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Research report

# Where action impairs visual encoding: an event-related fMRI study

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**Abstract**

Behavioral experiments revealed an impairment in a perceptual task when a motor task has to be planned in parallel. In two event-related fMRI experiments healthy participants performed a GO–NOGO motor task and a visual identification task. Thus, we were able to investigate the influence of a motor task on visual identification. The paradigm allowed to compare visually identical trials with and without a concurrently performed motor response. In Experiment 1, the visual task focused on shape identification, whereas in Experiment 2, the visual task focused on color identification. We found an action-dependent BOLD response modulation in extrastriate visual areas (V3, V3A in Experiment 1 and additionally V4 in Experiment 2). Thus, results demonstrate that the planning of an action has modulatory effects in brain areas concerned with early processes in visual encoding.


*Theme:* Sensory systems
*Topic:* Visual cortex: extrastriate

*Keywords:* Extrastriate cortex; V3; V3A; V4; Visual identification; Visual impairment; fMRI; Action planning; Dual tasks

## 1. Introduction

It is well known that reaction times are prolonged when participants are confronted with two tasks in parallel [so-called Psychological Refractory Period (PRP)]. This impairment is generally assumed to emerge from capacity limitations to perform more than one response (selection and/or execution) simultaneously [20]. However, in recent studies an impairment has also been observed when the second task required only the visual identification of a stimulus [12,17,29]. Thus, even visual encoding seems to suffer from the need to share limited processing capacities with another task. In a variant of the PRP paradigm, Müsseler and Wühr [17] focused on the influence of action planning in a first task on visual encoding in a second task. Their participants had to accomplish a GO–NOGO motor task (S1–R1) followed by a visual identification task (S2–R2). In the visual task, masked left- or right-pointing arrows were presented with a variable stimulus onset asynchrony (SOA) after the presentation of the motor task. The results showed that visual identification was impaired in GO trials compared to NOGO trials, especially at short SOAs when both tasks overlapped in time. This can be taken as evidence that the selection and/or execution processes of R1, which was only required in GO trials, impaired the visual encoding of S2 [action-induced blindness (AIB)].

From a neuroanatomical perspective, the interesting question is how and where this interference between action and perception emerges. One possibility is that action planning and visual encoding interfere at higher brain areas crucial for both processes, resulting in a reduced visual performance. Alternatively, one can think of the possibility that the motor planning demands of the GO–NOGO task reduce the activity in visual areas and thus impair early visual processes. Thus, the observed impairment in visual identification might be due to a BOLD response modulation either in primary visual areas, in extrastriate visual areas or at higher integrating brain areas. The present fMRI experiments aimed to identify these neural processes.

We conducted two event-related fMRI experiments with an adapted version of the experimental procedure of

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Müsseler and Wühr [17]. GO trials, requiring a motor response and visual identification, were contrasted with NOGO trials, which require only visual identification. Thus, while the visual task remained constant over trials, we aimed to compare identification processes with and without an accompanying motor response. Visual areas being influenced by the motor task should show a modulated hemodynamic response. The two experiments differed only with respect to the visual stimulus material (shape and color). In the first experiment we used left- and right-pointing arrows, in the second experiment red and green rings were used as visual stimuli. This was to investigate whether the location of BOLD modulation is stimulus-dependent.

## 2. Method

### 2.1. Participants

Sixteen neurologically healthy volunteers participated in the Experiment 1 (mean age 25 years, 21–32 years of age, all right-handed, six females). In Experiment 2, 18 volunteers took part (mean age 26 years, 22–36 years of age, all right-handed, 11 males). All participants had normal or corrected-to-normal vision and normal hearing. Participants gave informed written consent prior to scanning. No participant had a history of neurological, major medical or psychiatric disorder; none were taking medication at the time of measurement.

### 2.2. Psychophysical procedures

Each trial combined two tasks: a speeded GO–NOGO task (S1–R1 task) followed by an unspeeded visual identification task (S2–R2 task) with an SOA of 200, 400, or 1000 ms (see Fig. 1). Participants responded to both tasks by pressing keys on a five-button box.

The experimental procedure was the same in both experiments. They differed only with respect to the visual identification task: In Experiment 1, participants identified the direction of arrows, whereas in Experiment 2, they identified the color of rings.

#### 2.2.1. The GO–NOGO motor task

The motor task (S1–R1 task) was a 2 × 2 choice reaction time task with two GO and two NOGO signals and, in case of GO, two different motor responses. Therefore, four types of tones, presented binaurally with headphones (Commander XG, Magnetic Resonance Technologies, Northridge, USA), were used as auditory stimuli (S1): two sine tones of 400 Hz (single and double low-pitch tones) and two sine tones of 2000 Hz (single and double high-pitch tones). Single tones had a duration of 70 ms and signalized a GO condition. Double tones consisted of two 10 ms tones with an interstimulus interval of 50 ms in between. They signa-

