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Response Conflict and Affective Responses in the Control and Expression of Race Bias

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Abstract

Models of racial attitudes traditionally have assumed that individual differences in the strength of underlying, ‘implicit’ associations between racial categories and stereotypical traits are the primary determinant of the expression of race bias. Thus, individual differences in performance on laboratory tasks designed to assess implicit race bias tend to be interpreted in terms of association strength. Here, we argue that such associations tell only part of the story, and probably the least interesting part. We posit that response conflict and its regulation are critical to understanding the need for control, and that affect-related processes help to determine the extent to which control resources will be implemented to overcome biased associations. We present data from a number of recent behavioral and psychophysiological studies in support of this idea, as well as conceptual accounts that point toward a model of race bias regulation that depends upon processes identified as important for regulation of thought, affect and action more generally.

In the 1960s, Walter Mischel and his colleagues began studying arguably the most interesting scientific question to date involving marshmallows (e.g., Mischel & Ebbesen, 1970; Mischel, Ebbesen, & Zeiss, 1972; Mischel, & Metzner, 1962). These researchers recruited a sample of 4-year-olds to assess their ability to delay gratification. Mischel et al. found that some children were able to delay eating a marshmallow so that they could eat more marshmallows later, whereas others were unable to wait. Furthermore, these children’s individual variations delaying gratification predicted their ability to self-regulate well into adolescence and early adulthood (Shoda, Mischel, & Peake, 1990; Mischel & Ayduk, 2002; Eigsti et al., in press). In a nutshell, Mischel and colleagues showed that individuals differ in their ability to engage in self-regulatory control, that these abilities generalize across domains, and that individual variability remains fairly stable across many years and developmental stages.

Although it seems intuitive that self-regulatory abilities differ between individuals (cf., Miyake et al., 2000), it has not been apparent until quite recently that such individual differences might play an important role in a host of social behaviors (see Muraven & Baumeister, 2000). In particular, the dominant, social-cognitive approach to the study of stereotyping and prejudice has been characterized by a focus on automatic associations, stored in long-term memory, between racial categories (e.g., Black) and stereotypic traits and/or evaluations (e.g., Dovidio, Evans, & Tyler, 1986; Dovidio, Kawakami, Johnson, Howard, 1997). In this context, race bias generally is understood as the expression of the association between a group category and (generally negative) affective evaluations. For example, if an individual responds more quickly to a word such as LAZY than to a word such as AMBITIOUS following a Black face prime, it is assumed that the ‘Black’ category is more strongly associated in memory with laziness than ambition. Moreover, individual

differences in the magnitude of this associative bias are assumed to reflect differences in strength of the link between the category and the evaluative construct (i.e., the strength of the underlying, implicit attitude; Fazio, Jackson, Dunton, & Williams, 1995).¹

The primary thesis of this article is that individual differences in the expression of race bias are determined at least as much by variability in self-regulatory processes as by potential differences in the strength of associations between racial categories and affective evaluations. Admittedly, this is not an entirely novel idea, as numerous other theorists recently have argued that cognitive control plays an important role in determining the extent to which underlying biases will be expressed (see Amodio, Devine, & Harmon-Jones, 2008; Conrey et al., 2005; Payne, 2005; Sherman et al., 2008; von Hippel, 2007). However, the framework proposed here goes a step further by specifying the role of response conflict and its regulation, by underscoring the interplay between the ‘cognitive’ and ‘affective’ aspects of self-regulatory control, and by considering how affect-related responses are important in determining whether and to what extent cognitive control processes are implemented.

Race Bias as Association Strength

A fundamental tenet of the dominant social cognition approach to understanding race bias has been that racial attitudes are represented by associations between racial categories and evaluative constructs (see Greenwald et al., 2002; Macrae & Bodenhausen, 2000; Wheeler & Petty, 2001). When a racial category cue is encountered, associated evaluative constructs are automatically made more accessible, leading to facilitated responses (i.e., faster RT in laboratory tasks) to stimuli representing those constructs. Based on the general idea that priming an evaluative category automatically increases accessibility of evaluatively-related constructs (e.g., Fazio, Sanbonmatsu, Powell, & Kardes, 1986), numerous researchers have shown that priming a racial category (e.g., by briefly presenting a face or category label) facilitates responses to trait stereotypically associated with that category (e.g., Devine, 1989; Dovidio et al., 1986, 1997; Bargh & Burrows, 1996; Bargh, 1999; Wittenbrink, Judd, & Park, 1997). Moreover, Fazio and colleagues (e.g., Fazio, Jackson, Dunton, and Williams, 1995) have argued that individual differences in the magnitude of such priming effects reflect variability in the valence and/or strength of underlying racial attitudes (see also Fazio & Dunton, 1997; Fazio, 2007).

