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ScienceDirect
Journal of Experimental Social Psychology 43 (2007) 698–711
Journal of Experimental Social Psychology
www.elsevier.com/locate/jesp

Automatic emotion regulation during anger provocation ☆

Iris B. Mauss [a,*], Crystal L. Cook [b], James J. Gross [b]

[a] *Department of Psychology, University of Denver, 2155 South Race Street, Denver, CO 80208, USA*
[b] *Jordan Hall, Stanford University, Stanford, CA 94305, USA*

Received 16 December 2005; revised 27 July 2006
Available online 20 September 2006

Abstract

Individuals frequently have to regulate their emotions, especially negative ones, to function successfully. However, deliberate emotion regulation can have significant costs for the individual. Are there less costly ways to achieve emotion regulatory goals? In two studies, we test the hypothesis that more automatic types of emotion regulation might provide the benefits of deliberate emotion regulation without the costs. Study 1 introduces a priming technique that manipulates automatic emotion regulation. Using this priming technique, we show that relative to priming emotion expression, priming emotion control leads to less anger experience in response to a laboratory anger provocation. Study 2 examines the experiential and physiological consequences of automatic emotion regulation. Results suggest that relative to priming emotion expression, priming emotion control reduces negative emotion experience without maladaptive cardiovascular responding. Together, these findings suggest that automatic emotion regulation may provide an effective means of controlling powerful negative emotions.
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Keywords: Emotion regulation; Control; Anger; Experience; Priming; Physiology; Automatic emotion regulation

Introduction

Emotional impulses, especially potentially destructive ones such as anger, regularly present us with the question of how we ought to respond to them. Should we openly express them or attempt to control them? On the one hand, frequent expression of anger has costs for individuals' well-being, social functioning, and physical health (Baumeister & Exline, 2000; Booth-Kewley & Friedman, 1987; Mayer & Salovey, 1995; Tavris, 1984), suggesting that it is important to regulate negative emotions. On the other hand, emotion regulation often seems to come at a price for individuals' well-being, social and cognitive functioning, and even physical health (e.g., Bonanno, Papa, Lalande, Westphal, & Coifman, 2004; Gross & John, 2003; Muraven, Tice, & Baumeister, 1998; Polivy, 1998), suggesting that emotion regulation may not be a satisfactory solution either. Ideally, there would be a way for individuals to exert the emotion control that they need without "paying a price." Is that possible?

One way to address this question is to re-examine how emotion regulation has been conceptualized in the past. Thus far, interest in emotion regulation has centered principally on *deliberate*, *response-focused* emotion regulation (e.g., Bonanno et al., 2004; Gross & Levenson, 1997; Muraven et al., 1998; Wegner, Erber, & Zanakos, 1993), whose costs may arise from the conscious effort involved in suppressing emotion-related responses. Less attention has been given to automatic (largely unconscious) regulatory processes such as those involved in overlearned habits or culturally transmitted norms (e.g., Cohen, 1997; Fitzsimons & Bargh, 2004; Gollwitzer, 1999). This is unfortunate, because such automatic regulatory processes might operate with less cost to the individual, as they are executed relatively effortlessly, and might thus provide a solution to the dilemma of how

☆ This research was supported by Grant MH58147 to the last author. The authors thank Serena Borja, Jennifer Cheng, Tania Davila, Rebecca Ray, Frank Wilhelm, Chien-Ying Yu, Robert Zajonc, and members of the Stanford Psychophysiology Laboratory for their help with this research.

* Corresponding author.
E-mail address: imauss@psy.du.edu (I.B. Mauss).

0022-1031/$ - see front matter © 2006 Elsevier Inc. All rights reserved.
doi:10.1016/j.jesp.2006.07.003

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negative emotional impulses can be managed. However, at present, only correlational evidence is available to support this hypothesis (Jackson et al., 2003; Mauss, Evers, Wilhelm, & Gross, 2006). In the present studies, we use an experimental manipulation to test whether automatic emotion regulation is an effective means of reducing anger.

Because the literatures on emotion regulation (e.g., Davidson, Jackson, & Kalin, 2000; Gross, 1998; Thompson, 1994) and on automaticity (e.g., Bargh, 1994) are both fraught with conceptual complexities (e.g., Cole, Martin, & Dennis, 2004; Gross, 1998), it is essential to clarify our terms at the outset. We use the term "emotion regulation" to refer to the modification of any aspect of an emotional response, including experience, physiology, and expressive behavior (cf. Eisenberg & Spinrad, 2004; Goldsmith & Davidson, 2004; Gross, 1998; Gross & John, 2003). In the present context, our focus is on processes that reduce one or more aspects of emotion. "Automatic" emotion regulation includes two types of processes: first, implicitly (largely unconsciously) represented *ideas* or *goals* that individuals have regarding emotion regulation, and, second, automatic (largely unconscious and effortless) emotion regulation *behaviors* that individuals engage in during emotional situations (cf. Bargh & Chartrand, 2000; Bargh, Gollwitzer, Lee-Chai, Barndollar, & Troetschel, 2001). It is likely that emotion regulation goals prompt emotion regulation behavior (e.g., Bargh et al., 2001; Shah & Kruglanski, 2003). However, because this cannot be *presumed*, we empirically test whether our priming manipulation is associated with observable responses to an emotional situation.

To set the stage for our studies, we first review the repressive coping literature and the automaticity literature which, as we will see, offer contradictory perspectives on the likely impact of automatic emotion regulation. This review suggests a number of limitations in the existing research that make it difficult to come to firm conclusions about the consequences of automatic emotion regulation. These limitations motivate two studies, in which we experimentally manipulate automatic emotion regulation using a priming technique, and then assess affective responses during an experimental anger provocation. Results from these studies raise the intriguing possibility that automatic emotion control relative to emotion expression leads to effective reduction of feelings of anger but is not accompanied by the experiential "cost" of negative emotion experience (e.g., shame or sadness) or the cardiovascular "cost" of heightened levels of maladaptive cardiovascular activation.

The repressive coping literature: Automatic emotion regulation is costly

As formulated by Freud, defensive inhibition of negative emotional experiences, or *repression*, is a form of automatic emotion control that is motivated by the individual's need to remain unaware of emotions that are intolerably painful or incompatible with the ideal self (Freud, 1930/1961). Freud took a negative view of this type of emotion regulation, postulating that this defensive "work" would come at the cost of expenditure of "psychic energy."

More recently, repression has been examined empirically, and quantitative measures of repressive tendencies have been developed (e.g., Byrne, Golightly, & Sheffield, 1965; Erdelyi, 2001; Paulhus, Fridhandler, & Hayes, 1997; Weinberger, 1995). When tested in laboratory inductions of negative emotions such as stress or frustration, participants high in repression tend to report experiencing lesser negative emotion, but exhibit impaired cognitive and social skills, as well as greater physiological reactivity (e.g., Asendorpf & Scherer, 1983; Brosschot & Janssen, 1998; Schwartz, 1995; Weinberger, 1995). Together, these studies suggest that automatic emotion regulation is associated with lesser negative emotion experience, but that this reduction in negative emotion comes at a cost.