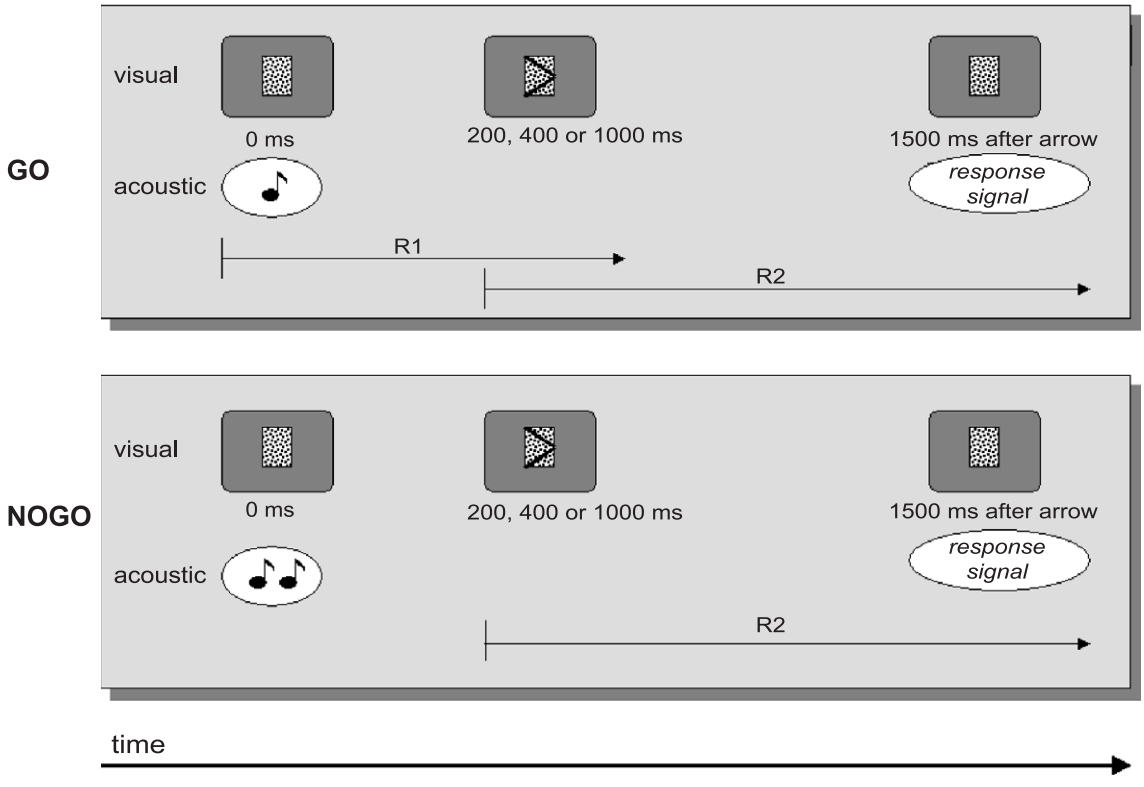


Fig. 1. Task sequence of the experiments. An acoustic signal indicated if a motor response had to be executed or not. Three different SOAs provided different amount of overlap between the motor and the visual task. An onset masking procedure was used in the visual task.

lized a NOGO condition. A single high-pitch tone required a key press with the middle finger of the right hand, a single low-pitch tone required a key press with both the index finger and the ring finger of the right hand (R1).

*2.2.2. The visual identification task in Experiment 1*

Visual stimuli (S2), consisting of black arrowheads presented against a background image (randomly arranged small black lines), were shown with a variable SOA after the presentation of the tones. To reduce BOLD responses in visual areas caused by the mask, we decided to use onset masking instead of backward masking. With onset masking the visual mask remains on the screen at least as long as the stimulus is presented [5]. In the present experiment, the background image served as an onset mask and remained on the screen for the whole experimental run. To strengthen the masking effect, additional small black lines with the same orientation and thickness like the arrowheads were presented simultaneously with the arrow, randomly arranged around it.

Presentation duration of the arrows depended on the participants' performance, with a minimum of 17 ms and a maximum of 306 ms. After each trial, the percentage of correct judgments was calculated. If more than 85% of judgments were correct, presentation duration of the arrow was reduced by one frame (17 ms); if less than 65% of judgments were correct, presentation duration was increased by one frame. Mean presentation duration across all participants was 177 ms (S.D.: 94 ms).

Participants responded with their left hand to the visual task after an acoustic white noise appeared as a response indicator for S2. Therefore, they waited at least 1500 ms before responding to S2 (unspeeded response). Participants indicated a left- or right-pointing arrow with two vertically arranged response buttons using thumb and index finger. Stimulus–response matching was balanced over participants.

*2.2.3. The visual identification task in Experiment 2*

In Experiment 2, red or green rings served as visual stimuli. For onset masking, we used a background image consisting of randomly arranged small red and green squares. Mean adjusted presentation duration across all participants was 39 ms (S.D.: 27 ms). Other parameters were the same as in Experiment 1.

Visual stimuli of both experiments were presented with VisuaStim (Magnetic Resonance Technologies), which consists of two small TFT-monitors placed directly in front of the eyes simulating a distance to a normal computer screen of about 100 cm.

*2.2.4. Design*

Experiment 1 consisted of 240 pseudorandomized trials and 60 nonevents. Half of the trials were GO trials, the other half were NOGO trials. Of each trial type one third had an SOA of 200 ms, one third of 400 ms and one third had an SOA of 1000 ms. Fifty percent of the presented arrowheads pointed to the left side, the others to the right side, equally balanced over GO and NOGO conditions and different SOAs. Trials were presented every 6 s on average. During nonevents the visual mask remained on the screen.

Experiment 2 consisted of 288 pseudo-randomized trials and 72 nonevents. Half of the trials were GO trials, the other half were NOGO trials. Because the results of Experiment 1 indicated that the trials with the shortest SOA of 200 ms are the most interesting with regard to the interference between both tasks we used more SOA-200 ms trials. Fifty percent of both trial types had an SOA of 200 ms, 25% had an SOA of 400 ms and 25% had an SOA of 1000 ms. Fifty percent of the presented arrowheads pointed to the left side, the others to the right side, equally balanced over GO and NOGO conditions and different SOAs. Trials were presented every 5 s on average. During nonevents, the visual mask changed to another image of randomly arranged red and green squares to prevent that participants adapt to the pattern of the mask.

