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Dissociating Local and Global Levels of Perceptuo-Motor Control in Masked Priming

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Masked prime stimuli presented near the threshold of conscious awareness affect responses to subsequent targets. The direction of these priming effects depends on the interval between masked prime and target. With short intervals, benefits for compatible trials (primes and targets mapped to the same response) and costs for incompatible trials are observed. This pattern reverses with longer intervals. We argue (a) that these effects reflect the initial activation and subsequent self-inhibition of the primed response, and the corresponding inhibition and subsequent disinhibition of the nonprimed response, and (b) that they are generated at dissociable local (within response channels) and global (between channels) levels of motor control. In two experiments, global-level priming effects were modulated by changing the number of response alternatives, whereas local-level effects remained unaffected. These experiments suggest that low-level motor control mechanisms can be successfully decomposed into separable subcomponents, operating at different levels within the motor system.

Keywords: low-level motor control, masked priming, motor activation, inhibition

The question of whether stimuli that are inaccessible to conscious awareness can have systematic effects on behavior and whether any such subliminal effects are qualitatively different from the effects of consciously perceived, supraliminal stimuli, has always been one of the most controversial issues in psychology (see Holender, 1986; and Holender & Duscherer, 2004, for critical reviews). However, in recent years, a number of response priming studies have provided unequivocal evidence that stimuli presented near or even below the threshold of conscious awareness can, under certain well-defined conditions, activate their corresponding motor responses (e.g., Dehaene et al., 1998; Eimer, 1999; Eimer & Schlaghecken, 1998; Leuthold & Kopp, 1998; Neumann & Klotz, 1994; Schlaghecken & Eimer, 2000, 2002, 2004). However, very little is known about the mechanisms underlying this type of response priming, except that it appears to reflect processes at relatively low-level stages of direct perceptuo-motor control (e.g., Neumann & Klotz, 1994; Schlaghecken & Eimer, 2001; Schlaghecken, Münchau, Bloem, Rothwell, & Eimer, 2003). The present study was conducted to investigate in more detail the mechanisms underlying low-level response priming, in particular to test the hypothesis that such priming effects are produced at dissociable "local" (within an individual response channel) and "global" (between two or more response channels) levels of perceptuo-motor control.

In the standard masked prime experiment, on each trial a backward-masked prime stimulus is presented before the response-relevant target. Primes and targets are mapped to the same response in *compatible* trials, and to different responses in *incompatible* trials. Although participants usually are unable to identify the masked primes with more than chance accuracy, behavioral as well as electrophysiological measures indicate that prime-target compatibility affects the target-related response. Typically, *positive compatibility effects* (PCEs) are observed when primes and targets are presented in close temporal succession: relative to neutral trials (where the prime is not mapped to any response), responses are faster and more accurate in compatible trials, whereas responses in incompatible trials are slower and less accurate (e.g., Dehaene et al., 1998; Leuthold & Kopp, 1998; Neumann & Klotz, 1994). The Lateralized Readiness Potential, an index of selective response preparation and activation (Eimer, 1998; Eimer & Coles, 2003), indicates that these behavioral benefits and costs are caused by the masked primes' impact on the motor system: the primes trigger an activation of their corresponding response, which facilitates responding to compatible targets,

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and impairs responding to incompatible targets (Dehaene et al., 1998; Leuthold & Kopp, 1998). Such findings suggest the existence of direct perceptuo-motor links (Neumann, 1990), which allow sensory information access to motor stages even if it has not been accessible to conscious awareness.

However, the masked prime task does not always produce PCEs. In fact, when the interstimulus interval (ISI) between prime offset/mask onset and target onset (prime/mask-target ISI)[1] is increased to approximately 100 ms or longer, PCEs turn into *negative compatibility effects* (NCEs), with performance benefits on incompatible trials and costs on compatible trials, relative to neutral trial (for an overview, see Eimer & Schlaghecken, 2003). Behavioral (Eimer, Schubö, & Schlaghecken, 2002; Klapp, 2005; Klapp & Haas, 2005; Klapp & Hinkley, 2002; Lingnau & Vorberg, 1995; Schlaghecken & Eimer, 2002) and electrophysiological (Eimer, 1999; Eimer & Schlaghecken, 1998; Seiss & Praamstra, 2004; Praamstra & Seiss, 2005) evidence suggests that this inverse priming effect is caused by the active inhibition of the primed response. Thus, the observed pattern of PCEs at short prime/mask-target ISIs followed by NCEs at longer prime/mask-target ISIs can be interpreted as reflecting an activation-followed-by-inhibition sequence. Initially, the prime-induced motor activation facilitates the prime-compatible response and impairs the prime-incompatible response. However, when the mask removes the supporting sensory evidence for the primed response, this initial activation should quickly decrease. In fact, evidence has been provided to suggest that if the prime has a relatively strong perceptual impact (presumably causing a correspondingly strong motor activation that even might result in an overt response), the initial activation not only decays passively, but is actively inhibited, thereby leaving the opposite response relatively more active. If a target is presented and a response elicited during this subsequent inhibition phase, then performance on compatible trials is impaired, and performance on incompatible trials is facilitated.[2]

We have argued (Bowman, Schlaghecken, & Eimer, 2006; Schlaghecken & Eimer, 2000, 2001, 2002) that the inhibitory mechanism responsible for NCEs does not represent top-down or executive control but rather a low-level self-inhibition process acting as an "emergency brake" mechanism in motor control. According to this account, response activation and inhibition act as opponent processes, with inhibition as a direct consequence of response activation whenever a relatively strong activation—which might interfere with overt performance—is suddenly no longer supported by sensory evidence. Empirical support for this account has been obtained in studies in which the prime's potential impact on the motor system was manipulated by varying its perceptual strength (Eimer & Schlaghecken, 2002; Schlaghecken & Eimer, 2000, 2001, 2002). NCEs—indicating the presence of self-inhibition processes—were obtained only with perceptually "strong" near-threshold primes, likely to have a correspondingly strong impact on the motor system. In contrast, only PCEs—indicating the absence of self-inhibition—were observed when the perceptual strength of the masked primes (and thus their potential impact on the motor system) was further reduced. Unsurprisingly, only PCEs also occurred when, at the other extreme, primes were unmasked and thus perceptual support for the corresponding response remained present throughout response selection and activation (Klapp & Hinkley, 2002).

To account for positive and negative compatibility effects in masked priming, we recently have proposed a functional model of low-level perceptuo-motor control processes (Schlaghecken & Eimer, 2002; a detailed computational model is provided in Bowman et al., 2006). Here, the activation-followed-by-inhibition sequence is interpreted as reflecting the operation of a nested opponent network. Similar opponent-process systems have been used repeatedly in neural network models of inhibitory control (e.g., Houghton & Tipper, 1994; Houghton, Tipper, Weaver, & Shore, 1996). A simple schematic of such a network is shown in Figure 1a for an experimental situation with two possible response alternatives (left and right hand). At the core of this model is the assumption that inhibitory motor control includes both global and local levels, in which the global level describes interactions across response channels and the local level describes interactions within single response channels. At the *local level*, each response channel consists of two asymmetrically linked nodes: an activation or ON node, which is directly activated by input from the perceptual system, and an inhibition or OFF node. Response execution starts when the ON node activation level exceeds threshold. Of importance, ON and OFF nodes form a self-inhibition circuit, where the ON node has an excitatory connection to the OFF node, which feeds inhibitory input back to the ON node. Because response inhibition appears to be linked to the perceptual strength of masked primes (as mentioned previously in this article), the additional assumption is made that the OFF node will inhibit the ON node only when the OFF node activation level exceeds a critical threshold (see, e.g., Hagenzieker & van der Heijden, 1990, and Hagenzieker, van der Heijden, & Hagenaar, 1990, for a similar concept of inhibition thresholds).

Consequently, local activity within a single response channel critically depends on the factors influencing activation of the OFF node. The model assumes that the presence of perceptual evidence for a particular response will increase activity in this response channel by simultaneously exciting the ON node of this local circuit and inhibiting the OFF node. Presenting a masking stimulus immediately after the prime will remove the sensory evidence supporting activity in this response channel, thereby abruptly removing both the excitation of the ON node and the inhibition of

[1] In earlier studies of the temporal characteristics of priming effects in the masked prime paradigm (e.g., Schlaghecken & Eimer, 2000), prime-mask ISI was kept constant at 0 ms to ensure optimal masking. Thus, these studies could not provide a direct answer to the question whether the direction of these priming effects is determined primarily by prime-target ISIs or by mask-target ISIs. According to the model put forward in Schlaghecken and Eimer (2002), mask-target ISIs are critical. Although the presentation of the prime triggers an activation of the corresponding response, the sudden removal of the prime—caused by the mask—is viewed as responsible for the inhibition of the primed response. However, given that there have not yet been any studies investigating the relative importance of prime-target ISI and mask-target ISI, we use the neutral term prime/mask-target ISI in the present paper.