The automaticity literature: Automatic emotion regulation is cost-free

In contrast with the literature on repressive coping, recent research on automaticity suggests that automatic emotion regulation may operate at little cost. These studies have shown that complex judgments, social behaviors, and even the pursuit of higher-level goals (e.g., to cooperate with another person in a competitive game) can be executed automatically (e.g., Bargh et al., 2001; Bodenhausen, Macrae, & Hugenberg, 2003; Kihlstrom, 1987; Nosek, Greenwald, & Banaji, 2005).

Three features of automatic goal pursuit suggest that if automatic emotion regulation operates in a similar fashion to automatic goal pursuit, one would expect it to be effective for controlling feelings and behaviors, and to occur with little or no psychological and physiological cost. First, automatic goal pursuit can occur without subjective awareness, and thereby may consume little or no attentional capacity or subjective effort (Bargh et al., 2001; Chartrand & Jefferis, 2003; Fitzsimons & Bargh, 2004; Koole & Jostmann, 2004). Second, automatic processes presumably are activated quickly and operate efficiently (Bargh, 1994; Kihlstrom, 1987; Webb & Sheeran, 2003; Wilson & Schooler, 1991). Automatic emotion control might thus be antecedent to the emotional response, effectively interrupting the development of an emotional impulse *before* it unfolds and resulting in adaptive experiential and physiological responding (Gross, 1998). Third, automatic emotion control might avoid some of the "side effects" of deliberate emotion control that result from one's conscious awareness of controlling one's emotions. Such side effects of conscious emotion control might emerge from individuals feeling "inauthentic" (not expressing feelings goes against North American notions of expressing oneself; Gross & John, 2003), or from "ironic effects" of control (consciously focusing on the anger to down regulate it might make it more salient, leading to more anger; Wegner, 1994). By avoiding conscious awareness of emotion control, thus, automatic emotion control should presumably also avoid its negative concomitants.

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Automatic emotion regulation: Costly or cost-free?

While the literature on repressive coping suggests that automatic emotion regulation should be costly, the literature on automaticity predicts that automatic emotion regulation should be cost-free. How can we explain these two literatures' opposite conclusions? One explanation is that both literatures contain limitations that make it difficult to come to *any* firm conclusions about automatic emotion regulation. Three such limitations of prior studies stand out. First, automatic emotion regulation mainly has been investigated in correlational designs (e.g., Jackson et al., 2003; Koole & Jostmann, 2004; Mauss et al., 2006; Weinberger, 1995). This makes it difficult to derive causal accounts about the effects of automatic emotion regulation. Second, evidence from the correlational studies is tentative, because it has been difficult to validly and reliably measure individual differences in automatic emotion control. Existing measures that tap automatic emotion regulation and related constructs (e.g., repression, defensiveness, or alexithymia) (1) often rely on explicit self-reports, which might be inappropriate to assess automatic processes (e.g., in the case of alexithymia; Bagby, Parker, & Taylor, 1994), and (2) often have relatively low face validity (e.g., in the case of repression, a combination of high social desirability and low self-reported trait anxiety; cf. Holmes, 1990). Third, few studies have assessed the effects of automatic emotion control in intense, ecologically valid emotional situations, while measuring key affective responses (including experience, behavior, and physiological responding).

The present research

The limitations outlined above draw into sharp relief how little is known about automatic emotion regulation. In the present studies, we sought to address two key questions. First, can automatic emotion regulation be experimentally manipulated? Second, what are the affective consequences of automatic emotion regulation? In Study 1, we experimentally manipulated automatic emotion regulation by priming emotion control versus emotion expression with an adaptation of the Sentence Unscrambling Task (Srull & Wyer, 1979). After the priming task, anger was induced in the laboratory, and participants' anger experience was measured to assess the extent to which the primes influenced anger experience. In Study 2, we used a similar anger provocation to assess the effects of priming emotion control versus emotion expression on anger experience, more general negative emotion experience, and cardiovascular responses. The goal of Study 2 was to establish whether automatic emotion regulation would reduce anger experience without the 'cost' of greater negative emotion or maladaptive physiological responding.

Study 1: Assessing effects of priming emotion regulation on anger experience and behavior

An adaptation of the Sentence Unscrambling Task (Srull & Wyer, 1979) was chosen to manipulate automatic emotion regulation for two reasons. First, it *implicitly* activates (primes) concepts and goals, a feature important to the conclusion that automatic rather than deliberate emotion control was manipulated. Second, this task seemed to be a promising candidate for manipulating a quite abstract goal such as emotion regulation, because versions of this task have been used to prime other types of "high-level" goals (e.g., Bargh, Chen, & Burrows, 1996; Bargh et al., 2001). Careful debriefing allowed us to conclude that participants were not aware of the nature of the primes, ensuring that the regulatory goals were really activated implicitly. Together, these features make the Sentence Unscrambling Task an ideal candidate for priming emotion regulatory goals.

An anger provocation was chosen as the emotional context in which to examine the effects of automatic emotion regulation for two reasons. First, anger is an emotion that frequently arises in everyday life (e.g., Stearns & Stearns, 1986). Second, anger is seen as a negative emotion that must, at times, be controlled (e.g., Ayduk, Mischel, & Downey, 2002; Davidson, MacGregor, Stuhr, Dixon, & MacLean, 2000; Timmers, Fischer, & Manstead, 1998) and at times be expressed (Stearns & Stearns, 1986; Tiedens, 2000). Anger thus seems to be an ideal context for studying the activation of emotion regulatory processes. To minimize confounds due to the influence of impression management, limited introspective insight, and memory biases (e.g., Feldman Barrett, 1997), we chose a standardized laboratory anger provocation rather than descriptions of anger-provoking events. Because anger-related emotion regulatory goals appear to apply with particular force to women (anger expression is seen as more inappropriate for women than for men; Kring, 2000; Timmers et al., 1998), and to eliminate variance due to gender differences, only female participants were used. To assess the effects of the primes, anger experience was assessed via self-reports. Our goal was to test whether emotion regulation primes would affect emotional responses to an anger provocation.

Method

Participants

Thirty-four female students (mean age = 20.6, $SD = 5.6$) participated in this study. The ethnic composition of the sample was mixed: 2% African-American, 5% Asian-American, 58% Caucasian-American, 18% Latino-American, and 17% with multiple ethnic identities.

