

Implicit Anxiety Measure Predicts Cardiovascular Reactivity to an Evaluated Speaking Task

Boris Egloff

Johannes Gutenberg–Universität Mainz

Frank H. Wilhelm, Dana H. Neubauer,

Iris B. Mauss, and James J. Gross

Stanford University

Explicit personality tests assess introspectively accessible self-descriptions. By contrast, implicit personality tests assess introspectively inaccessible processes that operate outside of awareness. Despite their inaccessibility, implicit processes are presumed to influence a variety of current responses. This study tested the hypothesis that an implicit anxiety test should predict cardiovascular reactivity during a speech stressor task. In all, 97 participants completed a measure of attention allocation toward threat (implicit test) and an anxiety questionnaire (explicit test) 1 week before giving an evaluated speech. Whereas the explicit test showed modest relations within only 1 measure of cardiovascular reactivity, the implicit test predicted heart rate and blood pressure reactivity during preparation and delivery of the speech. These findings encourage the broader use of implicit measures to assess cardiovascular responses to threat.

Nonconscious processes shape a wide range of psychological responses (Kihlstrom, 1987). Increased appreciation of this fact has led to a boom in research on the implicit measurement of attitudes and personality variables. Implicit tests refer to introspectively unidentified traces of past experiences that influence current responses (Greenwald et al., 2002). Explicit tests, by contrast, assess introspectively accessible self-descriptions and self-evaluations with questionnaires. Thus, the accessibility to conscious awareness

is the primary distinction between implicit and explicit measures.

Typically, implicit and explicit measures are unrelated or only weakly associated: Low or no correlations have been reported in the field of stereotypes (Banaji & Hardin, 1996), motives (McClelland, Koestner, & Weinberger, 1989), attitudes (Greenwald et al., 2002), and self-esteem (Spalding & Hardin, 1999). Although dissociations of explicit and implicit measures per se are of great theoretical importance, they do not provide sufficient evidence for the validity of implicit tests. What is needed are studies that link implicit measures to outcome variables of interest and demonstrate that implicit measures predict relevant criteria independently from explicit ones.

It has been shown, for example, that implicit racial attitudes predict participants' nonverbal behavior toward Black and White experimenters better than explicit measures (McConnell & Leibold, 2001). Also, in contrast to explicit self-esteem, implicit self-esteem has been associated with apparent anxiety during a self-relevant interview (Spalding & Hardin, 1999). Moreover, implicit prejudice exhibited by White participants correlated with functional magnetic resonance imaging (fMRI) assessed amygdala activation and eye-blink startle responses when participants were shown Black compared with White faces (Phelps et al., 2000). Taken together, implicit measures seem especially promising for the prediction of

Boris Egloff, Department of Psychology, Johannes Gutenberg–Universität Mainz, Mainz, Germany; Frank H. Wilhelm, Department of Psychiatry and Behavioral Sciences, Stanford University; Dana H. Neubauer, Iris B. Mauss, and James J. Gross, Department of Psychology, Stanford University.

This research was supported by National Institute of Mental Health Grants MH56094 and MH58147, National Science Foundation Grant SBR-9728989, and by Johannes Gutenberg–Universität Mainz Grant B6. We thank Brian B. Jones and Ajay S. Kochar for helping with data collection. We also thank members of the Stanford Psychophysiology Laboratory for their help with this research and members of the Mainz Personality Research Unit for their comments on earlier versions of this article.

Correspondence concerning this article should be addressed to Boris Egloff, Department of Psychology, Johannes Gutenberg–Universität Mainz, D-55099 Mainz, Germany. E-mail: egloff@mail.uni-mainz.de

behaviors that are not normally subject to conscious control such as nonverbal behavior and physiological responses.

The Present Study

The present study examined the utility of implicit and explicit anxiety measures in the prediction of cardiovascular responses to a threatening event. Cardiovascular responses are among the most prominent features of anxiety (Wilhelm & Roth, 1998). They have attracted particular attention because over the long term, sustained and exaggerated cardiovascular responses may increase risk for coronary artery disease and hypertension (Blascovich & Katkin, 1993; Lovallo & Wilson, 1992). Consequently, considerable research has focused on identifying antecedents and concomitants of cardiovascular reactivity to threat (Houston, 1989; Kubzansky & Kawachi, 2000). Unfortunately, the use of self-report measures of anxiety as predictors of cardiovascular reactivity has produced mixed and inconclusive findings (Baggett, Saab, & Carver, 1996; Schwebel & Suls, 1999; Witvliet & Vrana, 1995). In light of increasing evidence for both conscious and nonconscious routes to emotion activation (LeDoux, 1995), we reasoned that the study of psychophysiology of anxiety might be particularly well suited for implicit measures to complement explicit ones.