In order to increase the temporal resolution, trials were presented with onset delays of 0, 400, 800, 1200, or 1500 ms in Experiment 1. This produced an oversampling of the actual image acquisition time of 2 s by a factor of 5 [16]. In Experiment 2, onset delays of 0, 330, or 660 ms were used. This produced an oversampling of the actual image acquisition time of 2 s by a factor of 6, because trials were presented every 5 s on average.

*2.3. MRI scanning procedure*

A 3-T whole body scanner (Bruker Medspec 30/100, Ettlingen) was used for data acquisition. In Experiment 1, 14 axial slices (19.2 cm FOV, 64 × 64 matrix, 5 mm thickness, 1 mm spacing), parallel to the AC-PC plane and covering the whole brain were acquired using a single shot, gradient recalled EPI sequence (TR 2 s, TE 30 ms, 90° flip angle). Totally, 900 time points were measured during two experimental runs, with each time point sampling over the 14 slices. Prior to the functional runs, 14 corresponding anatomical slices (data matrix 256 × 256, TR 1.3 s, TE 10 ms) and 14 T1-weighted EPI images were acquired.

In Experiment 2, we acquired 16 axial slices (4 mm thickness, 1 mm spacing) parallel to the AC-PC plane covering the whole temporal and occipital cortex up to the ventral border of the paracentral lobule. Nine hundred time points were measured during one experimental run. All other parameters remained as in Experiment 1.

*2.4. Data analysis*

FMRI data analyses of both experiments were performed with LIPSIA software [13]. Functional data were corrected for motion using a matching metric based on linear correlation, and for slice acquisition time using a cubic-spline interpolation [24]. A temporal highpass filter with a cutoff frequency of 1/144 Hz was used and a spatial Gaussian filter

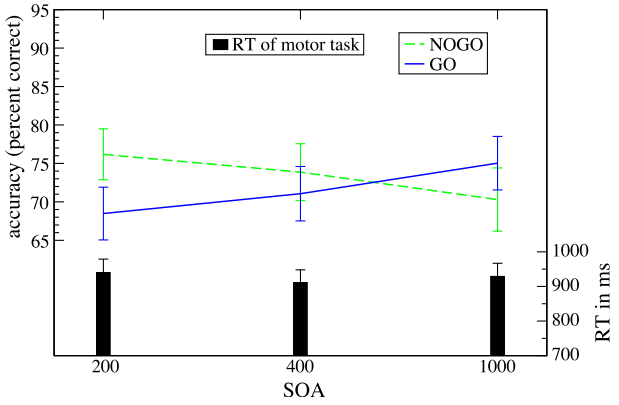


Fig. 2. Mean identification accuracies and standard errors (S.E.M. across participants) of the identification task for the three SOAs and the GO/NOGO conditions. Additionally, reaction times and standard errors (S.E.M.) of the motor task are plotted (Experiment 1).

with $\sigma = 0.6$ was applied. Functional data sets were coregistered with high-resolution 3D structural images standardized to the Talairach stereotactic space [22]. The transformation parameters were then applied to the functional slices using trilinear interpolation, so that the resulting functional slices were aligned with the stereotactic coordinate system. The linear normalization process was improved by a subsequent processing step that performs an additional nonlinear normalization [25].

We excluded those trials from the statistical analysis where participants made errors in the GO–NOGO task. In Experiment 1, this was the case in 13.7% of all trials. In Experiment 2, we excluded 3.3% of all trials due to R1 errors.

Statistical analysis was accomplished using the general linear model for serially autocorrelated observations [8,28]. The design matrix was generated utilizing a synthetic hemodynamic response and its first and second derivative [9]. The model equation, including the observation data, the design matrix and the error term, was convolved with a Gaussian kernel with a dispersion of 4 s FWHM. Contrast maps, i.e., estimates of the raw-score differences between specified conditions, were generated for each participant. The random effects analysis was effected as a one-sample $t$ test on these contrast images across participants [11].

The underlying signal time courses of the brain areas significantly activated in the NOGO–GO contrast were also analyzed. The percent signal change was calculated in relation to the mean signal intensity across all time steps. The signal change was averaged for each condition for the period of 3–8 s after trial onset collapsed over all participants.

## 3. Experiment 1

### 3.1. Behavioral results

The identification accuracy (percentage of correct responses) in the visual discrimination task was of main interest (Fig. 2). A repeated measurement ANOVA revealed a significant interaction of trial type and SOA, $F(2,14) = 12.7$, $p = 0.001$. Paired $t$ tests showed that with a SOA 200 ms identification performance was more impaired in GO trials than in NOGO trials, $t(15) = 4.3$, $p = 0.001$. Contrary, identification performance was somewhat better in GO trials than in NOGO trials with a SOA 1000 ms, $t(15) = -2.2$, $p = 0.041$.

