Sequential dependencies in the Eriksen flanker task: A direct comparison of two competing accounts

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In the conflict/control loop theory proposed by Botvinick, Braver, Barch, Carter, and Cohen (2001), conflict monitored in a trial leads to an increase in cognitive control on the subsequent trial. The critical data pattern supporting this assertion is the so-called *Gratton effect*—the decrease in flanker interference following incongruent trials—which was initially observed in the Eriksen flanker task. Recently, however, the validity of the idea that this pattern supports a general conflict/control mechanism has been questioned on the grounds that the Gratton effect is only observed with stimulus repetition. We present an experiment testing whether the Gratton effect reflects a stimulus-independent increase in cognitive control or stimulus-specific repetition priming. Although our results support the latter hypothesis, the priming effect is modulated by the congruency of the previous trial. We discuss a new mechanism through which monitored conflict is used to exert executive control by modulating stimulus–response associations.

The Eriksen flanker task (Eriksen & Eriksen, 1974) has been frequently used to investigate attentional control processes. The basic task requires a fast response to a centrally presented target stimulus, which is flanked by several distractor stimuli that also activate response channels. A congruent trial is one in which the flankers are associated with the same response as the target (e.g., <<<<<), whereas in an incongruent trial, the flankers are associated with a competing response (e.g.,<<><<). Reaction times (RTs) are faster for congruent than for incongruent trials. The dominant explanation for this finding is that the presentation of flankers results in automatic activation of the response channel associated with the flanker stimuli (Gratton, Coles, & Donchin, 1992; Gratton, Coles, Sirevaag, Eriksen, & Donchin, 1988), leading to fast correct responses in the congruent condition (i.e., the flankers are identical to the target and facilitate a correct and fast response). In the incongruent condition, the automatic activation leads to fast incorrect responses, necessitating attentional control processes to overrule the incorrect automatic activation, resulting in slow correct responses.

This generally accepted view has been implemented in sophisticated computational models to investigate the dynamics of the control processes. One such model is the conflict-monitoring theory of Botvinick, Braver, Barch, Carter, and Cohen (2001), in which the automatic activation of the incorrect response channel competes with activation of the correct response channel at the level of response selection. Resolving this response competi-

tion requires time and thus underlies the main effect of congruency—the flanker effect.

One of the important contributions made by the Botvinick et al. (2001) model is that the amount of response competition, or conflict, is monitored and used to adjust top-down attentional control—the more conflict, the more control. In the case of the flanker task, increased control results in an increased attentional focus to the central target stimulus. Botvinick et al. proposed that conflict monitored on the previous trial is used to modulate cognitive control on the current trial and that this sequential dependency across trials underlies the so-called *Gratton effect* (Gratton et al., 1992). This effect is an interaction between the trial type (congruent vs. incongruent) of the current trial and the trial type of the previous trial (see Figure 1). In this article, we will use the notation xX, where the first letter—in lowercase—indicates the congruency of the previous trial and the second letter—in uppercase—indicates the congruency of the current trial. The Gratton effect is a pattern of slower RTs for congruent trials that follow incongruent trials (iC), as compared with those that follow congruent trials (cC), and faster RTs for incongruent trials that follow incongruent trials (iI), as compared with those that follow congruent trials (cI). To prime the reader, the RT difference between cI and iI trials tends to be larger than the difference between iC and cC trials.

The conflict-monitoring hypothesis explains the Gratton effect as follows (Figure 1, black arrows). An incongruent trial produces more response conflict than does a congruent trial. This monitored conflict is used to sharpen