We assessed explicit anxiety by using a standard anxiety measure (the trait form of the State-Trait Anxiety Inventory [STAI; Spielberger, Gorsuch, & Luchene, 1970]). We assessed implicit anxiety by means of the attentional dot probe task, which measures automatic attention allocation toward threatening stimuli (MacLeod, Mathews, & Tata, 1986). This task was adapted from cognitive psychology paradigms that indicated that spatial attention can be assessed from the speed of manual responses to visual probes (Posner, Snyder, & Davidson, 1980). It enables direct measurement of the distribution of visual attention toward threat (see *Method* section).

To elicit anxiety in the laboratory, we used an evaluated speaking task. This task is closely related to the anxiety-inducing stressors that occur in everyday life and is known to produce substantial increases in state anxiety, heart rate, and blood pressure (al'Absi et al., 1997). To permit us to test the generalizability of our findings, we also examined the prespeech preparation period (which is also known to elicit high levels of anxiety), as well as a neutral resting baseline period.

We hypothesized that the implicit anxiety measure would predict cardiovascular reactivity during preparation and delivery of the speech, but not during the resting baseline period. In light of inconsistent prior findings, we made no predictions concerning the association between the explicit anxiety measure and cardiovascular responding during baseline, preparation, and speech periods.

Method

Participants

As part of a larger project on anxiety and physiological responding, 97 female undergraduates from Stanford University participated in individual experimental sessions. They were told that the experiment was a research project on emotion and that we were interested "to learn more about the physiological and subjective qualities of emotion." Their mean age was 19.1 years ($SD = 1.1$ years). They received either course credit or money in exchange for participation.

Procedure

Session 1. Upon arrival at the laboratory, participants completed an explicit anxiety measure (described below). Afterward, attention allocation toward threat-related stimuli was assessed. This constituted our implicit anxiety measure.

Session 2. Approximately 1 week later ($M = 6.5$ days), participants returned to the laboratory for the speaking task. Physiological sensors were attached, and participants viewed a 3-min nature film. After viewing this baseline film, participants rated how anxious they had felt during the film. Participants then were informed that they had to give an impromptu speech on the topic "Is it wrong for the government to execute people?" They received the following instructions:

You will have 3 minutes to prepare with pen and paper, and then you'll have 3 minutes to deliver your speech. Your speech will be videotaped and later scored by a panel of judges who will rate and compare your speech to others given under the same circumstances. The judges will rate your speech on its overall persuasiveness, so it is very important that you try to be as thorough and persuasive as possible, talking for the full 3 minutes.

A videocamera was positioned directly in front of the participant and care was taken to maximize the evaluative nature of this task. During all phases of physiological measurements (baseline film, speech preparation, and delivery), the experimenter was not

present in the room. Communication during these experimental phases was possible through an intercom. Participants remained seated throughout the complete task. After delivering their speech, participants responded to the state measures described below.

Measures

Explicit anxiety measure. The trait form of the STAI contains 20 items that assess enduring symptoms of anxiety on a 4-point Likert scale ranging from 1 (*almost never*) to 4 (*almost always*). The trait anxiety score (computed as item average) in our sample was 2.10 ($SD = 0.55$, Cronbach's $\alpha = .93$).

Implicit anxiety measure. We reasoned that attention allocation toward threat is a defining, but pre-conscious, feature of the anxiety construct (Mogg & Bradley, 1998). To assess automatic attention allocation to threatening stimuli, we used a modified version of the attentional dot probe task (MacLeod et al., 1986). It enables direct measurement of the distribution of visual attention toward threat. A threatening and a neutral word were simultaneously presented on different areas of a computer screen. Attention was measured by a secondary task that involved the detection of a dot, which appeared in the spatial location of either word, immediately after the display of that word was terminated. By examining the impact of the threat versus the neutral word on the relative dot detection tendencies, an index of attention allocation toward or away from threat—independently from general vigilance—was derived.

