Active vision modulates early cortical stages of visual

processing: Evidence from MEG and eye-tracking

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Abstract

It has been hypothesized that the visual system anticipates upcoming visual input contingent on the execution of saccadic eye movements. In line with this idea, it has been shown that changes in visual input across saccades elicit stronger post-saccadic fixation-locked neural responses compared to no-change conditions; an effect known as the *preview effect*. In the present study, we demonstrate that this preview effect depends on active vision and cannot be explained by either classical or spatiotopic adaptation. In a gaze-contingent experiment, in which participants were cued to make saccades to object stimuli, we concurrently recorded magnetoencephalography (MEG) and eye-tracking data. The source-localized MEG signal was deconvolved with stimulus onset and eye movement events in order to account for systematic variation in gaze behavior related to the no-change (valid preview) and change (invalid preview) conditions. Crucially, preview effects in the primary visual and in ventral-occipital cortices were markedly larger when participants made saccades compared to replay blocks in which we simulated the visual consequences of saccades absent saccade execution, demonstrating that the preview effect cannot be simply explained by classic adaptation. A further control condition ruled out spatiotopic adaptation. Our results show that early visual cortical areas which are not known to exhibit saccadic remapping neurons still show signs of sequential neural history effects across saccades. Previous research might have overlooked this influence of active vision because of the lack of an invalid preview condition which breaks the correspondence between pre-saccadic extrafoveal and post-saccadic foveal stimulation.

36 **Introduction**

Humans make about three to four saccadic eye movements per second in daily life and it

has been hypothesized that, across each of these saccades, the visual system predicts

upcoming visual input (1–17). This anticipatory process is thought to be based on input from

extrafoveal regions of the visual field, the motor command for an eye movement, and

learning about the change in visual appearance from pre-saccadic extrafoveal to post-

saccadic foveal input (10,17–25). Consistent with this idea, we and others have previously

shown that changing a stimulus during the saccade that is directed to that stimulus, aka an

invalid preview condition, leads to discrimination performance decrements and a larger

post-saccadic neural response compared to a no-change, *valid* preview, condition (26–28).

This *preview effect* is reminiscent of the preview effect in reading research (29–37) and

could be interpreted in terms of neural surprise within a predictive processing framework

(7,11,16).

At present, it is, however, unclear how the neural preview effect comes about.

Previous research suggests that neurons in V1 and higher up along the ventral visual stream

respond largely in the same way to visual input that is brought about by a saccade

compared to conditions in which the eyes remain fixed and saccade-like control conditions

are achieved through various types of simulated saccade and fixation onsets (38–41).

Studies that find differences between simulated and actual saccade conditions (e.g. 42,43)

did not always control perfectly for intra-saccadic stimulation (1,44). Consequently,

saccade-independent repetition suppression (45,46), visual mismatch responses (47–51),

and neural adaptation (52–54) within the visual cortex could well explain the more

pronounced post-saccadic response that was observed in invalid compared to valid

preview conditions, meaning that the preview effect would be independent from active

vision.

In particular two types of adaptation could explain the neural preview effect: first,

adaptation of neurons with very large receptive fields, here called classic adaptation (e.g.

52), and second, craniotopic or temporarily-spatiotopic adaptation (6,55) (see also 56).

Although strictly retinotopic adaptation can be ruled out a priori because of the different

retinal locations of the pre-saccadic peripheral and the post-saccadic foveal stimulus

locations, neurons in particular along the ventral visual stream show receptive fields that are

large enough to span both pre- and post-saccadic locations in many preview designs. For

instance, neurons in the inferotemporal cortex (IT) of macaque monkeys show large

variation in receptive field sizes from a few degrees up to 30-40° (57–60). In humans,

evidence from population receptive field mapping using fMRI suggests receptive field sizes

of 2-12° (61; in particular Figure 6) or 4-8° (62) in lateral occipital and ventral visual areas.

Thus, adaptation of neurons with large receptive fields could explain the reduced response

for valid compared to invalid previews.

For spatiotopic adaptation, the adaptor and test stimuli have to appear in the same

spatiotopic coordinates before and after a saccade and exactly that is the case for pre- and

post-saccadic stimuli in preview designs and in ecologically valid contexts. Evidence for

spatiotopic adaptation comes primarily from perceptual studies using tilt aftereffects (55)

and the neural basis for this effect has been located in ventral visual areas (63) which are

close to or even overlap with the above-mentioned visual areas exhibiting very large

receptive fields. However, compared to retinotopic adaptation effects, spatiotopic

adaptation shows a distinct feature: it needs time, meaning that spatiotopic adaptation

increases with increased time in the form of a blank screen between adaptor and test stimuli

(55,63,64).

To examine whether the sequential history effects elicited by a trans-saccadic

preview could be explained by classic adaptation or spatiotopic adaptation, we conducted

a gaze-contingent experiment in which participants made cued saccades to objects in three

different blocked viewing conditions while we coregistered magnetoencephalography (MEG)

and eye-tracking data. In the *saccade* viewing blocks, participants actively made saccades

to an extrafoveally presented object. In the *replay* viewing blocks, participants kept their

gaze fixed at the center of the screen and instead of moving their eyes, the object moved to

the center of the screen. This condition mimicked the visual input obtained in the saccade

blocks as well as possible without actual saccade execution. If the preview effect was due

to adaptation of neurons with very large receptive fields, it should be the same in these

replay blocks compared to the saccade blocks, because the same visually responsive

neurons would be triggered in both viewing conditions. In order to make the visual input

temporally predictable, as it was in the saccade blocks because of the oculomotor

efference copy, object motion was always played-back after a fixed delay. The third type of

viewing blocks was the same as the saccade blocks, except that we presented a blank

screen between the preview and the target onset and called them *blank* blocks. The preview

effect should be larger in these blank blocks compared to the saccade blocks, if it resulted

from spatiotopic adaptation, because spatiotopic adaptation increases with a blank interval

between adaptor and test stimuli (55). Given that perception and visual action in the form of

eye movements are tightly intertwined (21,44,65), it would not be surprising if the preview

effect could not completely be explained by adaptation but depended on saccade

execution.

Our experimental design contains active eye movements or simulated replay

equivalents and gaze-contingent trans-saccadic changes which means that one trial

contains more than one stimulus onset with potential follow-up saccades and fixations.

Each event triggers an MEG-response and to separate these temporally overlapping

responses we used temporal deconvolution (66,67,see also 68,69). This method provides

the additional advantage of being able to explicitly model any systematic variation in gaze

behavior across conditions which we observed in our experiment.

To preview our preview results, we found a more pronounced difference between

valid and invalid preview conditions in the saccade blocks than in the replay blocks and no

preview effects in the blank blocks in the primary visual (V1) and the ventral occipital (VO)

cortex. These results suggest that early visual cortices are more sensitive to pre-saccadic

extrafoveal information when participants make saccades, compared to when the same

visual input is provided without making a saccade.

Results

Preview effects in eye movements and behavior necessitate temporal

deconvolution of MEG data

To determine whether the neural preview effect could be explained by classic adaptation,

spatiotopic adaptation, or active vision, we measured the source-localized MEG signal time-

locked to target foveation in three different types of viewing blocks (Figure 1). However, the

preview conditions and viewing blocks did not only affect the MEG signal but also the

participants' eye movements. In the saccade and blank blocks, where participants made a

cued saccade to a pre-saccadic object (valid preview) or phase-scrambled and blurred

version of the same object (invalid preview) (Figure 1A, 1C, 1D), the saccade amplitudes

were minimally (by 0.22°), but very consistently, greater for valid (8.13°) than for invalid

previews (7.91°, $F(1,35) = 39.71$, $p < .001$, Figure 1F). We, therefore, included each trial's

saccade amplitude as covariate in the MEG temporal deconvolution model to explicitly

capture the effect of saccade amplitudes on the first post-saccadic EEG/MEG component,

aka the lambda response (66,67,70–72), because the lambda response extends into the

time window of 100-300 ms where preview effects are usually found.

Interestingly, the preview conditions did not only affect saccade amplitudes but also

the follow-up gaze behavior after the initial target fixation and, crucially, this effect varied

across viewing blocks. With an invalid preview, participants were more likely to make a

follow-up saccade than with a valid preview (Figure 1G), but only in the saccade block ($t(35)$

$= 3.11$, $p < .010$). In the replay block, this effect was reversed (interaction $F(1,35) = 11.04$, p

$= .002$). In the blank block, the effect was comparable to the saccade condition (interaction

$F(1,35) = 0.01$, $p = .912$) considering first the onset of the blank screen. The subsequent

target onset did not appear to further modulate follow-up saccades (interaction with

saccade condition, F(1,35) = 6.44, p = .016, invalid 875 ms, valid 873 ms, t(35) = 0.14, p =

.890). If a follow-up saccade was made, the time until this saccade happened, that is the

duration of the first fixation on the target (Figure 1H), showed the opposite pattern in the

saccade (t(35) = 9.56, p < .001) compared to the replay block (t(35) = 4.21, p < .001,

interaction F(1,35) = 107.99, p < .001). In the blank blocks, there are two first fixation onsets

because of the intermediate blank screen, one on the blank screen and one on the target.

For the blank screen onset, the timing of follow-up saccades was similar as in the saccade

blocks but less extreme (interaction F(1,35) = 62.50, p < .001). After the onset of the target,

which followed the 400-ms blank screen, there was no evidence anymore for an effect of

the preview (t(35) = 0.79, p = .436, interaction with saccade blocks F(1,35) = 54.77, p <

.001). Because of these patterns in eye movements, we included follow-up saccades as

additional events in our deconvolution model. This allowed us to separate the MEG

response to the initial fixation form the response elicited by follow-up saccades (66,67).

Besides making a saccade (Figure 1B and 1D) the extrafoveally presented objects or

watching the replay (Figure 1C), participants had to report in each trial whether the object

was tilted left or right (Figure 1). The manual responses in this tilt discrimination task on the

target object also showed a behavioral preview effect (Figure 1E). Response times were

faster with a valid (471 ms) compared to an invalid (573 ms) preview (F(1,35) = 116.64, p <

.001) and there was no evidence for a modulation by the viewing blocks (both interaction p

> .823). Some participants, however, showed on average very early response times, even

within the time period of a neural preview effect around 200-250 ms (cf. Figure 1E). In order

to avoid that these occasionally very early responses could confound our conclusions from

the MEG signal, we added manual response as separate events to our temporal

deconvolution model. Error rates did not provide any evidence for preview effects or

differences between viewing blocks (all p > .076).