Priming stimuli

To manipulate automatic emotion regulation, we adapted the Sentence Unscrambling Task, in which participants have to construct grammatical four-word sentences from five-word jumbles. Embedded in 19 of the 42 sentences were either emotion control (e.g., "restrains," "stable," "covered") or emotion expression terms (e.g., "impulsively," "volatile," and "boiled") to prime participants. These words were arrived at by asking two groups of

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Table 1
Mean emotion experience (*SEM*) for baseline, priming task, and anger provocation by priming condition (emotion control versus emotion expression; Study 1)

Measure	Priming condition	Task		
		Baseline	Priming task	Anger provocation
Self-reported anger (0–10)	Emotion control	1.31 (0.40)[a]	1.38 (0.43)[a]	2.44 (0.62)[b]
	Emotion expression	0.73 (0.41)[a]	0.73 (0.45)[a]	3.47 (0.64)[c]

Note. [abc] Cells that do no share a superscript are significantly different from each other at $p < .05$, $ns = 18, 16$.

undergraduate students (34% male, 66% female) to "list the 20 words that come to your mind when you think of the concepts "emotion control" ($N = 90$) or "emotion expression" ($N = 105$). From the resulting lists, words were selected that could be arranged in sentences matched with respect to the other words in each sentence (see Appendix A for all words used in the priming tasks).

Procedure

Participants were recruited for a one-hour study on mood and cognitive performance. In the beginning of the laboratory session, participants watched an emotionally neutral film for 5 min, and then reported on their anger experience (embedded in a set of distractor terms) using a Likert scale ranging from 0 (none at all) to 10 (extremely). The stem for these ratings was "Please use the following scale to indicate the amount of each feeling you were experiencing during the film you just watched" (for the baseline) and "…during the cognitive performance task you just completed" (for the anger provocation).

Participants were then randomly assigned to one of the Sentence Unscrambling Tasks described above. Seventeen participants were in each condition, and the experimenter was blind as to which condition participants were in. On average, participants in the emotion expression prime group took 562 s, $SD = 114$, and participants in the emotion control prime group took 501 seconds, $SD = 209$, $p = .31$, to complete this task. After this priming task, participants again rated their anger experience.

Anger was then induced by having participants perform a tedious task that was based on the d2 concentration endurance test (Spreen & Strauss, 1991), requiring participants to quickly count letters with minor differences on a blurry copy. The experimenter interrupted the participant multiple times with scripted remarks on the participant's performance and cooperation, delivered in an increasingly impatient tone of voice. Participants were told that they were working too slowly and were eventually told to just stop with the task in an irritated tone that implied that the whole session had not gone properly. Participants then completed another anger experience questionnaire. At this point, participants were informed that the experiment was over.

A funneled debriefing procedure was used to assess the extent to which participants were aware of the true nature of the "linguistic task" (the priming task) and the "cognitive performance task" (the anger provocation). Of the 34 participants, none had any suspicion about the priming task. In regards to the anger provocation, 16 (47.1%) did not report any suspicion at all, 14 (41.2%) reported some suspicion, and 4 (11.7%) reported strong suspicion. In secondary analyses, we examined the effects of participants' reported suspicion about the nature of the anger provocation by (1) assessing the correlations between suspicion and anger experience, and (2) entering suspicion as a covariate in analyses. These analyses indicated that (1) anger experience was not significantly associated with suspicion ($r = .06$, $p = .76$), and (2) significance of results when controlling for suspicion was comparable to primary analyses. Therefore, results presented are based on all participants. After the funneled debriefing questions, participants were thanked and fully debriefed.

Results and discussion

The following sections (a) show the effectiveness of our anger provocation, (b) establish that there were no differences between experimental groups before the anger induction, and (c) address our hypothesis regarding automatic emotion regulation.

Effectiveness of the anger provocation

To assess whether the anger provocation was successful in inducing anger relative to the neutral baseline and the non-provocative priming task, an ANOVA with task as a repeated measures factor (baseline, priming task, anger provocation) was conducted. This test revealed a significant effect of task, $F(2, 31) = 53.88$, $p < .001$, $\eta^2 = .64$. Pairwise t tests indicated the anger provocation led to greater experience of anger than the baseline and the priming task, $ps < .001$, $\eta^2 s > .43$.[1] There were no differences between priming task and baseline, $p = .71$, $\eta^2 < .01$ (see Table 1).

[1] The fact that participants experienced "real" anger is illustrated by the following comments, recorded after the study (before the debriefing): "[The experimenter] was stressed out because things were running late was crinkling damn food bags (very annoying when testing concentration);" "[The experimenter] was rude, unfriendly and got annoyed when I asked a question. Very unprofessional and mean;" "I was very annoyed with [the experimenter] because she was impersonal and didn't explain the instructions very well. I felt she blamed me."

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Randomization check

As Table 1 indicates, the two priming conditions did not differ after the baseline or the priming task with respect to their anger experience, $ps > .45$.

Effects of priming emotion regulation

To test the hypothesis that the emotion control prime would lead to lesser increases in anger experience than the emotion expression prime, we conducted an ANCOVA with priming condition as a group factor. To control for individual differences in baseline anger experience, baseline anger experience was entered as a covariate. This test revealed a significant main effect of priming condition, $F(1, 33) = 5.38$, $p = .03$, $\eta^2 = .15$, on self-reported anger experience such that participants primed with emotion control reported less anger experience than participants primed with emotion expression (see Table 1).[2]

Study 2: Assessing the emotional cost of automatic emotion regulation

Study 1 suggests that emotion regulatory goals can be implicitly activated in a laboratory setting. Participants primed with emotion control reported less anger experience after the anger provocation than did those primed with emotion expression. Importantly, the goal to regulate emotions was not operative in an emotionally neutral situation (the priming task itself), but only in an emotional situation (the anger provocation). This is consistent with research indicating that concepts, once activated, lead to corresponding behaviors and feelings only in applicable situations (e.g., Higgins, 1996).

The goal of Study 2 was to address whether automatic anger regulation would be associated with a "cost." Two types of cost were considered: first, negative emotions other than anger,[3] and second, maladaptive physiological responding. It is possible that automatic emotion regulation leads to lessened anger but greater negative emotion such as shame, sadness, or anxiety, as is suggested by psychoanalytically based notion of "anger turned inward" (Freud, 1917/1984; cf. Johnston, Rogers, & Searight, 1991). Likewise, it is possible that automatic emotion regulation leads to maladaptive physiological responding, similar to that associated with emotion suppression (e.g., Gross, 1998) and repression (e.g., Weinberger, 1995).

In Study 2 we employed the same general design as Study 1, again using a Sentence Unscrambling Task to prime emotion control versus emotion expression in the context of an anger provocation. Three domains of affective responding were measured, including anger experience, negative emotion experience, and cardiovascular responses. Study 2 more broadly sampled emotion experience than Study 1, including sadness, anxiety, guilt, shame, worry, fear, nervousness, as well as reverse-scored happiness, joy, pleasantness, and amusement. This broad measure of global emotion experience allowed us to more broadly index potential effects of automatic emotion regulation. Measures of cardiovascular responding broadly sampled indices of cardiovascular activation, allowing us to (1) maximize chances of uncovering potential deleterious effects of automatic emotion control, (2) differentiate not just activation from deactivation (cf. Lacey, 1967) but more versus less adaptive *patterns* of cardiovascular responding, including (more adaptive) challenge versus (less adaptive) threat patterns (Mendes, Reis, Seery, & Blascovich, 2003; Tomaka, Blascovich, Kelsey, & Leitten, 1993), as well as the degree of parasympathetic withdrawal (Porges, 1995).