Similar arguments have been made with respect to performance on other reaction time-based measures, such as the implicit association test (IAT; Greenwald, McGhee, & Schwartz, 1998). The IAT requires participants to assign both racial categories and valenced words to one of two response keys as quickly as possible. For most participants, responses are faster when the Black category and negative words are assigned to the same key – the congruent condition – than when Black and positive words are to be categorized with the same key – the incongruent condition. The difference in RT between these two conditions (the “IAT effect”) is said to represent the valence and strength of the respondent’s underlying, implicit racial preferences (see Greenwald et al., 2002; Greenwald & Farnham, 2000; Greenwald, Nosek & Banaji, 2003; Nosek, Greenwald, & Banaji, 2005).

Another widely used race bias task relies on stereotypic associations between young Black men and violence (e.g., Correll, Park, Judd, & Wittenbrink, 2002; Devine & Elliot, 1995; Sagar & Schofield, 1980). In the Weapons Identification Task (WIT; Payne, 2001) White

¹Note that in the social-cognitive and person-perception literatures the concept of “evaluation” tends to be used to denote an affect-related judgment (e.g., a stereotypical association of Blacks with negative personal traits), whereas in the cognitive and cognitive neuroscience literatures this concept tends to refer to a more general, value-neutral process associated with monitoring or gleaned information (e.g., deciding whether a given stimulus is a target or nontarget). Here, we use “affective evaluation” to refer to the concept as used in person perception.

and Black face primes appear just before briefly-presented target objects (guns and tools) that must be categorized as quickly as possible. Research consistently shows that responses to guns are faster and more accurate following Black compared to White face primes (e.g., Payne, 2001, 2005; Payne, Shimizu, & Jacoby, 2005). Payne and colleagues have shown that this pattern is due largely to a stereotypic accessibility bias. In other words, the effect of the primes does not alter participants' ability to accurately discriminate guns from tools, but rather Black primes produce a biased tendency to activate the "gun" response (Payne et al., 2005). Moreover, process dissociation analyses, which provide separate estimates of the influence of automatic and controlled processes on performance (see Jacoby, 1991), consistently have shown that prime race (Black vs. White) influences the "automatic" parameter rather than the "controlled" parameter (e.g., Payne, 2001, 2005).

In general, then, the degree to which an individual expresses bias on reaction-time-based laboratory tasks is said to reflect the strength of stereotypic associations in her or his long-term memory. For example, in touting the benefits of the IAT, Greenwald et al. (2009) noted, "A useful property of IAT measures is their presumed reliance on associative processes that can operate automatically" (p. 18). However, as we will review in the next section, evidence recently has been accumulating that calls such assumptions into question by indicating a major role for control-related processes in determining performance on standard laboratory-based measures of implicit race bias.

Race Bias as Control Failure

Interest in the dynamic interplay between automatic and controlled processes in stereotyping and prejudice goes back decades (e.g., Devine, 1989; see Devine & Monteith, 1999). In her seminal work on this topic, Devine found that while activation of stereotypic associations appears to automatically occur in virtually all people upon exposure to racial category cues, expression of prejudice-related behavior is subject to the influence of controlled processing. Devine's (1989) work was particularly important in suggesting that expression of bias might depend more on what occurs once automatic associations are activated than on the strength of those associations.

In-line with this perspective, a number of recent models have emphasized the role of cognitive control processes whose influence occurs at the level of controlling expression rather than moderating activation of associations (e.g., Conrey, Sherman, Gawronski, Hugenberg, & Groom, 2005; Macrae et al., 1999; Mierke & Klauer, 2003; Payne, 2005; Rothermund & Wentura, 2004; Sherman et al., 2008; von Hippel et al., 2000). For example, in applying their "Quad model" formulation, which provides estimates of the extent to which performance on implicit measures is due to several automatic and controlled aspects of processing, to data from a standard Black-White racial IAT, Conrey et al. (2005) found that IAT performance was determined by both automatic associations (i.e., between race categories and affective evaluations) and by control-related processes, especially "overcoming bias," which represents the extent to which the influence of stereotypic associations can be overcome in order to respond correctly on incongruent trials. Thus, IAT performance reflects more than the strength of one's implicit associations, and importantly depends upon the (controlled) ability to overcome biased response tendencies stemming from those associations.

Of course, this issue is not specific to the IAT, in that analyses like those used by Conrey et al. (2005) can be applied to other race bias tasks as well. For example, Bartholow, Dickter, and Sestir (2006) tested the role of self-regulatory control in bias expression by having participants consume alcohol (or a placebo beverage) prior to completing a sequential racial priming task (see Dovidio et al., 1986). On a number of the trials a visual cue signaled

participants to withhold their categorization response (i.e., “stop” trials). Participants who had consumed alcohol expressed more bias in the task in terms of greater difficulty withholding responses on stereotype-congruent stop trials.

