# THE ROLE OF PRIMARY VISUAL CORTEX IN UNCONSCIOUS VISUAL PROCESSING

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## Tiivistelmä

Aivovaurio primaarilla näköaivokuorella (V1) aiheuttaa sokeuden siihen osaan näkökenttää, jonka prosessoinnista vaurioitunut aivokuoren osa vastaa. Jotkut tällaisista primaarin näköaivokuoren vauriosta johtuvasta sokeudesta kärsivistä potilaista pystyvät kuitenkin tiedostamattomasti prosessoimaan visuaalisia ärsykkeitä, jotka on esitetty sokealle näkökentälle. Tällaista prosessointia voidaan mitata esimerkiksi pyytämällä koehenkilö esittämään paras arvaus visuaalisen ärsykkeestä, jota hän ei raportoi nähneensä. Mikäli vastaustarkkuus on arvaustodennäköisyyttä parempi, voidaan todeta, että visuaalinen ärsyke vaikutti henkilön käyttäytymiseen tiedostamattomalla tasolla. Tästä ilmiöstä käytetään yleisesti nimeä sokeanäkö. Neurologisilla potilailla tehdyistä havainnoista ei kuitenkaan voida suoraan tehdä päätelmiä, jotka yleistyisivät koskemaan kaikkia ihmisiä. Potilaiden aivoissa tapahtuu plastisiteetin vuoksi muutoksia, jotka voivat selittää näitä säästyneitä visuaalisia kykyjä. Transkraniaalisella magneettistimulaatiolla (TMS) voidaan tutkia neurologisesti terveiden koehenkilöiden aivojen osien funktioita, sillä TMS häiritsee hetkellisesti aivojen normaalia viestinvälitystä. Primaarille näköaivokuorelle kohdistetut TMS-pulssit häiritsevät tietoista näkemistä. Tässä tutkielmassa selvitin, onko tiedostamaton prosessointi mahdollista, kun ärsykkeen tietoista havaintoa on häiritty primaarin näköaivokuoren stimulaatiolla. Tutkimuksissamme havaitsimme, että sekä tietoinen että tiedostamaton näköinformaation prosessointi vaativat primaarin näköaivokuoren toimintaa neurologisesti terveillä. Tiedostamatonta näköinformaation prosessointia havaittiin ainoastaan yksinkertaisimmassa tehtävässä, jossa koehenkilö reagoi ärsykkeen ilmestymiseen. Silloinkin ainoastaan, kun aikaista prosessointia primaarilla näköaivokuorella ei häiritty. Liikkeen ja värin prosessointia mittaavissa kokeissa tiedostamatonta prosessointia V1-stimulaation aikana ei havaittu. Tiedostamaton näköinformaation prosessointi riippuu siis primaarin näköaivokuoren toiminnasta neurologisesti terveillä koehenkilöillä.

Avainsanat: sokeanäkö, primaari näköaivokuori, tiedostamaton prosessointi, transkraniaalinen magneettistimulaatio

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## Abstract

Lesion in primary visual cortex (V1) causes blindness in the visual field that is processed by the damaged area. Some patients with V1 lesion can unconsciously process the stimuli presented in their blind visual field. This kind of processing can be observed, for example, using a paradigm in which a patient is required to make a guess about the visual stimulus that he or she reports not seeing. If the accuracy of the responses is better than chance, it can be interpreted as unconscious processing, which is a measurable effect on behavior without conscious perception. This phenomenon of unconscious processing without V1 is commonly referred to as blindsight. The findings of unconscious processing without V1 in blindsight patients cannot be straightforwardly generalized to neurologically healthy people. Neural plasticity changes these patients' brains; therefore, blindsight could be explained by these neural changes. Transcranial magnetic stimulation (TMS) is a method used to study the function of a cortical area in neurologically healthy people. With TMS, it is possible to interfere with the normal functioning of the brain. TMS of V1 can suppress the conscious perception of a visual stimulus. In this thesis, I studied whether the unconscious processing of visual stimuli is possible in neurologically healthy participants when the conscious perception of the stimulus is suppressed by TMS of V1. I found that both conscious and unconscious processing of visual information depend on V1 in neurologically healthy participants. Unconscious processing was observed only with the simplest task, in which participants responded to stimulus appearance. In this task, unconscious processing was observed only when the early activation of V1 was intact. The unconscious processing of chromaticity and motion was not observed when the stimulus was suppressed by TMS of V1. Therefore, I conclude that unconscious visual processing depends on V1 in neurologically healthy participants.

Keywords: blindsight, primary visual cortex, unconscious processing, transcranial magnetic stimulation

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I thank everyone that has helped me along the way.

15.11.2019
*Mikko Hurme*

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## List of original publications

This thesis is based on three original articles. The studies are referred to in the text by the following roman numerals.