Figure 1. Trial procedure and behavioral results. (A) Stable fixation for 500 ms triggered the presentation of one
out of 80 objects, here a garden shovel. In the valid condition, the preview object was the same as the target
object; in the invalid condition, the preview was a blurred version of the object. In the saccade blocks (B), the
participant made a cued saccade to the preview object which appeared as intact, non-blurred object in both the
valid and invalid preview conditions. In the replay blocks (C), the participants maintained stable fixation
throughout the trial and the preview object moved into the foveal visual field after a saccadic latency determined
per participant depending on their practice trial performance. The blank blocks (D) were the same as the saccade
blocks except that a blank screen was presented for 400 ms before the target onset. In all three viewing

conditions, participants reported whether the final target object was tilted left or right. (E) Manual response times
showed a significant preview effect in all three viewing conditions. (F) Saccades were significantly larger with
valid than with invalid preview for both saccade and blank viewing. (G) In the saccade blocks, the proportion of
trials with a follow-up eye movement after the initial target fixation was larger with an invalid compared to with a
valid preview. (H) In the saccade blocks, the first fixation on the target (or blank) was longer with a valid than with
an invalid preview. This effect was the same for fixations on the blank screen, was reversed for the replay blocks,
and was gone after the final target onset in the blank blocks. The black horizontal line indicates target stimulus
offsets (800 ms) in all blocks and the end of the blank screen (400 ms) for fixation durations following the blank
onset.

The preview effect in the early visual cortex depends on active vision

To test whether the neural preview effect could be explained by adaptation of neurons in

with large receptive fields, presumably in mid-to-higher level visual processing, we

compared the neural preview effect in an active saccade condition to the neural preview

effect in a passive replay condition (Figure 1). We reasoned that having the same visual

input in active and passive viewing conditions should trigger the same visually-responsive

neurons and therefore the preview effect should be the same in both active and passive

viewing conditions only if it resulted from adaptation of visually-responsive neurons.

Figure 2. MEG source regions of interest (ROI) based on (73) illustrated on the fsaverage freesurfer brain. We
included all visual areas V1, V2, and V3 plus V4 merged into area V34 in order to exhibit a similar number of
vertices compared to V1 and V2. In addition, we defined two mid-higher level visual areas: a lateral occipital area
(LO) encompassing regions relevant for object processing (74) and a ventral occipital area (VO) with areas
associated with spatiotopic adaptation (63). ROIs for the left and right hemispheres were kept separate to
capture any lateralized visual activity due to the lateralized preview stimulus. For further details see section
Materials and Methods, MEG and eye-tracking data processing, Regions of interest.

We source-localized the MEG signal to five visual cortical surface regions of interest

per hemisphere: V1, V2, V34, ventral occipital (VO), and lateral occipital (LO) (Figure 2).

Using temporal deconvolution we isolated the MEG response time-locked to foveating the

target object at the participant-level (67). For the right-hemisphere ROIs, cluster-based

permutation tests at the group level showed a clear preview effect in V1 and in VO after

foveating the target object in the saccade blocks (Figure 3A and 3B). In the replay blocks,

there was some evidence for a preview effect only in V1 (Figure 3C and 3D). Crucially, the

cluster-based permutation tests of the planned interaction contrast preview (valid, invalid) x

viewing block (saccade, replay) indicated that the preview effect was larger, i.e. more

negative, in the saccade than in the replay condition in particular in right V1 and right VO

around the time period during which preview effects have been observed in the past using

EEG, that is around 200 ms after fixation onset (Figure 3G and 3H) (26–28,31). This

interaction demonstrates that the preview effect cannot be explained by classic neural

adaptation alone. Moreover, within the saccade blocks, the preview-effect cluster appeared

numerically before the preview-effect cluster within the replay blocks, which further

supports the idea that active vision modulates neural processing in the striate and

extrastriate visual cortex.

Note, that in the saccade viewing blocks, the invalid preview showed a more

negative source-localized MEG signal than the valid preview condition. This result mimics

the direction of the preview effect in the EEG (28,31) suggesting more neural spiking activity

in the invalid compared to the valid preview condition.

Figure 3. Grand average source-localized and temporally deconvolved MEG signal in the primary visual (V1, left panels) and ventral occipital cortices (VO, right panels) of the right hemisphere. Panels A and B show fixation-locked responses in the valid and invalid preview conditions for the active saccade blocks, panels C and D the target-locked responses for the passive replay, and E and F the target-locked responses for the blocks with the blank screen between preview and target stimuli. Panels G and H compare the preview effect contrast invalid minus valid across the three types of viewing blocks. Horizontal bars indicate significant cluster effects: For both V1 and VO, there was a preview effect in the saccade condition (A and B) which was significantly different from the preview effect in the replay condition (C and D; interaction effect clusters in G and H). The contrast comparing the saccade preview effect to the preview effect in blocks with a blank screen between preview and target stimuli did not show any significant clusters (no corresponding clusters in G and H). The histograms along the x-axis count the number of follow-up saccades per 10 ms time bins, averaged across participants.

Potentially overlapping activity from before the saccade in the replay

blocks further supports an active-vision interpretation

Comparing the preview effect in the saccade blocks to the preview effect in the

replay blocks showed three significant preview (valid, invalid) x viewing block (saccade,

replay) interaction clusters in right V1 which can probably not all be explained by the same

process (Figure 3G). Whereas the middle (~200 ms) and later clusters (~300 ms) can be

explained by differential responses in V1 time-locked to target foveation, the early cluster

seems to be too early to be related only to the target because it takes some tens of

milliseconds for neural activity from the retina to arrive at V1 (75,76). This early effect rather

stems from visual input before the saccade, which can be explained as follows: In the

saccade blocks, the time interval between preview and target depended gaze-contingently

on saccade execution which made the target onset temporally predictable. The replay

blocks had a fixed time interval between preview and target in order to make the target

onset temporally predictable as well despite the lack of saccades. This fixed preview-to-

target interval meant, however, that the neural response to the preview stimulus could not

be deconvolved from the neural response to the target stimulus, because deconvolution

relies on variation in timing between events (see Methods). As a consequence, the target-

locked signal in the replay blocks could in theory still contain overlapping activity from the

pre-saccadic preview stimulus (Figure 1A and 1C). Importantly, however, any overlapping

activity triggered by the preview stimulus is expected to *exacerbate* target-locked preview

effects, but our results show that, even with a potentially exacerbated preview effect in the

replay blocks, there is still a difference to the saccade blocks, which further supports our

conclusion that classic adaptation cannot explain the preview effect in the early visual

cortex.

Spatiotopic adaptation cannot explain the preview effect in early visual

cortex

If the preview effect resulted from spatiotopic adaptation, it would become larger as the

time between preview (adaptor) and target (test) stimuli increases (63). To investigate that,

we added a 400 ms blank screen before the target onset in the blank viewing blocks (Figure

1D). In all other respects the blank viewing blocks were the same as the saccade blocks.

We statistically tested whether the blank screen increased the preview effect through the

planned preview (valid, invalid) x viewing block (saccade, blank) interaction contrast. This

interaction effect did not show any significant clusters in any of the ROIs (all cluster p > .05).

As can be seen from Figure 3, the preview effects in the blank blocks in right V1 (Figure 3G)

and right VO (Figure 3H) were numerically even smaller than the corresponding significant

preview effects in the saccade blocks. We, therefore, conclude that the preview effect can

also not be explained by spatiotopic adaptation.

Left V1 shows a complementary signal to right V1

Overall, the evidence for the preview effect was more concentrated in the right hemisphere,

which is not completely unexpected. Before saccade onset, the left-lateralized preview

stimulus projects to the right hemisphere and the left hemisphere receives input from a

uniform screen background. This uniform background is the same for valid and invalid

preview conditions, thus, for the left hemisphere there is actually no difference between

valid and invalid previews; such a difference exists per design only for the right hemisphere.

Although the preview effect is evaluated after the saccade when the object is foveated and

processed bilaterally, the pre-saccadic contrast between left and right hemispheres still

counts, because the preview effect consists per definition in a match between pre- and

post-saccadic visual input. From all ROIs in the left hemisphere, only left V1 showed one

significant preview (valid, invalid) x viewing (saccade, replay) interaction cluster very early

after fixation/target onset (Figure 4D). This interaction contrast showed the opposite polarity

compared to the same very early effect in right V1 (Figure 3G), which suggests that left V1

shows a complementary signal of the same neural source. The preview effects within the

saccade blocks and within the replay blocks were not significant in left V1 (Figure 4A and

4B). Regarding the blank blocks, the pattern of results in the left hemisphere was the same

as in the right hemisphere. Left V1 did not show any evidence for a modulation of the

preview effect by the blank screen compared to the saccade blocks (Figure 4D).

Figure 4. Grand average source-localized and temporally deconvolved MEG signal in the left primary visual cortex (V1). Panel A shows fixation-locked responses in the valid and invalid preview conditions for the active saccade blocks, panel B the target-locked response for the passive replay, and C the target-locked response for the blocks with the blank screen between preview and target stimuli. Panel D compares the preview effect (contrast invalid minus valid) across the three types of viewing blocks. Horizontal bars indicate significant cluster effects: There was one early significant cluster indicating that the preview effect was different between saccade and replay blocks. In contrast, the preview effect in the saccade compared to the blank blocks was not significantly different.

The blank-screen response supports inferences about neural spiking activity from MEG source activations

As can be seen from Figure 3E, in the blank blocks, the target onset was preceded by a very clear and large positive signal across both valid and invalid preview conditions in right V1. This response was obviously triggered by the onset of the blank screen which appeared 400 ms before the target. In general, we baseline-corrected the MEG signal with respect to

the final part of the preview/cue stimulus period in all blocks (Figure 1A, see Methods).