But did this effect occur because alcohol increased the accessibility of stereotypic associations, impaired response control, or both? Sherman et al. (2008) addressed this question by analyzing Bartholow et al.’s (2006) stop-trial error data using their Quad model formulation. Their analyses indicated that, compared to the placebo beverage, the alcohol beverage impaired the parameter associated with overcoming bias, but had no effect on the association activation parameter (or the other parameters).

The findings of Conrey et al. (2005) and Sherman et al. (2008) have important implications for interpreting IAT scores in terms of individual differences in racial “preferences” (see Nosek, Greenwald, & Banaji, 2005). Consider the case of two hypothetical individuals, both of whom hold strong negative attitudes toward Blacks, but one of whom has superior self-regulatory cognitive control abilities relative to the other. Although both of these people would be assumed to have strong automatic associations between the Black racial category and negative evaluations, Conrey et al.’s (2005) findings suggest that the individual with stronger self-regulatory skills would be less likely to overtly express bias. Importantly, this would occur not because the poor self-regulator is any more ‘racist’ in terms of the strength of her underlying negative associations, but because she is less able to keep responses betraying those associations from bubbling to the surface.

According to numerous models of the development of implicit racial attitudes (e.g., Arkes & Tetlock, 2004; Karpinski & Hilton, 2001), the seeds of bias are planted simply by growing up in a culture in which some groups are stereotypically associated with negative traits, regardless of the valence of one’s explicit, overtly-expressed attitudes or one’s desire to be egalitarian. Under such circumstances it is reasonable to expect that most if not all people in the culture have developed negative automatic associations for stigmatized groups (see Devine, 1989). Therefore, differences in the expression of those negative associations (e.g., on laboratory measures of race bias) probably stem not from differences in the types or strength of those associations but rather from variation in the ability and/or desire to control their expression (cf., Payne, 2005; von Hippel, 2007).

Several lines of research recently have investigated links between general control abilities and expression of race bias. For example, Payne (2005) showed that bias scores on the race IAT are significantly correlated with performance on the anti-saccade task, a commonly used measure of inhibitory control. Similarly, von Hippel and colleagues (e.g., Stewart, von Hippel, & Radvansky, 2009; von Hippel et al., 2000; von Hippel, Silver, & Lynch, 2007) have shown that the increase in race bias that tends to occur with aging (see Firebaugh & Davis, 1988; Nosek, Banaji, & Greenwald, 2002) reflects age-related deficits in control of inhibition, not to differences in the strength of automatic associations (also see Sherman et al., 2008), cohort effects (e.g., having grown up in a time when overt race prejudice was commonplace), or to reduced self-presentation concerns.

In addition to inhibitory abilities, other work strongly implicates general task-switching abilities in explaining IAT performance. Task switching refers to the ability to switch or shift attention from one task or goal set to another (e.g., Rogers & Monsell, 1995). A model proposed by Klauer and colleagues (e.g., Klauer & Mierke, 2005; Mierke & Klauer, 2001, 2003; see also Klauer et al., 2007, in press) predicts that people who generally are good at switching should show similar magnitude IAT effects regardless of the attribute a given IAT is supposed to measure (also see McFarland & Crouch, 2002). Consistent with this idea, Mierke and Klauer (2001, 2003) found that performance on different IATs is correlated even

when they ostensibly measure conceptually disparate attitude categories (e.g., politics and insects).

But...Control of What, Exactly?

Within the context of such evidence, it is important to consider more specifically *why* control should be important in determining performance on laboratory bias tasks. That is, what are cognitive control abilities controlling? Addressing this question requires us to consider how bias is operationalized in most laboratory tasks. In general, bias is defined in terms of enhanced performance (in terms of RT and/or error rates) on stereotype-congruent trials (e.g., when 'BLACK' and negative are assigned to the same response key within the IAT) and/or reduced performance on stereotype-incongruent trials. As discussed eloquently by Payne (e.g., 2001), quick and accurate responding on stereotype-congruent trials should be relatively easy because automatic associations and task requirements (i.e., controlled processes) both pull for the same response. In contrast, on stereotype-incongruent trials the response driven by automatic associations conflicts with the goal-driven correct response, and therefore control is needed to overcome the tendency to simply rely on automatic associations.

Research by Amodio and colleagues has been instrumental in outlining the role of detection and regulation of conflict in the expression and control of race bias. In several studies (e.g., Amodio et al., 2004; Amodio, Devine, & Harmon-Jones, 2008; Amodio, Kubota, Harmon-Jones, & Devine; 2006), Amodio and colleagues have shown that the error-related negativity (ERN), a component of the ERP generated in the anterior cingulate cortex (ACC) and linked to response conflict monitoring (see Yeung, Botvinick, & Cohen, 2004), is particularly large when participants erroneously categorize tools as guns following Black face primes in the WIT (i.e. race-bias errors). Moreover, the larger an individual's ERN on race-bias errors the more control he or she displays throughout the task (using Jacoby's [1991] process dissociation estimate of control). These data lend support to the idea that stereotype-incongruent trials are associated with enhanced conflict as participants struggle to activate the correct, counter-stereotypic response in the face of readily accessible stereotypic associations, but also suggest that the processing engendered by this internal struggle ultimately leads to better performance.