[2] To present targets during this hypothetical inhibition phase that is assumed to be triggered by the mask, masks and targets have to be distinct stimuli that are presented successively, with targets presented after mask onset (see also Lingnau & Vorberg, 2005). In metacontrast priming experiments, where the mask itself acts as a target, relatively long prime-mask/target ISIs still result in PCEs (e.g., Neumann & Klotz, 1994).

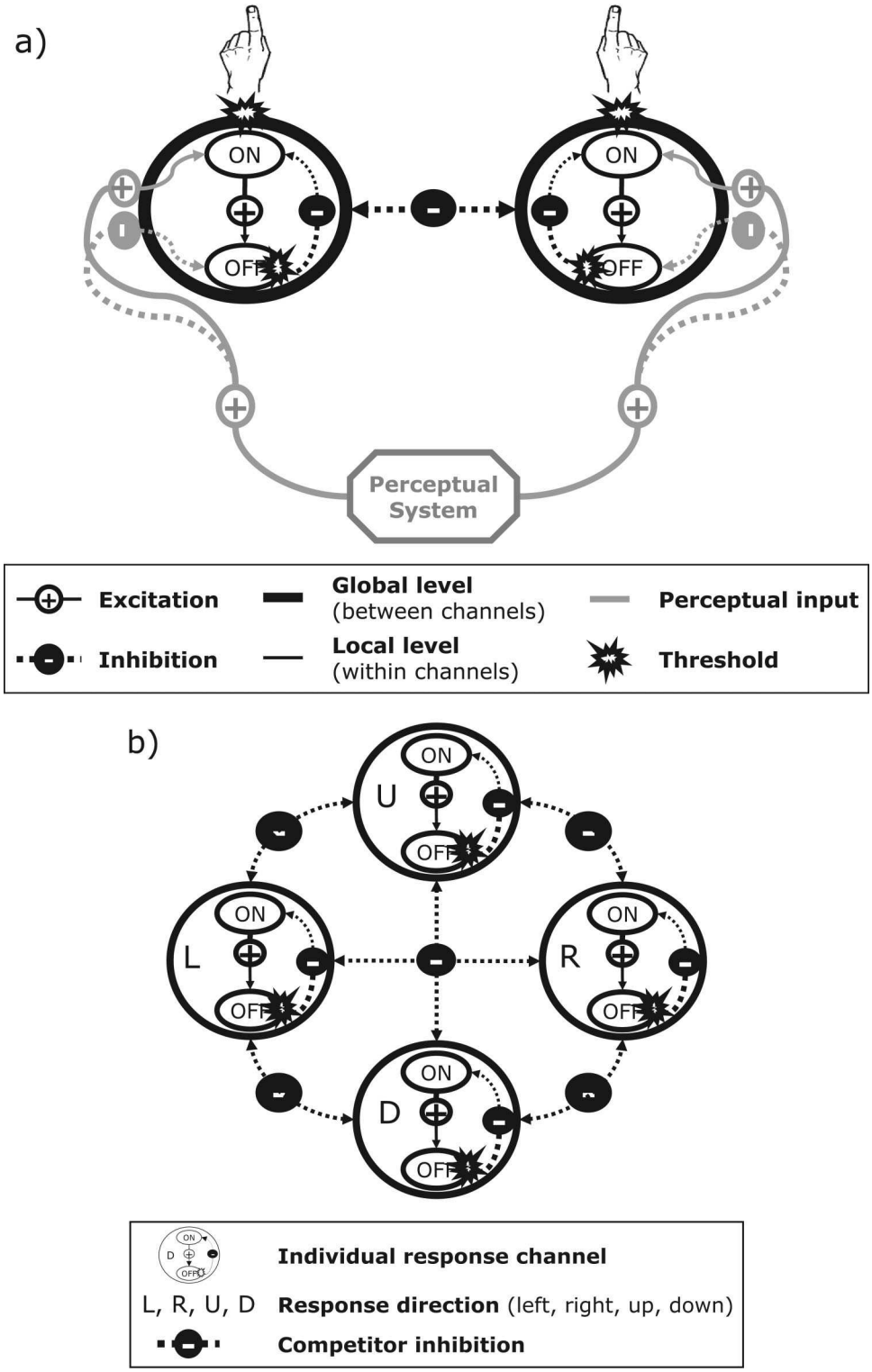

Figure 1. (a) A schematic model of global and local levels of low-level motor control in a standard situation with only two response alternatives (left hand or right hand). Black: motor system; gray: perceptual system. Excitatory connections (increasing activation levels of the target structure) are indicated by solid lines and plus signs; inhibitory connections (decreasing activation levels of the target structure) are indicated by dotted lines and minus signs. Starburst-signs indicate output thresholds. (b) The same system with four response alternatives (leftward, rightward, upward, or downward movement). For greater clarity, the perceptual system component and hand symbols have been omitted from this figure.

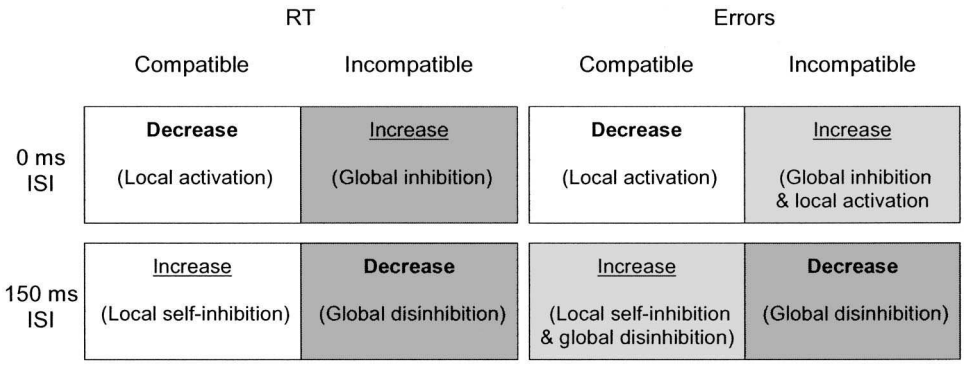

Figure 2. Summary of Local-Level and Global-Level Effects in Masked Priming With Short (0 ms) and Long (150 ms) Prime/Mask-Target Interstimulus Interval (ISI). White cells = pure local-level effects; dark gray cells = pure global-level effects; light gray cells = mixed global/local-level effects. Behavioral benefits are printed in bold, behavioral costs with underlines. Note that although behavioral costs and benefits are symmetrical for response times (RTs) and error rates, the levels that generate these effects are different.

the OFF node.[3] However, ON node activation will not decrease immediately to its resting level (if this were the case, no inhibition process would be necessary), but will decrease gradually after excitatory input is removed (e.g., Feng, Hall, & Gooler, 1990; Rolls, Tovée, & Panzeri, 1999). This means that for a short time, the OFF node will be released from inhibitory perceptual input while still receiving excitatory input from the ON node. If this excitation is sufficiently strong (because of high ON node activation levels), it is likely to cause above-threshold OFF node activation, which then inhibits its corresponding ON node (i.e., self-inhibition of the initially primed response channel). In contrast, if the excitatory input is only weak (because of low ON node activation levels with weak perceptual stimulation), activation of the OFF node is likely to remain below threshold, and no self-inhibition will occur. Of course, if excitatory input to the OFF node remains counterbalanced by inhibitory input from sustained perceptual stimulation (i.e., with unmasked stimuli), OFF node activation also will remain below threshold.

At the *global level*, competing response channels are linked symmetrically by reciprocal inhibitory connections, so that increased activation in one response channel results in a corresponding decrease of activation in the alternative response channel. This intuitively plausible concept of reciprocal inhibitory links between competing response alternatives (competitor inhibition) recently has gained additional experimental support. A series of studies using a variety of physiological measures has demonstrated that as activation levels in one response channel increase, activation levels in the alternative channel decrease correspondingly (for a review, see Burle, Vidal, Tandonnet, & Hasbroucq, 2004). A crucial assumption within the present model is that when inhibitory links between response alternatives are not activated (i.e., when one response channel fails to inhibit its competitor), alternative channels will become disinhibited and will be able to reach above-baseline activation levels even without additional sensory input. Such disinhibition or postinhibitory rebound effects frequently are observed with reciprocally linked neurons (e.g., Calabrese, 1995; Perkel & Mulloney, 1974), and have been described in some detail for primate basal ganglia-thalamo-cortical motor control circuits (e.g., Berns & Sejnowski, 1996; Chevalier & Deniau, 1990; Destexhe & Sejnowski, 1997). Thus, although at the local level, the system's performance critically depends on factors influencing activation levels of one particular OFF node, at the global level it depends on factors influencing the relative activation levels of potentially competing alternative response channels.