As in Study 1, we examined participants' responses to an anger-provoking interaction. A different anger provocation was used to assess whether findings from Study 1 would generalize to a more interactive context. In this provocation, participants counted backwards by 7 or 13 s from a large number. Participants were instructed to count out loud and were interrupted more frequently than in Study 1, allowing for more interactions between participant and the "annoying" experimenter. Unlike Study 1, all instructions were pre-recorded (they allegedly came over the intercom from the experimenter in the next room), rendering the procedure more standardized across participants. In addition, Study 2 included two separate anger provocations: one before the priming task, which functioned as the neutral (unprimed) control condition, and one after the priming task, which allowed assessing the effects of the primes.

Hypotheses

Based on results from Study 1, we predicted that the emotion control prime, relative to the emotion expression prime, would lead to lesser self-reported experience of anger. Our goal was additionally to test competing predictions as to whether automatic emotion control would be costly (repressive coping literature) or cost-free (automaticity literature) in terms of self-reported experience of negative emotions and cardiovascular responding during the anger provocation.

Method

Participants

Participants were 114 female college students (mean age = 20.8, $SD = 3.2$). The ethnic composition of the sample was mixed: 52.2% Caucasian, 22.6% Asian-American, 11.3% Hispanic, 3.5% African-American, and 0.9% Native American, with 6.1% of participants coming from mixed

[2] Preliminary analyses were conducted in which ethnicity was entered as an additional factor. Ethnicity did not interact with priming condition and thus was not considered further. The same results were obtained in Study 2.

[3] While Study 1 had not found effects of emotion regulation primes on negative emotion experience, it did not present an optimal test of this hypothesis, because it had relatively small sample sizes and did not sample negative emotions very broadly. Negative emotion terms assessed in Study 1 included sad, anxious, guilty, happy (reverse scored), joyful (reverse scored), and pleased (reverse scored).

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ethnic backgrounds, and 3.4% of participants electing not to declare their ethnicity. Three participants decreased (as opposed to increased or stayed at the same level) in anger experience from the neutral baseline to the anger provocation and were excluded from subsequent analyses. This left 111 participants for analyses, 55 in the emotion control condition and 56 in the emotion expression condition.[4]

Procedure

Participants were recruited for a one-hour study on mood and cognitive performance. Participants were greeted by a female research assistant, who attached a series of physiological sensors to the participant and then stated she would send the experimenter in. While the participant was waiting, an emotionally neutral, five-minute nature film was shown to induce relatively neutral mood across participants and to establish baseline cardiovascular activation. Once the video was over, participants rated their current emotional experience and the experimenter (a different person from the research assistant) entered the room. The experimenter was brisk with all participants and made little eye contact. She informed participants that they'd be participating in two cognitive tasks, one linguistic and one mathematic, and that the two would be communicating through an intercom system.

At this point, the anger provocation began. Pre-recorded questions and directions were played over the intercom to the participant. Participants were asked to count backwards quickly in increments from a large number (e.g. "Count backwards in steps of 7 from 18,652"). Between each counting task, they were told that they were moving too often, producing physiological artifacts and rendering the data useless, and that they were not speaking loudly enough. After three counting tasks (each lasting one minute), the experimenter informed the participant that they would have to return to the task later. Then, participants again rated their emotion experience.

After the first anger provocation, participants were shown a second five-minute neutral film to allow them to return to more neutral mood before receiving their second "cognitive task," the same Sentence Unscrambling Tasks as in Study 1. Assignment was done randomly, and the experimenter was blind as to which condition participants were in. Once participants were finished, they again rated their emotion experience. The experimenter then announced that they would continue with "the other task" to begin the second anger provocation. Participants then were brusquely instructed to continue the earlier counting task, which was stopped after two one-minute long counting attempts. Again, participants rated their emotion experience.

Then, sensors were removed and the same funneled debriefing procedure as in Study 1 was used to assess the extent to which participants were aware of the true nature of the "linguistic task" (the priming task) and the "cognitive performance task" (the anger provocation) (Bargh & Chartrand, 2000). Of the 111 participants, none had any suspicion about the priming task. About the anger provocation, 83 (74.8%) did not report any suspicion, 26 (23.4%) reported some suspicion, and 2 (1.8%) reported strong suspicion. After the funneled debriefing, participants were thanked and debriefed.

To examine the effects of participants' reported suspicion, we (1) assessed the correlations between suspicion and emotion experience, and (2) entered suspicion as a covariate in analyses. These analyses indicated that (1) emotion experience was not significantly associated with suspicion (all rs were $< .08$, all ps $> .41$), and (2) significance of results when controlling for suspicion was comparable to primary analyses. Therefore, results are based on all participants.

Measures

Emotion experience. Anger and other emotions were assessed after the baseline, the first anger provocation, the priming task, and the second anger provocation with ratings on 11-point Likert scales, ranging from 0 (*none at all*) to 10 (*extremely*). Anger experience was measured using one item. A global emotion experience composite was formed using the terms sad, anxious, guilty, ashamed, worried, afraid, nervous, happy (reverse scored), joyful (reverse scored), amused (reverse scored), and pleased (reverse scored) (Cronbach's $\alpha = .83$). To control for individual differences in baseline emotion experience, all analyses were performed on change scores from the baseline.

Cardiovascular responding. Six measures of cardiovascular responding were sampled at 400 Hz using laboratory software. Measures were selected that would allow us to broadly sample sympathetic and parasympathetic influences on various aspects of the cardiovascular response known to be relevant to anger responding (e.g., Herrald & Tomaka, 2002; Stemmler, 1997). These measures included: heart rate (beats per minute), mean arterial blood pressure (MAP), total peripheral resistance (TPR), peripheral sympathetic responding (as indexed by a composite of finger pulse transit time, finger pulse amplitude, ear pulse transit time, and finger temperature), central sympathetic responding (as indexed by pre-ejection period, PEP), and parasympathetic activation (as indexed by heart rate variability). In addition, somatic activity was assessed through the use of a piezo-electric device attached to the participant's chair, which generates an electrical signal proportional to the participant's overall body movement in any direction. This measure of activity was then used to control for the effects of body movement on cardiovascular activation.

[4] Technical problems led to faulty ECG measurements in six cases (three in each condition), to faulty blood pressure readings in ten cases (four in the emotion control condition and six in the emotion expression condition), to faulty ZCG readings in nine cases (six in the emotion control condition and three in the emotion expression condition), and to faulty somatic activity measurements in nine cases (four in the emotion control condition and six in the emotion expression condition).