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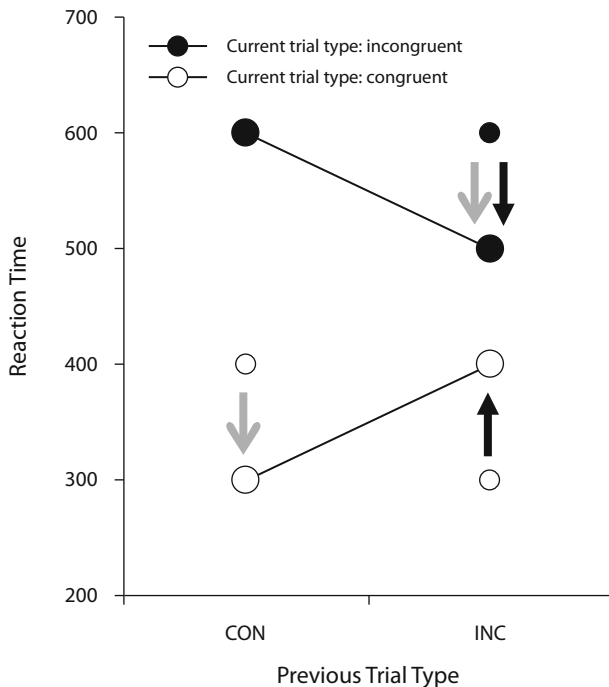
Figure 1. Hypothetical pattern of the Gratton effect. The basic pattern consists of faster reaction times (RTs) in iI trials relative to cI trials and slower RTs in iC trials relative to cC trials. The black arrows indicate the explanation given by the conflict/control theory, in that after an incongruent (INC) trial, the monitored conflict is used to sharpen the attentional focus, leading to faster RTs for incongruent trials and slower RTs for congruent (CON) trials. The gray arrows indicate the stimulus-priming explanation: Repetition of the same stimulus (target + flankers) leads to faster responding, producing the same interaction.

the attentional focus on the subsequent trial, which leads to better exclusion of the flankers and thus to a smaller flanker effect.

The conflict-monitoring model has been challenged (Mayr, Awh, & Laurey, 2003; Nieuwenhuis et al., 2006) on the basis that it captures results that are typical for only half of the data. In particular, the Gratton effect is observed when the target/response repeats across trials. Mayr et al. conducted a flanker experiment and analyzed the data separately for trial pairs in which the target/response repeated and for pairs of trials in which the target/response changed. The Gratton effect was observed only when the target/response repeated. Because the cC and iI trials included repetitions of the full stimulus (target + flankers), Mayr et al. concluded from their data that priming speeds up cC and iI trials (Figure 1, gray arrows), producing the Gratton effect. The priming occurs as a direct result of reactivating an association made (during the previous trial) between the full stimulus and the response that it invoked.

Mayr et al.'s (2003) findings have been disputed by Ullsperger, Bylsma, and Botvinick (2005), who obtained a Gratton effect in nonrepetition trials (see also Notebaert & Verguts, 2006). This discrepancy could be due to methodological differences between Mayr et al.'s study and that of Ullsperger et al. (especially relating to the temporal sequence of trials), as well as between Mayr et al.'s study

and that of Notebaert and Verguts (who used a numerical flanker task). It is yet unclear how these methodological differences affect the Gratton effect × repetition interaction, but Nieuwenhuis et al. (2006) replicated Mayr et al.'s findings, testing over 900 participants.

Here, we adopt the arrow-flanker paradigm used by Mayr et al. (2003) and Nieuwenhuis et al. (2006) and contrast the two main competing hypotheses—that the Gratton effect is due to modulation of attentional control or to repetition priming. The rationale for using this paradigm is its recent use in assessing specific deficits in cognitive control in clinical groups (Ridderinkhof, Scheres, Oosterlaan, & Sergeant, 2005; Wylie, Ridderinkhof, Eckerle, & Maning, 2007). To avoid misclassification of patients as "cognitive control impaired," debates relating to the construct validity of this particular task need to be resolved. This is not to say that variations of the flanker task (using brief stimulus presentations or using numbers as stimuli) are uninteresting, but the Gratton × repetition interaction does not occur for those variations or for the Stroop task (we will return to the Stroop task in the Discussion section). Mayr et al.'s task analysis highlighted an anomaly that led to an important reinterpretation of the Gratton effect in the flanker paradigm. By constraining the experimental method to this particular (arrow-flanker) paradigm, we aimed to contribute additional observations to understand this theoretically critical dissociation. Note that this particular paradigm confounds target repetition with response repetition (we will return to this issue in the Discussion section).