Compared to this baseline period, the onset of the blank screen marks an abrupt decrease

in visual input which probably leads to a decrease in neuronal spiking activity in early visual

cortices. Conversely, a negative deflection in V1 can probably be interpreted as an increase

in neuronal firing which matches our interpretation regarding the preview effect in V1 where

the more *negative* response in the invalid compared to the valid preview condition is

regarded as an *increase* in neuronal spiking activity. Interestingly, the blank screen response

was absent in VO (Figure 3F), which suits the general notion that slightly higher visual areas

are less affected by sudden low-level visual changes.

Discussion

In the present study, we investigated whether the sequential history effects elicited by a

trans-saccadic preview could be explained by classic adaptation of neurons with large

receptive fields (52–54) or by craniotopic or transiently-spatiotopic adaptation (55,63) (see

also 56) in the visual cortex. For this purpose, participants performed a gaze-contingent

task in which they made saccades to extrafoveally presented objects and we compared this

active viewing blocks to passive replay blocks where the extrafoveal object moved to the

foveal field of view while the participants maintained stable gaze. In the active saccade

viewing blocks, we found a robust preview effect in the early visual cortex and this preview

effect was significantly smaller in the replay blocks, which demonstrates that the reduced

activity in the early visual cortex for a valid preview compared to an invalid preview cannot

be explained by adaptation alone; in particular not by adaptation of neurons with receptive

fields that are large enough to cover the pre-saccadic extrafoveal and post-saccadic foveal

stimulus locations.

To rule out that the preview effect was the result of spatiotopic adaptation, we

contrasted the active viewing blocks to a blank control condition in which a blank screen

was inserted between the pre-saccadic preview and the post-saccadic target stimulus.

Such a blank screen increases spatiotopic adaptation effects (55,63), but the preview effect

in the blank blocks was numerically smaller than the preview effect in the saccade blocks. In

sum, our results demonstrate that the preview effect cannot be driven by spatiotopic

adaptation and instead depends on the oculomotor and saccade-specific processes

implied by active vision.

Previous research has suggested largely similar responses of neurons in V1 and

further downstream in ventral visual cortices for stimuli that appear in the visual field

because of a saccade compared to saccade-like simulated stimulus onsets (38–41,77).

These findings are contrasted by two exceptions (42,43). For these two studies, however,

differences between saccade and saccade-like control conditions can in theory be

explained by differences in intra-saccadic visual stimulation because of the employed

experimental design (1,44,78–80). Intra-saccadic visual stimulation affects the post-

saccadic neurophysiological response (81) and can therefore be confounded with an effect

from active saccade execution. Apart from this limitation, previous studies on neural

responses in visual cortices did not pay much attention to the availability of pre-saccadic

preview information which is inherent to active vision (39,82,83). For instance, (39)

compared sudden stimulus onsets during fixation to onsets after saccades without

corresponding previews. One of these study accounted for the complete dynamics of active

vision through a passive replay condition, however, they did not focus on sequential trans-

saccadic effect but examined particular types of neural coding principles which did not

appear to differ much between active and passive vision conditions (84). Previous research

has, thus, not yet been able to provide compelling evidence for the idea that the visual

cortex is affected by active vision.

In contrast to the abovementioned studies that found largely similar responses for

active saccade and proper passive control conditions, we harnessed a preview effect

design with a passive replay control condition and with this setup we could clearly see that

active vision modulated the influence of pre-saccadic information on post-saccadic

processing. In particular, the invalid preview condition might have been crucial to reveal this

type of trans-saccadic neural history effect because it is the only way that completely

breaks the correspondence between pre-saccadic extrafoveal and post-saccadic foveal

stimulation. The lack of an invalid preview, or trans-saccadic change, might actually have

been the reason why previous studies failed to determine viewing-history effects even in

very comprehensive single neuron recordings (e.g. 85). Moreover, our preview effect design

has the advantage that any imperfect simulation of visual input in the passive replay

condition which could lead to uncontrolled intra-saccadic stimulation (44) cancels out in the

valid-invalid contrast.

The same logic of a preview effect design with active and passive viewing conditions

has recently been applied by Zhang and colleagues (86) to direct recordings from the

macaque superior colliculus with remarkably similar results to ours. In that study, macaques

made saccades to gratings that could change their spatial frequency (invalid preview) or

remain the same (valid preview) during the saccade. In invalid preview conditions, single

neuron and population activity increased after fixation onset but only if the saccade had

been actively made and not if the monkey had maintained fixation and the grating had

moved from the extrafoveal to the foveal location. Importantly, this effect was recorded from

receptive fields that did not overlap the extrafoveal but only the foveal target location which

means that these neurons responded to stimuli that they had not been exposed to (86). This

remarkably similar result shows that saccade-contingent history effects are not limited to

the visual cortex.

Interestingly, using a preview effect design we found trans-saccadic history effects in

the primary and ventral visual cortices which are not among the typical brain areas

associated with modulations of visual activity by eye-movements. Traditionally, parietal

areas, like the lateral intraparietal area (LIP) in monkeys (87), the frontal eye fields (FEF) (88),

the superior colliculus (SC), and the mediodorsal thalamus (MD) (89) have been shown to

exhibit neurons that change their receptive fields to match the location of upcoming stimuli

in the face of an impending saccade (24,for reviews see 90–98). This phenomenon of

predictive remapping has also been reported for earlier visual areas like V2, V3A, and V4

(99–101), is considered to be the basis for perceptual aftereffects across saccades (102–

106), and underlies the impression of visual stability despite saccades (20). The idea here is

that a copy of the motor signal for a saccade, the efference copy, is sent from the SC via

MD to the FEF and further to visual areas in a way that allows to anticipate the visual

consequences of the upcoming saccade, i.e. the corollary discharge (107,108), making

these visual consequences translucent to subjective experience. However, although being

somehow involved in this process, the primary visual cortex and ventral visual areas do not

seem to exhibit neurons that show predictive remapping (85,100). Why did we then find a

modulation of preview effects by active vision in particular in V1 and ventral occipital

cortices?

To us it seems plausible that V1 and ventral occipital areas receive signals about an

impending saccade from typical remapping-related areas further downstream, e.g. as close

as V2 (99) or further away like LIP or FEF, or from subcortical structures like the SC or MD

which leads to a transfer of neural activity that was before the saccade elicited by the

extrafoveal stimulus to neurons with foveal receptive fields that cover the post-saccadic

target. In other words, although we ruled out classical adaptation and spatiotopic

adaptation as possible explanations for the preview effect, it is not impossible that

adaptation *in combination with remapping* leads to the reduced post-saccadic activity in

valid compared to invalid preview trials that we observed under active vision conditions.

Crucially, this type of remapped retinotopic adaptation cannot result from neural fatigue

(109), has to act at the timescale of the preview effect around 200 ms after fixation onset,

and relies on a surprisingly fast neural activation transfer at the timescale of saccades within

tens of milliseconds. Eventually, V1 might contain retinotopic internal model neurons that

represent the current environment and which are updated by means of saccade execution

and the efference copy to match the newly incoming bottom-up input after saccade offset

(108,110). Interestingly, this type of remapping implies a transfer of information from

extrafoveal to foveal cortices which could in theory take place in any experimental context

where eye movements are not completely controlled and lead to the presence of extrafoveal

visual information in foveal cortical regions (e.g. 111) (see 112 for a perceptual correlate that

matchs this proposed neural effect).

418   Future research could disentangle several ways of how this type of remapping-

related adaptation could come about. Neural activity could be transferred across retinotopic

neural populations through relays to subcortical structures, through areas further

downstream (e.g. 113), or horizontally within the visual hierarchy directly across retinotopic

cortices. All three possibilities of how retinotopic neural activity could be transferred across

saccades make different predictions in terms of bottom-up and top-down information

transfer which implies contrasting predictions regarding laminar activation profiles. Bottom-

up connections to pyramidal neurons arrive primarily at supragranular layers (2 and 3)

whereas top-down connections arrive at infragranular layers (5 and 6) (114–116). The

laminar activation profile in V1 is therefore expected to vary depending on how neural

activity is remapped across saccades.

429   Apart from alterations in bottom-up and top-down processing, the crucial

computational principle by which active vision modulates the preview effect could also be of

a temporal nature. Temporal expectations are known to enhance effects of expectations

(e.g. 117) and one might interpret the preview effect as an effect of expectations with a valid

preview entailing an expected target in contrast to an invalid preview entailing an

unexpected target. Although we made the target temporally predictable also in the passive

replay blocks by including a fixed delay between cue and replay onset, the target might still

have been more temporally predictable in the saccade blocks simply because saccade

execution directly yields the target onset.

438   Our finding that active vision affects early cortical stages of visual processing aligns

well with a long history of research showing that perception and visual action in the form of

eye movements are tightly intertwined (21,44,65). These ideas date back to academics like

(118) and (119) and their conceptual precursors might even be found in the works of

medieval and ancient western scholars (cf. 120). Besides these historic ideas, contemporary

empirical evidence provides many examples for influences of eye movements on perceptual

processing (e.g. 79,80,121) and for the flexible adjustment of these processes according to

sensorimotor contingencies (122,123). At the neural level, it is clear that the brain accounts

for non-retinal signals of oculomotor origin during eye movements which has recently been

elegantly demonstrated in a compelling study in mice showing that the primary visual cortex

combines visual and non-visual but eye-movement specific signals of subcortical origin in a

way that allows to distinguish sensory input due to self-generated actions, i.e. the reafferent

response, from visual motion in the external environment (124) (see also 125–127). In non-

human primates, there is evidence that the excitability of neurons in V1 is timed with the

perpetual cycle of saccades and fixations showing larger excitability following fixation

onsets (128,129) which has been associated with rhythmic and oscillatory neural activity

more generally in the context of active sampling (130,131). In addition to these pieces of

evidence, we show that active vision leads to trans-saccadic history effects, in terms of

neural preview effects, in visual cortices which are not typically associated with saccadic

remapping.