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Heart rate (HR; beats/Min) was calculated from RR intervals in the electrocardiogram. *Mean arterial blood pressure* (MAP; mm Hg) was obtained from the third finger of the non-dominant hand by means of the Finapres™ 2300 (Ohmeda, Madison, WI) system. From this signal, beat-to-beat stroke volume was measured using Wesseling's pulse-contour analysis method (BEATFAST, TNO-Biomedical Instrumentation, Amsterdam). *Cardiac output* (CO; l/Min) was calculated as stroke volume × heart rate. *Total peripheral resistance* (TPR) was calculated as (MAP × 80)/CO. *Finger pulse amplitude* (FPA; A-D units) was assessed using a plethysmograph transducer attached to the tip of the participant's second finger. *Finger pulse transit time* (FPTT; msec) was indexed by the time (in ms) elapsed between the closest previous R-wave from the ECG and the upstroke of the peripheral pulse at the finger. *Ear pulse transit time* (EPTT; ms) was determined similarly using a UFI plethysmograph transducer attached to the participant's left ear. *Finger temperature* (degrees Fahrenheit) was measured with a thermistor attached to the palmar surface of the tip of the fourth finger.

Peripheral sympathetic activation was assessed with a composite used previously (Gross & Levenson, 1997) consisting of reversed and *z*-scored FPA, FPTT, EPTT, and finger temperature (Cronbach's $\alpha = .49$ for the baseline, .58 for the first anger provocation, and .64 for the second anger provocation). *Central sympathetic activation*, as indexed by pre-ejection period (PEP; with smaller values of pre-ejection period indexing greater central sympathetic activation), was derived from the ECG and the ZCG waves. The ZCG signal was obtained with an HIC-2000 Bio-Impedance Cardiograph (Bio-Impedance Technology, Inc.) using a set of four spot electrodes, applied at the front of the neck above the collar bone, the nape of the neck, the xiphisternal junction, and the lower back. A 4 mA AC 400 kHz current was sent through the two back sensors and transthoracic impedance as well as the first derivative of basal impedance (or the change of impedance over time), were obtained from the two front sensors. Pre-ejection period is identified as the time (in ms) elapsed between the Q point on the ECG wave (the left ventricle contracting) and the B inflection on the ZCG wave (the opening of the aortic valve). *Parasympathetic activation* was indexed by heart rate variability (ms^2), a measure derived from the ln-transformed high-frequency (0.13–0.5 Hz) $R-R$ interval power from the ECG.

Customized analysis software (Wilhelm, Grossman, & Roth, 1999) was used for physiological data reduction, artifact control, and computation of average physiological scores for each participant for the initial 5-min baseline, across the three 1-min counting tasks for the first anger provocation, for the priming task (average time for emotion expression prime = 510 s, $SD = 171$; average time for emotion control prime = 534 s, $SD = 235$; $p = .55$), and across the two 1-min counting tasks for the second anger provocation. To control for individual differences in baseline activation, all analyses were performed on change scores from the baseline.

Data analysis

The following data analytic strategy was used. (a) We conducted omnibus ANOVAs for each DV, with priming condition (emotion control versus emotion expression) as a group factor and task (baseline, anger provocation 1, priming task, anger provocation 2) as a repeated-measures factor. (b) To assess the effectiveness of the anger provocations, we followed up on main effects of task by using paired *t* tests. (c) To ascertain that randomization was successful, groupwise *t* tests comparing the two priming conditions were performed. (d) To test our hypotheses, we followed up on priming condition by task interactions with ANCOVAs for each DV, with priming condition (emotion control versus emotion expression) as a group factor, task (anger provocation 1 versus anger provocation 2) as a repeated-measures factor, and baseline responding as the covariate. Priming condition by task interactions in these ANCOVAs were followed up on with focused univariate ANCOVAs.

Results and discussion

The following sections show (a) results of omnibus ANOVAs, (b) tests for the effectiveness of the anger provocation, (c) randomization checks, and (d) tests of our hypotheses.

Results of the omnibus ANOVAs

For anger experience, the ANOVA revealed no significant effect of priming condition, $p = .91$, a significant effect of task, $F(3, 330) = 38.78$, $p < .001$, $\eta^2 = .27$, and a significant priming condition by task interaction, $F(3, 330) = 2.54$, $p = .05$, $\eta^2 = .03$. For global emotion experience, the ANOVA revealed no significant effect of priming condition, $p = .20$, a significant effect of task, $F(3, 330) = 80.54$, $p < .001$, $\eta^2 = .43$, and a trend for the priming condition by task interaction, $F(3, 330) = 2.15$, $p = .07$, $\eta^2 = .02$. For the six measures of cardiovascular responding, the ANOVAs revealed significant effects of task, all ps < .02, all η^2s > .05, and no significant effects of priming condition or of the interaction between priming condition and task (all ps > .29, all η^2s < .01).[5]

Effectiveness of the anger provocations

To assess the effectiveness of the anger provocations, pairwise *t* tests were used to follow up on main effects of task. As is evident in Tables 2 and 3, both anger provocations were successful in inducing anger experience, shifts in global emotion experience, and greater cardiovascular responding relative to the baseline and the non-provocative priming task (with the exception of total peripheral resistance; all ps < .05).

[5] To control for potential effects of somatic activity, secondary analyses were performed in which somatic activity was entered as a covariate in analyses. Results were comparable to those of primary analyses.

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Table 2
Mean emotion experience (*SEM*) for baseline, first anger provocation, priming task, and second anger provocation by priming condition (emotion control versus emotion expression; Study 2)

Measure	Priming condition	Task			
		Baseline	Anger provocation 1	Priming task	Anger provocation 2
Anger (0–10)	Emotion control	0.57 (0.16)[a]	2.18 (0.37)[b]	0.84 (0.23)[a]	1.66 (0.36)[c]
	Emotion expression	0.40 (1.15)[a]	1.93 (0.34)[b]	0.58 (0.22)[a]	2.29 (0.34)[b]
Negative emotion (0–10)	Emotion control	2.80 (0.15)[a]	4.68 (0.22)[b]	3.02 (0.16)[a,c]	3.96 (0.20)[d]
	Emotion expression	2.89 (0.14)[a]	4.58 (0.22)[b,e]	3.18 (0.15)[c]	4.40 (0.19)[d,e]

Note. [abcde] Within each measure, cells that do no share a superscript are significantly different from each other at $p < .05$, $ns = 55, 56$.

