

# Conflict adaptation effects in the absence of executive control

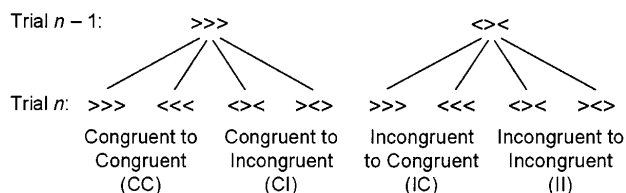
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According to the 'conflict-monitoring' model, a leading theory of cognitive control<sup>1–4</sup>, information-processing conflict registered in the anterior cingulate cortex (ACC) triggers the prefrontal cortex to reduce conflict susceptibility. Here we show that the existing empirical support for an online modulation of susceptibility to conflict through immediately preceding conflict, the 'conflict-adaptation effect'<sup>1,5</sup>, needs to be reevaluated. In a human cognitive control task, we found that it was not the stimulus-independent level of conflict that was responsible for the conflict-adaptation effect but rather an episodic memory phenomenon: stimulus-specific priming<sup>6</sup>.

The so-called flanker task<sup>7,8</sup> is frequently used to study cognitive control. Subjects respond with a left or right key press to a central target arrow while ignoring congruent (>>>) or incongruent (<><) flanker arrows. The presumed role of cognitive control in this situation is to enhance target processing and/or exclude flanker processing. Control efficiency is indexed by the congruency effect, the performance decline on incongruent compared to congruent trials. An open question is how control itself is controlled in such a situation<sup>1</sup>. That is, how does the cognitive system determine when regulation becomes necessary? The conflict-monitoring model suggests that control is modulated through a relatively 'dumb' system, situated in the ACC, which constantly extracts from ongoing information processing an



**Fig. 1.** Critical trial-to-trial transitions in the flanker task. Note that only for CC and II trials, probability of target + flanker repetitions is 0.5.

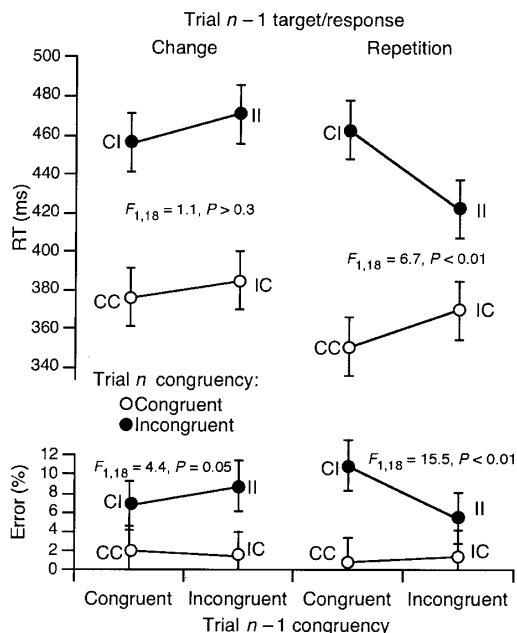
abstract index of information-processing conflict<sup>1</sup>. A high value on this index triggers regulative control sites (such as prefrontal cortex) to boost control activity.

It has been suggested that this conflict-monitoring device can be observed at work by looking at the congruency effect as a function of the congruency effect on the previous trial (trial  $n-1$ )<sup>1,5</sup>. Conflict on trial  $n-1$  should trigger tightening of control, thereby reducing susceptibility to conflict on trial  $n$ . Consistent with this prediction, incongruent-trial response times (RTs) are usually faster after incongruent trials (II) than after congruent trials (CI) and congruent-trial RTs are faster after congruent (CC) than after incongruent trials (IC)<sup>2,5</sup>.

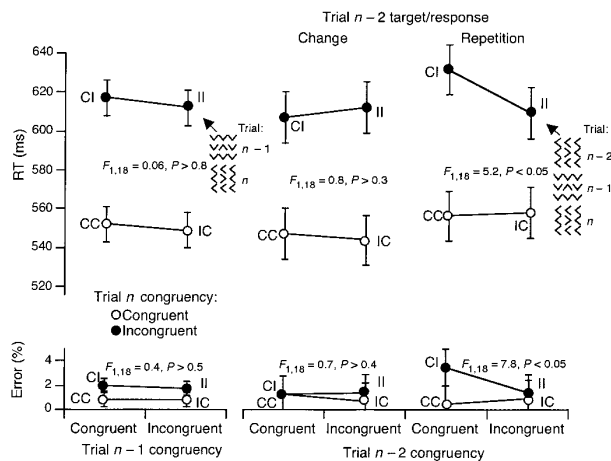
This conflict-adaptation effect is used as empirical support for the conflict-monitoring model. It is the only evidence in favor of the model's key assumption that bottom-up and dynamic modulation of control is accomplished by means of an abstract measure of most recent conflict. In contrast to this view, we suggest an explanation of the conflict-adaptation effect that requires no record of response conflict. Specifically, we propose that the conflict-adaptation effect is a consequence of episodic memory during attentional selection<sup>6</sup>, which results in stimulus-specific repetition priming.