Research with this task showed that clinically anxious patients often display a tendency to focus on threatening stimuli in their environment, thus showing an attentional bias toward threat (Mineka & Sutton, 1992). However, studies using nonclinical populations have produced inconsistent findings (Mansell, Clark, Ehlers, & Chen, 1999; Mogg & Bradley, 1998). Thus, in the normal range of anxiety, attention allocation toward threat and self-reported trait anxiety show only weak or no associations. These findings mirror the dissociations of explicit and implicit measures that were observed in the domains of stereotypes, attitudes, and self-esteem (Greenwald et al., 2002).

The 20 socially threatening words (e.g., nervous, blush, worried) used in this study were drawn from previous research of the current authors. Each threat word was paired with a neutral word matched for length and frequency to constitute 20 word pairs. The word pairs were presented using a microcomputer. The program (MEL; Schneider, 1988) first presented

a fixation cross for 500 ms and then each word pair for a duration of 1,000 ms, with the words separated on the vertical axis of the computer monitor by a distance of 3 cm (visual angle less than 2°). Then a small dot was presented in the location of one of the word stimuli and remained on the screen until the participants responded. Half the participants used the computer key "M" to indicate when the dot replaced the word in the "up" position and the key "C" for the "down" position, while the other half used the opposite key configuration. After a pause of 1,000 ms, the program continued with the next trial. The trials were presented randomly for each participant. The program started with five practice trials that consisted of neutral word pairs.

The threat word appeared in the up and down positions with equal probability, and the dot followed in the same location, or in the location of the neutral word, with equal probability. Thus, by varying the position of the threat word and the dot independently, four configurations could arise (average reaction times in parentheses): threat word and dot in the up position ($M = 479$ ms, $SD = 91$ ms), threat word and dot in the down position ($M = 467$ ms, $SD = 89$ ms), threat word up and dot down ($M = 461$ ms, $SD = 95$ ms), and threat word down and dot up ($M = 467$ ms, $SD = 93$ ms). False responses and outliers were excluded. Then we computed an index indicating attention allocation toward threatening stimuli by computing the following equation:

$$[(\text{threat word up and dot down} + \text{threat word down and dot up}) - (\text{threat word up and dot up} + \text{threat word down and dot down})]/2.$$

Thus, by subtracting the two configurations where threat word and dot position were identical (faster reactions in case of attention allocation toward threat) from those two configurations where they were not identical (slower reactions in case of attention allocation toward threat), we created an implicit anxiety score where higher values indicate attention allocation toward threatening stimuli. For our sample, there was a slight tendency to withdraw attention from threat ($M = -9$ ms, $SD = 38$ ms).

State measures. To evaluate the impact of the experimental manipulation and to permit the examination of possible alternative interpretations, participants indicated their state anxiety, their state anger, and their task engagement during the speaking task. Participants used an 11-point Likert scale ranging

Table 1
Means and Standard Deviations of the Cardiovascular Variables During Different Experimental Phases

Cardiovascular variable	Experimental phase					
	Neutral film		Speech preparation		Speech delivery	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Systolic blood pressure (mmHg)	126.64 _a	21.30	151.46 _b	30.26	167.46 _c	31.52
Diastolic blood pressure (mmHg)	74.30 _a	15.96	91.71 _b	22.19	104.15 _c	23.72
Heart rate (bpm)	73.98 _a	9.57	81.63 _b	11.58	87.27 _c	12.97

Note. $N = 97$. mmHg = millimeters of mercury; bpm = beats per minute. Physiology scores with different subscripts in each row are different at $p < .01$ (results of paired t tests [two-tailed] with Bonferroni correction for the number of comparisons).

from 0 (*none at all*) to 10 (*extremely*) to indicate how much state anxiety they had felt during the baseline film and the speech. This scale consisted of six items (Anxious, Self-Conscious, Relaxed [reverse scored], Afraid, Worried, and Tense) and showed good internal consistencies: $\alpha = .90$ (speech) and $.76$ (baseline). State anger during baseline and speech was assessed by one item (Angry) using the Likert scale described above. Task engagement was measured by asking participants “How hard did you try during the task?” using an 11-point Likert scale ranging from 0 (*didn't try at all*) to 10 (*tried my hardest*).