Conclusion

The present study highlights the tight link between neural activity in early visual areas

and active vision in the form of saccadic eye movements. We demonstrated that the

influence of pre-saccadic visual input from extrafoveal regions on post-saccadic processing

cannot be explained by classic adaptation or spatiotopic adaptation and instead crucially

hinges on saccade execution. Our results suggest that retinotopic neural activity in early

visual cortices, in particular in V1 and in ventral-occipital areas, is being transferred from

extrafoveal to foveal cortical areas through the execution of saccadic eye movements in

combination with an efference copy signal that arrives at V1 and VO either from subcortical

or higher-level visual and oculomotor areas. This trans-saccadic updating process suggests

that vision unfolds in a profoundly different way, in particular with respect to bottom-up and

top-down signaling between early visual cortices and higher-level or subcortical regions,

under naturalistic viewing conditions where the visual world is explored with active eye

movements in contrast to classic stable-gaze experiments. As a consequence, the growing

field of naturalistic neuroscience (132–136) will probably have to focus as much on neural

activity as on eye movements and gaze behavior (e.g. 137–139).

Materials and Methods

Participants

Coregistered MEG and eye-tracking data was collected from 39 participants. Written informed consent was obtained from all subjects prior to participating in the study which had been approved by the local ethics committee (CMO region Arnhem/Nijmegen). The data of three participants had to be excluded; for one participant because of problems with the head localization coils, for another because of a magnetic artifact, and for one participant because only a fourth of the whole dataset could have been included for the deconvolution analysis based on the trial exclusion criteria described below.

Stimuli

Eighty objects were selected from the stimulus set of (140), available at https://bradylab.ucsd.edu/stimuli/ObjectsAll.zip, considering that the objects should have horizontal or vertical elements to be useful for the main behavioral task, which was a tilt discrimination task. Additional 10 objects were selected for practice trials. The image background was rendered transparent and, if necessary, the original object orientation was adjusted with the image editor GIMP (https://www.gimp.org) to create an upright view of the object. Images were converted to greyscale with the Python module PIL (https://pypi.org/project/pillow/). Subsequently, mean image luminance across all non-background pixels was reduced to $1/8^{th}$ of the maximum possible luminance, i.e. pixel value 32 out of 255, and pixel luminance standard deviation was set to 16, with the help of the SHINE toolbox (141).

For each object, we created a phase-scrambled version which served as invalid preview. Note, that we did not maintain the background-foreground distinction for the scramble object, because this would have made the object easier to recognize, but created a two-dimensional gaussian window in the alpha channel that blended the phase-scrambled object with the background and made the object's outline harder to recognize. Finally, the

luminance of the scrambled images was adjusted to the same value as the luminance of the

intact images. All image processing code is available with the experiment code (see section

Data and code availability).

The objects had a size of 4° visual angle and were placed with their center at 8°

eccentricity to the left of the screen center. Stimulus size and location in visual angels were

keep constant across participants by automatically adjusting actual size for each participant

individually based on the actually measured screen-eye distance.

A digital light processing (DLP) projector (PROPixx; VPixx Technologies, Saint-

Bruno, QC, Canada) rendered the visual stimuli at a refresh rate of 120 Hz on a translucent

screen placed 80 cm before the subject. To precisely monitor stimulus onsets, a photodiode

was placed in the bottom-right corner of the screen. At that location, a white square was

presented with the onset of each stimulus to create a light flash which was recorded via the

photodiode into a MISC channel of the MEG system. This procedure revealed the well-

known delay of one frame between MEG stimulus onset triggers and the actual change on

screen and the time stamps of all stimulus onset triggers events were offline adjusted by the

amount of that delay.

Procedure

The temporal sequence of events within a trial was similar to previous gaze-contingent

experiments with the preview effect using face images (28,142). In the present study, the

experiment consisted of three blocked viewing conditions: a *saccade*, a *replay*, and a *blank*

condition (Figure 1). The Participants were informed about which viewing block was coming

up at the start of each block. In each of these viewing blocks, a trial started with the

presentation of a small fixation dot of 0.1° visual angle presented at screen center and an

equally sized placeholder dot located at 8° eccentricity to the left. Stable gaze at screen

center for 500 ms triggered the onset of the preview object which had a maximum diameter

of 4° and could be either an intact object (*valid* preview) or its phase-scrambled and

Gaussian-blurred version (*invalid* preview). The preview object was tilted to the left or right

by 2°, which was decided randomly in each trial. Stable gaze was defined in terms of the

fixation-update events provided online by the Eyelink eye-tracker. A fixation-update event

contains the average gaze position across 50 ms of individual gaze samples. If this position

was within 2° visual angle from screen center, it counted as stable gaze. If the participants

moved their eyes out of this area, the stable-fixation timer restarted. After 500 ms of stable

fixation with the preview object on screen, the saccade cue appeared which consisted in

the fixation dot turning slightly darker (Figure 1A).

In the saccade blocks (Figure 1B), participants were instructed to monitor the

centrally presented dot and as soon as they noticed the saccade cue they had to look at the

extrafoveally presented object. The moment at which the eyes started to move was

detected online through a heuristic by calculating the difference in screen pixels between

two subsequent gaze samples. If this difference was greater than 8 pixels, it counted as a

saccade. This threshold value was determined in pilot testing for this specific lab setting.

The detected saccade onset triggered a transient image at the target location which was the

scrambled and blurred version of a randomly chosen different object. If the eyes had moved

too early, i.e. before the saccade cue, the current trial was appended at the end of the

program's trial queue and the next trial started. The transient image was presented for one

frame only and its purpose was to equalize the amount of visual change between valid and

invalid conditions. The transient image was followed by the target object which was always

an intact object.

The blank blocks were the same as the saccade blocks, expect that instead of the

target object a blank screen containing only the placeholder and fixation dots was

presented for 400 ms (Figure 1D). Thus, in this condition, the participants saw a blank

screen after the saccade. They were instructed to maintain their gaze at the peripheral

placeholder location until the target was presented.

The replay blocks were different from the saccade blocks in terms of gaze behavior

(Figure 1C). In that condition, participants were instructed to maintain fixation at the screen

center throughout the experiment which was controlled by the eye-tracker with the same

stable-gaze and saccade detection criteria mentioned above for the saccade blocks. If

fixation was too far from screen center or if a saccade was detected, the trial was appended

at the end of the trial queue and the next trial started. To match visual input as well as

possible to the saccade blocks, the extrafoveal preview object started to move towards the

screen center after a fixed time period which was determined for each participant from the

saccade latencies that were recorded during the initial practice trials (see below). We call

this the *replay* or *replay sequence*. We took the median saccade latency across saccade

and blank blocks and subtracted 50 ms to account for the minor speed-up in saccadic

latencies that took place throughout the experiment. This adjustment was based on pilot

data and previous studies (28,143). The preview object moved in equally spaced steps, one

per frame, from the extrafoveal preview location to the screen center. The number of frames

for this replay sequence was determined based on how many complete frame durations

could fit into a participant's median saccade duration calculated from the practice trial data.

The median saccade duration was not adjusted because it does not change much

throughout an experiment. The first couple of frames of the replay sequence contained the

preview object and only the last frame of the replay sequence contained the transient image

to account for the delay between saccade detection and transient presentation in the

saccade and blank blocks which also contained only one frame of the transient image. After

the replay sequence, the target object was presented at screen center where the

participants were still foveating. As in the other viewing blocks, the target object was always

intact, that is, not phase scrambled.

Introducing a proper replay sequence in the replay blocks was important in order to

minimize visual stimulation differences compared to the saccade blocks because, although

we are usually not aware of intra-saccadic visual input, the visual system clearly processes

visual input from within a saccadic eye movement (80). However, it is in practice impossible

to perfectly mimic the visual input arising from a saccadic eye movement. This would

require rotating the whole world around a resting eye. To deal with any remaining

differences between saccade and replay blocks, our experiment was designed to cancel out

any such remaining differences through the preview effect contrast.

In all three viewing blocks, as soon as the participants had foveate the target object,

they had to indicate whether it was tilted left (counter-clockwise) or right (clockwise). The tilt

of the post-saccadic target object was 2° and always the same for target and pre-saccadic

preview object (illustrated in Figure 1B). In all three viewing blocks, the target object

disappeared 800 ms after its onset. Manual responses were given with MEG-compatible

button-boxes connected to a DataPixx device (VPixx Technologies, Saint-Bruno, QC,

Canada).

Each of the 80 objects appeared once in each condition for each participant. With

two preview (valid, invalid) and three types of viewing blocks (saccade, replay, blank), this

design resulted in 480 trials which were administered in 15 blocks of 32 trials each. Preview

condition trials occurred randomly intermixed within each block. The viewing condition was

blocked and the order of blocks was pseudorandom in the sense that before a viewing

condition could be repeated both of the other two viewing conditions had to have occurred

at least equally often. This constraint was set to balance sequence and practice effects that

can result from repeated presentation of the same type of contextual condition in trans-

saccadic perception studies (123,cf. 143). Between viewing blocks, participants could take

self-paced breaks. If necessary, the experimenter could also halt the experiment during the

task, had the participant moved or had the eye-tracker to be recalibrated.

At the start of an experimental session, the participant received a step-by-step

explanation of the sequence of event within a trial and they were walked through one trial

per viewing block as example. Afterwards they conducted practice trials in each viewing

block with the replay condition being the last one in order to obtain the gaze parameters for

the replay sequence from the preceding saccade and blank blocks. During these practice

trials, participants received feedback about the correctness of their manual responses in the

object tilt discrimination task. If the response was incorrect, the placeholder dot turned red

for 300 ms. This feedback was omitted during the experiment proper.

The experiment concluded with one minute of a separate gaze task which had the

purpose to obtain MEG data free from task-relevant visual input. This data was added to the

training data for the ICA which was conducted to attenuate artifactual eye-movement

components primarily related to eyeball motion (see section MEG and eye-tracking data

processing and deconvolution analysis, Preprocessing). During this gaze task participants

looked back and forth between the fixation dot at screen center and the placeholder dot to

the left of fixation. Their gaze was cued by the placeholder and fixation dots alternatingly

turning slightly darker in the same way as the saccade cue in the proper experiment.

MEG and eye-tracking data recording and synchronization

MEG data was collected with a 275-channel CTF system with axial gradiometers in an

electromagnetically shielded room at a sampling rate of 1200 Hz. Throughout the recording,

the participants' head position was monitored online with the help of three positioning coils

which had been placed at cardinal landmarks at the nasion, left ear, and right ear according

to the standard operating procedure in the lab. If the participant had moved too far away

from their starting position, they were verbally guided back into their original position in the

break between experimental blocks (144). In addition to MEG data, horizontal and vertical

EOG was collected for the first couple of participants in order to rule out gaze data

synchronization issues between MEG and eye-tracking computers. After the experimental

session, a set of 250 to 300 head shape points was recorded with a motion tracking system.