In the present investigation, we included neutral trials (e.g., $--\!>\!--$) to construct baseline conditions (i.e., nX). We reasoned that a weaker version of the conflict/control theory would assume that the change in target/response destroys the increase in attentional focus and would thereby account for the Gratton × repetition interaction without loss of its core assumptions about conflict-induced control. Although such an extension merely perpetuates the debate, the accounts do make divergent predictions with regard to the difference in RTs between the experimental conditions and the corresponding baseline conditions. The inclusion of a baseline condition has not featured in the aforementioned literature. If the Gratton effect is due to an increased focus of attention after an incongruent trial, performance on trials following a neutral trial is expected to be the same as performance on trials following a congruent trial (inasmuch as conflict on these trials is sufficiently similar), resulting in the following two orderings of RTs: $RT_{cI} = RT_{nI} > RT_{iI}$ and $RT_{cC} = RT_{nC} < RT_{iC}$ (Figure 2A). As can be seen in Figure 2, for the strong version of the conflict/control hypothesis, this ordering would be expected independent of target/response repetition (a weaker version in which target/response alternation disrupts the increase in attentional focus would predict this same ordering only for target/response repetition). If, however, the Gratton effect is due to positive priming of the repeated stimuli (target + flankers), the expectation would be that the performance on exact repetition trials (cC and iI) would be faster than on the corresponding baseline trials (nC and nI), resulting
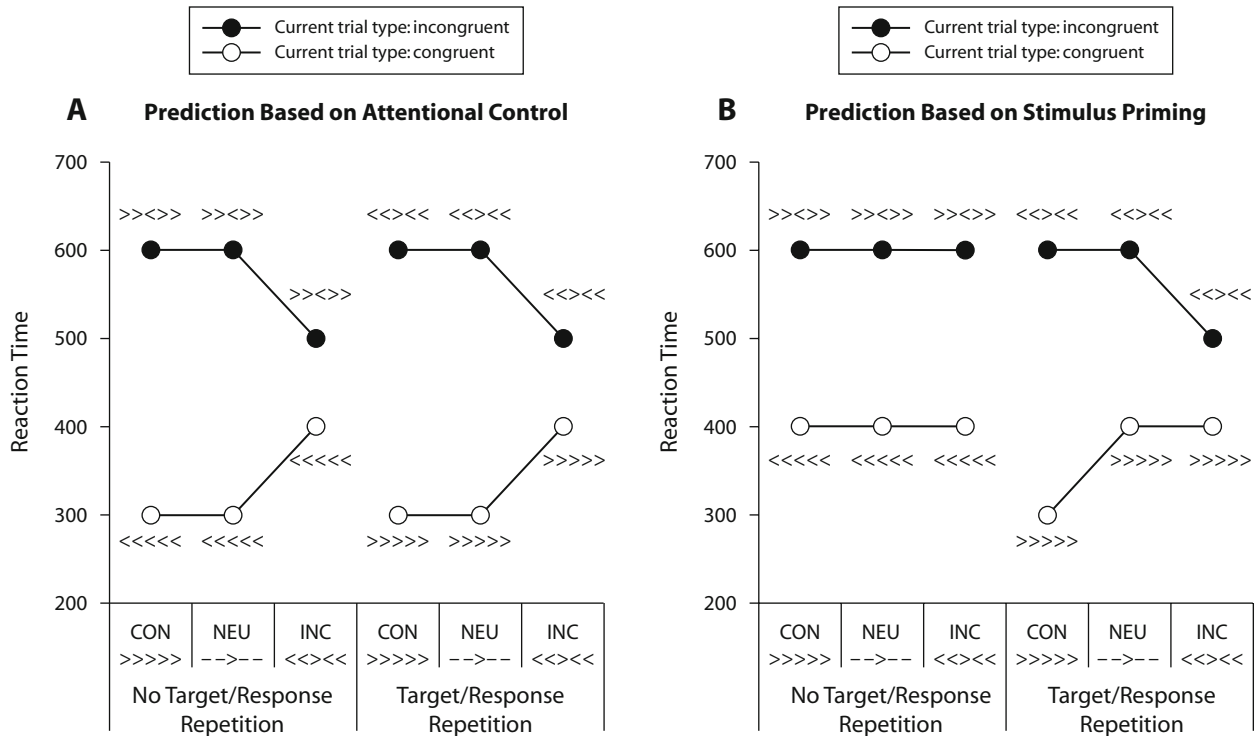
Figure 2. Predictions based on an attentional-control account (A) and a stimulus-priming account (B) for the effect of previous trial type (CON, congruent; NEU, neutral; INC, incongruent) and target/response repetition. Examples of stimuli are given in the panels.

in the following two orderings of RTs: $RT_{cI} = RT_{nI} > RT_{iI}$ and $RT_{cC} < RT_{nC} = RT_{iC}$ (Figure 2B). Note that the priming account predicts that priming is absent for non-repetition trials. In the following experiment, we aimed to test these predictions by adding a baseline condition to the flanker task, thereby providing converging evidence for a contribution of priming to the Gratton effect.

METHOD

Participants

Twenty-two participants (16 women; mean age = 29 years) from the University of London were tested individually in this experiment and received a remuneration of £7 for their time.