Bartholow and Dickter (2008) provided additional evidence that response conflict plays a role in expression of race bias by showing that competing responses lead to performance decrements on stereotype-incongruent trials. These authors measured the lateralized readiness potential (LRP), a component of the ERP emanating from motor cortex that reflects preparation of overt responses (Coles, 1989), as participants performed a race bias task. The LRP waveforms elicited on correct response trials indicated that, prior to making the correct overt response participants tended to initially activate the incorrect categorization response on stereotype-incongruent trials. Moreover, the magnitude of this response activation effect was correlated with the RT slowing seen on stereotype-incongruent trials, indicating that greater conflict between responses driven by stereotypic associations and goal-driven responses slows the emission of overt responses.²

From this brief review, it appears that one important reason that control is needed in race bias tasks is to overcome the conflict that occurs when activated associations interfere with the goal to make counter-stereotypic responses. Botvinick and colleagues (Botvinick, Braver, Carter, Barch, & Cohen, 2001; Botvinick, Cohen, & Carter, 2004) proposed an influential two-part model of cognitive control that provides an excellent conceptual framework for understanding this process. According to their model, an *evaluative component*, localized to the ACC (see Botvinick, Nystrom, Fissel, Carter, & Cohen, 1999;

Carter et al., 1998; Yeung et al., 2004), continuously monitors ongoing responses for instances of conflict. When conflict is detected, the ACC signals a second, *regulative component*, located in dorsolateral prefrontal cortex (dlPFC; see Kerns et al., 2004), to implement increased control. Sherman et al. (2008) similarly proposed that bias control relies on separate but complimentary processes, one to detect when control is needed and another to overcome inappropriate associations to permit correct or unbiased responding.

Although typically not considered in terms of affect per se, the evaluative component of the Botvinick et al. (2001) control model hints at an important role for affect-related processes in self regulation in general, and in regulating responses associated with bias in particular. In the next section, we consider this possibility in detail by reviewing research pointing to synergy in the neural circuitry involved in conflict monitoring, evaluation of pain and distress, and adjustment of ongoing performance.

A Role for Distress in the Regulation of Biased Responding

The notion that affect-related processes are important for guiding social behavior in general, and responses to racial outgroups in particular, certainly is not new (see Allport, 1954; Amodio, Harmon-Jones, & Devine, 2003; Hamilton & Mackie, 1993; Mackie & Smith, 1998). Currently, however, most models of bias expression do not directly address the possibility that affect could be an important factor signaling the need for control (but see Monteith, Arthur, & McQueary, 2010). As just described, in the Botvinick et al. (2001, 2004) model the ACC is critical to evaluating the need for control. Additionally, Amodio et al.'s (2004, 2006, 2008) work points clearly to a role for ACC activation in bias control. Thus, to better understand the psychological mechanisms driving bias control, it is useful to further consider the functional significance of ACC activation for information processing.

Several lines of evidence provide insights on this issue. First, both ERP (e.g., Hermann, Römmler, Ehrlis, Heidrich & Fallgatter, 2004; O'Connell et al., 2007) and fMRI studies (e.g., Carter et al., 2000; Kiehl et al., 2000) have shown that the ACC is particularly active when participants make errors in speeded RT tasks. Other work (e.g., Dehaene et al., 1994; Lütcke & Frahm, 2008; van Veen & Carter, 2002) has shown that the ACC is highly sensitive to instances of correctly-resolved response conflict as well as errors. However, long before it was associated with errors or response conflict the ACC was known to play an important role in the evaluation of pain. For example, Talbot et al. (1991) described how painful heat causes significant activation of the ACC, and noted the "unexpectedly specific representation of pain" (p. 1355) in this region. Building on previous reports that patients with cingulate lesions still feel pain but report it as less bothersome (e.g., Corkin & Hebben, 1981; Foltz & White, 1962; Hurt & Ballantine, 1974), subsequent work by Rainville and colleagues (1997) showed that the ACC is sensitive to the perceived unpleasantness of noxious stimuli. Importantly, Rainville et al. distinguished the somatosensory experience of pain (i.e., whether a stimulus is painful) from the evaluation of whether that pain is distressing (i.e., pain affect), noting that the ACC is involved only in the latter, evaluative process. A meta-analysis (Peyron, Laurent, & García-Larrea, 2000) confirmed that the ACC