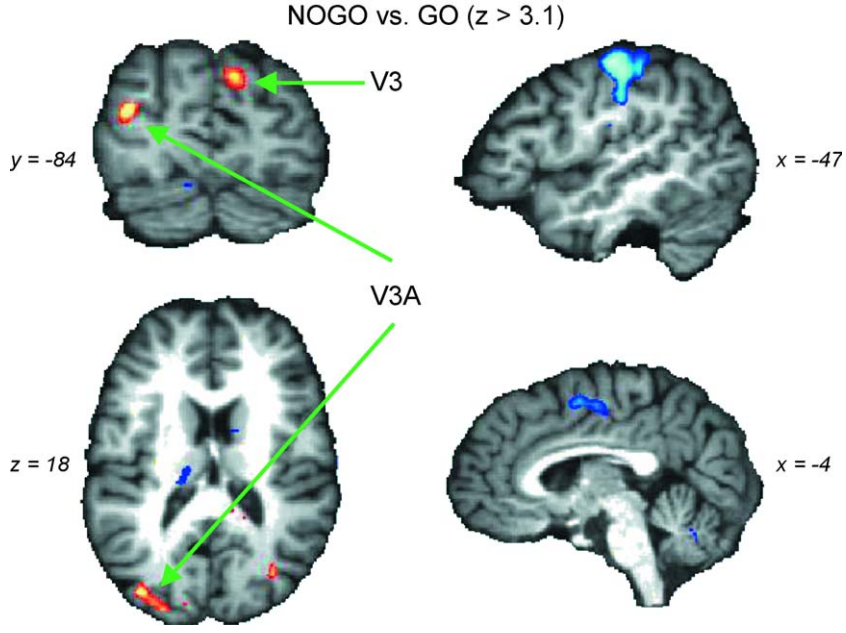


Fig. 3. Averaged contrast images of the NOGO vs. GO comparison with SOA 200 ms mapped on a standard brain. Red blobs indicate a stronger activation in NOGO trials than in GO trials. Blue blobs indicate stronger GO activations (Experiment 1).

This could indicate that visual processing is facilitated immediately after the release of the motor response. However, the effect is weak and a corresponding finding was not present in the data of Müsseler and Wühr [17]. Therefore, we have to treat this finding with care.

There was neither a significant main effect of trial type nor SOA ($p \geq 0.25$). Mean reaction times in the motor task of GO trials differed significantly across SOA, $F(2,14) = 8.4$, $p \leq 0.004$. Reaction times were 940 ms with SOA 200 ms, 911 ms with SOA 400 ms, and 929 ms with SOA 1000 ms.

## 4. fMRI results

In order to reveal the basic task-related network, we initially contrasted GO trials against nonevents and NOGO trials against nonevents collapsed over all three SOAs. Each trial type activated a distributed network across fronto-parietal and temporal regions. However, as the direct contrast between GO and NOGO trials demonstrated (see below) there were no significantly stronger NOGO activations than GO activations in frontal or parietal cortex areas. This is most likely the case, because in contrast to other GO–NOGO paradigms we used an equal percentage of GO and NOGO trials. Usually, in order to investigate response inhibition, a higher percentage of GO trials is used to establish a response bias.

The main contrast of interest is between the NOGO trials and the GO trials with the SOA of 200 ms (see Fig. 3 and Table 1). With this SOA, identification data indicated the most pronounced action-induced impairment. This contrast revealed stronger NOGO activations than GO activations in the transverse occipital sulcus (TOS) bilaterally and in the right cuneus. Despite some individual differences in the location of the visual areas V3 and V3A, Tootell et al. [26] report that in most subjects V3A is located around the crossing of the TOS and the intraoccipital sulcus (IOS). Thus, the observed TOS activation in this experiment coincided most likely with visual area V3A [26] and the right cuneus activation was located in the medial part of V3 [30]. Both V3 and V3A are contrast and orientation sensitive visual areas [26].

GO trial activations identified a motor circuit consisting of left primary motor cortex, SMA proper, thalamic and ipsilateral right cerebellar regions. The left SMA activation is located in the arm region of the SMA proper [21] which projects directly to the primary motor cortex and to the spinal cord. Additionally, somatosensory areas (left SI/SII, right postcentral sulcus) and the left frontal eye field were active in GO trials. An activation of the right sensorimotor cortex did not emerge here, because left hand responses (response to the visual task) were required in every trial, independent from GO and NOGO.

The NOGO vs. GO contrasts with longer SOAs (400 and 1000 ms) showed no GO trial associated reductions, but only GO trial activations in the same brain areas described in the contrast with SOA 200 ms. With SOA 1000 ms, GO activations were more widespread than with other SOAs.

ROI analyses of the voxel with peak significance in the NOGO activations were conducted. For the left and right TOS trial averaged time courses of the BOLD response reflected a reduced signal change in GO trials compared to NOGO trials with the 200 ms SOA (Fig. 4). Thus, the profile of the signal change in extrastriate visual brain areas reflects the behavioral interaction of GO/NOGO and SOA. The ROI analyses at the bottom of Fig. 4 (central sulcus and SMA) demonstrate, that this pattern of BOLD responses was specific for the TOS region. As expected, in motor areas only the GO trials