Although the distinction between local and global levels of inhibitory control is central to the functional model shown in Figure 1a, previous studies of masked priming have focused primarily on local level processes and have not yet explicitly investigated competitive interactions between response channels at the global level.[4] However, as illustrated in Figure 2, local control mechanisms are insufficient to fully explain the overall pattern of effects obtained in many of these studies. Consider first PCEs that are observed when prime/mask-target ISIs are short (0 ms ISI in Figure 2). Here, performance benefits on compatible trials (shorter reaction times, reduced error rates) can be explained by local

[3] In the model described in Bowman et al. (2006), OFF nodes are isolated from perceptual input, and masks directly affect ON node activity instead. This is functionally equivalent to the model described here: in both cases, successful masking of the prime results in an imbalance of the local self-inhibition circuit, which allows the OFF node to reach threshold activation levels and thus to inhibit its corresponding ON node. For the present purposes, depicting this mechanism as described above represents a simple way to conceptualize the relationship between perceptual evidence for a given response and activation levels within the corresponding response channel.

[4] An exception is a recent study by Praamstra and Seiss (2005), providing electrophysiological evidence that in a masked prime task, activation of one response channel is accompanied by simultaneous inhibition of the opposite response channel. However, this study focused exclusively on activity within the primary motor cortex. Although there is reason to believe that competing motor responses have reciprocal inhibitory connections at the level of the motor cortex, it seems likely that the activation and inhibition processes studied in the context of the masked prime task are generated at earlier—presumably subcortical—stages, and that the corresponding motor cortical activation patterns only reflect these earlier processes (Schlaghecken et al., 2003).

prime-induced response activations within response channels. In contrast, performance costs on incompatible trials are likely to result from both local-level activation of the primed response and global-level inhibition of competing response channels. The inhibition of the competing response reduces its activation level, thus making its execution less likely and increasing reaction times (RTs) when it has to be executed. At the same time, local-level activation has made the execution of the primed response more likely, which will be reflected by a higher percentage of incorrect (prime-compatible) responses on incompatible trials. Next, consider the condition in which prime/mask-target ISIs are longer and NCEs typically are observed (150 ms ISI in Figure 2). Here, the situation is reversed, as local self-inhibition of the primed response reduces its activation level, making its execution less likely and increasing RTs when it has to be executed on compatible trials. At the same time, global-level disinhibition of the alternative response channel has made it more likely that this response is executed, which will decrease RTs and error rates on incompatible trials and increase the percentage of incorrect (prime-incompatible) responses on compatible trials. A similar argument has been put forward by Verleger, Jaśkowski, Aydemir, van der Lubbe, and Groen (2004), who suggest that behavioral benefits for incompatible trials reflect a mechanism of "balanced competition" (i.e., at the global level), whereas costs on compatible trials reflect genuine inhibition (i.e., at the local level).

The aim of the present study was to obtain experimental evidence in support of these assumptions. One important implication of the model's architecture is that global and local levels should differ with respect to their sensitivity to variations of task set, such as manipulations of the number of response alternatives in a choice reaction time task. Primed response activation and subsequent self-inhibition are modeled as strictly local processes, which operate within specific response channels. These processes should therefore be entirely "blind" to any factors beyond the level of individual channels. In contrast, interactions between response channels at the global level should be sensitive to variations in the number of alternative responses: To the extent that competitor inhibition and disinhibition operate via distributed reciprocal inhibitory links between all potentially task-relevant response channels, increasing the number of response alternatives involved in a task set will have direct consequences for such global interactions. Figure 1b illustrates this for a task in which the number of response alternatives has been increased from two to four. Because each response in a task set competes with all other responses, the total number of inhibitory links increases from 2 to 12, as each response channel now receives inhibitory input from three competitors.

If the performance costs and benefits observed in previous masked prime studies are produced at different levels of motor control (global vs. local), then altering the number of response alternatives—and thus manipulating global, but not local levels—should have asymmetrical effects on those costs and benefits. This should be particularly the case for RTs, which (unlike error rates, see Figure 2) are assumed to be influenced purely by either the local or the global level. These predictions were tested in two experiments, which used standard masked prime procedures, except for the fact that the number of alternative responses (two vs. four) was varied across blocks. In Experiment 1, the interval separating primes and targets was 0 ms (i.e., targets were presented at the same time as the mask), which was expected to result in RT benefits (reflecting local response facilitation without subsequent inhibition) for compatible trials, and RT costs (reflecting the effects of global competitor inhibition) for incompatible trials. According to the model outlined in Figure 1, increasing the number of response alternatives should affect globally produced costs but should have no impact on (local) benefits. In Experiment 2, the interval separating primes and targets was increased to 150 ms, which should result in RT costs (reflecting local response inhibition) for compatible trials and RT benefits (reflecting the effects of global competitor disinhibition) for incompatible trials. Here, the model's predictions are exactly opposite to the predictions for Experiment 1: Increasing the number of response alternatives should now affect globally produced benefits but not (local) costs.

It is important to note that our activation-followed-by-inhibition framework represents just one possible account of PCEs and NCEs observed in masked prime experiments and that important alternative theoretical interpretations have been put forward recently. In particular, Verleger et al., (2004) have attributed the NCE to specific perceptual interactions of primes and masks, whereas Lleras and Enns (2004) have attributed it to the updating of object representations, including their perceptuo-motor links. In either case, the NCE is regarded as reflecting a sequence of initial activation of the prime-related perceptuo-motor representation followed by subsequent activation of the opposite perceptuo-motor representation, without invoking any response inhibition processes. The implications of such alternative accounts for the interpretation of behavioral costs and benefits produced by masked priming, and of the effects of the number of alternative responses, will be addressed in detail in the General Discussion.

Experiment 1

This experiment investigated the impact of manipulating the number of response alternatives on the costs and benefits obtained for compatible and incompatible relative to neutral trials when primes and targets followed each other immediately. Under such conditions, RT benefits for compatible trials (indicative of primed response activation at the local level without subsequent inhibition) and RT costs for incompatible trials (caused by global inhibition of competing responses) of similar size previously have been observed in a task with two response alternatives (e.g., Aron et al., 2003).

The present model predicts that increasing the number of response alternatives should not affect benefits for compatible relative to neutral trials because these benefits result directly from the (local) activation of the response channel corresponding to the prime. In contrast, the costs observed for incompatible relative to neutral trials are assumed to reflect the inhibition of competing responses, which is mediated by (global) inhibitory links. Thus, the size of these costs should be influenced by the number of response alternatives. With only two response alternatives, the primed activation of one response channel will strongly inhibit the only other response channel, resulting in performance costs when this response has to be executed. With four response alternatives, each channel receives lateral inhibitory input from three competitors (see Figure 1b). Activation of the primed response channel will increase the inhibition this channel exerts on its competitors, thus

reducing their activation levels. However, this implies that these channels will now be less able to inhibit each other.

Consequently, any decrease in their activation levels (as the result of stronger inhibitory signals from the primed response channel) should be partly counteracted by reduced inhibitory input from the other two nonprimed channels. In other words, the average strength of the total inhibitory input received by a nonprimed response channel should be reduced with four as compared with two response alternatives. As a result, the RT costs observed on incompatible trials should be smaller with four response alternatives. Furthermore, error rate benefits on compatible trials should show the same pattern as RT benefits because they similarly result from local-level processes only. In contrast, error rate costs on incompatible trials might be less affected than RT costs, because they are assumed to reflect both local- and global-level processes (see Figure 2).

Method

Participants. Twenty volunteers (three men), aged 18–38 years (mean age, 23.0 years), participated in the experiment for either course credit or payment of £5. According to self-report, all but four participants were right-handed, and all had normal or corrected-to-normal vision. Two participants, who produced exceptionally high error rates or RTs (more than two standard deviations above the mean) were replaced.