The potential role of repetition priming becomes apparent when looking at the space of possible trial-to-trial transitions (Fig. 1). Importantly, 50% of the CC and II transitions but none of the IC and CI transitions involve exact target + flanker repetitions. This is critical in light of reports of substantial RT benefits for exact stimulus–response repetitions<sup>9,10</sup>. Accordingly, RTs in II and CC trials may be fast because of a repetition benefit that is completely absent from CI or IC trials.

To examine the role of stimulus repetitions, we assessed the conflict-adaptation effect in a standard flanker experiment. We obtained the typical conflict-adaptation pattern, with II RTs faster than CI RTs (448 ms versus 460 ms,  $t_{18} = 2.5$ ,  $P < 0.05$ ) and IC RTs slower than CC RTs (378 ms versus 364 ms,  $t_{18} = 3.2$ ,  $P < 0.01$ ) plus corresponding error effects. When we examined



**Fig. 2.** Mean response times and error percentages as a function of conflict on trial  $n$  and conflict on trial  $n-1$ , broken down by the target/response repetition factor plus  $F$ -statistics for each relevant conflict-adaptation interaction. Error bars reflect 95% within-subject confidence intervals for the critical interaction between all shown design factors. After giving informed written consent, 19 subjects (age 20–24, right-handed, 12 females) worked through one practice block and eight 64-trial test blocks. Each stimulus ensemble (a single central target arrow plus three flanker arrows on either side) was presented randomly either 2° above or below center and was 11.1° wide and 1.3° high (individual targets or flankers were 1.3° wide). Left versus right index fingers were used for arrow-compatible keypress responses. Error trials and trials following errors were eliminated from analysis. Experiments were approved by the University of Oregon Institutional Review Board.



**Fig. 3.** Mean response times and error percentages as a function of trial  $n - 1$  conflict and trial  $n$  conflict (left) and as a function of trial  $n - 2$  conflict, trial  $n$  conflict and  $n - 2$  response repetitions (right). A new group of 19 subjects (age 19–24, right-handed, 14 females) worked through three practice blocks followed by seven 64-trial test blocks. The stimulus ensemble was shown centrally and was  $4.4^\circ$  wide and high (individual targets or flankers were  $1.3^\circ$  wide and high). Alternating across trials, arrows differed in the vertical or the horizontal dimension and responses compatible with arrow directions had to be executed with the preferred-hand index finger on keys arranged in a cross-wise manner. Sample displays for representative trial sequences are shown for two conditions.

the same data separately for target/response changes and repetitions (Fig. 2), however, the conflict-adaptation pattern was present only for repetition trials with target + flanker repetitions producing particularly large benefits. Thus, stimulus-specific repetition priming can provide a complete explanation of the conflict-adaptation pattern observed here.

Of course, we cannot rule out that with variants of the flanker paradigm, a stimulus-independent conflict-adaptation pattern may be obtained. The original study<sup>5</sup> reported a small conflict-modulation effect after eliminating stimulus repetitions, but statistical reliability of this numerically reduced effect was not established. Also, even in cases of apparent stimulus-independent conflict adaptation, repetition effects could easily be based on higher-order perceptual features (for example, the degree to which display items can be grouped by similarity repeats in CC and II trials but not in CI or IC trials).

Nevertheless, to provide a broader empirical basis for the repetition-priming account, we conducted another experiment in which immediate stimulus–response repetitions were eliminated: targets and flanker arrows alternated in a trial-by-trial manner between the  $x$  or  $y$  dimensions and required corresponding left–right or up–down responses. For the conflict-monitoring account, absence of stimulus repetitions should be irrelevant. However, the priming account predicts that elimination of repetitions eliminates the conflict-adaptation pattern. Consistent with this prediction, no conflict adaptation as a function of conflict on trial  $n - 1$  was observed (Fig. 3, left).

Although all immediate stimulus or response repetitions were eliminated, such repetitions did occur from trial  $n - 2$  to trial  $n$ . The priming account predicts that the conflict effect on trial  $n$  should be affected by conflict on trial  $n - 2$ , although again limited to complete target + flanker repetitions. This prediction was confirmed in our data (Fig. 3, right). A memory-based priming account can easily handle ‘adaptation’ effects across intermediate trials, whereas the conflict-monitoring model does not provide a mechanism that allows adaptation across intermediate trials when there is no trial-to-trial adaptation.

The idea of conflict-triggered modulation of control is attractive because of its simplicity and its fit to certain characteristics of ACC functioning<sup>11</sup>. However, the present data challenge a key assumption of this model by showing that degree of conflict per se may not determine control on subsequent trials. Thus, the brain imaging results consistent with the ACC as a simple conflict-detection device<sup>2,3</sup> also deserve a reevaluation. According to the conflict-monitoring model, there is a relative increase in regulation on II compared to CI trials, which in turn leads to reduction of flanker-induced conflict. Thus, the finding of less ACC activation on II than on CI trials seems to suggest that the ACC does not itself regulate, but simply registers conflict<sup>2</sup>. According to the repetition-priming account, however, conflict-triggered regulation is not necessary to explain the conflict-adaptation effect. This reopens the possibility that conflict-related brain activity in the ACC does reflect regulation<sup>12</sup>. Specifically, there may be less to regulate and therefore also less ACC activity during II trials, simply because on a large proportion of these trials (50%), regulative demands are bypassed through stimulus-driven repetitions of just-executed responses<sup>10</sup>.

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**Competing interests statement**

The authors declare that they have no competing financial interests.

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