These head shape points served the offline coregistration of the MEG and the MRI

coordinate system.

An Eyelink 1000 plus eye-tracker (SR research, Ontario, Canada) recorded eye gaze

concurrently to the MEG at a sampling rate of 1000 Hz in the default eye-tracker data file

format (SR research's EDF). In addition, the gaze data from the eye-tracker was streamed

online via an analog output into dedicated MISC channels of the MEG system. Eye gaze

events (fixation start/end, saccade start/end, blink start/end) were provided by the SR

research gaze parsing algorithm with a saccade velocity threshold of 35 deg/s and a

saccade acceleration threshold of 9500 deg/s^2.

To synchronize MEG and eye-tracking data, trigger events were sent from Psychopy

via the python module serial (https://pyserial.readthedocs.io/en/latest/pyserial.html) and a

splitter cable connection to the MEG computer and the eye-tracker at the same time. These

events were used to offline merge the MEG data with the eye-movement events that had

been parsed by the SR research algorithm. The eye-movement events were then used for

temporal deconvolution to obtain eye-fixation-locked and stimulus-locked MEG responses.

Anatomical MRI processing

Anatomical T1-weighted MRI data for each participant was either already available at

the institute or had been obtained after the MEG experiment with the exception of two

participants. For these two participants, it we uses freesurfer's *fsaverage* template brain,

skull, and skin meshes warped to the individual participants's head shape points. Apart

from using the anatomical templates all other processing steps were the same as for the

other participants. The T1 images were automatically segmented and cortical as well as

skull and skin surfaces were reconstructed with freesurfer (version 7.4.1, command) and

fastsurfer (https://github.com/Deep-MI/FastSurfer).

According to the mne python standard processing pipeline, the outer skin surface

model was used to coregister the anatomical MRI data with the MEG sensor locations by

matching the locations of the three fiducials (nasion, left, right ear) and the head shape

points to the outer skin surface with an Iterative Closest Point (ICP) fitting algorithm.

MEG and eye-tracking data processing and deconvolution analysis

Preprocessing

Both the MEG and eye-tracking data was processed with mne python (version 1.6)

(145). The raw MEG signal had been visually inspected for channels with aberrant signals

during data collection and any such bad channels were removed and interpolated with

spherical spline interpolation during offline data analysis. A third-order gradient

compensation was applied to the MEG data before it was filtered with a 0.1 Hz high-pass

and a 40 Hz low-pass Hann-windowed finite impulse response filter using mne python's

default filter option arguments.

In order to remove heartbeat as well as corneoretinal and myogenic eye and eyeblink

artefacts in the MEG signal we ran an independent component analysis (ICA) in a separate

pipeline which featured an additional 1 Hz high-pass filter to provide the required largely

stationary signal for the ICA. An ICA infomax solution was obtained from all successfully

completed trials' data, that is from all trials which had not been aborted because of

problems with the trial procedure, and from the separate eye movement task at the end of

the experiment. Saccade-related components were automatically labelled by applying the

method introduced by (146). In short, if the variance ratio of a component's activation during

saccades compared to fixations was larger than 1.1, the component was flagged as

saccade related. For most of the participants, visual inspection confirmed the selected

components and additionally identified heartbeat components. Three to six components

were rejected per participant with the exception of eight components for one participant.

The obtained ICA solution weights were then applied to the MEG data in the original

pipeline.

In order to integrate the MEG and eye-tracking data in a temporally precise way, the

eye-movement events from the Eyelink eye-tracking data file (EDF) were added to the MEG

data events array considering the shared experiment triggers which were present in both

eye-tracking and MEG data streams (cf. 30). Following this procedure, we obtained a

maximum coregistration error of 2 ms for all common trigger events. After the coregistration

with eye-tracking data events, the MEG data was downsampled to 400 Hz.

We ensured that only data from trials where the participants had properly followed

the gaze procedure and where the stimulus onsets had happened in time was selected for

further processing. In addition, the response time in the speeded object tilt discrimination

task had to be within three median absolute deviations per participant and response option

(correct, incorrect). In the saccade and blank conditions, the onset of the participant's

fixation on the target/blank had to be within 50 ms from target/blank onset and not earlier.

In the replay condition, any occasional saccades during the constant fixation period had to

be smaller than 3°. There was no eye blink from the stable fixation at trial start up until

500 ms after target onset, including the blank period in the blank blocks.

Source space transformation

The continuous MEG data was transformed into individual participants source

spaces with minimum norm estimation (MNE). We used surface source spaces based on

the freesurfer white matter segmentation with dipole locations obtained from a recursively

subdivided icosahedron downsampled to 5124 locations and a three-layer boundary

element model based on the shells between brain, inner skull, outer skull, and outer skin

surfaces with default conductivity values of 0.3, 0.006, and 0.3, respectively. Dipole

orientations of the source model were restricted to the direction perpendicular to the

cortical surface. The noise covariance matrix for the inverse operator was estimated from

the different baseline periods that were relevant within a trial. We obtained a noise estimate

from the baseline periods with respect to two events per trial that were later relevant for

deconvolution: One main event of interest for all three viewing conditions was the onset of

the preview object. For the saccade condition, the second event was the onset of the

fixation on the target object. For the replay and blank conditions, the second event was the

onset of the target object. The baseline period duration was set to the interval of -275 to -

75 ms in order to omit the saccadic spike that was present in the saccade and blank

conditions. Correspondingly, from this point on we ran two separate pipelines, one with the

noise covariance matrix based on the preview onset event and another one where the noise

covariance matrix was based on the fixation/target onset. For each event of interest, we

finally report the source space results based on the source space projection with the noise

covariance from the baseline period of the respective event. We also ensured that all noise

covariance matrices were based on the same number of trials per condition in order to

avoid that any condition difference in the source localization could have resulted from

imbalances in the number of trials.

Regions of interest (ROI)

After source space projection, the signal was summarized across the vertices of

each anatomical region of interest (ROI). Individual participant's regions of interest had been

obtained according to the multimodal parcellation of the Human Connectome Project (HCP)

(73). The HCP parcellation, which is available for the freesurfer subject _fsaverage_, was

transformed to each participant's individual anatomical space using the freesufer

mri_label2label function. For each individual ROI, the summary source space signal across

vertices was computed by finding the dominant orientation across vertices and flipping the

sign of vertex activations to match the dominant orientation (see mne python's _mean_flip_

label summary function).

Based on our hypothesis that the preview effect might be explained by different

types of adaptation in visual areas, we selected a set of ROIs for statistical analyses from

the Glasser atlas (Figure 2). ROIs with considerably smaller numbers of vertices were

merged in order to obtain roughly comparable regions. The selected ROIs were: The visual

areas _V1_ and _V2_. Area V3 merged with V4 to obtain a region called _V34_. The smaller lateral-

occipital areas LO1, LO2, LO3 and V4t – V4t is also known as LO2 – merged to form _LO,_

because lateral-occipital areas are implicated in object processing and sensory prediction

effects (e.g. 74). The ventral occipital areas V8, VMV3, and the ventral visual complex (VVC)

merged to form one region called *VO*, because ventral-occipital cortex is a strong candidate

for neurons with large receptive fields (58) and has been identified as a key region for

spatiotopic adaptation (63). For further details about the location and differentiation of these

areas the reader is referred to the Supplementary Neuroanatomical Results of (73).

Temporal deconvolution

The source activities summarized per region of interest were eventually deconvolved

with experiment and eye-movement events using the Unfold.jl toolbox (67) implemented in

the Julia programming language (https://julialang.org, version 1.10.0) and access from

within python through the juliacall package (version 0.9.15). Temporal deconvolution

requires continuous data at the level of single trials. Sections of the continuous data from in

between trials or from trials that were not eligible (see preprocessing above) were set to

missing data to exclude them from deconvolution.

We used the maximum number of events for deconvolution given our experimental

design, considering that only events which vary in their relative timing across trials can be

deconvolved. In line with this constraint, we defined as first event of main interest the onset

of the preview image in the saccade and blank blocks. For the saccade block, the second

event of main interest was the onset of the first fixation on the target. We did not model the

preceding saccade onset separately because the time interval from saccade onset to

fixation onset was almost constant. In the blank blocks, the next event of interest was the

onset of the blank screen. However, the blank screen and the subsequent target onset had

a fixed timing which meant that only one of the events could have been selected for the

deconvolution. We selected the target onset because our main hypothesis was about the

response to the target stimulus. However, the event basis functions were defined for a time

range that was long enough to encompass the onset of the preceding blank screen to

additionally obtain insights into potential neural omission responses triggered by that screen

onset. Similarly, in the replay blocks the onset of the first event of interest, i.e. the preview

object, and the onset of the second event of interest, i.e. the onset of the target, occurred at

a fixed timing. This fixed timing served the purpose to render the target temporally

predictable because a temporally predictable target was expected to enhance any potential

preview effects in this passive replay condition which would make our comparison to the

active saccade condition more conservative. For the deconvolution model, this important

experimental feature meant that we could only include either the preview onset or the target

onset. We opted again for the target onset because the response to the target was of main

interest and the deconvolution model was extended in time far enough to encompass the

preview onset in order to also compare the neural responses to the preview onsets between

all conditions.

Besides the preview onset and the fixation/target onset, we defined four other types

of events in the deconvolution model in order to correct for the overlap of their

corresponding neural signals: Follow-up saccades after target onsets, additional saccades

during the replay sequence which had to be smaller than 3°, additional saccades during

presentation of the blank, and the event of the manual response. Participants showed

substantial individual variation in the numbers of these three types of additional saccades

which happened very likely unintentionally despite the strict gaze-contingent experimental

procedure.

The model formulas for each event consisted in the same two predictors preview

condition (valid, invalid) and viewing condition (saccade, replay, blank), with the exception of

the occasional saccades during the replay sequence and during presentation of the blank

screen because these two types of events only occurred within their respective viewing

condition and therefore the viewing condition factor was omitted from their model formula.