²It is interesting to consider that such findings are at odds with the long-held notion that spreading of activation accounts for differences in RT on stereotype-congruent and -incongruent trials on race bias tasks. That is, spreading activation theory holds that response facilitation or slowing stems from differences in the accessibility of associations (see Fazio et al., 1995). It follows from this theory that only a single response is activated on a given trial, the latency of which is determined by how quickly activation spreads from the primed category node to the associated target construct. In contrast, response conflict theory hypothesizes that multiple responses can be activated either simultaneously or sequentially on any given trial, and that slower responses on stereotype-incongruent trials result from opposing responses competing for overt expression, which slows the ultimate behavioral expression of the "winning" response (see Coles, Smid, Scheffers, & Otten, 1995). The possibility that slow responses on stereotype-incongruent trials on laboratory measures of bias are due to response conflict rather than to relatively weak associations per se suggests a somewhat more complex model of stimulus processing and response output dynamics than is typically assumed.

is not involved in coding the intensity of pain but participates in both the affective and attentional concomitants of pain sensation, as well as in response selection. Experimental lesion studies using animal models have shown similar results (e.g., Donahue, LaGraize, & Fuchs, 2001). Finally, a recent review (Eisenberger & Lieberman, 2004) noted overlap of effects in dorsal ACC responding to both conflict monitoring and to unexpected pain.

Taken together, the considerable evidence linking ACC activity with conflict and error processing (e.g., Botvinick et al., 2004; O'Connell et al., 2007), and the research showing the involvement of the ACC in the evaluation of distress (e.g., Peyron et al., 2000), suggests that the experience of conflict, including the conflict inherent in incorrect responding, is distressing. A number of lines of evidence are consistent with this idea. For example, Mandler's influential constructivist theory of emotion (e.g., Mandler, 1975e.g., Mandler, 1990) suggests that goal interruptions (analogous to errors) and/or conflict elicit negative affective responses, consistent with recent psychophysiological work showing that error commission is aversive (Hajcak & Foti, 2008). Additionally, several studies have shown that the ERN is sensitive to the motivational significance of errors, such that more meaningful errors produce larger responses (Gehring et al., 1993; Gehring & Taylor, 2004; Hajcak, Moser, Yeung, & Simons, 2005). Also, the fact that ERNs tend to be larger among people who have difficulty regulating negative affect, such as those high in neuroticism (e.g., Pailing & Segalowitz, 2004) or anxiety (e.g., Hajcak, McDonald & Simons, 2003) and those with obsessive-compulsive disorder (Gehring, Himle, & Nissenon, 2000; Hajcak & Simons, 2002), further supports this idea. Thus, understanding the role of the ACC in models of cognitive control (e.g., Botvinick et al., 2001) appears to depend critically upon consideration of affective processes (see Hajcak, McDonald, & Simons, 2004), particularly the apparent distress associated with control failures (errors) or potential failures (conflict).

Moreover, enhanced ACC activation during error commission leads to increased control on subsequent trials (Kerns et al., 2004; see also Botvinick et al., 2004), suggesting that evaluating mistakes as distressful is adaptive for self-regulation. This idea is consistent with an influential theory linking errors with a response in the mesencephalic dopamine system, linked to the ACC, which is important for learning from mistakes (see Holroyd & Coles, 2002). This view also is consistent with other work indicating that pain and distress are highly effective at capturing attention, disrupting ongoing behavior, and motivating a new course of action (see Eisenberger & Lieberman, 2004; Wall, 1999), all of which are integral to interrupting automatic processes and engaging controlled processes. Thus, we contend that the distress signal emitted from the ACC during conflict and errors is critical to engaging increased attention and response control, such as that needed to overcome race-based response tendencies (Sherman et al., 2008).

In placing a distress-related response at the heart of a system of bias control, it is important that we attempt to clarify the nature of this response. Importantly, we do not intend to suggest that a role for distress in understanding ACC function should supplant the importance of conflict per se in bias regulation. Rather, in our view bringing the distress-evaluating function of the ACC (e.g., Peyron et al., 2000; Rainville et al., 1997) to bear provides a plausible account for why conflict captures attention, disrupts ongoing behavior and motivates enhancement of control. That is, we contend that the conflict reflected in ERNs elicited by race-bias errors (see Amodio et al., 2004, 2008), for example, is not affectively neutral, and thus should motivate attempts to increase control. Indeed, it seems difficult to reconcile the finding that more meaningful errors elicit larger ERNs (Gehring & Taylor, 2004; Hajcak et al., 2005) with a view that ACC function reflects only (affectless) levels of conflict; why would some errors be associated with more conflict than others?

In our view it also is not necessary that the distress registered by the ACC be experienced subjectively by the individual, consistent with the widely-held view that numerous cognitive and affect-related processes occur below the level of conscious awareness (e.g., Bargh & Ferguson, 2000). Following this logic, it also is not necessary that ACC activity represent conscious awareness of errors or conflict. Previous research (Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001) has indicated that the initial ACC response to an error reflected in the ERN is not associated with error awareness, but that subjective awareness might be reflected in a later-developing component, the error positivity (Pe), thought to be generated in the rostral ACC and other medial prefrontal areas. It could be that the Pe represents a conscious, subjective evaluation of the distress associated with the response seen in the ERN.