Cardiovascular variables. We concentrated on three key cardiovascular measures: heart rate, systolic blood pressure, and diastolic blood pressure (Vander, Sherman, & Luciano, 1998). These three measures are known to be among the most prominent features of the anxiety response and are of high relevance because of their association with cardiovascular disease. Heart rate was calculated by means of RR intervals that were measured from an electrocardiogram. Systolic and diastolic blood pressure were measured continuously from the index finger of the nondominant hand by means of the Finapres 2300 system (Ohmeda, Madison, WI). Period averages of heart rate and blood pressure were computed for the baseline film, speech preparation, and speech delivery. Change scores were then computed for each cardiovascular variable during speech preparation and speech delivery by subtracting the respective values from baseline. The data were screened for multivariate outliers.

Results

Manipulation Check

State anxiety changes from baseline film watching ($M = 1.03$, $SD = 0.96$) to speech delivery ($M = 4.45$, $SD = 2.31$; all reported values are item averages) showed that participants were subjectively af-

ected by the experimental manipulation, $t(96) = 16.2$, $p < .001$. To examine whether the speaking task produced the anticipated responses of the cardiovascular system, we conducted three multivariate analyses of variance with experimental phase as repeated factor and heart rate, systolic blood pressure, and diastolic blood pressure as dependent variables, respectively. These analyses yielded highly significant results, Wilks's $\Lambda < .39$, $F_s(2, 95) > 70$, $ps < .001$. As can be seen in Table 1, preparation and especially the delivery of the speech resulted in substantial increases in blood pressure and heart rate. The implicit and the explicit anxiety measures were unrelated ($r = .08$, *ns*).

Prediction of Cardiovascular Variables by Implicit and Explicit Anxiety Measures

We first computed zero-order correlations between cardiovascular variables and the anxiety measures separately for each experimental phase (see Table 2). To analyze our main research question, we then computed regression analyses where both anxiety measures were entered simultaneously as predictors. These analyses were conducted separately for each experimental phase (neutral film, speech preparation, and speech delivery) and with each cardiovascular variable (systolic blood pressure, diastolic blood pressure, and heart rate) as separate criterion. Thus, we computed altogether nine regression analyses where the implicit anxiety measure and the explicit anxiety measure competed with each other for explaining variance in the cardiovascular variables.¹

¹ To control for the issue of multiple comparisons, reviewers suggested to combine across our cardiovascular measures. For this reason, we computed z scores of each physiological variable and aggregated the three cardiovascular variables within each experimental phase to derive a composite of cardiovascular responding. We then computed

Table 2
Correlations Between Cardiovascular Variables and Anxiety Measures During Different Experimental Phases

Cardiovascular variable	Experimental phase					
	Neutral film		Speech preparation		Speech delivery	
	IA	EA	IA	EA	IA	EA
Systolic blood pressure	.13	.03	.32**	.22*	.34**	.22*
Diastolic blood pressure	.01	-.06	.32**	.18†	.29**	.15
Heart rate	-.07	-.01	.23*	.05	.19†	.05

Note. $N = 97$. IA = implicit anxiety measure; EA = explicit anxiety measure. Cardiovascular variables for the speech preparation and the speech delivery phase are change scores from baseline. † $p < .1$. * $p < .05$. ** $p < .01$.

The first set of regressions involved the prediction of the cardiovascular variables during the baseline film. As could be expected from the zero-order correlations, each of the three regressions showed that cardiovascular variables at baseline were not predicted by the anxiety measures, R^2 s $< .014$, F s(2, 94) < 1 , β s $< .14$, *ns*. In contrast, the second and the third set of regressions showed that change scores in cardiovascular variables during speech preparation and speech delivery could be predicted by the anxiety measures in the expected way (see Table 3). The results of these analyses can be summarized as follows: (a) As hypothesized, during speech preparation and delivery, the implicit anxiety measure consistently predicted reactivity in systolic and diastolic blood pressure as well as in heart rate. (b) The explicit anxiety measure showed inconsistent associations with cardiovascular reactivity. It was unrelated to diastolic blood pressure and heart rate during both phases of the speech. In contrast, it was significantly related to

systolic blood pressure reactivity during speech preparation and delivery.

Secondary Analyses

In this section, we examined several possible alternative explanations for our central findings. After all, cardiovascular responses are not only a prominent feature of anxiety, but are also well-known concomitants of anger (Drummond & Quah, 2001) and task engagement (Cohen et al., 2000). Might state changes in anger and task engagement explain our findings? In addition, our primary analyses did not take into account state changes in anxiety. Might state changes in anxiety also play a critical role in our primary analyses?