Stimuli and Procedure

Stimuli consisted of five horizontally arranged arrowheads or minus signs, making up congruent (e.g., $>>>>>$), incongruent (e.g., $<<><<$), and neutral (e.g., $--->--$) trials. A pilot study using zeroes ($00>00$) produced slow RTs—hence, the decision to use minus signs instead, which also provided some level of perceptual load and kept the RTs fast. Stimuli were presented in a black font on a white background. Participants were instructed to respond to the central target arrowhead by pressing the "z" or the "/" key on the keyboard when the arrow pointed to the left or the right, respectively. The instruction was followed by a practice block of 36 pairs of trials consisting of all possible combinations of stimuli. Each pair of trials started with a blank interval of 1,000 msec succeeded by the first flanker stimulus, presented for a maximum of 1,500 msec, followed by another blank interval and finally the second flanker stimulus. From the viewpoint of the participant, each block consisted of 72 independent trials. Participants were given feedback on their accuracy and average RT after the practice block and after

each experimental block. Participants were instructed to aim for an average RT of less than 1 sec and to maintain accuracy above 80% correct. After the practice block, participants completed 18 experimental blocks, testing each unique condition 36 times.

RESULTS

In accordance with previous studies, Figure 3 shows the correct mean RTs and error rates for each combination of current trial type and previous trial type separately for target/response change trials (left panels) and target/response repetition trials (right panels). Trials following an error were excluded.

Besides the standard finding of slower RTs for incongruent relative to congruent trials, three critical patterns can be discerned from Figure 3. First, we replicated the finding that the Gratton effect occurs for the trial pairs in which the target/response repeats (i.e., $RT_{cI} > RT_{iI}$ and $RT_{cC} < RT_{iC}$), but not when there is no repetition (i.e., parallel lines). Second, for the trial pairs with the same response (right panel), the RTs for the baseline trials, nC and nI, were slower than those for the full repetition trials, cC and iI. This pattern is in accordance with a priming account. Third, the size of the priming effect (right panel) was larger after an incongruent than after a congruent trial (i.e., $RT_{nI} - RT_{iI}$ was larger than $RT_{nC} - RT_{cC}$). Each of these observations was borne out by the following statistical tests.

A repeated measures ANOVA with the factors of current trial type (congruent, incongruent, or neutral), previous trial type (congruent, incongruent, or neutral) and target/
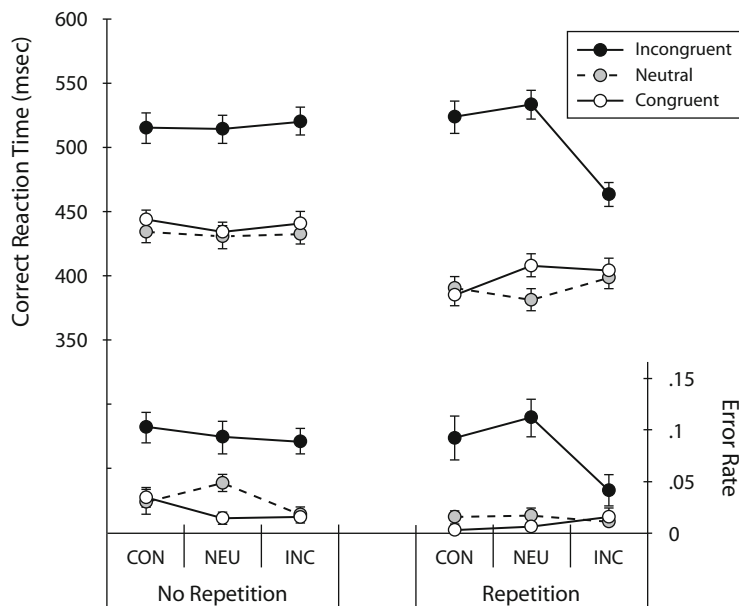
Figure 3. Mean correct reaction times and error rates as functions of current trial type and previous trial type (CON, congruent; NEU, neutral; INC, incongruent). The error bars indicate standard errors of the means.

response repetition (repetition or change) revealed a significant three-way interaction in the RT data [$F(4,84) = 33.79$, $MS_e = 261.95$, $p < .001$, $\eta^2 = .62$] and in the error data [$F(4,84) = 9.31$, $MS_e = 0.001$, $p < .001$, $\eta^2 = .31$]. Statistical analyses on the correct RT data revealed a significant interaction between current trial type and previous trial type for repetition trials [$F(4,84) = 58.04$, $MS_e = 267.91$, $p < .001$, $\eta^2 = .73$] but not for nonrepetition trials ($p > .54$). For the error data, there was a current × previous trial type interaction for repetition [$F(4,84) = 13.23$, $MS_e = 0.001$, $p < .001$, $\eta^2 = .39$] and for nonrepetition [$F(4,84) = 3.15$, $MS_e = 0.001$, $p < .05$, $\eta^2 = .13$] trials. This latter interaction was due to more errors occurring in the nN trials than in the nC trials [$t(21) = 5.38$, $p < .001$] and was eliminated when the ANOVA was conducted excluding the pairs with a neutral condition ($p > .66$).