An Experimental Test of the Distress Hypothesis

To the extent that experiencing errors as distressing is important for controlling bias, it could be the case that levels of negative affect during a bias task will determine both the magnitude of ACC activity to errors and the degree to which behavioral adjustments are made following self-regulation failure (i.e., errors). Bartholow, Henry, Lust, Sauls and Wood (2010) recently tested this idea in the context of a larger investigation of alcohol's effects on performance monitoring and adjustment. Previous work (Ridderinkhof et al., 2002) had already established that alcohol reduces ERN amplitude and interferes with post-error behavioral adjustment. However, those previous results had been interpreted in terms of alcohol impairing the brain's ability to detect errors; Ridderinkhof et al. did not consider alcohol's known affect-modulatory effects (e.g., Greeley & Oei, 1999) in explaining their results. Following the dopamine theory of the ERN (Holroyd & Coles, 2002), and given that alcohol is known to increase resting levels of dopamine in the mesencephalic dopamine system (see Koob et al., 1998), Bartholow et al. hypothesized that alcohol reduces the ERN by reducing distress over error commission, presumably due to increased dopaminergic activity, and that this reduced distress accounts for alcohol's impairment of post-error behavioral adjustment by disrupting the alarm signal sent by the ACC to dlPFC.

Bartholow et al. (2010) had participants consume alcohol, an alcohol placebo or a control beverage prior to completing the WIT. Participants also completed the Positive and Negative Affect Scales (Watson, Clark, & Tellegen, 1988), which provide separate indices of positive affect (PA) and negative affect (NA), at baseline and several post-drinking intervals. Findings supported the authors' hypotheses. The ERN was much larger in the placebo group than in the alcohol group (cf., Ridderinkhof et al., 2002). Post-response accuracy judgments (see Payne et al., 2005) indicated that alcohol participants were just as aware of their errors as participants in the other groups. As predicted, NA was reduced following alcohol consumption and the significant effect of beverage on ERN amplitude was significantly mediated by post-drinking changes in NA, suggesting that alcohol's affect-regulatory effects influenced error-related ACC activation. Also, a structural equation model showed that alcohol's impairment of post-error adjustments in bias regulation was fully mediated by effects on NA change and ERN amplitude. Although alcohol also reduced the Pe component this effect was unrelated to post-error adjustment. Finally, whereas the size of the race bias ERN significantly predicted use of controlled processing for placebo and control participants, this typical association (see Amodio et al., 2004) was absent for alcohol participants.

Taken together, these findings support a critical role for affect-related processes in regulating the control and expression of bias. The fact that intoxicated participants were not unaware of their errors but simply appeared less bothered by them is consistent with a role for the ACC in evaluating distress (see Foltz & White, 1962; Hurt & Ballantine, 1974;

Rainville et al., 1997), and with previous research indicating that error awareness is reflected in the Pe and not the ERN (e.g., Nieuwenhuis et al., 2001). More generally, these findings support the view that the ERN represents a distress-related response to errors, generated in the ACC, which serves to signal other areas of PFC (e.g., dlPFC) that increased control is needed.³ When this distress or “alarm signal” (Eisenberger & Lieberman, 2004, p. 294) is reduced, whether by alcohol or due to insufficient motivation (e.g., Hajcak, Moser, Yeung, & Simons, 2005), control resources are not brought to bear, leading to increased expression of race bias. These data also are consistent with other studies showing greater ACC and dlPFC activity when participants need to override responses based on negative associations with racial outgroup members (see Beer et al., 2008; Chee et al., 2000; Knutson et al., 2007; Luo et al., 2006).

A Potential Role for Individual Differences in Affect Regulation

Just as cognitive control abilities vary widely between individuals (see Friedman et al., 2008), people also vary in their affect-regulatory skills and strategies. Recent neuroendocrine data indicate that failure to effectively regulate negative affective reactions elicited by outgroup members, indicated by heightened cortisol responses during an interracial interaction, results in decreased controlled processing during the WIT (Amodio, 2009). Thus, effectively regulating the affective experience elicited by outgroup cues appears vital for ensuring control of race bias.

Emotion regulation (EmReg) refers to the ways in which individuals attempt to influence the emotions they experience, when they experience them, and how they are expressed (Gross, 1989). According to Gross and colleagues (e.g., Gross & John, 2003; John & Gross, 2007), two commonly used EmReg strategies are cognitive reappraisal and expressive suppression. *Cognitive reappraisal* is considered an antecedent-focused strategy, meaning it occurs early in the emotion-generative process, allowing it to intervene before an emotional response has been fully generated. Thus, using cognitive reappraisal allows the ensuing course of emotion to be altered. For example, an individual using reappraisal might view a loss (e.g., in a game) as an opportunity to learn from mistakes and improve. *Expressive suppression*, however, is considered a response-focused strategy because it occurs later in the emotion-generative process, modifying the behavioral expression of emotional response. Continuing the game analogy, individuals using expressive suppression regulate emotions by keeping a “poker face” regardless of the cards they hold, thereby not allowing the emotion they feel to be visibly expressed.