Correlational analyses showed that these state measures were only slightly—and nonsignificantly—related to changes in systolic blood pressure, diastolic blood pressure, and heart rate (r s were .07, .12, and .15, respectively, for task engagement; .08, .08, and .03 for state anger; and -.01, -.02, and .06 for state anxiety). We then conducted three stepwise regressions with change scores in systolic blood pressure, diastolic blood pressure, and heart rate as criterion, respectively. In these regression equations, change scores in state anger ($M = 1.26$, $SD = 2.44$ [baseline: $M = 0.10$, $SD = 0.37$; speech delivery: $M = 1.36$, $SD = 2.41$]), change in state anxiety ($M = 3.41$, $SD = 2.07$), and task engagement ($M = 6.81$, $SD = 1.97$) were entered as predictors in Step 1; trait anxiety was entered in Step 2; and the implicit anxiety measure was entered in Step 3 (see Table 4). Thus, we were able to estimate the portion of variance that the implicit anxiety measure shared with the cardiovascular variables when all other measures were controlled for.

In summary, these secondary analyses revealed that

three regressions (one for each phase) with the cardiovascular composite variable as criterion and both anxiety measures as simultaneous predictors. These analyses yielded results similar to those reported in the text: During speech delivery ($R^2 = .139$, $F[2, 94] = 7.57$, $p = .001$), the implicit anxiety measure predicted cardiovascular reactivity ($\beta = .33$, $p = .001$), and the explicit anxiety measure was only slightly related with the criterion ($\beta = .15$, $p = .12$). The same pattern of results was found during speech preparation ($R^2 = .169$, $F[2, 94] = 9.56$, $p < .001$), implicit anxiety measure ($\beta = .36$, $p < .001$), and explicit anxiety measure ($\beta = .17$, $p = .07$). In contrast, at baseline the composite cardiovascular variable was unrelated to both anxiety measures ($R^2 = .002$, $F[2, 94] < 1$, *ns*), implicit anxiety measure ($\beta = .04$, *ns*), and explicit anxiety measure ($\beta = -.02$, *ns*).

Table 3
Results of the Main Analyses: Regressions of Cardiovascular Variables on Anxiety Measures During Speech Preparation and Speech Delivery

Variable/predictor	β
Speech preparation	
Δ Systolic blood pressure: $R^2 = .14, F(2, 94) = 7.84^{**}$	
Implicit anxiety measure	.31 ^{**}
Explicit anxiety measure	.20 [*]
Δ Diastolic blood pressure: $R^2 = .13, F(2, 94) = 6.23^{**}$	
Implicit anxiety measure	.31 ^{**}
Explicit anxiety measure	.15
Δ Heart rate: $R^2 = .05, F(2, 94) = 2.68$	
Implicit anxiety measure	.23 [*]
Explicit anxiety measure	.03
Speech delivery	
Δ Systolic blood pressure: $R^2 = .15, F(2, 94) = 8.35^{**}$	
Implicit anxiety measure	.32 ^{**}
Explicit anxiety measure	.19 [*]
Δ Diastolic blood pressure: $R^2 = .10, F(2, 94) = 5.11^{**}$	
Implicit anxiety measure	.28 ^{**}
Explicit anxiety measure	.13
Δ Heart rate: $R^2 = .04, F(2, 94) = 1.88$	
Implicit anxiety measure	.19 [†]
Explicit anxiety measure	.04

Note. $N = 97$. In each of the six regression analyses, the implicit anxiety measure and the explicit anxiety measure were entered simultaneously. Cardiovascular variables for the speech preparation phase and the speech delivery phase are change scores from baseline.

[†] $p < .1$. ^{*} $p < .05$. ^{**} $p < .01$.

(a) anxiety was the dominant emotion during the speaking task in terms of absolute levels and change scores, (b) state measures were only weakly related to cardiovascular variables, and (c) the implicit anxiety measure remained the most important predictor of cardiovascular reactivity to the speaking task even when explicit anxiety measures (state and trait) and self-reports of task engagement and anger were controlled for.