Table 1
Talairach coordinates, maximum $z$-value of the local maxima and volume (in $mm^3$) of the activated regions for the contrast NOGO vs. GO [$z$-values were thresholded at $z > 3.1$ and clusters had a minimum size of 90 $mm^3$ (two voxels) in Experiment 1 and 108 $mm^3$ (three voxels) in Experiment 2]

| NOGO vs. GO | | Volume | $z$-Max | Talairach coordinates | | |
|---|---|---|---|---|---|---|
| | | | | $x$ | y | z |
| *Experiment 1* | | | | | | |
| NOGO | L. Transverse Occipital Sulcus[a] | 649 | 4.13 | −37 | −85 | 18 |
| | R. Transverse Occipital Sulcus[a] | 121 | 3.65 | 31 | −75 | 18 |
| | R. Cuneus | 237 | 3.82 | 7 | −85 | 32 |
| GO | L. Central S., Postcentral G., and FEF | 9964 | −4.94 | −49 | −22 | 53 |
| | L. MI | | −4.51 | −37 | −22 | 52 |
| | L. FEF | | −4.59 | −25 | −7 | 47 |
| | R. Postcentral Sulcus | 110 | −3.54 | 37 | −34 | 47 |
| | L. SMA proper | 1000 | −4.06 | −4 | −1 | 50 |
| | L. Thalamus | 475 | −4.08 | −13 | −19 | 12 |
| | R. Cerebellum | 4875 | −5.57 | 19 | −55 | −15 |
| *Experiment 2* | | | | | | |
| NOGO | L. Transverse Occipital Sulcus[a] | 926 | 4.64 | −31 | −88 | 21 |
| | R. Transverse Occipital Sulcus[a] | 1082 | 4.58 | 31 | −82 | 15 |
| | L. Lateral Occipitotemporal Sulcus[b] | 148 | 3.54 | −43 | −64 | 0 |
| | R. Lateral Occipitotemporal Sulcus[b] | 258 | 3.81 | 37 | −58 | −6 |
| GO | R. Posterior Rostral Cingulate Zone | 567 | −3.82 | 4 | 20 | 32 |
| | L. SMA proper | 2830 | −3.41 | −5 | −16 | 47 |
| | L. Precentral Gyrus | 335 | −4.00 | −56 | 2 | 32 |
| | L. Anterior Superior Insula | 432 | −3.89 | −22 | 26 | 18 |
| | L. Postcentral Sulcus | 16746 | −5.86 | −50 | −22 | 38 |
| | L. Superior Precuneus | 288 | −3.62 | −1 | −67 | 47 |
| | R. Inferior Parietal Lobule | 173 | −3.55 | 58 | −28 | 27 |
| | L. Thalamus | 3303 | −5.27 | −14 | −25 | 12 |
| | R. Cerebellum | 10152 | −6.15 | 22 | −52 | −12 |
| | L. Cerebellum | 412 | −3.75 | −20 | −67 | −15 |
| | | 396 | −4.21 | −38 | −49 | −21 |
| | Pedunculi | 343 | −4.17 | 1 | −22 | −9 |

[a] These coordinates fall within the region denoted V3A (see Refs. [10,26]).
[b] These coordinates fall within the region denoted V4 complex (see Ref. [1]).

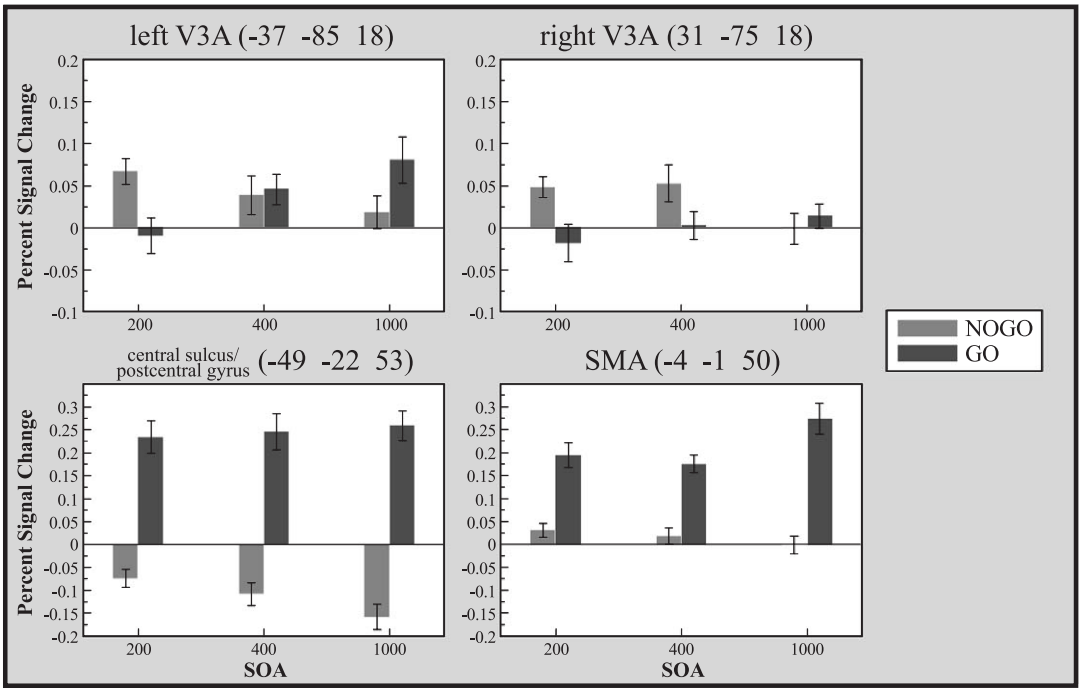


Fig. 4. Averaged percent signal change of BOLD responses (3–8 s after stimulus onset) in V3A, SI and SMA in voxels with highest z-scores in the GO/NOGO contrast and standard errors (S.E.M.). The left and right transverse occipital sulci were more activated in NOGO trials than in GO trials at the SOA 200 ms. The other areas (central sulcus/postcentral gyrus, SMA proper) showed more percent signal change in GO trials independent of SOA (Experiment 1).

produced a high amplitude of signal change, independent of SOA.