Of importance for the current discussion, research has shown that differences in these EmReg strategies are related to the extent to which emotional stimuli impact cognitive performance (Gross & Richards, 2000; Gross & John, 2003), and that suppression and reappraisal are associated with distinct neural signatures (e.g., McRae et al., 2010). Such findings suggest that EmReg strategies are likely to influence the regulation of bias, perhaps due to differences in the extent to which neural circuits important for conveying affect-

³Of course, the ACC is not the only brain structure implicated in affect-related responses in the regulation of bias. In particular, the amygdalae, subcortical structures often implicated in determining negative affective response (e.g., Ledoux, 2000; Zald, 2003) and motivational significance more generally (see Anderson & Phelps, 2001; Whalen, 1998), appear highly sensitive to perception of outgroups and might play a role in bias expression (see Amodio et al., 2003; Hart et al., 2000). Also, although there is little evidence supporting direct neural connections between the PFC and the amygdalae, research evidence suggests that these structures work in concert to affect regulation of bias. For example, in a sample of White participants reporting strong motivation to control prejudice (Cunningham et al., 2004), greater amygdala activity was elicited by Black than White faces when faces were shown too briefly to be consciously detected (30 ms), but no racial difference in amygdala activity was observed when faces were shown for a longer duration (525 ms). Rather, Black faces presented at this longer duration elicited greater activity in the ACC and right ventrolateral PFC and dlPFC, possibly reflecting enhanced control over implicit negative evaluations.

related responses to prefrontal control-related areas are engaged in the presence of outgroup cues.

Although not a study of bias regulation, recent work by Henry, Bartholow, Becker, and Kerns (2010) demonstrates that individual differences in EmReg strategies have important implications for how affective information influences cognitive control. Participants were classified as either “suppressors” or “reappraisers” on the basis of their responses to an individual-differences measure of EmReg (see Gross & John, 2003). ERPs were recorded as participants completed a Stroop color-naming task (Stroop, 1935) in which each word was preceded by a positive, negative, or neutral picture. The ERP data showed that the N200 component -- often linked to the evaluative, conflict monitoring aspect of cognitive control (see Botvinick et al., 2001; Liotti et al., 2000) and likely generated by the dorsal ACC (see Huster, Westerhausen, Pantev, & Konrad, 2010) -- was reduced among suppressors relative to reappraisers on incongruent trials preceded by negative images, suggesting that suppressors were less effective at regulating response conflict on those trials. Following Botvinick et al.’s (2001) control model, this ineffective conflict regulation should have two downstream consequences: (1) suppressors’ behavioral performance on these negative-incongruent trials should suffer, and (2) there should be increased conflict during response output, seen as increased amplitude of the ERN on errors. Both of these predictions were borne out by the data. Relative to reappraisers, suppressors’ accuracy on negative-incongruent trials was impaired, and suppressors showed enhanced ERN responses on error trials, indicative of heightened distress response in the ACC as a result of control failure. According to Botvinick et al., this pattern suggests that conflict not resolved during stimulus processing (i.e., the N200) results in heightened conflict during response activation (ERN).

Henry et al.’s (2010) findings increase understanding of how individual differences in EmReg can influence cognitive control processes, particularly when stimuli have clear motivational relevance. More specifically, however, an analogy can be drawn between the effects of the negative images used by Henry et al. and the presentation of outgroup faces in most laboratory measures of race bias. First, both arguably elicit negative affect that needs to be regulated (see Cunningham et al., 2004). Difficulty at this stage (e.g., using an ineffective EmReg strategy) apparently interferes with recruitment of cognitive control resources needed to respond in a goal-directed manner, whether to an incongruent Stroop stimulus or to a stereotype-incongruent target (e.g., Black-tool trials in the WIT). In either case the affect-eliciting stimulus is not task-relevant for the respondent’s goals, but effective regulation of the automatic response it elicits helps to ensure proper behavioral control.