Stimuli and apparatus. Left-pointing, right-pointing, upward-pointing, and downward-pointing double arrow heads, subtending a visual angle of approximately 0.9° × 0.3°, served as primes and targets, and a + sign served as neutral prime (Figure 3). Masking stimuli were constructed on the basis of a 6 × 5 matrix, randomly filled with overlapping horizontal, vertical, and oblique lines of different length (0.06° to 0.3°; width 0.06°), resulting in a roughly rectangular array of about 1.4° × 0.9°. To minimize the possibility that the masks share identical features with primes and targets (see Lleras & Enns, 2004; Verleger et al., 2004), none of the lines used to construct the masking stimuli was tilted at the same angle as the lines making up the prime and target arrows. On each trial, a new random mask was created to avoid perceptual learning of the mask, which could result in an increased ability to ignore the mask and a correspondingly increased ability to consciously perceive the prime (Schubö, Schlaghecken, & Meinecke, 2001). With this type of mask and procedure, prime visibility has been shown to be drastically reduced, with prime identification performance in forced choice staircase procedures reaching above-chance levels only when prime duration was prolonged to 50 ms and more (e.g., Eimer & Schlaghecken, 2002).

Procedure. Participants were seated at a table in a dimly lit chamber, in front of a computer screen at a distance of 100 cm. A computer keyboard was placed on the table in front of them. Participants were instructed to

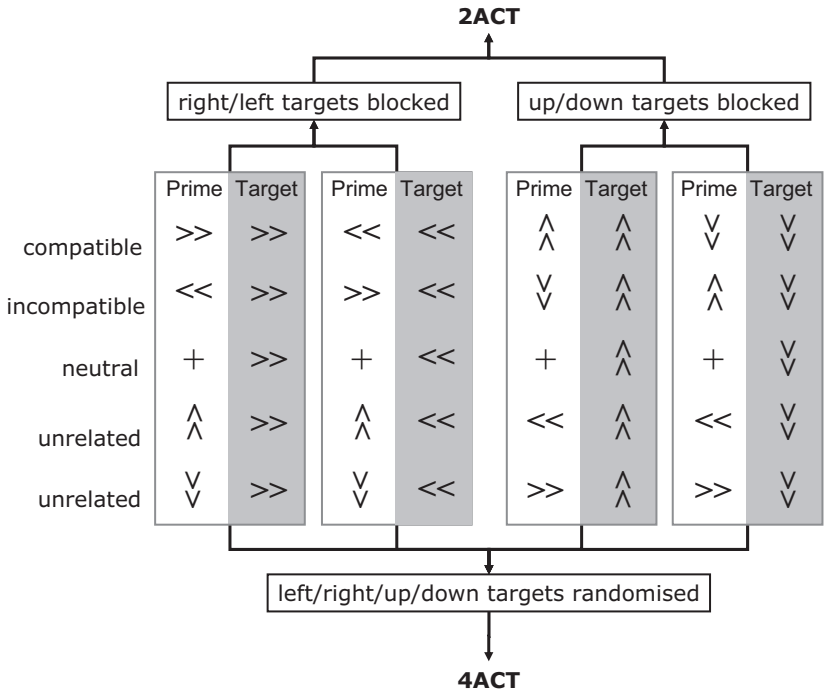

Figure 3. General experimental design: complete list of all trial types, and trial composition of the three tasks. Each of the four vertical boxes depicts all possible priming conditions (compatible, incompatible, neutral, and two unrelated conditions) for one of the four target types (right target, left target, up target, and down target). In the two-alternative choice tasks (2ACTs), only trials from either the two left-hand boxes (right target and left target) or the two right-hand boxes (up target and down target) were presented within a given experimental block. In the four-alternative choice task (4ACT), all trials from all four boxes were presented in each experimental block. Note that regardless of the number of response alternatives, each experimental block contained all primes and, consequently, all five types of prime-target relationships. Further note that in the 4ACT condition, "unrelated" trials were incompatible in that the prime indicate a different response from the target. However, as the status of these trials differed between 2ACT and 4ACT blocks, they were not included in the analysis.

maintain central eye fixation. Responses were given with the right middle finger on the right-side number pad of the keyboard. At the beginning of the trial, the finger rested on the central *5* key. In response to each arrow target, a spatially compatible movement had to be executed as fast and accurate as possible, that is, an upward movement (to the *8* key) in response to an upward-pointing arrow, a downward movement (to the *2* key) in response to a downward-pointing arrow, and a left- or right-going movement (to the *4* or the *6* key) in response to left- or rightward-pointing arrows, respectively. The necessity to return to the central key at the end of each trial was particularly stressed during instruction, and was practiced at the beginning of the experiment under the supervision of the experimenter.

The experiment consisted of two parts, which differed with respect to the number of different targets presented within a given block (two vs. four) but were identical in all other respects of stimulus presentation. In particular, all five different primes (left, right, up, down, and neutral) were presented in each block. In the first part (two-alternative choice task, 2ACT), targets within a given block were all from the same spatial dimension, that is, either all horizontal (left/right) or all vertical (up/down). Each of the two targets was presented randomly and with equal probability in each block. Participants performed 3 blocks of 80 trials each for each spatial dimension. Half of the participants started with three horizontal blocks followed by three vertical blocks; for the other half, this sequence was reversed. At the end of this part, participants were given a short rest period. In the second part (four-alternative choice task, 4ACT), consisting of 10 blocks (80 trials each), all four targets (left, right, up and down) were presented randomly and with equal probability in each block. Each part started with a 100-trial practice phase.[5] An overview of all 20 possible prime-target combinations (5 primes × 4 targets) and their distribution across conditions is provided in Figure 3.

Each trial consisted of a prime (16.7-ms duration), immediately followed by mask and simultaneously presented target (100 ms). Targets consisted of two identical double arrows, presented randomly and with equal probability either directly above or below the mask or directly left and right of the mask. The intertrial interval (ITI) was 1300 ms (Figure 4). Trials were termed compatible when prime and target arrows pointed in the same direction and incompatible when they pointed in opposite directions. On neutral trials, the prime was a + sign, which had no response assignment and never occurred as a target. To make primes completely uninformative with respect to the upcoming target, we included two types of "unrelated" prime/target trials, where prime and target were from different spatial dimensions (e.g., left prime with up target, right prime with up target, and correspondingly two unrelated trials for each of the other three targets). These five conditions (compatible, neutral, incompatible, unrelated1, and unrelated2) were equiprobable and randomized within each block and were used in both the 2ACT and the 4ACT part of the experiment. Therefore, 2ACT and 4ACT conditions only differed with respect to target presentation but were identical with respect to prime presentation (cf. Figure 3).

Data analysis. No data trimming procedure was used. Because primes on "unrelated" trials were response relevant in 4ACT blocks but not in 2ACT blocks, these trials were not fully equivalent across conditions and therefore were not formally analyzed. For compatible, neutral, and incompatible trials, repeated measures analyses of variance were computed on mean correct RTs and error rates for the factors Alternatives (2ACT, 4ACT) and Compatibility (Compatible, Neutral, Incompatible). Greenhouse-Geisser adjustments to the degrees of freedom were performed where appropriate (indicated in the results section by ε), and corrected p values are reported. Follow-up directional analyses of costs and benefits were conducted with one-tailed t tests.

Results

Figure 5 shows response times and error rates obtained for compatible, neutral, and incompatible trials in the 2ACT and 4ACT conditions. Error rates were higher in the 2ACT condition (6.2% on average) than in the 4ACT condition (4.1%), $F(1, 19) = 8.18$, $p = .010$.[6] Errors were more frequent on incompatible trials (6.1%) than on neutral and compatible trials (4.8% and 4.3%, respectively), $F(2, 38) = 10.12$, $p = .001$, $\varepsilon = .806$, but there was no interaction between Alternatives and Compatibility, $F < 1$. Because this might have been the result of a floor-effect of low error rates in the second experimental half, error data were reanalyzed taking only participants with an error rate of 5% or more in the 4ACT task into account ($n = 7$). For these participants, error rates in the 4ACT task compared to the 2ACT task showed a numerical effect in the expected direction, because costs were slightly reduced (by 1.3%), whereas benefits remained unaffected. However, this difference failed to result in a significant Alternatives by Compatibility interaction, $F < 1$.