Taken together, the results of Experiment 1 show that the GO response in the motor task led to a reduced BOLD signal in extrastriate visual areas compared to NOGO trials, in which only the visual task had to be accomplished.

## 5. Experiment 2

This experiment aimed to clarify why in the previous experiment the GO–NOGO difference just occurred in V3/V3A and not in other visual areas. Different hypotheses can be made with regard to this issue. The action-induced BOLD response modulation could be a general modulation at the level of extrastriate visual brain areas—but can only be observed in those areas which are crucial for encoding the respective stimulus. This hypothesis is based on the observation that V3/V3A are contrast and orientation sensitive visual areas and therefore should be mainly involved in encoding the masked arrowheads. Alternatively, the activated area itself (V3/V3A) is especially sensitive to action-induced interferences, independent of its role in visual encoding.

To further examine whether the location of BOLD modulation is stimulus-dependent or area-dependent, visual stimuli were now masked red and green rings instead of black arrowheads. The task was to identify the color of the rings. If the brain location of the AIB effect is stimulus dependent, a GO–NOGO difference should occur in color sensitive brain areas.

### 5.1. Behavioral results

A repeated measurement ANOVA revealed a significant SOA effect in the identification task with $F(2,16) = 5.5$, $p = 0.015$. At SOA 200 ms identification performance was more impaired than at the SOAs 400 and 1000 ms, paired $t$ test, $p \leq 0.014$ (Fig. 5). The interaction between GO/NOGO and SOA was marginally significant, $F(2,16) = 3.3$, $p = 0.061$. There was no significant main effect of GO/NOGO ($F \leq 1$)[1]. However, a paired $t$ test revealed that with SOA 200 ms identification performance was slightly more impaired in GO trials than in NOGO trials, $t(17) = 1.7$, $p = 0.099$.

Mean reaction times were 757 ms (with SOA 200 ms), 746 ms (SOA 400 ms), and 729 ms (SOA 1000 ms). Reaction times in the motor task of GO trials did not differ significantly across SOAs, $p \geq 0.15$.

In contrast to Experiment 1, we found a behavioral AIB effect at SOA 200 ms that was only marginally significant. The AIB effect most likely failed to reach conventional significance levels, because the color identification task

[1] Because of the nonsignificant GO/NOGO effect in this fMRI experiment, we tested another 20 participants behaviorally with this task. Here we found a significant main effect of GO/NOGO with $F(1,19) = 5.2$, $p = 0.035$ and an SOA effect, $F(2,18) = 4.8$, $p = 0.021$.

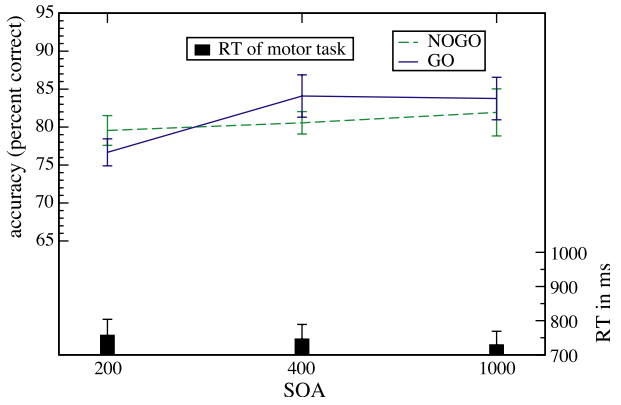


Fig. 5. Mean identification accuracies and standard errors (S.E.M.) of the identification task for the three SOAs and the GO/NOGO condition. Additionally, reaction times and standard errors of the motor task are plotted (Experiment 2).

was easier than the arrow identification task in Experiment 1. That is, at SOA 200 ms presentation times were shorter (39 vs. 177 ms in Experiment 1), RTs were faster (757 vs. 940 ms in Experiment 1), and error rates were lower (76.67% and 79.55% in GO and NOGO trials, respectively, vs. 68.48% and 76.18% in Experiment 1). Importantly, due to the refresh rate of the VisuaStim, stimulus presentation times could only be increased or decreased by 17 ms, what unfortunately prevented a fine-grained adaptation to the performance of the subjects in the color identification task.

## 6. fMRI results

In Experiment 2, the NOGO vs. GO contrast with the 200 ms SOA revealed stronger NOGO activations in the lateral occipitotemporal sulcus (LOTS) bilaterally (V4 complex; for an overview, see Ref. [1]) and in the transverse occipital sulcus (TOS) bilaterally (V3/V3A) [26] (Fig. 6).

With respect to GO trial activations one must take into account that the acquired slices covered the cortex only up to the ventral border of the paracentral lobule (because of reduced slice thickness). Thus, the activation in the primary motor cortex was not collected in Experiment 2. Brain areas that were more activated in GO trials than in NOGO trials were detected in the left SMA proper, right posterior rostral cingulate zone, left precentral gyrus, left postcentral sulcus, right inferior parietal lobule, left superior precuneus, left anterior superior insula, left thalamus, cerebellum and pedunculi.

As in Experiment 1, the NOGO vs. GO contrasts with longer SOAs (400 and 1000 ms) did not show GO trial reductions, but GO trial activations in the same brain areas described in the contrast with SOA 200 ms.