All saccade event formulas and the fixation onset events included saccade amplitude as

tenth order spline predictor to account for the non-linear effect of saccade amplitude on the

post-saccadic evoked potential (67). To make the replay condition comparable to the

saccade condition despite the lack of an actual saccade, we included the simulated

saccade amplitude of 8 dva as continuous predictor for the target onset event in that

condition.

796   All events were modelled with finite impulse response basis functions but the

temporal extent of these basis functions, also known as estimation time window length,

varied across event types in line with the time point at which an event could occur within a

trial, additionally considering that we wanted to capture all possible overlapping responses.

The preview onset time window was -275 ms to 2,000 ms after, the fixation/target onset

time window was -1,495 ms to 800 ms, for follow-up saccades, saccades during the replay

sequence and during blank presentation it was -275 ms to 800 ms, and for the manual

response it was -875 ms to 800 ms. The time windows were based on the maximum time

intervals between time-varying events across all participants.

805   The temporal deconvolution was conducted two times, separately for both source

806   space pipelines, for the one with the noise covariance matrix based on the baseline period

807   before preview image onsets and for the one where the noise covariance matrix was based

808   on the period before fixation/target onsets.

809   The deconvolution analysis was conducted separately for each participant.[1] From

810   the resulting regression coefficients, we calculated the predicted means (using the Julia

811   package *Effects*) per condition, region of interest, and time point. Statistical significance

812   was assessed at the group level by applying cluster-based permutation statistics in the time

813   domain using a significance threshold of p = .05 (147).

814   **Preregistration**

815   This study was preregistered (https://osf.io/uxtqn). We eventually deviated from the

816   preregistration in the sense that we did not go into the source space for individual vertices

817   but selected regions of interest. This step was necessary for computational reasons related

818   to temporal deconvolution. Initially, we did not consider deconvolution for the

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[1] We would have preferred to model the data of all participants in one mixed model with random factors for participants and items. This procedure was, however, computationally infeasible, because for only a single data channel the calculations exceeded the maximum resources available on our compute cluster.

preregistration because, based on previous research (28,142), we had assumed to get

largely the same types of eye movements in both valid and invalid viewing conditions.

However, it turned out that the invalid preview condition with a heavily degraded object

eventually led to slightly different saccade characteristics compared to the valid preview

condition. To deal with these different characteristics we employed deconvolution which

removes the overlapping effect of neural responses to subsequent eye movement events.

However, with deconvolution, projecting sensor data into source space becomes less

straightforward, because to go into source space with minimum norm estimation we need a

noise-covariance matrix which has to be estimated from single-trial data. Per definition we

do not have any single-trial data anymore after deconvolution, only the effects aggregated

across trials per conditions per participant. Deconvolution is a type of temporally extended

regression which means that the result are regression coefficients which summarize the

effect across single-trial data. To get source space results with deconvolution, we had to

run the deconvolution analysis on data that had already been projected into source space.

There are two options of running unfold on source-projected data: Run it for each vertex or

run it for ROIs with signal summarized across vertices within one ROI. We chose the second

option, because running deconvolution on each vertex would have been computationally

infeasible.

Data and code availability

839   All raw data and the code to generate the results from the raw data can be obtained

840   from the Radboud Data Repository under https://doi.org/10.34973/sqkg-xm80.

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References

1. Binda P, Morrone MC. Vision during saccadic eye movements. Annu Rev Vis Sci. 2018;4:193–213.

2. Bosco A, Lappe M, Fattori P. Adaptation of saccades and perceived size after trans-saccadic changes of object size. J Neurosci. 2015;35(43):14448–56.

3. Edwards G, Vetter P, McGruer F, Petro LS, Muckli L. Predictive feedback to V1 dynamically updates with sensory input. Sci Rep. 2017;7(1):1–12.

4. Edwards G, VanRullen R, Cavanagh P. Decoding trans-saccadic memory. J Neurosci. 2018;38(5):1114–23.

5. Fabius JH, Fracasso A, Acunzo DJ, Van der Stigchel S, Melcher D. Low-level visual information is maintained across saccades, allowing for a postsaccadic hand-off between visual areas. J Neurosci [Internet]. 2020 Oct 28; Available from: http://www.jneurosci.org/lookup/doi/10.1523/JNEUROSCI.1169-20.2020

6. Fairhall SL, Schwarzbach J, Lingnau A, Van Koningsbruggen MG, Melcher D. Spatiotopic updating across saccades revealed by spatially-specific fMRI adaptation. NeuroImage. 2017;147(November 2016):339–45.

7. Friston K, Adams RA, Perrinet L, Breakspear M. Perceptions as hypotheses: Saccades as experiments. Front Psychol. 2012;3:1–20.

8. Grzeczkowski L, Deubel H, Szinte M. Stimulus blanking reveals contrast-dependent transsaccadic feature transfer. Sci Rep [Internet]. 2020 Oct 29 [cited 2025 July 11];10(1). Available from: https://www.nature.com/articles/s41598-020-75717-y

9. Grzeczkowski L, van Leeuwen J, Belopolsky AV, Deubel H. Spatiotopic and saccade-specific transsaccadic memory for object detail. J Vis. 2020 July 6;20(7):2.

10. Herwig A, Schneider WX. Predicting object features across saccades: Evidence from object recognition and visual search. J Exp Psychol Gen. 2014;143(5):1903–22.

11.   Huber-Huber C, Buonocore A, Melcher D. The extrafoveal preview paradigm as a
measure of predictive, active sampling in visual perception. J Vis. 2021 July
20;21(7):12.

12.   Hübner C, Schütz AC. A bias in saccadic suppression of shape change. Vision Res.
2021 Sept;186:112–23.

13.   Johnston P, Robinson J, Kokkinakis A, Ridgeway S, Simpson M, Johnson S, et al.
Temporal and spatial localization of prediction-error signals in the visual brain. Biol
Psychol. 2017;125:45–57.

14.   Melcher D, Colby CL. Trans-saccadic perception. Trends Cogn Sci. 2008;12:466–73.

15.   Parr T, Friston KJ. The active construction of the visual world. Neuropsychologia.
2017;104(July):92–101.

16.   Parr T, Friston KJ. Active inference and the anatomy of oculomotion.
Neuropsychologia. 2018;111(October 2017):334–43.

17.   Valsecchi M, Gegenfurtner KR. Dynamic re-calibration of perceived size in fovea and
periphery through predictable size changes. Curr Biol. 2016;26(1):59–63.

18.   Bompas A, O'Regan JK. More evidence for sensorimotor adaptation in color
perception. J Vis. 2006;6(2):145–53.

19.   Bosco A, Rifai K, Wahl S, Fattori P, Lappe M. Trans-saccadic adaptation of perceived
size independent of saccadic adaptation. J Vis. 2020;20(7):1–11.

20.   Cavanaugh J, Berman RA, Joiner WM, Wurtz RH. Saccadic corollary discharge
underlies stable visual perception. J Neurosci. 2016;36:31–42.

21.   Herwig A. Linking perception and action by structure or process? Toward an
integrative perspective. Neurosci Biobehav Rev. 2015 May;52:105–16.

22.   Paeye C, Collins T, Cavanagh P, Herwig A. Calibration of peripheral perception of
shape with and without saccadic eye movements. Atten Percept Psychophys.
2018;80(3):723–37.

23.   Stewart EEM, Valsecchi M, Schütz AC. A review of interactions between peripheral and
foveal vision. J Vis. 2020 Nov 3;20(12):2.

24.   Sun LD, Goldberg ME. Corollary discharge and oculomotor proprioception: Cortical
mechanisms for spatially accurate vision. Annu Rev Vis Sci. 2016 Oct 14;2(1):61–84.

25.   Xie XY, Morrone MC, Burr DC. Serial dependence in orientation judgments at the time
of saccades. J Vis. 2023 July 10;23(7):7.

26.   Buonocore A, Dimigen O, Melcher D. Post-saccadic face processing is modulated by
pre-saccadic preview: Evidence from fixation-related potentials. J Neurosci. 2020 Jan
30;0861–19.

27.   de Lissa P, McArthur G, Hawelka S, Palermo R, Mahajan Y, Degno F, et al. Peripheral
preview abolishes N170 face-sensitivity at fixation: Using fixation-related potentials to
investigate dynamic face processing. Vis Cogn. 2019 Nov 26;27(9–10):740–59.

28.   Huber-Huber C, Buonocore A, Dimigen O, Hickey C, Melcher D. The peripheral
preview effect with faces: Combined EEG and eye-tracking suggests multiple stages of
trans-saccadic predictive and non-predictive processing. NeuroImage. 2019;200:344–
62.

29.   Degno F, Loberg O, Zang C, Zhang M, Donnelly N, Liversedge SP. Parafoveal previews
and lexical frequency in natural reading: Evidence from eye movements and fixation-
related potentials. J Exp Psychol Gen. 2019;148(3):453–74.

30.   Dimigen O, Sommer W, Hohlfeld A, Jacobs AM, Kliegl R. Coregistration of eye
movements and EEG in natural reading: Analyses and review. J Exp Psychol Gen.
2011;140:552–72.

31.   Dimigen O, Kliegl R, Sommer W. Trans-saccadic parafoveal preview benefits in fluent
reading: A study with fixation-related brain potentials. NeuroImage. 2012;62:381–93.

32.   Hutzler F, Braun M, Võ MLH, Engl V, Hofmann M, Dambacher M, et al. Welcome to the
real world: Validating fixation-related brain potentials for ecologically valid settings.
Brain Res. 2007;1172(1):124–9.

33.   Kornrumpf B, Niefind F, Sommer W, Dimigen O. Neural correlates of word recognition:
A systematic comparison of natural reading and rapid serial visual presentation. J
Cogn Neurosci. 2016;28:1374–91.

34.   Rayner K. The perceptual span and peripheral cues in reading. Cognit Psychol.
1975;7:65–81.

35.   Rayner K. Eye movements in reading and information processing: 20 years of research.
Psychol Bull. 1998 Nov;124(3):372–422.

36.   Schotter ER. Reading ahead by hedging our bets on seeing the future: Eye tracking
and electrophysiology evidence for parafoveal lexical processing and saccadic control
by partial word recognition. In: Federmeier KD, Watson DG, editors. Current Topics in
Language [Internet]. Academic Press; 2018. p. 263–98. Available from:
https://doi.org/10.1016/bs.plm.2018.08.011

37.   Schotter ER, Angele B, Rayner K. Parafoveal processing in reading. Atten Percept
Psychophys. 2012;74:5–35.

38.   Amthor FR, Tootle JS, Gawne TJ. Retinal ganglion cell coding in simulated active
vision. Vis Neurosci. 2005 Nov;22(6):789–806.