We conducted contrast analyses on the repetition trials only to address the specific predictions made by an attentional-control account and by a priming account. These revealed that the nI trials were significantly slower [$F(1,21) = 86.24$, $MS_e = 542.33$, $p < .001$, $\eta^2 = .80$] and less accurate [$F(1,21) = 36.93$, $MS_e = 0.004$, $p < .001$, $\eta^2 = .64$] than the iI trials and that the nC trials were significantly slower [$F(1,21) = 57.31$, $MS_e = 209.55$, $p < .001$, $\eta^2 = .73$] than the cC trials. Critically, for all 22 participants, $RT_{cC} \leq RT_{nC}$. Except for marginal effects between nI and cI trials (for RT; $p = .06$) and between nC and iC trials (for error rate; $p = .10$), all other contrasts were nonsignificant (all $ps > .15$). This pattern of results supports the priming account and counters the prediction of the attentional-control account as presented in Figures 1 and 2.

Finally, the priming effect (for repetition trials) was larger after an incongruent trial [iI vs. nI, $M = 70.3$ msec,

$t(21) = 9.287$, $p < .001$] than after a congruent trial [cC vs. nC, $M = 23.4$ msec, $t(21) = 7.57$, $p < .001$; interaction, $F(1,21) = 46.72$, $MS_e = 259.78$, $p < .001$]. This was also the case when the relative priming effect (absolute priming effect divided by the RT of the baseline condition) was tested [$t(21) = 6.238$, $p < .001$].

DISCUSSION

We replicated the findings of Mayr et al. (2003), obtaining the triple interaction with the Gratton effect only when the target/response repeated across trials. We also observed that the Gratton effect itself was due to faster RTs (and lower error rates) for those trials in which the *full* stimulus repeats (not just the target character), as shown through comparing the critical trials (cC and iI, with target/response repetition) with the appropriate baseline trials. Because previous reports did not include baseline conditions, the debate primarily focused on finding a Gratton effect for nonrepetition trials. By including baseline conditions, we obtained converging evidence for the view that priming contributes to the Gratton effect. We now turn to our third finding, the size of the priming effect, which underlies our rationale for extending the conflict-monitoring hypothesis.

Mayr et al. (2003) rejected the general stimulus-independent conflict/control loop theory and instead appealed to Hommel's (2004) notion of event files that store stimulus–response associations. Our results support an event-file-type explanation and show unequivocally that the strong version of stimulus-independent cognitive control (Botvinick et al., 2001) is difficult to uphold. This makes intuitive sense, given that control is exerted in response to some sort of event-induced conflict. However,

the extreme version of noncontrol/stimulus priming is not supported by our data, either. We found that the amount of repetition priming is modulated by the previous trial type, such that the priming effect is larger after an incongruent than after a congruent trial. This conflict-dependent priming effect does not support a bottom-up stimulus priming account. We therefore propose a third, intermediate account, which addresses the underlying question of whether the Gratton effect × repetition interaction falsifies the conflict/control theory. The original theory stated that conflict in the previous trial affects performance in the current trial. Although Botvinick et al. focused on the hypothesis that control is exerted by sharpening spatial attention, this is not the only possible implementation of cognitive control. In a recent article, Blais, Robidoux, Risko, and Besner (2007) suggested that monitored conflict modulates associative strengths within information-processing channels (see also Verguts & Notebaert, 2008). Our proposal is formulated along similar lines.

Our proposed extension of the conflict/control loop theory is that the amount of conflict monitored on a trial is used as a modulator for associating the current stimulus with the response. In computational terms, the associative connection between the stimulus (i.e., the whole stimulus array, target + flankers) and the response is adjusted in proportion to the amount of conflict monitored. The sign of the adjustment depends on the accuracy of that trial; after a correct response to a high-conflict trial, the associative strength will strongly increase, whereas after an incorrect response to a high-conflict trial, the associative strength will strongly decrease. Correct and error responses following a low-conflict trial will lead to small increases or decreases, respectively. When the stimulus is repeated, the increased stimulus–response association leads to fast correct responses and is thus observed as the Gratton effect. In our experiment, this was seen as faster RTs for iI trials than for nI trials and faster RTs for cC trials than for nC trials. This result captures the Gratton × repetition interaction and also explains the larger positive priming after incongruent trials as due to a larger increase in associative strength.