RTs were less than 3 ms longer in the 4ACT condition than in the 2ACT condition ($F < 1$), indicating that the extensive practice had been successful. A positive compatibility effect on RTs, $F(2, 38) = 32.46$, $p < .001$, $\varepsilon = .803$, was modified by the number of response alternatives, reflected in a Compatibility × Alternatives interaction, $F(2, 38) = 3.77$, $p = .034$, $\varepsilon = .955$. Figure 5 shows that RTs were shorter on compatible than on neutral trials (behavioral benefits) and longer on incompatible than on neutral trials (behavioral costs). However, whereas benefits were indistinguishable between the two conditions, $t(19) < 0.02$, costs were significantly reduced in the 4ACT condition compared with the 2ACT condition, $t(19) = 2.3$, $p = .016$. Two additional t tests, comparing neutral trials with compatible trials and incompatible trials, respectively, confirmed that both costs and benefits were significant in both conditions (2ACT: benefits 9.4 ms, costs 14.2 ms; 4ACT: 9.3 ms and 4.6 ms, respectively; all ts(19) > 2.5, all p values $< .012$).

Discussion

The results of Experiment 1 are in line with the predictions derived from the functional model of perceptuo-motor control shown in Figure 1. RT benefits and costs of masked priming were of equal size when there were only two response alternatives. Increasing the number of response alternatives to four left the benefits for compatible trials entirely unaffected, as would be expected if these benefits were caused by a strictly local process of

[5] Unlike the subsequent self-inhibition phase studied in Experiment 2, the initial prime activation phase is transient and turns into self-inhibition if overt responses are not executed quickly (e.g., Eimer, 1999). Extensive practice was therefore given to avoid long overall reaction times, and in particular to avoid an additional RT lengthening in the 4ACT compared with the 2ACT condition, which otherwise would compromise any interpretation of effects.

[6] This unexpected decrease in error rates is most likely due to the extensive practice given before the 4ACT task. Note that the relevant S-R mappings (left/right and up/down) had already been used throughout the first half of the experiment. Thus, during the first few practice trials, participants tended to responded much slower, but not to make more errors, than during the preceding 2ACT trials. As practice progressed, both RTs and error rates decreased, until RTs became comparable with those in the 2ACT task—and error rates, correspondingly, became considerably less than in the 2ACT task.

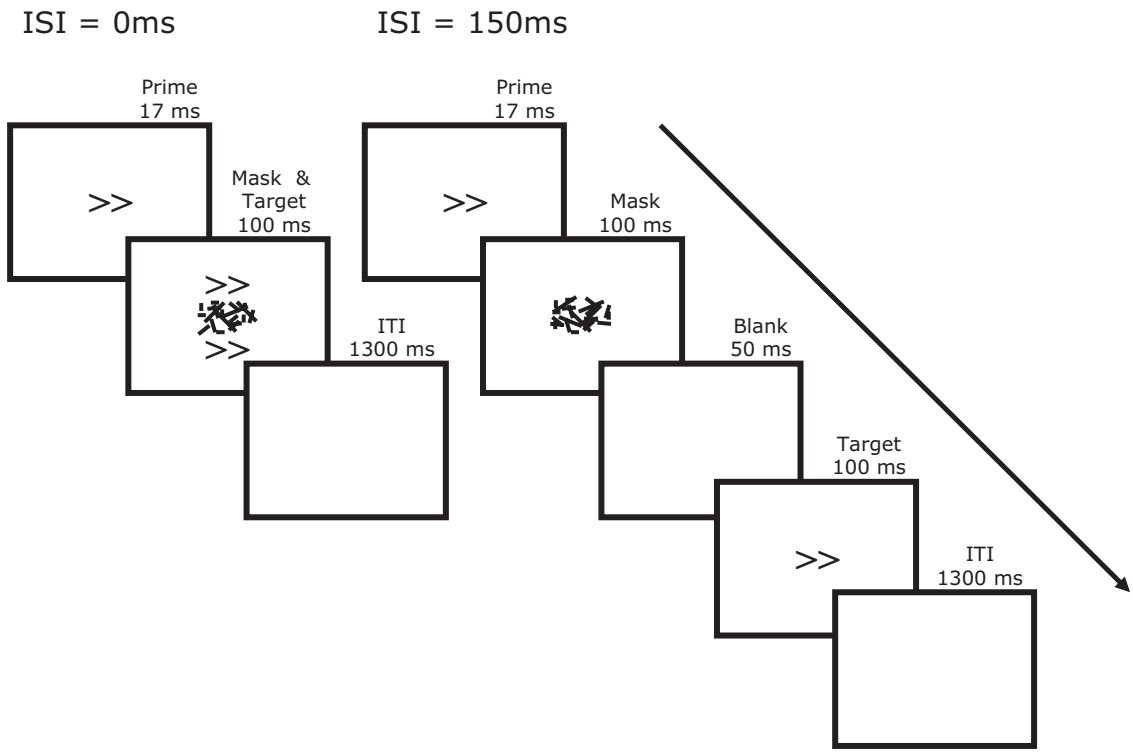

Figure 4. Trial structure in Experiment 1 (interstimulus interval [ISI] = 0 ms) and Experiment 2 (ISI = 150 ms). The two examples depict compatible trials.

primed response activation, independent of any changes in task set. In contrast, RT costs on incompatible trials were markedly reduced (albeit still significant) with four response alternatives relative to two response alternatives.

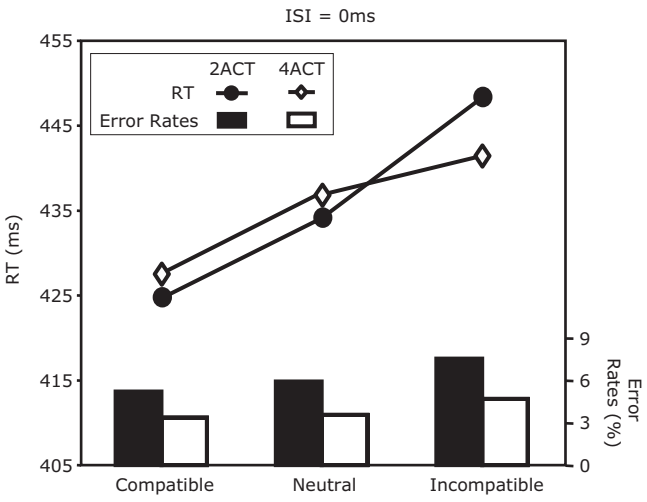

Figure 5. Results of Experiment 1: Reaction times (RTs, line graphs) and error rates (bar graphs) in compatible, neutral, and incompatible trials under two-alternative choice task (2ACT) conditions (black circles, black bars) and under four-alternative choice task (4ACT) conditions (white diamonds, white bars).

This result fits with the hypothesis that these costs result from competitive interactions between response channels at the global level. Primed activation of one response channel increases this channel's inhibitory influence on its competitors. When there is only one competing response, this channel will become strongly inhibited, resulting in behavioral costs on incompatible trials (i.e., on trials requiring the execution of this inhibited response). With four response alternatives, increasing the inhibitory input from the primed response channel to its three competitors will still reduce activation within these response channels. However, this is at least partly compensated by the corresponding reduction in the lateral inhibition between these three channels, resulting in an average inhibitory input to each nonprimed channel that is smaller with four than with two response alternatives. Consequently, the RT costs on incompatible trials should be smaller with four as compared with two response alternatives, which was exactly what was observed.

One might argue that the present results simply indicate that the capacity of a response channel to inhibit its competitors is limited, such that with larger numbers of response alternatives, each competing channel will receive a correspondingly smaller proportion of inhibitory input from the primed channel. Although this interpretation is equally consistent with the local/global model of low-level motor control, it seems less plausible than the "interacting inhibition" account outlined previously in this article: in general, the strength of lateral competitor inhibition is assumed to depend only on two factors (e.g., Arbuthnott, 1995), the activation level of the inhibiting node, and the degree of relatedness between

inhibiting and inhibited node—it is not assumed to depend on the number of competing nodes.

Experiment 2

Experiment 2 was similar to Experiment 1 (with exceptions as outlined herein) but with a prime/mask-target ISI of 150 ms, which was expected to result in NCEs on performance. According to the model shown in Figure 1, RT costs for compatible relative to neutral trials should be unaffected by the number of response alternatives because they result directly from the (local) self-inhibition of a primed response. In contrast, benefits observed for incompatible relative to neutral trials should become smaller when the number of response alternatives is increased. These benefits are assumed to result from the disinhibition of competing response channel(s), which is mediated by the global-level inhibitory links between task-relevant response alternatives. With only two response alternatives, self-inhibition of the primed response results in disinhibition of the only competing response, allowing it to reach substantial activation levels. This is reflected in performance benefits when this response has to be executed on incompatible trials. With four response alternatives, self-inhibition of the primed response channel will still remove the inhibition this channel exerts on its competitors. However, this will not remove the reciprocal inhibition these three nonprimed channels exert on each other (cf. Figure 1b). Because of this continued reciprocal inhibition, each of these three channels can be expected to remain at a relatively low activation level even after the primed response channel is inhibited. As a result, performance benefits on incompatible trials should be small or possibly even completely absent.

