of inferring false positive conclusions from the results, we assume a conservative statistical criterion of $p<0.025$.

3. Effect of self-efficacy and incentive value on anxiety seems to be additive, and so specific perceived control seems to affect self-reported anxiety and physiological responsiveness in a different way. Moreover, anxiety-state does not seem to be a mediator of the influence exerted by perceived control on psychophysiological reactivity.

In our opinion, the strategy used here to explore the relationship between specific perceived control and physiological reactivity yields a point of view that could contribute to the clarification of some contradictory results from previous research on this matter, since positive or negative slopes can be found when examining different parts from all the rank of self-efficacy, and also, when both factors influencing physiological reactivity (sense of control and effort to control, in Gerin's terms) are simultaneously present.

In the same way, it must be taken into account that the influence exerted by perceived control on physiological reactivity has been postulated as one of the two major ways by which cognitive factors can influence health status (Bandura, 1997; Haidt and Rodin, 1999; O'Leary, 1990, 1992). In regard to this, our results would indicate that extreme degrees of self-efficacy (very low and very high) jointly with a sustained, high worry about performance outcomes (i.e., high incentive value) would lead to greater cardiovascular reactivity, and therefore, to disease. Maybe the deleterious effects attributed to enhanced cardiovascular reactivity, which are still controversial (Linden et al., 2003; Lovallo and Gerin, 2003), could be due to a juxtaposition of several factors. One of them would be the maintenance over time of expectancies with respect to important personal areas (work, study, interpersonal relationships) at levels that lead to a sustained, chronic increase of physiological arousal. This point of view is entirely compatible with the conception of the physiological reactivity as a trait dimension, which could also partially contribute to health status.

4.1. Limitations and prospective

In line with Gerin's proposal, we have explained enhanced cardiovascular reactivity in very low self-efficacy subjects in terms of greater threat appraisal, but in terms of a greater commitment with task and effort in very high self-efficacy subjects. Concerning this, we have measured anxiety-state, but we have neither demonstrated its role as an underlying mechanism, nor measured commitment with goal or intention to effort. Therefore, in order to quantify the relative contribution of these two aspects on peripheral physiological reactivity, future studies should also take into account (1) a methodology specifically designed to study the mediator effects of anxiety and effort, and (2) the measurement of intention to effort and/or related variables: mental workload, commitment with goal, or commitment with task.

Moreover, it would be important to elucidate which autonomic and hormonal pathways are mediating the complex cognitive regulation of peripheral arousal in active coping conditions. In any case, results obtained both in this study and in previous research show a directional fractioning of responses, in Lacey's terms (Lacey, 1967; Cacioppo, 1994; Waldstein et al., 1997, for a review), suggesting the existence of several mediating mechanisms. As it was pointed out above, in regard to the possible role of perceived control on disease, it could be useful identifying the cognitive control pathways (sympathetic, parasympathetic, and hormonal) of physiological reactivity since, as Heponiemi et al. (2004) have stressed, "*health-related significance of…* [physiological] *reactivity may differ substantially depending on its autonomic origin.*"

Also, in regard to methodological limitations, this line of research would be strengthened if in the future a within-subjects design was used, since a great amount of non-explained variance would disappear.

Finally, it is worth underlining that nothing can be said about the causal direction of the found relationships, taking into account the quantitative design of this study. It is assumed in congruence with Bandura (1986) that specific control beliefs and psychophysiological reactivity maintain a reciprocal causation, although we tacitly assume, concerning the present study, that specific control beliefs influence psychophysiological reactivity. In order to provide new evidences to this postulate (yet supported by previous research, but very controversial), an experimental, non-quantitative design must to be employed in future works.

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