Although the present experiment was not designed to address other variants of the conflict and priming accounts, we mention two of these variants that a reader could think of as providing alternative explanations for the data. The first possibility is that neutral stimuli produce more conflict than do congruent stimuli (e.g., Roelofs, van Turennout, & Coles, 2006) and therefore that the larger RT for nC relative to cC trials reflects a conflict adaptation effect. Although we agree that neutral trials may elicit more conflict than do congruent trials (for a model, see Davelaar, 2008), we also see that such a mechanism could not explain the full data set, since one would then have expected the RTs for nI trials to be faster than those for cI trials in the repeated sequences. Instead, the former RTs were marginally *slower*. Moreover, in the no-repetition trial pairs, there is no evidence whatsoever of cross-trial conflict adaptation, neither with incongruent nor with neutral trials. The second possibility is that the faster RTs for cC and iI trials relative to nC and nI trials,

respectively, are due to repetition of the flankers for the cC and iI trials. In a recent article, Frings, Rothermund, and Wentura (2007) provided support for their variant of Hommel's (1998) theory of event files, showing (in a negative-priming paradigm) an RT benefit when distractors *and* responses repeat across trials. Although Frings et al.'s variant and Hommel's (1998) original theory can account for the Gratton × repetition interaction, they do not capture the association between the *size* of the priming effect and the congruency of the previous trial. Instead of looking at every possible variant of conflict and priming models, we believe that the data are more consistent with a combined conflict-modulated priming account.

An interesting prediction of this account is that after an error, the adjustment should be negative, with the participants thereby unlearning the association between the presented stimulus and the incorrect response. However, in this experiment, participants did not make sufficient errors to test this prediction.

As mentioned in the introduction, we chose the arrow-flanker paradigm because this is used in the clinical literature on cognitive control. In doing so, we necessarily confounded target repetition with response repetition. To address whether the Gratton effect occurs as a result of repeating the target character, the response alone, or the full stimulus (target + flankers), we reanalyzed an unpublished study in which 22 participants made consonant–vowel judgments about a target letter (cf. Davelaar, 2008). With two vowels (E and A) and two consonants (B and K), cI trials can be compared with iI trials involving repetition of responses (e.g., BBBBB vs. BBEBB followed by KKAKK), repetition of the target character (e.g., BBBBB vs. BBEBB followed by KKEKK), or repetition of the whole stimulus (i.e., BBBBB vs. BBEBB followed by BBEBB). Analyses revealed that when the whole stimulus repeats, RTs for the iI trial are faster than those for the cI trial [RT difference = 56 msec; $t(21) = 2.67$, $p = .014$], suggesting that both stimulus and response need to be repeated (cf. Frings et al., 2007). All other comparisons were nonsignificant (and there were no differences among iI trials, including one in which only the flankers repeated—i.e., BBABB).

Although the debate regarding the role of conflict in accounting for the Gratton effect has focused on the flanker task, such discussion is absent for the Stroop task. This may be due to the fact that both tasks differ critically in their underlying processing demands and sensitivity to conflict. For example, Spieler, Balota, and Faust (2000) showed that in the Stroop task, but not in the flanker task, the exponential component of the RT distribution is affected by the congruency of the trial. This was related to the differential requirement of processing competing information from the same (Stroop) or a different (flanker) location. Indeed, when the word and color information were spatially separated, the congruency effect on the exponential part of the RT distribution disappeared (Spieler et al., 2000, Experiment 5).

Note that although our experiment replicated the findings of Mayr et al. (2003), it did not falsify the hypothesis that conflict monitored within a trial is used to control

information processing. The results do falsify a general stimulus-independent conflict/control mechanism, but we propose an explanation in which stimulus-induced conflict modulates the associative strength between stimulus and response. Further empirical and computational research is needed to address the precise relation between conflict-induced cognitive control and priming; the details of what aspects of information processing are controlled using monitored conflict (spatial attention, stimulus–response mapping); when this control is exerted (within a trial or between trials); and how it is exerted (lingering activations or updating connections). The proposal of conflict-modulated priming remains to be tested computationally, and it has the potential to unify the previously competing accounts, furthering our efforts to understand the mechanisms of cognitive control.

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