Method

Participants. Twenty volunteers (four men) from the departmental subject panel, aged 18–47 years (mean age, 24.7 years), participated in the experiment for course credit. According to self-report, all but two participants were right-handed, and all had normal or corrected-to-normal vision.

Stimuli and apparatus. These were identical to Experiment 1.

Procedure. The procedure in Experiment 2 was identical to Experiment 1 with the following exceptions (see Figure 4). Masks were now followed by a 50-ms blank screen, after which the target was presented, resulting in a prime/mask-target ISI of 150 ms. Targets were not presented near fixation, as in Experiment 1, but at fixation, in the same location as primes and masks. This change was introduced to make the procedure identical to NCE conditions used in almost all previous masked prime experiments. The number of practice trials was reduced to 20, as pilot data had indicated that satisfactory performance levels could be achieved without the need for excessive training.[7]

Data analysis. Analysis of data was identical to Experiment 1.

Results

Figure 6 shows reaction times and error rates obtained for compatible, neutral, and incompatible trials in the 2ACT and 4ACT conditions. Error rates were lower in the 4ACT condition (2.0% on average) than in the 2ACT condition (3.3%), $F(1, 19) = 7.15$, $p = .015$, but there was no significant main effect of compatibility, nor an interaction of Alternatives x Compatibility (both F values < 2.7, both p values $> .1$).

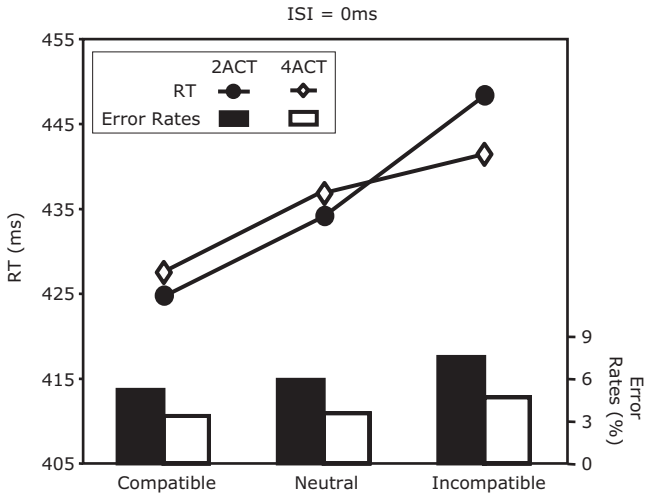

Figure 6. Results of Experiment 2. Reaction times (RTs, line graphs) and error rates (bar graphs) in compatible, neutral, and incompatible trials under two-alternative choice task (2ACT) conditions (black circles, black bars) and under four-alternative choice task (4ACT) conditions (white diamonds, white bars).

RTs were about 11 ms longer in the 4ACT condition than in the 2ACT condition, $F(1, 19) = 8.55$, $p = .009$, reflecting the difference in task difficulty. A negative compatibility effect on RTs, $F(2, 38) = 33.80$, $p < .001$, $\varepsilon = .798$, was found to be modified by the number of response alternatives, reflected in an Alternatives $\times$ Compatibility interaction, $F(2, 38) = 6.87$, $p = .003$, $\varepsilon = .942$. As can be seen from Figure 6, RTs were longer on compatible than on neutral trials (behavioral costs) in both conditions. In contrast, RTs were shorter on incompatible than on neutral trials (behavioral benefits) in the 2ACT condition, but not in the 4ACT condition (compatible, neutral, and incompatible trials: 457 ms, 448 ms, and 439 ms in 2ACT, and 466 ms, 456 ms, and 458 ms in 4ACT, respectively).

Whereas costs were of similar size in the two conditions, $t(19) < 0.5$, benefits were significantly reduced in the 4ACT condition compared with the 2ACT condition, $t(19) = 3.9$, $p < .001$. Additional t tests, comparing compatible with neutral trials and incompatible with neutral trials, confirmed that in the 2ACT condition, both costs and benefits were significant (costs: $t(19) = 3.08$, $p = .003$; benefits: $t(19) = 4.43$, $p < .001$). In the 4ACT condition, costs were significant, $t(19) = 5.59$, $p < .001$, but there was no indication of any significant benefits for incompatible relative to neutral trials, $t(19) < 1$.

Discussion

The results of Experiment 2 provide further evidence in favor of the model of low-level perceptuo-motor control shown in Figure 1,

[7] Note that under NCE conditions, in contrast to short-ISI PCE conditions, longer RTs do not pose a problem for data interpretation because robust NCEs have been observed even with (a) prime/mask-target ISIs of up to 200 ms (Schlaghecken & Eimer, 2000) and (b) RTs of 500 ms and longer (Eimer, 1999).

and in particular for the core assumption of this model that such control processes operate on separable global and local levels. As in Experiment 1, there were substantial RT benefits and costs of masked primes with only two response alternatives. Increasing the number of response alternatives from two to four left the costs for compatible trials unaffected, as would be expected if these costs were caused by the self-inhibition of primed response activations at a strictly local level. In contrast, RT benefits for incompatible relative to neutral trials were eliminated completely with four response alternatives. This finding is fully in line with the model's assumption that these benefits originate at the global level and thus will be affected by manipulations of task set. With two response alternatives, self-inhibition of the primed response channel eliminates this channel's inhibitory influence on the nonprimed channel. Because there is no sustained inhibitory input from any other channels, RT benefits will be observed for incompatible trials. With four response alternatives, inhibition of the prime response channel still results in disinhibition of the other channels but is counteracted by sustained inhibitory interactions between the three nonprimed channels. As a result, RT benefits on incompatible trials will be reduced or even (as demonstrated by Experiment 2) completely absent.

It has to be noted that Klapp and Hinkley (2002; Exp. 3) reported that NCEs did not differ between a two- and a three-alternative choice RT task. However, according to the model discussed here, increasing the number of response alternatives from two to three should have a less pronounced effect than doubling the number of response alternatives to four (in the former case, the number of global inhibitory links increases from two to six; in the latter, it increases to 12). Furthermore, in Klapp and Hinkley's experiment, no neutral trials were included, and the number of response alternatives was varied in a between-subjects design. Therefore, it was not possible to conduct a direct cost/benefit analysis, and baseline differences in effect size between experimental groups might have further obscured any potential reduction of benefits on incompatible trials. Finally, although Klapp and Hinkley did not report statistically significant differences, there was at least a numerical reduction of the NCE in the 3ACT compared to the 2ACT condition, in line with the findings reported above.

In contrast to the clear-cut costs and benefits observed for RTs in Experiment 2, there were no main effects or interactions involving compatibility on error rates. Even numerically, error rates on compatible trials in the 2ACT condition did not exceed those on neutral trials. The most likely explanation for this unexpected finding is that, similar to Experiment 1, there was a floor effect for error rates. Only 4 of 20 participants had error rates of 5% or more in the 2ACT condition, and only one produced more than 5% errors in the 4ACT condition.

General Discussion

The aim of the present study was to gain further insights into the mechanisms underlying response priming effects in the masked prime task by testing the hypothesis—derived from our recent functional model of low-level motor control (Schlaghecken & Eimer, 2002; Bowman et al., 2006)—that these effects reflect dissociable local and global processes of low-level motor control. Local processes operate within response channels and are therefore "blind" to any experimental manipulations beyond the level of individual channels. In contrast, global processes operate between response channels and should therefore be affected by manipulations of task set, such as varying the number of alternative responses.

To test these assumptions, we compared the costs and benefits of masked priming in compatible and incompatible relative to neutral trials under conditions in which there were either two or four alternative responses. In addition, prime-target intervals were either short (0 ms; Experiment 1) or long (150 ms; Experiment 2), to produce overall response priming effects of opposite polarity (PCEs vs. NCEs). Previous studies have demonstrated that masked primes trigger an initial activation of their corresponding motor response that is later followed by an inhibition of this primed response tendency (Eimer, 1999; Eimer & Schlaghecken, 1998; Klapp & Hinkley, 2002; Praamstra & Seiss, 2005; Schlaghecken & Eimer, 2000). According to our model, these prime-induced response activation and self-inhibition processes operate strictly locally at the level of individual response channels. Local activation and self-inhibition can account for the presence of RT benefits (with short prime-target intervals) and costs (with longer intervals) for compatible relative to neutral trials. However, these within-channel processes are insufficient to fully account for the corresponding pattern of error rate effects, and they cannot explain why performance on incompatible trials should be impaired (with short intervals) or facilitated (with longer intervals) when compared with neutral trials. To account for the complete set of priming effects, the model postulates an additional global level where activation in one response channel influences activation levels of competing response channels via lateral inhibitory links.