Discussion

The cardiovascular system is exquisitely tuned to adjust blood flow when we are confronted with physical threats or psychological stressors. Usually, these cardiovascular adjustments are adaptive. However, sustained activation may increase “wear and tear” on the cardiovascular system. Indeed, there is growing evidence that chronically elevated physiological responses to psychological stress may increase risk of cardiovascular disease (Blascovich & Katkin, 1993; Lovallo & Wilson, 1992). These findings have prompted efforts to better understand who is at risk for increased cardiovascular responses to threat.

Prior efforts to predict cardiovascular responses to threatening circumstances have met with modest success. In this study, we complemented the explicit anxiety measures that traditionally have been used with an implicit measure of automatic attention allocation toward threat. As hypothesized, the implicit anxiety measure predicted heart rate and blood pressure changes both during speech preparation and delivery. It is important that our implicit anxiety measure did not predict cardiovascular responding during the neutral baseline period, suggesting that it taps implicit processes relevant to responding to acutely stressful events rather than a broader affective or cognitive set.

Another important feature of our study was that the anxiety measures were taken 1 week apart from the assessment of cardiovascular reactivity to the speech stressor. This provides an indication of the temporal stability of the observed relationship between cardiovascular reactivity and the implicit anxiety measure. Thus, the implicit anxiety measure seems to predict over some length of time, validating the trait character of what is being tested.

Table 4
Results of the Secondary Analyses: Hierarchical Regressions of Cardiovascular Variables on State Measures, Explicit Anxiety, and Implicit Anxiety During Speech Delivery

Step/predictor	β	ΔR^2	R^2
Δ Systolic blood pressure			
Step 1: State measures		.014	.014
State anxiety change	-.06		
State anger change	.10		
Task engagement	.08		
Step 2: Explicit anxiety measure	.28*	.063*	.077
Step 3: Implicit anxiety measure	.31**	.095**	.172**
Δ Diastolic blood pressure			
Step 1: State measures		.025	.025
State anxiety change	-.08		
State anger change	.11		
Task engagement	.12		
Step 2: Explicit anxiety measure	.22†	.037†	.062
Step 3: Implicit anxiety measure	.26**	.068**	.129*
Δ Heart rate			
Step 1: State measures		.023	.023
State anxiety change	.04		
State anger change	.01		
Task engagement	.14		
Step 2: Explicit anxiety measure	.07	.004	.027
Step 3: Implicit anxiety measure	.19†	.034†	.061

Note. $N = 97$. Cardiovascular variables are change scores from baseline. Betas are standardized regression weights at the entry into the equation.
 † $p < .1$. * $p < .05$. ** $p < .01$.

We interpret our findings as suggesting that the implicit anxiety measure taps other sources of common variance with cardiovascular reactivity to threat than self-report measures. How might we explain their relative independent predictive power? First, participants might not be able to perceive (and then report) the activity of the autonomic nervous system because these processes occur without awareness. By contrast, attentional and physiological reactions to threat may be triggered by common circuits (LeDoux, 1995), leading to an association between both types of measures. Second, some participants might be able but not willing to accurately report the anxiety-related thoughts and sensations they experience because of self-presentational concerns or cultural norms. Implicit measures—which may also be called *objective tests of personality* (Cattell, 1950)—are free of response biases and faking tendencies and, thus, might be a good addition to explicit ones.

Several limitations to the present study should be noted. First, because our sample consisted of women it will be important to replicate this finding in a male or mixed sample. Second, although we regard the

speaking task as a well-suited paradigm for investigating coping with threat, replications using other stressors are clearly warranted. Third, we need to learn more about the exact mechanisms by which affective and cognitive responses are associated (Berntson, Sarter, & Cacioppo, 1998; Compton, Heller, Banich, Palmieri, & Miller, 2000). Fourth, before establishing cognitive measures as implicit or objective tests of personality or clinical syndromes, we need to analyze their psychometric properties in terms of stability and convergent validity (Cunningham, Preacher, & Banaji, 2001; Wilhelm & Roth, 2001). Fifth, future studies might benefit from the cardiovascular differentiation of threat versus challenge appraisals (Tomaka, Blascovich, Kelsey, & Leitten, 1993) and explore their relations to implicit measures.

To the best of our knowledge, this study represents the first study of the association between an implicit measure of anxiety and cardiovascular reactivity to a threatening task. Our findings suggest that an index of cognitive processing of threat was able to predict blood pressure and heart rate responses to an evaluated speaking task. We regard this study as an initial

foray into an exciting new research domain at the intersection of emotion, cognitive, personality, and biological psychology.