Table 3
Mean cardiovascular responses (*SEM*) for baseline, first anger provocation, priming task, and second anger provocation by priming condition (emotion control versus emotion expression; Study 2)

Measure	Priming condition	Task			
		Baseline	Anger provocation 1	Priming task	Anger provocation 2
Heart rate (beats/min)	Emotion control	70.9 (1.7)[a]	84.9 (2.3)[b]	75.3 (1.8)[c]	84.9 (2.3)[b]
	Emotion expression	72.7 (1.7)[a]	86.6 (2.3)[b]	77.4 (1.8)[c]	86.7 (2.3)[b]
Mean arterial blood pressure (mm Hg)	Emotion control	81.7 (1.9)[a]	103.4 (2.4)[b]	98.6 (2.4)[c]	110.8 (2.5)[d]
	Emotion expression	80.8 (1.9)[a]	99.1 (2.4)[b]	96.5 (2.4)[c]	106.8 (2.5)[d]
Total peripheral resistance (dyne s/cm^5)	Emotion control	21.1 (0.8)[a,b]	20.4 (0.8)[a]	21.5 (0.7)[b]	21.3 (0.9)[b]
	Emotion expression	20.4 (0.8)[a,b]	19.1 (0.8)[a]	19.6 (0.7)[a,b]	20.2 (1.0)[b]
Peripheral sympathetic activation (composite, standardized units)$_x$	Emotion control	0.00 (0.08)[a]	0.78 (0.10)[b]	0.65 (0.10)[c]	1.12 (0.10)[d]
	Emotion expression	0.00 (0.08)[a]	0.75 (0.09)[b]	0.69 (0.10)[c]	1.07 (0.10)[d]
Central sympathetic activation (Pre-ejection period, ms)	Emotion control	108.6 (2.1)[a]	101.4 (2.3)[b]	105.5 (2.1)[c]	101.2 (2.5)[b]
	Emotion expression	108.6 (2.0)[a]	104.5 (2.3)[b]	107.6 (2.0)[c]	102.8 (2.5)[b]
Parasympathetic activation (heart rate variability, msec^2)	Emotion control	13.8 (0.16)[a]	13.1 (0.16)[b]	13.5 (0.14)[a]	13.2 (0.17)[b]
	Emotion expression	13.6 (0.15)[a]	13.2 (0.15)[b]	13.4 (0.13)[a,b,c]	13.2 (0.20)[b]

Note. [abcd] Within each measure, cells that do no share a superscript are significantly different from each other at $p < .05$, $ns = 49–56$ (depending on missing values).
$_x$Composite of reverse scored finger pulse amplitude, finger pulse transit time, ear pulse transit time, and finger temperature. To preserve change across tasks, each measure was standardized with the baseline's mean and standard deviation.

Randomization check

To ascertain that randomization was successful, group-wise *t* tests were used to compare the two priming conditions before the second anger provocation. As Tables 2 and 3 indicate, none of the effects of priming condition were significant (all $ps > .09$),[6] indicating that there were no group differences in emotion experience or cardiovascular responding before or during the priming manipulation.

Effects of priming emotion regulation

To examine whether priming led to differences in emotional responding, we followed up on the priming condition by task interactions from the omnibus ANOVAs by using ANCOVAs with priming condition (emotion control versus emotion expression) as the group factor, task (anger provocation 1 versus 2) as a repeated-measures factor, and baseline emotion experience as the covariate.

Emotion experience. For anger experience, this analysis revealed a significant interaction between priming condition and task, $F(1, 109) = 5.23$, $p = .02$, $\eta^2 = .05$ (see Fig. 1a). To follow up on this interaction, we conducted two repeated-measures ANCOVAs (comparing anger provocation 1 with anger provocation 2 for both priming conditions separately, using baseline anger experience as the covariate) and one one-way ANCOVA (comparing the emotion control to the emotion expression priming condition during anger provocation 2, using baseline anger experience as the covariate). These tests revealed that there was a significant decrease in anger experience from anger provocation 1 to anger provocation 2 in the emotion control condition, $p = .05$. There was no significant increase in anger experience from anger provocation 1 to anger provocation 2 in the emotion expression condition, $p = .16$. Individuals primed with emotion control reported lesser anger experience during anger provocation 2 than those primed with emotion expression, $p = .04$.

For global emotion experience, the ANCOVA also revealed a significant interaction between priming condition and task, $F(1, 109) = 4.25$, $p = .04$, $\eta^2 = .04$ (see Fig. 1b). To follow up on this interaction, we used the same tests as for anger experience. These tests revealed that there was a significant decrease in global negative emotion experience from anger provocation 1 to anger provocation 2 in the

[6] The marginal effect was in found for MAP for the first anger provocation. All other *p*s are >.16.

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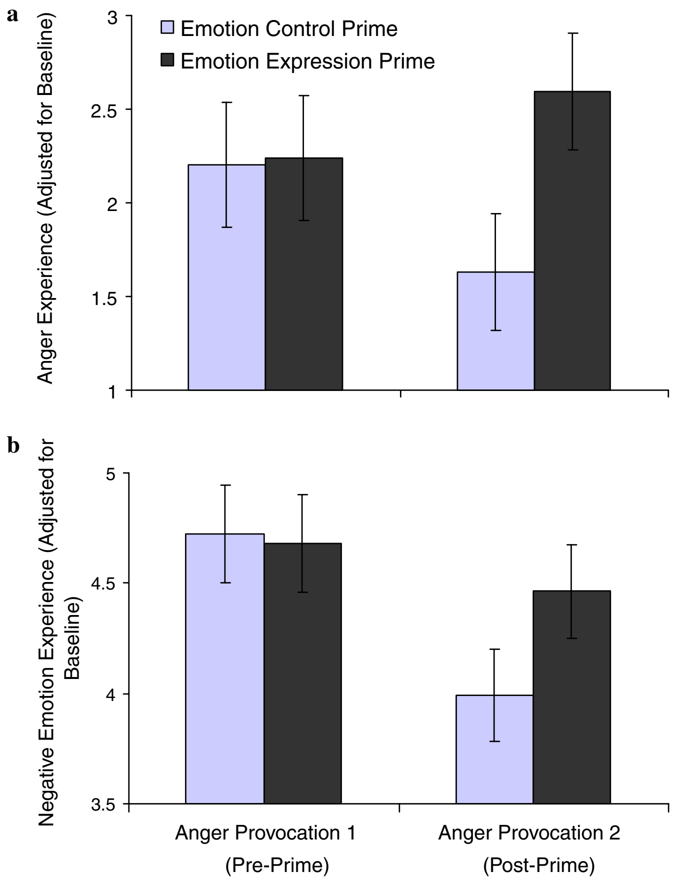

Fig. 1. (a and b) Mean (*SEM*) (a) anger experience and (b) negative emotion experience for the first and the second anger provocation (adjusted for baseline responding) by priming condition (emotion control versus emotion expression; Study 2).

emotion control condition, $p < .001$. There were no differences between anger provocation 1 and anger provocation 2 for the emotion expression condition, and the ANCOVA comparing the two priming conditions during anger provocation 2 was not significant, $ps > .12$.

Cardiovascular responding. Despite the fact that interactions between priming condition and task were not significant, we used the same more focused ANCOVAs to minimize type II error. These analyses again revealed no interactions between priming condition and task (all $ps > .43$, all η^2s $< .01$). This result, together with the fact that cell sizes are adequate ($ns = 49$–56) and that a broad range of cardiovascular measures was sampled, suggests that the priming manipulation did not affect cardiovascular responding.