The two experiments reported here provide supportive evidence for these assumptions. For compatible trials, RT benefits observed under conditions where targets were presented immediately after the primes (Experiment 1) were replaced by RT costs when the prime-target interval was 150 ms (Experiment 2). These effects were completely unaffected by manipulating the number of response alternatives, as would be expected if they were generated at a local within-channel level of motor control. In contrast, the priming effects observed for incompatible trials were strongly modulated by the number of alternative responses. In Experiment 1, RT costs for incompatible relative to neutral trials were substantially smaller (although still statistically significant), with four response alternatives than with just two alternative responses. In Experiment 2, RT benefits for incompatible trials were only present when there were two alternative responses. In contrast, no such benefits were observed when the task set included four possible responses. These results are fully in line with the hypothesis that performance costs and benefits for incompatible trials are generated at a global (between-channel) level, and are therefore affected by the number of alternative responses included in a task set.

Error rates at least numerically followed the expected pattern in Experiment 1, but failed to show any appreciable priming effects in Experiment 2. This result, which differs from most previous masked priming studies, where compatibility effects were usually observed both for RTs and error rates, is most likely attributable to the fact that errors were very rare in both experiments, particularly

in Experiment 2. Overall, the differential impact of the number of response alternatives on the pattern of priming effects observed for compatible and incompatible trials strongly suggests that these effects are produced at different and dissociable levels of motor control, with effects on compatible trials generated at a local (within-channel) level, and effects on incompatible trials at a global (between-channel) level.

However, it should be noted that these levels are not assumed to differ with respect to their sequential (during "earlier" stages of motor processing vs. during "later" stages of motor processing), hierarchical ("low-level" vs. "high-level"), structural ("subcortical" vs. "cortical"), or processing ("automatic" vs. "controlled") characteristics. In contrast, the finding that priming effects on compatible and incompatible trials are already of equal size with a 0 ms prime/mask-target ISI, and that they seem to reverse polarity in parallel, being again of equal (though inverse) size with 150 ms ISIs strongly suggests that these effects reflect closely related, fast acting, and relatively early processes. This is further supported by results from a study comparing masked priming effects before and after prolonged repetitive transcranial magnetic stimulation of the left motor cortex (Schlaghecken et al., 2003). Despite an overall selective slowing of the right (stimulated) hand, priming effects were entirely unaffected. Similar results were obtained in experiments investigating masked priming effects under conditions where response readiness was manipulated by varying between blocks the relative a priori probability of go versus no-go trials, or of trials with targets requiring a left-hand versus right-hand response (Schlaghecken & Eimer, 2001). Although these probability manipulations had a pronounced effect on overall RTs, neither prime-induced costs nor benefits were affected.[8]

Furthermore, the fact that these response priming effects occur in the absence of reliable conscious awareness of the prime stimuli (with prime identification performance at chance level; see Eimer & Schlaghecken, 1998, 2002, 2002; Schlaghecken & Eimer, 1997, 2004) implies that the underlying processes are low level and automatic rather than high level and cognitively controlled (i.e., they are reliably activated by a particular input, without requiring "active control" from the participant). This is supported by evidence from a functional magnetic resonance imaging (fMRI) study of healthy participants (Aron et al., 2003) indicating that the activation and inhibition processes underlying NCE and PCE are mediated by subcortical structures rather than by frontal cortical areas traditionally associated with cognitive inhibitory control (e.g., Faw, 2003). Moreover, studies with patients suffering from subcortical neurodegenerative motor diseases (Aron et al., 2003; Seiss & Praamstra, 2004) have provided converging evidence that PCE and NCE are mediated by subcortical—in particular, basal ganglia-thalamic—circuits. This is in line with recent findings suggesting that more automatic, less effortful inhibition processes (as opposed to more difficult, cognitively controlled processes) recruit subcortical structures, in particular the striatum (Heyder, Suchan, & Daum, 2004; Kelly et al., 2004).

Taken together, these results suggest that motor control in the context of the masked prime paradigm is influenced by low-level, automatic processes mediated by subcortical (presumably basal ganglia-thalamic) control circuits. The present experiments provide evidence for the assumption that these circuits contain local-level and global-level subcomponents, which jointly produce the overall priming effects but which can be dissociated using the appropriate experimental procedure. This does of course not imply that these effects are entirely independent of cognitive control. On the contrary, there is strong evidence suggesting that subliminal priming effects will only be obtained when participants intentionally activate the relevant stimulus–response (S-R) mapping (e.g., Klapp & Haas, 2005; Klapp & Hinkley, 2002; Kunde, Kiesel, & Hoffmann, 2003; Schlaghecken & Eimer, 2004), which results in a transient task-specific configuration of the perceptuo-motor system. However, once this task-defined configuration has been set up, further activity in the perceptuo-motor system might be determined by it in an automatic fashion, without the need for higher-level control. Future studies could investigate whether such an intentional presetting of S-R assignments is possible only with relatively simple mappings, and whether more complex and flexible mappings (e.g., context-dependent mappings) might require constant cognitive control, and hence would not result in subliminal priming effects.

Within this context, it is interesting to compare the present results with findings reported by Jaśkowski, Skalska, and Verleger (2003). Using a modified masked prime paradigm, these authors varied the relative proportions of compatible and incompatible trials (Experiment 3), with 20% incompatible trials in one half of the experiment, and 80% incompatible trials in the other. Priming effects on RTs and error rates were substantially smaller in the latter condition than in the former. Furthermore, evidence from electrophysiological recordings (Experiment 4) suggested that both perceptual processing of the primes and prime-induced motor activation were affected by the frequency manipulation. The authors argued that this reflected a nonconscious strategic adaptation: in the 80% incompatible condition, prime-induced motor activation would substantially increase error rate (as the response opposite to the one required by the target would be primed—and hence would be more likely to be executed—on the majority of trials). Therefore, it would be advantageous to "protect" the response system from these dysfunctional influences by reducing perceptual prime processing and prime-related motor activations (Jaśkowski et al., 2003). One might argue that a similar process was operative in the present experiments because they contained a similar shift in the proportion of incompatible trials. In the 2ACT condition, 20% of all trials were incompatible (i.e., used a prime that indicated the incorrect response), whereas in the 4ACT condition, this number increased to 60% (because now the formerly "unrelated" primes

[8] This insensitivity of costs and benefits to obvious differences in response or response side probabilities between blocks observed by Schlaghecken and Eimer (2001) is relevant for the interpretation of the present findings because it suggests that these effects should likewise be unaffected by any variation in the a priori probability of trial types between task conditions. There was in fact a difference in the probability of incompatible trials in the 4ACT condition (where primes and targets were mapped to different responses in 60% of all trials), and in the 2ACT condition (where this was the case for only 20% of all trials because each block also contained 40% prime-unrelated trials). Introducing such probability differences was an inevitable consequence of the necessity to avoid any predictive link between primes and subsequent targets, together with the requirement to employ the same types of trials in the 2ACT and 4ACT conditions.

were in fact mapped to a response alternative). Consequently, the risk of committing errors on incompatible and unrelated trials should have increased substantially, and a strategic process might have been implemented to protect the response system from maladaptive prime processing.

However, the present data does not support this interpretation. If such a protective mechanism had been in place, it would have impaired prime processing and motor activation in both incompatible and compatible conditions. This was not the case. The fact that behavioral benefits (in Experiment 1) and costs (Experiment 2) with compatible primes were the same for the 2ACT and the 4ACT condition strongly suggests that there was no quantitative difference in prime processing and prime-induced motor activations between these conditions. This apparent discrepancy between the present findings and the results reported in Jaśkowski et al. (2003) might be explained in terms of differences in the predictiveness of primes between the two studies. In the Jaśkowski et al. study, primes would predict the identity of the upcoming target (and hence the required response) with 80% accuracy. In contrast, in the experiments reported previously, primes were never predictive because they were followed by either of the possible targets with equal probability. The results thus might be taken as evidence that it is the predictiveness of the primes rather than the proportion of incompatible trials as such which has a potential effect on prime processing.