References

- al'Absi, M., Bongard, S., Buchanan, T., Pincomb, G. A., Licinio, J., & Lovallo, W. R. (1997). Cardiovascular and neuroendocrine adjustment to public speaking and mental arithmetic stressors. *Psychophysiology*, *34*, 266–275.
- Baggett, H. L., Saab, P. G., & Carver, C. S. (1996). Appraisal, task performance, and cardiovascular responses during the evaluated speaking task. *Personality and Social Psychology Bulletin*, *22*, 483–494.
- Banaji, M. R., & Hardin, C. D. (1996). Automatic stereotyping. *Psychological Science*, *7*, 136–141.
- Berntson, G. C., Sarter, M., & Cacioppo, J. T. (1998). Anxiety and cardiovascular reactivity: The basal forebrain cholinergic link. *Behavioural Brain Research*, *94*, 225–248.
- Blascovich, J., & Katkin, E. S. (Eds.). (1993). *Cardiovascular reactivity to psychological stress and disease*. Washington, DC: American Psychological Association.
- Cattell, R. B. (1950). *Personality*. New York: McGraw-Hill.
- Cohen, S., Hamrick, N., Rodriguez, M. S., Feldman, P. J., Rabin, B. S., & Manuck, S. B. (2000). The stability of and intercorrelations among cardiovascular, immune, endocrine, and psychological reactivity. *Annals of Behavioral Medicine*, *22*, 171–179.
- Compton, R. J., Heller, W., Banich, M. T., Palmieri, P. A., & Miller, G. A. (2000). Responding to threat: Hemispheric asymmetries and interhemispheric division of input. *Neuropsychology*, *14*, 254–264.
- Cunningham, W. A., Preacher, K. J., & Banaji, M. R. (2001). Implicit attitude measures: Consistency, stability, and convergent validity. *Psychological Science*, *12*, 163–170.
- Drummond, P. D., & Quah, S. H. (2001). The effect of expressing anger on cardiovascular reactivity and facial blood flow in Chinese and Caucasians. *Psychophysiology*, *38*, 190–196.
- Greenwald, A. G., Banaji, M. R., Rudman, L. A., Farnham, S. D., Nosek, B. A., & Mellott, D. S. (2002). A unified theory of implicit attitudes, stereotypes, self-esteem, and self-concept. *Psychological Review*, *109*, 3–25.
- Houston, B. K. (1989). Personality dimensions in reactivity and cardiovascular disease. In N. Schneiderman, S. M. Weiss, & P. G. Kaufmann (Eds.), *Handbook of research methods in cardiovascular behavioral medicine* (pp. 495–509). New York: Plenum.
- Kihlstrom, J. F. (1987, September). The cognitive unconscious. *Science*, *237*, 1445–1452.
- Kubzansky, L. D., & Kawachi, I. (2000). Going to the heart of the matter: Do negative emotions cause coronary heart disease? *Journal of Psychosomatic Research*, *48*, 323–337.
- LeDoux, J. E. (1995). Emotion: Clues from the brain. *Annual Review of Psychology*, *46*, 209–235.
- Lovallo, W. R., & Wilson, M. F. (1992). A biobehavioral model of hypertension development. In R. J. Turner, A. Sherwood, & K. C. Light (Eds.), *Individual differences in cardiovascular responses to stress* (pp. 265–280). New York: Plenum.
- MacLeod, C., Mathews, A., & Tata, P. (1986). Attentional bias in emotional disorders. *Journal of Abnormal Psychology*, *95*, 15–20.
- Mansell, W., Clark, D. M., Ehlers, A., & Chen, Y. P. (1999). Social anxiety and attention away from emotional faces. *Cognition and Emotion*, *13*, 673–690.
- McClelland, D. C., Koestner, R., & Weinberger, J. (1989). How do self-attributed and implicit motives differ? *Psychological Review*, *96*, 690–702.
- McConnell, A. R., & Leibold, J. M. (2001). Relations among the Implicit Association Test, discriminatory behavior, and explicit measures of racial attitudes. *Journal of Experimental Social Psychology*, *37*, 435–442.
- Mineka, S., & Sutton, S. K. (1992). Cognitive biases and the emotional disorders. *Psychological Science*, *3*, 65–69.
- Mogg, K., & Bradley, B. P. (1998). A cognitive-motivational analysis of anxiety. *Behaviour Research and Therapy*, *36*, 809–848.
- Phelps, E. A., O'Connor, K. J., Cunningham, W. A., Funayama, E. S., Gatenby, J. C., Gore, J. C., & Banaji, M. R. (2000). Performance on indirect measures of race evaluation predicts amygdala activation. *Journal of Cognitive Neuroscience*, *12*, 729–738.
- Posner, M., Snyder, C. R., & Davidson, B. J. (1980). Attention and the detection of signals. *Journal of Experimental Psychology: General*, *109*, 160–174.
- Schneider, W. (1988). Micro Experimental Laboratory: An integrated system for IBM PC compatibles. *Behavior Research Methods, Instruments & Computers*, *20*, 206–217.
- Schwebel, D. C., & Suls, J. (1999). Cardiovascular reactivity and neuroticism: Results from a laboratory and controlled ambulatory stress protocol. *Journal of Personality*, *67*, 67–92.
- Spalding, L. R., & Hardin, C. D. (1999). Unconscious unease and self-handicapping: Behavioral consequences of individual differences in implicit and explicit self-esteem. *Psychological Science*, *10*, 535–539.
- Spielberger, C. D., Gorsuch, R. L., & Luchene, R. E.