General discussion

Individuals frequently have to regulate their emotions, especially negative ones, to function in life. However, research suggests that deliberate emotion regulation can be costly (e.g., Gross & John, 2003; Muraven et al., 1998; Polivy, 1998). This presents a dilemma, namely whether to express emotions or pay the price of exerting costly control.

We have suggested that considering a type of emotion regulation that has been neglected in the past—automatic emotion regulation—may help to resolve this dilemma. After all, recent studies from personality and social psychology indicate that many goals may be pursued automatically, and often in ways that do not appear to be costly (Bargh et al., 2001; Mauss et al., 2006). However, it has been difficult to assess this hypothesis based on existing research, mainly because prior studies of automatic emotion regulation have relied principally on correlational designs. The present studies were designed to fill this gap by (1) providing an experimental manipulation of automatic emotion regulation by priming emotion control versus emotion expression (Study 1), and (2) assessing the emotional and physiological cost of automatic emotion control relative to automatic emotion expression (Study 2).

Results from Study 1 suggest that automatic emotion regulation leads to lesser anger experience after a laboratory anger provocation. Results from Study 2 show that automatic anger regulation leads to lesser anger experience after a more interactive anger provocation. Importantly, Study 2 also suggests that automatic anger regulation does not come at the cost of elevated negative emotions or maladaptive cardiovascular responding (e.g., increased sympathetic activation, threat pattern of physiological responding). Together, these results have implications for our understanding of emotion regulation, socio-cultural variation in emotion, and important domains of individuals' lives.

Implications for emotion regulation

While there is a growing body of literature concerning deliberate types of emotion regulation (e.g., Ayduk et al., 2002; Davidson et al., 2000, 2000; Gross, 1998), much less is known about relatively automatic types of emotion regulation.

One important implication of our findings is that emotion regulation goals—like other high-level goals—can be activated automatically. Psychologists have long assumed that "lower-level" processes such as affective responding or basic perceptual processes could take place automatically (Devine, 1989; Fazio & Olson, 2003; Greenwald & Banaji, 1995; Winkielman & Berridge, 2004; Zajonc, 1980). In addition, research on automaticity by Bargh and colleagues has suggested that even "higher-level" processes such as the execution of goals (e.g., performing well on a task or cooperating with someone in a competitive game) can be activated and executed automatically (e.g., Bargh et al., 2001). This development has lead to a reconceptualization of seemingly "willful" human activities such as goals, with more emphasis being placed on automatic processes. The present findings support this view. Like other important human activities that were thought to be solely in the realm of deliberate and conscious functioning, it seems that emotion regulation may also at times operate automatically.

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A second important implication of our findings is that predictions based on the automaticity literature may be correct regarding the cost-free nature of automatic emotion regulation. While the emotion control prime led to effective reduction of anger experience, it did not entail a cost in terms of negative emotion experience or maladaptive physiological responding. This suggests automatic emotion control as an adaptive regulatory strategy. That said, it bears noting that Study 2 showed *no* association between automatic emotion regulation and cardiovascular responding. Given that participants primed with emotion control (versus expression) showed lesser anger experience, one might expect them to also show *lesser* cardiovascular responding. One explanation for this lack of an effect is that physiological measures are multiply determined. For example, effortful involvement in the counting task (with strong cardiovascular effects; e.g., Bosch, de Geus, & Veerman, 2003) might have counteracted any beneficial effects of lesser anger experience and could thus have obscured effects of the experimental manipulation. However, the fact that there is *no* maladaptive effect of the emotion control prime on cardiovascular responding distinguishes this type of emotion control from other types of emotion control (e.g., suppression or repression), which have been reliably associated with maladaptive physiological responding (e.g., Demaree, Schmeichel, Robinson, & Berntson, 2006; Gross, 1998; Gross & Levenson, 1997; Weinberger, 1995).

Implications for socio-cultural variation in emotion

The present findings also have implications that extend beyond the individual. Which regulatory strategies an event evokes is not just a function of the individual but also the social and cultural context (Kitayama, Karasawa, & Mesquita, 2004; Markus & Kitayama, 1992; Tsai, Knutson, & Fung, 2006). Gender, ethnic background, and socioeconomic status are but examples of socio-cultural factors that systematically affect how a person thinks about emotions and emotional events (e.g., Cohen, Nisbett, Richard, & Bowdle, 1996; Mesquita, 2002; Timmers et al., 1998).

Admittedly, such social norms are rarely conveyed by flashing words in front of people. However, just like the primes used in the present study, social norms are often conveyed implicitly through reinforcement contingencies, social models, and individuals' engagement with cultural practices (Cohen, 1997; D'Andrade, 1984; Rudman, 2004). For example, individuals socialized to regulate emotions from early childhood would be more likely to engage in automatic emotion regulation, without this norm even entering their awareness. By extension, the present results suggest that cultural norms to control one's emotions should lead to automatic and hence cost-free emotion control. The fact that deeply ingrained, culturally transmitted norms are often inaccessible to introspection might explain why cultural differences have been difficult to understand using explicit measures and manipulations. Thus, by providing a framework for automatic regulatory goals regarding emotion, the present research can help shed light on the complex mechanisms by which socio-cultural factors affect emotional responding.

Implications for well-being, psychosocial functioning, and physical health

People encounter events that might provoke anger on a daily basis in many different situations. Often, these situations do not allow for free expression of anger, and how someone deals with them has implications for a wide range of domains, including well-being, psychosocial functioning, and physical health (e.g., Booth-Kewley & Friedman, 1987; Chemtob, Novaco, Hamada, Gross, & Smith, 1997; Howells, 2004). This suggests that greater automatic emotion control would be associated with positive outcomes in these domains.

Moreover, the present findings suggest how positive change in individuals' ability to reduce anger might be best achieved. A number of anger management interventions are currently used (e.g., Deffenbacher & McKay, 2000; Fein, 1993; Gerzina & Drummond, 2000; Zillmann, 1993), and while many seem to be quite successful, they often involve a number of different interventions, leaving it unclear which components of treatments are effective, and why they are effective. Our findings support the idea that anger control strategies might be most effective when they become automatic such as might be the case with highly overlearned strategies and habitual responses.

However, can we expect the automatic emotion regulatory processes described in the present studies to be at work in everyday life, when no words relating to emotion regulation happen to be presented to individuals? Under what conditions, if any, might individuals profit from this advantageous type of emotion control outside of laboratory contexts? Recent research suggests that social norms can be implicitly activated by social and situational cues encountered in everyday life, and affect individuals' behavior without the norm entering their awareness (Aarts & Dijksterhuis, 2000, 2003; Fitzsimons & Bargh, 2004). For example, Aarts and Dijksterhuis (2003) showed that the presentation of pictures of libraries (an environment associated with the norm to be quiet) led to automatic activation of the representation of silence, as well as to participants speaking more quietly in a seemingly unrelated word pronunciation task. These findings need to be followed up by studies directly relevant to emotion regulatory goals. However, they are consistent with the interpretation that emotion regulatory norms that are implicitly activated by social and situational cues would lead to automatic emotion control and, by extension, have beneficial effects.