Although the present findings are fully in line with the model of global and local levels of perceptuo-motor control, as illustrated in Figure 1, alternative interpretations are possible. In particular, different accounts of low-level motor priming effects in the masked prime task—and in particular the NCE—recently have been put forward by Lleras and Enns (2004) and by Verleger et al. (2004). Although these authors differ in their assumptions about the mechanisms responsible for the NCE (object updating vs. perceptual interactions between primes and masks), they agree that NCEs can be explained without invoking any form of inhibition. Instead, they propose that when masks share features with prime and target stimuli (e.g., when arrow-like stimuli are used to mask prime arrows), such mask features can trigger response activation processes. In particular, mask features that are novel (i.e., not already part of the prime) are necessarily linked to a different response than the response assigned to the prime and will cause the initial prime-induced motor activation to be replaced by a different mask-induced motor activation. According to this view, NCEs do not reflect any local self-inhibition of the response triggered by the prime, but instead positive response priming triggered by the prime, followed by positive response priming of opposite polarity, induced by masks that contain task-relevant features.

However, there is some evidence to suggest that this positive mask priming hypothesis might not be able to explain the full pattern of masked priming effects. Specifically, it can be demonstrated that NCEs can be reliably observed even when the mask does not contain any features that are similar to prime and target (e.g., when the mask is composed exclusively of horizontal and vertical lines, e.g., Klapp, 2005; Schlaghecken & Eimer, in press). In the present study, we explicitly aimed to avoid any obvious similarity between features of the mask and features of primes and targets by using masks composed of line segments that differed in their orientations from the prime and target arrows. These masks appear to be less likely to induce specific response activations, as suggested by Lleras and Enns (2004) and Verleger et al. (2004). Of course, one might use a more abstract notion of similarity, according to which the presence of overlapping diagonal lines—regardless of their actual orientation—is sufficient to create left- and right-pointing arrow-like shapes that are capable of triggering motor responses. This important issue has to wait for further clarification in future experiments, where the degree of feature overlap between prime/target and mask has to be systematically manipulated.

Along similar lines, one might want to argue that the change of priming direction between experiments (from PCEs in Experiment 1 to NCEs in Experiment 2) was not so much induced by lengthening of the prime/mask-target ISI from 0 to 150 ms but was at least partly the result of the simultaneous change in target position (above and below fixation in Experiment 1, at fixation in Experiment 2). In fact, Lleras and Enns (2005) found NCEs with nonarrow masks only when primes and targets were both presented at fixation, but obtained PCEs with central primes and noncentral targets. The authors interpreted this result as evidence that the NCE reflects "perceptual interactions among stimuli that appear in the same spatial location." Given these findings, one might speculate that no NCEs would have been observed in the present Experiment 2 if targets had been presented noncentrally.

Although this issue cannot be resolved on the basis of the present data, it is worth noting that in the Lleras and Enns (in press) study, prime identification performance was 80% correct with nonarrow masks. Therefore, their finding of PCEs with noncentrally presented targets is in line with earlier results indicating that with sufficiently high prime visibility, PCEs rather than NCEs are obtained (Eimer & Schlaghecken, 2002). Of importance, however, Lleras and Enns (in press) used the usual procedure and tested prime identification performance in blocks where no targets were presented. Thus, it seems likely that on those trials of the masked prime task where targets were presented at the same location as the masked primes, these targets effectively acted as a second masking stimulus and seriously impaired prime visibility. In fact, prime visibility under this condition might have been substantially below the levels indicated in the "prime-only" identification task,[9] and might have been sufficiently low to enable NCEs. If this is the case, than the findings of Lleras and Enns (in press) are fully in line with the self-inhibition account of masked priming.

At present, the debate as to whether NCEs reflect response inhibition or positive mask-induced response priming is far from settled. Therefore, it might be useful to speculate whether a positive mask priming account also might be able to explain the impact of increased numbers of response alternatives on masked priming effects observed in the present study. In fact, it seems that this alternative account might explain the observed effects just as well as the model illustrated in Figure 1.[10] Consider first the case

[9] In fact, pilot testing in our laboratory suggests that at least subjectively, prime visibility with nonarrow masks drops drastically when targets are presented at the same location.

[10] Our thanks to an anonymous reviewer for suggesting this alternative explanation.

of long prime/mask-target ISIs (150 ms). In the 2ACT condition, the prime (e.g., a right-pointing arrow) initially primes its corresponding response ("right") but is quickly replaced by the opposite response ("left") primed by the mask. A neutral prime, in contrast, does not prime any response, nor does the subsequent mask. Consequently, responding to a compatible (right-pointing) target will be impaired, and responding to an incompatible (left-pointing) target will be facilitated, relative to the neutral prime condition. In the 4ACT condition, the prime (again, a right-pointing arrow), also primes its corresponding response ("right"), but it is then replaced by three partial response activations ("left/up/down") that are simultaneously primed by the mask (provided that there is no a priori reason to assume that the mask would prime only the exact opposite of the prime arrow). Again, the neutral prime and its mask do not prime any response. Therefore, responding to a compatible (right-pointing) target will still be impaired, because "right" has not been primed. However, responding to an incompatible (left-pointing) target will be less facilitated (or not facilitated at all), because "left" is only one out of three equally primed responses. It is important to emphasize, however, that to explain the observed effects, this account still has to assume that competing response alternatives have to be connected by reciprocal inhibitory links. Otherwise, it would be unclear why responses on compatible trials should be delayed relative to neutral trials (note that in both cases, the response required by the target has not been activated—the only difference is that on neutral trials, no other response has been, either, whereas on compatible trials, the alternative responses are preactivated). Thus, although a positive mask priming hypothesis can account for the observed effects without the need for (local-level) self-inhibition, it still needs to assume the existence of (global-level) competitor inhibition.

The need to invoke global-level inhibition in the context of a positive mask priming hypothesis becomes even more apparent when trying to account for the findings of Experiment 1 (0 ms prime/mask-target ISI). The basic assumption is identical to the one implied by our model: PCEs are obtained with simultaneous onset of mask and target because target-related processes begin during the initial, prime-induced activity phase, that is, before subsequent mask-induced processes (self-inhibition in our model, opposite priming according to the positive mask priming hypotheses) can take effect. Again, one needs to assume that activated responses inhibit their competitors, so that although responding to a correctly primed (compatible) target is facilitated, responding to an incorrectly primed (incompatible) target is impaired relative to the neutral condition. Whereas the predictions derived from these assumptions are straightforward for the 2ACT condition, they are less clear for the 4ACT condition. To account for the fact that responses on incompatible trials are less delayed with four than with two response alternatives, the positive mask priming hypotheses need to postulate—as does the present model—that reciprocal inhibitory links between response channels are not simply two-way (i.e., between two opposite responses, as might be implemented by cortico-spinal or intrahemispheric cortico-cortical links, see Burle et al., 2004), but are multidimensional, so that activity in one channel will result in strong inhibition if there is only one competitor, but in considerably weaker net inhibition if there are several competitors.

Thus, it seems that although they do not assume the existence of local-level self-inhibition, the positive mask priming hypotheses of Verleger et al. (2004) and Lleras and Enns (2004) are more compatible with the model presented here than is obvious at first glance. This might suggest that this model could be more generally applicable to low-level motor control processes, and should not just be seen as an extension of the self-inhibition account of NCEs. Furthermore, it should be noted that whereas positive mask priming hypotheses can account for the findings of the present study, they do not specifically predict them. In contrast, the motor control model presented here is not merely consistent with these findings but specifically predicted the observed pattern of results and therefore would have been disproved if different results had been obtained.

At a more general level, the present experiments contribute to the study of cognitive control by demonstrating how low-level motor control mechanisms can be dissociated into component processes. Although different aspects of cognitive control have been studied for a long time, questions such as how control mechanisms can detect situations that require their involvement (e.g., Botvinick, Braver, Barch, Carter, & Cohen, 2001), or how they initiate appropriate adjustments in performance (e.g., Ridderinkhof, van den Wildenberg, Segalowitz, & Carter, 2004) have only recently begun to be addressed systematically. In order to gain further insights into the processes involved in cognitive control and their neural basis, globally defined control functions need to be decomposed into subprocesses that can then be studied independently with behavioral and neuroscientific methods (e.g., Friedman & Miyake, 2004; Garavan, Ross, Murphy, Roche, & Stein, 2002). The present study has demonstrated that this general strategy can be successfully applied to inhibitory motor control processes by showing that these processes can be decomposed into separate mechanisms that operate at different levels within the response system.

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