39.   DiCarlo JJ, Maunsell JHR. Form representation in monkey inferotemporal cortex is
virtually unaltered by free viewing. Nat Neurosci. 2000 Aug;3(8):814–21.

40.   Gawne TJ, Martin JM. Responses of Primate Visual Cortical Neurons to Stimuli
Presented by Flash, Saccade, Blink, and External Darkening. J Neurophysiol. 2002 Nov
1;88(5):2178–86.

41.   Richmond BJ, Hertz JA, Gawne TJ. The relation between V1 neuronal responses and
eye movement-like stimulus presentations. Neurocomputing. 1999 June;26–27:247–
54.

42.   MacEvoy SP, Hanks TD, Paradiso MA. Macaque V1 Activity During Natural Vision:
Effects of Natural Scenes and Saccades. J Neurophysiol. 2008 Feb;99(2):460–72.

43. McFarland JM, Bondy AG, Saunders RC, Cumming BG, Butts DA. Saccadic
modulation of stimulus processing in primary visual cortex. Nat Commun. 2015 Sept
15;6(1):8110.

44. Rolfs M, Schweitzer R. Coupling perception to action through incidental sensory
consequences of motor behaviour. Nat Rev Psychol. 2022;1(2):112–23.

45. Grill-Spector K, Henson R, Martin A. Repetition and the brain: Neural models of
stimulus-specific effects. Trends Cogn Sci. 2006;10:14–23.

46. Grotheer M, Kovács G. Can predictive coding explain repetition suppression? Cortex.
2016;80:113–24.

47. Czigler I. Visual mismatch negativity: Violation of nonattended environmental
regularities. J Psychophysiol. 2007;21:224–30.

48. Garrido MI, Kilner JM, Stephan KE, Friston KJ. The mismatch negativity: A review of
underlying mechanisms. Clin Neurophysiol. 2009;120:453–63.

49. Male AG. Predicting the unpredicted … brain response: A systematic review of the
feature-related visual mismatch negativity (vMMN) and the experimental parameters
that affect it. Bruns P, editor. PLOS ONE. 2025 Feb 27;20(2):e0314415.

50. Pazo-Alvarez P, Cadaveira F, Amenedo E. MMN in the visual modality: A review. Biol
Psychol. 2003;63:199–236.

51. Stefanics G, Kremláček J, Czigler I. Visual mismatch negativity: A predictive coding
view. Front Hum Neurosci. 2014;8(666):1–19.

52. Benda J. Neural adaptation. Curr Biol. 2021 Feb;31(3):R110–6.

53. Gawne TJ, Woods JM. The responses of visual cortical neurons encode differences
across saccades. NeuroReport. 2003;

54. Weber AI, Krishnamurthy K, Fairhall AL. Coding Principles in Adaptation. Annu Rev Vis
Sci. 2019 Sept 15;5(1):427–49.

55. Zimmermann E, Morrone MC, Fink GR, Burr D. Spatiotopic neural representations
develop slowly across saccades. Curr Biol. 2013;23:R193–4.

56. Cicchini GM, Binda P, Burr DC, Morrone MC. Transient spatiotopic integration across
saccadic eye movements mediates visual stability. J Neurophysiol. 2013;109(4):1117–
25.

57. De Beeck HO, Vogels R. Spatial sensitivity of macaque inferior temporal neurons. J
Comp Neurol. 2000;426(4):505–18.

58. DiCarlo JJ, Zoccolan D, Rust NC. How does the brain solve visual object recognition?
Neuron. 2012;73(3):415–34.

59. Ito M, Tamura H, Fujita I, Tanaka K. Size and position invariance of neuronal responses
in monkey inferotemporal cortex. J Neurophysiol. 1995;73(1):218–26.

60. Kobatake E, Tanaka K. Neuronal selectivities to complex object features in the ventral
visual pathway of the macaque cerebral cortex. J Neurophysiol. 1994;71(3):856–67.

61. Amano K, Wandell BA, Dumoulin SO. Visual Field Maps, Population Receptive Field
Sizes, and Visual Field Coverage in the Human MT+ Complex. J Neurophysiol. 2009
Nov;102(5):2704–18.

62. Dumoulin SO, Wandell BA. Population receptive field estimates in human visual cortex.
NeuroImage. 2008 Jan 15;39(2):647–60.

63. Zimmermann E, Weidner R, Abdollahi RO, Fink GR. Spatiotopic adaptation in visual
areas. J Neurosci. 2016;36(37):9526–34.

64. Zimmermann E, Weidner R, Fink GR. Spatiotopic updating of visual feature
information. J Vis. 2017;17(12):1–9.

65. Findlay JM, Gilchrist ID. Active Vision [Internet]. Active vision: The psychology of
looking and seeing. Findlay, John M.: U Durham, Dept of Psychology, Ctr for Vision &
Visual Cognition, Durham, United Kingdom: Oxford University Press; 2003. xiii, 220–
xiii, 220 p. Available from:
https://oxford.universitypressscholarship.com/view/10.1093/acprof:oso/978019852479
3.001.0001/acprof-9780198524793

66. Dimigen O, Ehinger BV. Regression-based analysis of combined EEG and eye-tracking
data: Theory and applications. J Vis. 2021 Jan 7;21(1):3.

67. Ehinger BV, Dimigen O. Unfold: an integrated toolbox for overlap correction, non-linear
modeling, and regression-based EEG analysis. PeerJ. 2019;1–33.

68. Smith NJ, Kutas M. Regression-based estimation of ERP waveforms: I. The rERP
framework. Psychophysiology. 2015;52:157–68.

69. Smith NJ, Kutas M. Regression-based estimation of ERP waveforms: II. Nonlinear
effects, overlap correction, and practical considerations. Psychophysiology.
2015;52:169–81.

70. Evans CC. Spontaneous excitation of the visual cortex and association areas —
Lambda waves. Electroencephalogr Clin Neurophysiol. 1953 Feb;5(1):69–74.

71. Kaunitz LN, Kamienkowski JE, Varatharajah A, Sigman M, Quiroga RQ, Ison MJ.
Looking for a face in the crowd: Fixation-related potentials in an eye-movement visual
search task. NeuroImage. 2014;89:297–305.

72. Yagi A. Saccade size and lambda complex in man. Physiol Psychol. 1979;7(4):370–6.

73. Glasser MF, Coalson TS, Robinson EC, Hacker CD, Harwell J, Yacoub E, et al. A multi-
modal parcellation of human cerebral cortex. Nature. 2016;536(7615):171–8.

74. Richter D, Ekman M, de Lange FP. Suppressed sensory response to predictable object
stimuli throughout the ventral visual stream. J Neurosci. 2018;38(34):7452–61.

75. Bullier J. Integrated model of visual processing. Brain Res Rev. 2001 Oct;36(2–3):96–
107.

76. Inui K, Kakigi R. Temporal Analysis of the Flow From V1 to the Extrastriate Cortex in
Humans. J Neurophysiol. 2006 Aug;96(2):775–84.

77. Ruiz O, Paradiso MA. Macaque V1 representations in natural and reduced visual
contexts: Spatial and temporal properties and influence of saccadic eye movements. J
Neurophysiol. 2012;108(1):324–33.

78. Boi M, Poletti M, Victor JD, Rucci M. Consequences of the Oculomotor Cycle for the
Dynamics of Perception. Curr Biol. 2017;27(9):1268–77.

79. Mostofi N, Zhao Z, Intoy J, Boi M, Victor JD, Rucci M. Spatiotemporal Content of
Saccade Transients. Curr Biol. 2020;30(20):3999-4008.e2.

80. Schweitzer R, Rolfs M. Intrasaccadic motion streaks jump-start gaze correction. Sci
Adv. 2021 July 23;7(30):eabf2218.

81. Nicolas G, Castet E, Rabier A, Kristensen E, Dojat M, Guérin-Dugué A. Neural
correlates of intra-saccadic motion perception. J Vis. 2021 Oct 26;21(11):19.

82. Gallant JL, Connor CE, Van Essen DC. Neural activity in areas V1, V2 and V4 during
free viewing of natural scenes compared to controlled viewing: NeuroReport. 1998
June;9(9):2153–8.

83. Kagan I, Gur M, Snodderly DM. Saccades and drifts differentially modulate neuronal
activity in V1: Effects of retinal image motion, position, and extraretinal influences. J
Vis. 2008 Nov 1;8(14):19–19.

84. Vinje WE, Gallant JL. Sparse Coding and Decorrelation in Primary Visual Cortex During
Natural Vision. Science. 2000 Feb 18;287(5456):1273–6.

85. Xiao W, Sharma S, Kreiman G, Livingstone MS. Feature-selective responses in
macaque visual cortex follow eye movements during natural vision. Nat Neurosci. 2024
Apr 29;1–10.

86. Zhang T, Bogadhi AR, Hafed ZM. Foveal neurons of the monkey superior colliculus
signal trans-saccadic prediction errors. Pack C, editor. PLOS Biol. 2025 June
23;23(6):e3003246.

87. Duhamel JR, Colby CL, Goldberg ME. The updating of the representation of visual
space in parietal cortex by intended eye movements. Science. 1992;255(5040):90–2.

88. Zirnsak M, Steinmetz N a, Noudoost B, Xu KZ, Moore T. Visual space is compressed in
prefrontal cortex before eye movements. Nature. 2014;507(7493):504–7.

89. Sommer MA, Wurtz RH. A Pathway in Primate Brain for Internal Monitoring of
Movements. Science. 2002 May 24;296(5572):1480–2.

90. Bisley JW, Mirpour K, Alkan Y. The functional roles of neural remapping in cortex. J
Vis. 2020;20(9):6.

91. Burr DC, Morrone MC. Spatiotopic coding and remapping in humans. Philos Trans R
Soc B Biol Sci. 2011 Feb 27;366(1564):504–15.