BOLD time course analyses of areas with reduced GO activations (V3/V3A and V4 voxels with strongest GO/

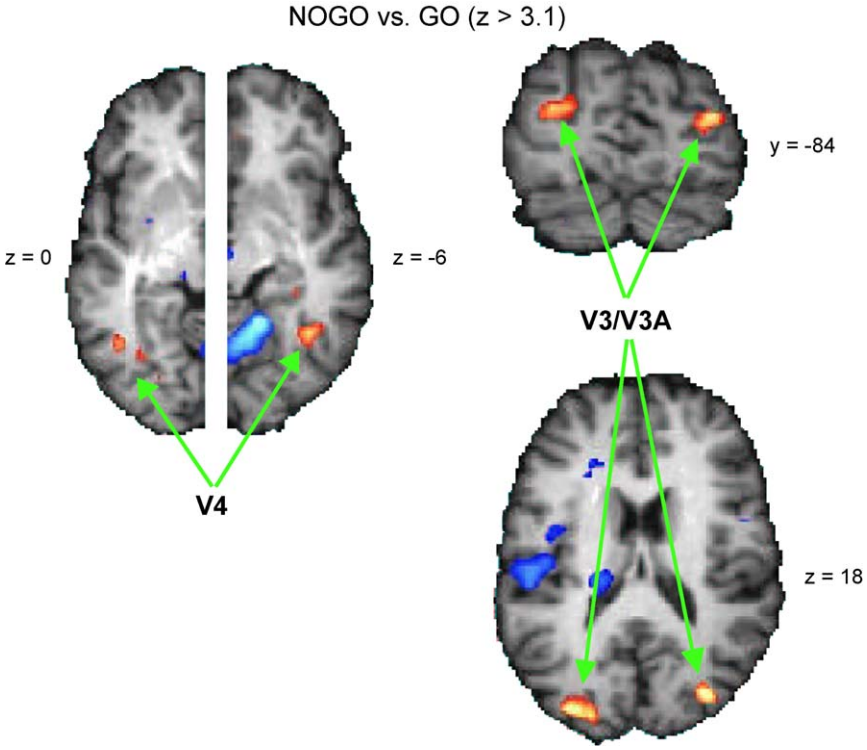


Fig. 6. Averaged contrast images of the NOGO vs. GO comparison with SOA 200 ms mapped on a standard brain. Red blobs indicate a stronger activation in NOGO trials than in GO trials. Blue blobs indicate stronger GO activations (Experiment 2).

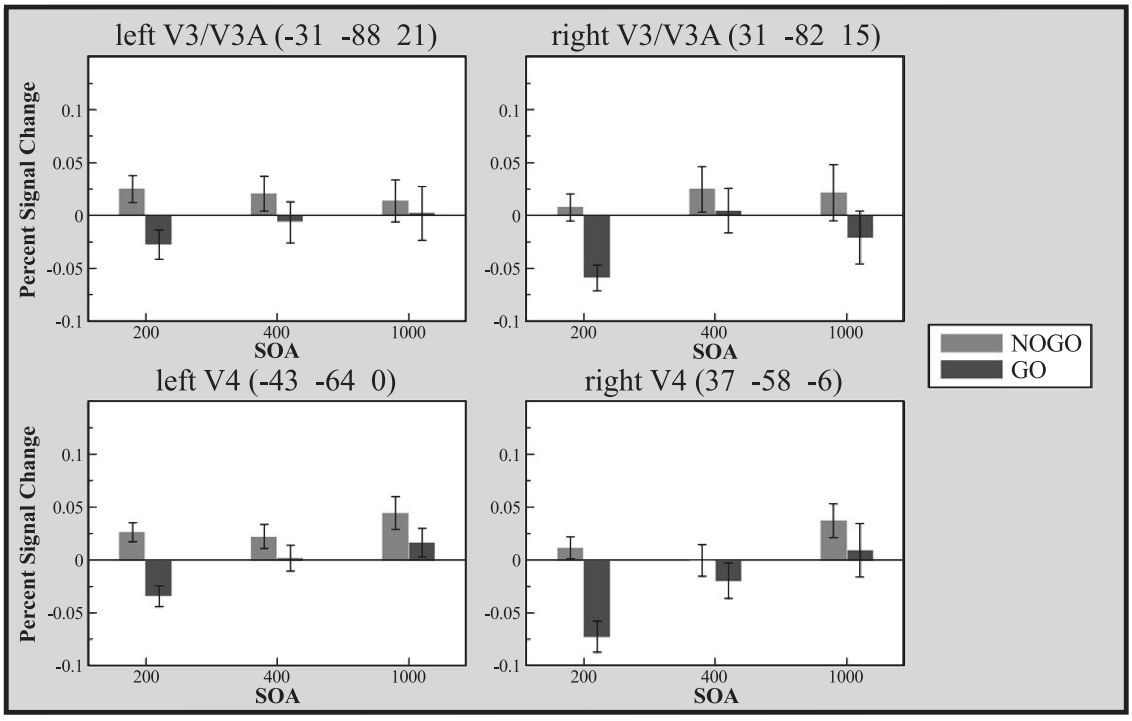


Fig. 7. Averaged percent signal change of BOLD responses (3–8 s after stimulus onset) in the V4 complex and V3/V3A in voxels with highest *z*-scores in NOGO activations and standard errors (S.E.M.). The BOLD response modulation is most pronounced with shortest SOAs (Experiment 2).

NOGO discrepancy) revealed a comparable activation pattern as in Experiment 1. With the shortest SOA the amplitude of signal change was reduced in GO trials compared to NOGO trials (Fig. 7). This result was found both in area V3/V3A and V4.