(1970). *State-Trait Anxiety Inventory*. Palo Alto, CA: Consulting Psychologists Press.

Tomaka, J., Blascovich, J., Kelsey, R. M., & Leitten, C. L. (1993). Subjective, physiological, and behavioral effects of threat and challenge appraisal. *Journal of Personality and Social Psychology, 65*, 248-260.

Vander, A., Sherman, S., & Luciano, D. (1998). *Human physiology: The mechanisms of body function* (7th ed.). New York: McGraw-Hill.

Wilhelm, F. H., & Roth, W. T. (1998). Taking the laboratory to the skies: Ambulatory assessment of self-report, autonomic, and respiratory responses in flying phobia. *Psychophysiology, 35*, 596-606.

Wilhelm, F. H., & Roth, W. T. (2001). The somatic symptom paradox in DSM-IV anxiety disorders: Suggestions for a clinical focus in psychophysiology. *Biological Psychology, 57*, 105-140.

Witvliet, C., & Vrana, S. (1995). Psychophysiological responses as indices of affective dimensions. *Psychophysiology, 32*, 436-443.

Received July 17, 2001
 Revision received November 23, 2001
 Accepted November 26, 2001 ■



AMERICAN PSYCHOLOGICAL ASSOCIATION

SUBSCRIPTION CLAIMS INFORMATION

Today's Date: _____

We provide this form to assist members, institutions, and nonmember individuals with any subscription problem. With the appropriate information we can begin a resolution. If you use the services of an agent, please do NOT duplicate claims through them and directly to us. PLEASE PRINT CLEARLY AND IN INK IF POSSIBLE.

PRINT FULL NAME OR KEY NAME OF INSTITUTION _____ ADDRESS _____ _____ CITY STATE/COUNTRY ZIP _____ YOUR NAME AND PHONE NUMBER _____ TITLE _____ _____ _____	MEMBER OR CUSTOMER NUMBER (MAY BE FOUND ON ANY PAST ISSUE LABEL) _____ DATE YOUR ORDER WAS MAILED (OR PHONED) _____ PREPAID <input type="checkbox"/> CHECK <input type="checkbox"/> CHARGE <input type="checkbox"/> CHECK/CARD CLEARED DATE: _____ (If possible, send a copy, front and back, of your cancelled check to help us in our research of your claim.) ISSUES: MISSING <input type="checkbox"/> DAMAGED <input type="checkbox"/> VOLUME OR YEAR NUMBER OR MONTH _____ _____ _____
--	---

(TO BE FILLED OUT BY APA STAFF)

DATE RECEIVED: _____	DATE OF ACTION: _____
ACTION TAKEN: _____	INV. NO. & DATE: _____
STAFF NAME: _____	LABEL NO. & DATE: _____

Send this form to APA Subscription Claims, 750 First Street, NE, Washington, DC 20002-4242
 or FAX a copy to (202) 336-5568.

PLEASE DO NOT REMOVE. A PHOTOCOPY MAY BE USED.