92. Colby CL, Goldberg ME. Space and attention in parietal cortex. Annu Rev Neurosci.
1999 Jan;22:319–49.

93. Golomb JD, Mazer JA. Visual Remapping. Annu Rev Vis Sci. 2021 Sept 15;7(1):257–77.

94. Mathôt S, Theeuwes J. Visual attention and stability. Philos Trans R Soc B Biol Sci.
2011;366:516–27.

95. Neupane S, Guitton D, Pack CC. Perisaccadic remapping: What? How? Why? Rev
Neurosci. 2020 July 28;31(5):505–20.

96. Sommer MA, Wurtz RH. Brain circuits for the internal monitoring of movements. Annu
Rev Neurosci. 2008 July;31:317–38.

97. Wurtz RH, Joiner WM, Berman RA. Neuronal mechanisms for visual stability: Progress
and problems. Philos Trans R Soc B Biol Sci. 2011;366(1564):492–503.

98. Zirnsak M, Moore T. Saccades and shifting receptive fields: Anticipating consequences
or selecting targets? Trends Cogn Sci. 2014;18(12):621–8.

99. Denagamage S, Morton MP, Hudson NV, Nandy AS. Widespread receptive field
remapping in early primate visual cortex. Cell Rep. 2024 Aug 27;43(8):114557.

100. Nakamura K, Colby CL. Updating of the visual representation in monkey striate and
extrastriate cortex during saccades. Proc Natl Acad Sci. 2002 Mar 19;99:4026–31.

101. Neupane S, Guitton D, Pack CC. Two distinct types of remapping in primate cortical
area V4. Nat Commun. 2016;7:10402.

102. He T, Fritsche M, de Lange FP. Predictive remapping of visual features beyond
saccadic targets. J Vis. 2018;18(13):1–16.

103. Mathôt S, Theeuwes J. Evidence for the predictive remapping of visual attention. Exp
Brain Res. 2010;200:117–22.

104. Melcher D. Predictive remapping of visual features precedes saccadic eye movements.
Nat Neurosci. 2007;10:903–7.

105. Rolfs M, Jonikaitis D, Deubel H, Cavanagh P. Predictive remapping of attention across
eye movements. Nat Neurosci. 2011 Feb;14:252–6.

106. Wolfe BA, Whitney D. Saccadic remapping of object-selective information. Atten
Percept Psychophys. 2015;77:2260–9.

107. Cont C, Zimmermann E. The Motor Representation of Sensory Experience. Curr Biol.
2021;31(5):1029-1036.e2.

108. Keller GB, Mrsic-Flogel TD. Predictive Processing: A Canonical Cortical Computation.
Neuron. 2018 Oct;100(2):424–35.

109. Whitmire CJ, Stanley GB. Rapid Sensory Adaptation Redux: A Circuit Perspective.
Neuron. 2016 Oct;92(2):298–315.

110. de Lange FP, Heilbron M, Kok P. How do expectations shape perception? Trends
Cogn Sci. 2018;22:764–79.

111. Williams MA, Baker CI, Op De Beeck HP, Mok Shim W, Dang S, Triantafyllou C, et al.
Feedback of visual object information to foveal retinotopic cortex. Nat Neurosci.
2008;11:1439–45.

112. Kroell LM, Rolfs M. Foveal vision anticipates defining features of eye movement
targets. eLife [Internet]. 2022 Sept 9;11. Available from:
https://elifesciences.org/articles/78106

113. Khayat PS, Spekreijse H, Roelfsema PR. Correlates of transsaccadic integration in the
primary visual cortex of the monkey. Proc Natl Acad Sci. 2004 Aug 24;101(34):12712–
7.

114. Bastos AM, Usrey WM, Adams RA, Mangun GR, Fries P, Friston KJ. Canonical
microcircuits for predictive coding. Neuron. 2012;76(4):695–711.

115. Bonaiuto JJ, Meyer SS, Little S, Rossiter H, Callaghan MF, Dick F, et al. Lamina-
specific cortical dynamics in human visual and sensorimotor cortices. eLife. 2018;7:1–
32.

116. Thomas ER, Haarsma J, Nicholson J, Yon D, Kok P, Press C. Predictions and errors
are distinctly represented across V1 layers. Curr Biol. 2024 May;34(10):2265-2271.e4.

117. Nobre A, Correa A, Coull J. The hazards of time. Curr Opin Neurobiol. 2007
Aug;17(4):465–70.

118. von Holst E, Mittelstaedt H. Das Reafferenzprinzip [The reafference principle].
Naturwissenschaften. 1950;37(20):464–76.

119. Helmholtz H von. Handbuch der physiologischen Optik [Handbook of physiological
optics]. Leipzig: Voss; 1867.

120. Silva JF, Yrjönsuuri M, editors. Active Perception in the History of Philosophy: From
Plato to Modern Philosophy [Internet]. Cham: Springer International Publishing; 2014
[cited 2025 Apr 30]. Available from: https://link.springer.com/10.1007/978-3-319-
04361-6

121. Grzeczkowski L, Stein A, Rolfs M. Trans-retinal predictive signals of visual features are
precise, saccade-specific and operate over a wide range of spatial frequencies. J
Neurophysiol. 2024;

122. Grzeczkowski L, Shi Z, Rolfs M, Deubel H. Perceptual learning across saccades:
Feature but not location specific. Proc Natl Acad Sci. 2023 Oct
24;120(43):e2303763120.

123. Scholes C, McGraw PV, Roach NW. Learning to silence saccadic suppression. Proc
Natl Acad Sci U S A. 2021;118(6).

124. Miura SK, Scanziani M. Distinguishing externally from saccade-induced motion in
visual cortex. Nature. 2022;610(7930):135–42.

125. Kleiser R, Skrandies W. Neural correlates of reafference: evoked brain activity during
motion perception and saccadic eye movements. Exp Brain Res. 2000 July
17;133(3):312–20.

126. Purpura KP, Kalik SF, Schiff ND. Analysis of perisaccadic field potentials in the
occipitotemporal pathway during active vision. J Neurophysiol. 2003;90(5):3455–78.

127. Troncoso XG, McCamy MB, Jazi AN, Cui J, Otero-Millan J, Macknik SL, et al. V1
neurons respond differently to object motion versus motion from eye movements. Nat

Commun [Internet]. 2015 Sept 15 [cited 2025 July 11];6(1). Available from: https://www.nature.com/articles/ncomms9114

128. Barczak A, Haegens S, Ross DA, McGinnis T, Lakatos P, Schroeder CE. Dynamic Modulation of Cortical Excitability during Visual Active Sensing. Cell Rep. 2019;27(12):3447-3459.e3.

129. Rajkai C, Lakatos P, Chen CM, Pincze Z, Karmos G, Schroeder CE. Transient cortical excitation at the onset of visual fixation. Cereb Cortex. 2008;18(1):200–9.

130. Leszczynski M, Schroeder CE. The Role of Neuronal Oscillations in Visual Active Sensing. Front Integr Neurosci [Internet]. 2019 July 23;13. Available from: https://www.frontiersin.org/article/10.3389/fnint.2019.00032/full

131. Schroeder CE, Wilson DA, Radman T, Scharfman H, Lakatos P. Dynamics of Active Sensing and perceptual selection. Curr Opin Neurobiol. 2010 Apr;20(2):172–6.

132. Hasson U. Uncovering a Timescale Hierarchy by Studying the Brain in a Natural Context. J Neurosci. 2025 Mar 19;45(12):e2368242025.

133. Leopold DA, Park SH. Studying the visual brain in its natural rhythm. NeuroImage. 2020;

134. Nastase SA, Goldstein A, Hasson U. Keep it real: rethinking the primacy of experimental control in cognitive neuroscience. NeuroImage. 2020 Nov 15;222:117254.

135. Singh VP, Li J, Dawson K, Mitchell JF, Miller CT. Active vision in freely moving marmosets using head-mounted eye tracking. Proc Natl Acad Sci. 2025 Feb 11;122(6):e2412954122.

136. Sonkusare S, Breakspear M, Guo C. Naturalistic Stimuli in Neuroscience: Critically Acclaimed. Trends Cogn Sci. 2019 Aug 1;23(8):699–714.

137. Li J, Singh V, Mitchell J, Huk A, Miller C. The role of active vision in the primary visual cortex of freely-moving marmosets. J Vis. 2025 July 15;25(9):2695.

138. Parker P. Neural coding and circuitry of active vision in mice. J Vis. 2025 July 15;25(9):1604.

139. Yates JL, Coop SH, Sarch GH, Wu RJ, Butts DA, Rucci M, et al. Detailed characterization of neural selectivity in free viewing primates. Nat Commun. 2023 June 20;14(1):3656.

140. Brady TF, Konkle T, Alvarez GA, Oliva A. Visual long-term memory has a massive storage capacity for object details. Proc Natl Acad Sci. 2008 Sept 23;105(38):14325–9.

141. Willenbockel V, Sadr J, Fiset D, Horne GO, Gosselin F, Tanaka JW. Controlling low-level image properties: the SHINE toolbox. Behav Res Methods. 2010;42:671–84.

142. Huber-Huber C, Melcher D. Saccade execution increases the preview effect with faces: An EEG and eye-tracking coregistration study. Atten Percept Psychophys [Internet]. 2023; Available from: https://doi.org/10.3758/s13414-023-02802-5

143. Huber-Huber C, Melcher D. The behavioural preview effect with faces is susceptible to
statistical regularities: Evidence for predictive processing across the saccade. Sci Rep.
2021 Dec 13;11(1):942.

144. Stolk A, Todorovic A, Schoffelen JM, Oostenveld R. Online and offline tools for head
movement compensation in MEG. NeuroImage. 2013 Mar;68:39–48.

145. Gramfort A, Luessi M, Larson E, Engemann DA, Strohmeier D, Brodbeck C, et al. MEG
and EEG data analysis with MNE-Python. Front Neurosci. 2013;7(7 DEC):1–13.

146. Plöchl M, Ossandón JP, König P. Combining EEG and eye tracking: Identification,
characterization, and correction of eye movement artifacts in electroencephalographic
data. Front Hum Neurosci. 2012 Jan;6:278.

147. Maris E, Oostenveld R. Nonparametric statistical testing of EEG- and MEG-data. J
Neurosci Methods. 2007;164(1):177–90.

1194