Limitations and future directions

The present studies suggest a number of compelling directions for future research. In the following section, we consider five of the most important limitations and future directions.

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First, it will be important to assess the extent to which automatic emotion control has comparable effects in other contexts. The words used in the Sentence Unscrambling Task were not specific to anger, suggesting that the current results might generalize to other emotional contexts. Automatic emotion control may even share operating principles with other forms of self-regulation. Just like anger, mental processes such as attention, and undesirable behaviors such as binge eating, are subject to the problem of how they can be controlled without the often ironic and deleterious effects of attempted deliberate control (e.g., Polivy, 1998; Wegner, 1994). The current research dovetails with other research showing that automatic self-control might be a very effective means of reaching desired mental processes and behaviors (e.g., Aarts & Dijksterhuis, 2000, 2003; Fitzsimons & Bargh, 2004; Gollwitzer, 1999). However, this hypothesis needs to be formally assessed in future studies.

Second, it will be important to examine differences in the effects of different types of anger regulation as a function of participants' age, gender, and culture (e.g., Cohen et al., 1996; Evers, Fischer, Rodriguez Mosquera, & Manstead, 2005; Seeley & Gardner, 2003). While ethnic background did not moderate the current results, small sample sizes did not allow for formal tests of this finding. Thus, it will be important to systematically assess diverse samples of both genders in future studies.

Third, although we have argued that lower levels of anger experience are adaptive in the present laboratory contexts, future studies will be needed to directly speak to the adaptiveness of automatic emotion regulation in various types of anger-provoking situations. Awareness and expression of anger, at least to a certain degree, are seen by some researchers as important to psychological health (e.g., Davey, Day, & Howells, 2005; Polivy, 1998; Roffman, 2004), as motivators to achieve social change (e.g., Campos, Frankel, & Camras, 2004; Tiedens, 2000), or as evolutionarily adaptive (e.g., Darwin, 1872/1965; Panksepp, 1994). Might the low anger levels associated with automatic emotion control thus be a disadvantage rather than an advantage in some situations? It is important to emphasize that participants who automatically controlled their anger did not completely deny feelings of anger—even they reported some measure of anger during the provocation. Furthermore, they did not report increased negative emotion—one of the predictions made by proponents of the "expression is healthy" argument. Together, these findings are consistent with the interpretation that the level of anger control achieved by automatic emotion control is quite adaptive. However, future studies that systematically manipulate the situational context are needed to test this conclusion, as are longitudinal studies designed to understand the longer-term effects of automatic emotion control for well-being, social functioning, and health.

Fourth, the present study did not provide a neutral (no regulation) condition, which makes it difficult to tell whether the emotion control condition led to decreased emotion, or whether only the emotion expression condition led to increased emotion (or delayed habituation) while the emotion control condition was "inactive." However, the fact that participants primed with emotion control exhibited a significant *decrease* in anger experience while those primed with emotion experience did not exhibit a significant *increase* from the first to the second anger provocation suggests that indeed the emotion control group was "active" in reducing anger experience. However, future studies that include a neutral condition will be able to more directly address this question.

Fifth, given how little is known about automatic emotion regulation processes, it will be important to assess additional outcome measures (e.g., startle magnitude; facial EMG; brain activation; social functioning). Such measures will allow a more conclusive assessment of which aspects of an emotion are altered by automatic control (e.g., only self-reported versus experienced emotion). Further, such measures will permit a clearer understanding of the mechanisms underlying the present effects. While existing research on automaticity suggests some mechanisms such as the activation of implicit goals, attitudes, and knowledge structures (e.g., Aarts & Dijksterhuis, 2000; Banaji, Blair, & Glaser, 1997; Fitzsimons & Bargh, 2004; Gollwitzer, 1999; Shah & Kruglanski, 2003), more research is needed to clarify how some people seem to be capable—without conscious effort—of remaining calm, cool, and collected in a powerfully negative situation.

Concluding comment

Much hinges on individuals' ability to regulate negative emotions, especially potentially destructive ones such as anger. Indeed, the control of anger is important in many domains of functioning, including well-being, psychosocial functioning, and physical health. However, effective anger control is difficult to achieve, and seems to be often associated with negative outcomes for the individual. The present results suggest that more automatic—effortless and largely unconscious—types of emotion regulation might present an effective answer to this problem. In intensely emotional situations, automatic emotion regulation was associated with less feelings of anger, without the cost of greater negative emotion or maladaptive cardiovascular activation. Extrapolating from these findings, it seems possible that automatic emotion control such as that engendered by habitual responses might lead to greater well-being, better social functioning, and better physical health. If so, it may help address the question as to how best to regulate powerful negative emotions such as anger.

Appendix A

Words used in the Sentence Unscrambling Task (target words were not highlighted in participants' forms)

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Emotion control condition	Emotion expression condition
cool weather whenever was the	hot weather whenever was the
drinking restrains she wine from	she drinks from wine impulsively
picture herself framed she the	picture herself framed she the
he none occasionally people watches	he none occasionally people watches
saw over train he the	saw over train he the
location limited there is access	location unlimited there is access
prices she the compared none	prices she the compared none
life water plan one should	life water savor one should
stable was although the stockmarket	volatile was although the stockmarket
pot was although covered the	pot over although boiled the
rode she bike her although	rode she bike her although
locked brakes weather were the	released brakes weather were the
money ago he the withheld	money ago he the spent
pear he a picked were	pear he a picked were
zoo animals confined throughout are	zoo animals liberated throughout were
skied repeated alone downhill he	skied repeated alone downhill he
ball the throw toss once	ball the throw toss once
his opinion kind hides he	action none she into burst
flowers had several once arrived	flowers had several once arrived
today car think clearly I	today car feel queasy I
send I mailed it over	send I mailed it over
walk for go path a	walk for go path a
what door closed is the	what door open is the
discussion disciplined far was the	discussion animated far was the
used commonly it pantry is	used commonly it pantry is
new was sudden movie the	new was sudden movie the
file concealed fact was the	file revealed fact was the
went the down when sun	went the down when sun
somewhat prepared I was retired	somewhat prepared I was retired
energy diet sugar restricts the	energy diet sugar discharges the
the push wash frequently clothes	the push wash frequently clothes
stifled the owner dog was	unleashed the owner dog was
watch gone she a found	watch gone she a found
the were beans none bottled	the were beans none boiled
fleas ago cat had the	fleas ago cat had the
better home were you go	better home were you go
the street blocked was also	the street exploded had also
haircut she over a got	haircut she over a got
day all sat we grown	day all sat we grown
curtain orange how was the	curtain orange how was the
picture took she a close-up	picture took she a close-up
file the contains bottle fluid	file the spilled was fluid

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