That this effect was present both in V3/V3A and V4, and not only in V4 is probably due to the fact that by introducing rings as visual stimuli in the second experiment, these stimuli were defined by both color and shape. Even if shape was not a relevant dimension, processing shape might have facilitated the identification of the visual stimulus against the colored background.

However, by using color as the important stimulus dimension a BOLD modulation occurs in V4. Thus, we can conclude that the action-induced BOLD modulation is not restricted to V3/V3A, but is a general phenomenon at the extrastriate level. The BOLD modulation is observable in those parts of the extrastriate cortex that are crucial for encoding the visual stimulus.

## 7. Discussion

The behavioral and imaging data of the present fMRI experiments showed that identification performance of a visual stimulus is more impaired when a motor response is planned concurrently than when no motor response is required (action-induced blindness). This interference between motor planning and visual identification is reflected at the level of extrastriate visual areas.

As the identification task was the same in GO and NOGO trials, the observed BOLD modulation in V3/V3A and V4 most likely originates from the action planning process in GO trials. Because the BOLD modulation was most pronounced with the shortest SOA of 200 ms and less distinct with the other SOAs, we can conclude that the modulation was most likely caused by the planning of an action and not by its execution. Mean reaction times with the shortest SOA were 940 and 757 ms in Experiment 1 and 2, respectively. Therefore, the arrow/color stimulus was presented about 740 or 557 ms before the execution of R1.

We found a GO–NOGO effect for the color task (Experiment 2) only in a behavioral experiment outside the scanner, and only a marginally behavioral effect within the fMRI experiment. Nevertheless, the action-induced blindness effect was reflected in the fMRI data. The BOLD modulation in V4 and V3/V3A of the second experiment most likely reflects an action-induced effect, because in the GO vs. NOGO contrast the only difference between trial types is the process of action preparation and execution. Since the purpose of Experiment 2 was to investigate whether the action-induced BOLD modulation is stimulus-or area-dependent, we had concrete anatomical hypotheses. Thus, although the behavioral results reached only marginally significance, the BOLD modulation in V4 rejects the hypothesis that the modulation is exclusively area-dependent (see Ref. [27] for a discussion about the interpretation of fMRI data without significant behavioral data).

As shown in Figs. 3 and 6, the planning of a GO response impairs mainly the orientation, direction, and contrast sensitive area V3A in Experiment 1 (e.g., Refs. [26,18]) and additionally the color sensitive area V4 in Experiment 2 (e.g., Ref. [1]). The GO–NOGO difference in V4 occurred only with color as important stimulus dimension in Experiment 2. When using left- or right-pointing arrowheads, only V3/V3A was modulated. As noted in the results section, the V3/V3A activation in the color identification task is most likely due to (additional) shape perception (colors had the shape of rings), even if this visual feature was not relevant for performing the task.

Therefore, we tentatively conclude that the observed modulation is stimulus-dependent. In future experiments, this hypothesis could be tested rigorously by presenting colored stimuli that are not projected onto a background but fill the complete projection area in order to prevent a special shape of stimuli. However, one has to think about the masking of these kind of stimuli.

Other studies also found modulatory influences on extrastriate visual areas. Macaluso et al. [14] found an increased response in the left lingual gyrus with concurrent tactile stimulation on the right side. Calvert et al. [2] reported a stronger activation in V5 during concurrent auditory speech perception. Thus, activity in extrastriate visual areas is not only influenced in a bottom-up manner, but also by activity of other brain areas which, primarily, are not involved in visual encoding. Our results show that the process of action planning is also able to modulate the extrastriate cortex.

It is known that V3A is modulated by cognitive processes (attention, memory, anticipation and saccadic eye movements) [18]. It receives major inputs from V2 and V3 and is interconnected to fronto-parietal cortices. In monkeys both the sensorimotor cortex, as well as the SMA, and V3A are interconnected with areas 7b and 7ip in the posterior parietal cortex [3,4,19]. Fries et al. [7] reported an influence of selective visual attention on the oscillatory neuronal synchronization in V4 in macaque monkeys. Thus, neuronal activity in both V3A and V4 seem to be subject to a cognitive modulation.

Furthermore, V3, V3A and V4 all receive input fibers from the lateral intraparietal area (LIP) [6]. Assuming that these fibers also exist in humans, the posterior parietal areas could provide a connection between motor and visual areas. Macaluso et al. [14] assumed that back-projections, running through the parietal lobe, account for BOLD response modulations in extrastriate visual areas. Such back-projections from sensorimotor areas to extrastriate visual areas could provide a connection between these functionally different areas and could give rise to a top-down modulation of visual processing.

The frontal eye fields (FEF)—significantly stronger activated in GO trials—also provide direct input to both V3/V3A and V4. On the one hand the FEF are sensitive to visual input [23], and on the other hand FEF have access to motor plans [15]. In this fMRI experiment the FEF show activity in both GO and NOGO trials. However, in GO trials the activation was significant stronger than in NOGO trials. Thus, the greater percent signal change in GO trials could result from the additional R1 motor preparation. Therefore, the visual encoding could interfere with motor preparation in the FEF, resulting in a reduced activation in V3/V3A and V4.

## 8. Conclusions

In both experiments, a modulation of the BOLD response was observed at the level of extrastriate visual brain areas. The activation in these areas proved to be decreased when a functionally independent action was planned concurrently. This modulation seemed to be stimulus-dependent; that is, when the task was to identify the color of a stimulus, modulations affected also color-sensitive areas.

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