Conclusions and Future Directions

In sum, although performance on race bias measures often is portrayed as demonstrating the strength of automatic associations between racial groups and evaluations (e.g., Fazio et al., 1995; Greenwald et al., 2002), it has become clear that cognitive control processes play a key role in determining whether bias will be expressed (see Conrey et al., 2005; Payne, 2005; Sherman et al., 2008). Some researchers have pointed to individual differences in specific cognitive control abilities, particularly inhibition (e.g., Payne, 2005; Stewart et al., 2009; von Hippel et al., 2000, 2007) and task switching (e.g., Klauer & Mierke, 2005; Mierke & Klauer, 2001, 2003), as important to understanding bias expression on laboratory tasks. We contend that such control abilities are important primarily for regulating the conflict that arises when responses driven by stereotypic associations compete with counter-stereotypic responses required on incongruent trials. Further, we argue that the extent to which cognitive control abilities are brought to bear is driven importantly by the distress that accompanies failures of control (i.e., errors), or potential failures (conflict), as reflected in the evaluative response of the ACC (see Botvinick et al., 2001, 2004). Finally, we posit that

affect-regulatory abilities and/or strategies also might play a role in this process, primarily by determining how the affective processing set in motion by the presence of outgroup cues will be managed by and integrated into the conflict and control system.

The hypothesis advanced here places affect-related processes at the heart of a conflict and control system responsible for the control of race bias, among other forms of self regulation. In so doing, it is important that we address previously proposed models in which “affective” and “cognitive” processes have been theorized as largely separable. Some prior theorizing (e.g., Bush, Luu, & Posner, 2000) has suggested that “emotional” and “cognitive” aspects of conflict can be dissociated, including at the level of underlying neural structures (rostral and dorsal aspects of ACC, respectively). Bush et al. presented meta-analytic findings suggesting that “emotional” tasks tend to activate the rostral (i.e., affective) division of ACC, whereas “cognitive” tasks tend to activate the dorsal (i.e., cognitive) division. However, a more recent, integrative account (Eisenberger & Lieberman, 2004) suggests that this dichotomy is likely overly general, noting that the Bush et al. (2000) analysis did not include studies of physical pain, which, in contrast to what the Bush et al. model would predict, tends to activate the dorsal ACC. Thus, following Eisenberger and Lieberman, we propose that both rostral and dorsal ACC can be involved in processing conflict because conflict is not affectively neutral, regardless of whether the task in which it is elicited is thought to be affective or “purely” cognitive in nature, and suggest that advancement of theory in this area is likely to benefit from integrative cognitive-affective accounts such as the one proposed by Eisenberger and Lieberman.

It is important to acknowledge, however, that some of the ideas presented here have not been fully vetted by empirical research. The primary purpose of this article is to stimulate further research investigating the processes that contribute to expression of bias as measured in the lab, and more generally to understand the factors that determine the implementation of controlled processing during intergroup interactions. One example is provided by ongoing, collaborative work involving our laboratory and others assessing the extent to which individual differences in specific executive functioning abilities, assessed at the level of latent variables (see Miyake et al., 2000; Friedman et al., 2008), is associated with differences in performance (and associated neural responses) on multiple laboratory bias tasks. This work holds promise to extend the work of Klauer and colleagues (e.g., Klauer & Mierke, 2005; Mierke & Klauer, 2001, 2003) and von Hippel and colleagues (Stewart et al., 2009; von Hippel et al., 2000, 2007) who have been focusing primarily on the role played by task switching and inhibitory abilities, respectively.

Another important issue for future research will be to further specify the nature of the relationship between negative affect elicited by outgroup cues, regulation of that affective experience, and expression of bias. Amodio (2009) has reported compelling evidence that failure to effectively regulate intergroup anxiety, indicated by increased cortisol response during an interracial interaction, is associated with reduced controlled processing during a subsequent race bias task. Also, preliminary evidence (Henry et al., 2010) suggests that the tendency to use a relatively ineffective emotion regulation strategy (suppression) is associated with reduced ability to regulate conflict following presentation of a negative visual cue. However, it remains unclear whether, for example, relatively ineffective emotion regulation strategies would predict poorer regulation of race bias conflict, possibly due to less effective regulation of intergroup anxiety.

It is important to underscore that the current proposal is not intended to impugn the predictive validity of laboratory measures of implicit bias (cf., Blanton & Jaccard, 2006). What is at issue here is not whether individual differences on such tasks predict expression of bias in other contexts (see Greenwald, Poehlman, Uhlmann, & Banaji, 2009), but whether

this predictive validity is due to differences in the strength of implicit, automatic associations as opposed to other factors that systematically vary with task performance. In our view, the predictive validity of these tasks is due largely to the fact that performance covaries with the strength of cognitive control abilities, EmReg propensities, or both.

As the field has become more collaborative in nature (see Cacioppo, 2007), and as knowledge from various sub-fields influences other sub-fields, it has become increasingly apparent that comprehensive understanding of a host of social behaviors, including race bias, requires consideration of multiple processes assessed at multiple (e.g., social, cognitive, and neural) levels of analysis (see Ochsner & Lieberman, 2001; Ochsner, 2004). While far from a formal model of race bias, it is our hope that the ideas presented here will stimulate further investigation into the integrative, reciprocal cognitive and affective processes – and their neural sources – that give rise to race bias and its control.

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