**I.** Hurme, M., Koivisto, M., Revonsuo, A., & Railo, H. (2017). Early processing in primary visual cortex is necessary for conscious and unconscious vision while late processing is necessary only for conscious vision in neurologically healthy humans. *NeuroImage, 150*, 230-238

**II.** Hurme, M., Koivisto, M., Revonsuo, A., & Railo, H. (2019). V1 activity during feedforward and early feedback processing is necessary for both conscious and unconscious motion perception. *NeuroImage, 185*, 313-321

**III.** Hurme, M., Koivisto, M., Henriksson, L., & Railo, H. (2020). Neuronavigated TMS of early visual cortex eliminates unconscious processing of chromatic stimuli, *Neuropsychologia*, 136, 107266

The studies are presented in the thesis in the order of the publication date.

# 1 Introduction

Lesion in primary visual cortex (V1, sometimes referred to as striate cortex) causes blindness in the visual field that is processed by the damaged area (Holmes, 1918). Therefore, patients with V1 lesion cannot consciously perceive anything presented in the blind visual field and if you present a visual stimulus to their blind field, they deny seeing it. However, it seems that all visual processing does not depend on V1 like conscious perception does. These visual processes are unconscious, meaning that although the patient denies seeing the stimulus, it has a measurable effect on the patient's behavior. In *blindsight*, a patient has a brain lesion in V1 that causes visual field loss, but they can still process the stimuli presented in their blind visual field to some extent (Pöppel, Held, & Frost, 1973; Weiskrantz, Warrington, Sanders, & Marshall, 1974). The stimulus-driven activation from the primary visual (geniculo-striate) pathway is absent in blindsight, but these patients still have various residual visual capacities. The question therefore is what are the visual pathways in the brain that enable these unconscious visual processes in these patients and can unconscious processing be found without the functioning of V1 in neurologically healthy people as well? In this thesis, I study whether unconscious processing independent of V1 is possible in neurologically healthy people or, alternatively, does unconscious processing depend on the stimulus-driven activation in V1.

## 1.1 Unconscious visual processes

Visual processes can be either conscious or unconscious if unconscious processing is understood simply as a visual process that does not reach consciousness. This distinction seems quite straightforward, but determining what processing is conscious and what is unconscious is not a trivial task. There is an ongoing debate about the nature of visual consciousness and whether there are conscious phenomenal experiences that cannot be reported (Dehaene & Naccache, 2001; Lamme, 2010; Lau & Rosenthal, 2011). In practice, the study of consciousness

heavily relies on subjective reports about consciousness; therefore, these phenomenal unreportable conscious experiences are difficult to grasp in experimental science. For that reason, in this thesis, conscious visual processing refers to the visual stimulus that the person is aware of and can report seeing.

To measure unconscious processing, one must first suppress the conscious perception of the stimulus and then measure the effects of the unconscious stimulus on behavior. There are multiple ways of interfering with conscious visual processing, for example, transcranial magnetic stimulation (TMS) or visual masking. These methods will be examined later (chapter 1.3); currently, I focus on measuring unconscious processing after the stimulus is suppressed. To ensure that the manipulation of the conscious visual experience was successful, one must somehow evaluate the conscious experience of the participants. This is achieved by asking the participants to report the content of their perception. One method of evaluation is asking if the participant saw the presented stimulus or not. However, this method of evaluation is based on an assumption that the suppression of conscious perception is working in an on–off fashion. Even though in some cases the suppression might be so effective that participants are satisfied with two awareness options (Sergent & Dehaene, 2004), all cases of conscious perception are not the same (Overgaard, Rote, Mouridsen, & Ramsøy, 2006). If the participants perceive the stimulus very weakly, they have difficulties in determining whether they saw the stimulus or not. Sometimes, the participant perceives features of the stimulus but not those that are relevant to the task. For example, when they are asked to report the shape of the stimulus, they might see a vague image but they have no idea if the stimulus was a square or a triangle. To take into account the fact that conscious perception is likely a gradual phenomenon at least in some cases, the four-point perceptional awareness scale (PAS) has been used (Overgaard et al., 2006). The participant selects whether he or she "did not see," "saw a weak glimpse," "saw an almost clear image," or "saw a clear image." Using PAS gives participants more options to select the description of the visual perception that more accurately represents their conscious experience. If, however, the suppression would work in a dichotomous manner, the participant could still report only "did not see" or "saw a clear image" as in two-point awareness scales. Another way to tackle this issue is to use confidence ratings (Sandberg, Timmermans, Overgaard, & Cleeremans, 2010), wherein the participants report how confident they were about their awareness report and only the trials in which the participant reports being confident of not seeing the stimulus are considered unconscious.

### 1.1.1 Forced-choice paradigm

One of the simplest measures of unconscious processing is the performance in a forced-choice task when the participant reports that he or she did not see the stimulus. If the performance in this task is above chance level, the conclusion is that the participant could unconsciously process the stimuli. This paradigm is, however, not without its limitations. The participant is required to make a best guess about the stimulus that he or she did not perceive. The task feels very counterintuitive to many and the participant may realize that the study is measuring unconscious processing. Another problem arises when a two-point awareness scale is used. The participants might change their response criterion (not deliberately) to the seen/unseen evaluation because they feel like they should be certain about their guess when they report that they saw something.

### 1.1.2 Redundant target effect

The measure of unconscious processing should optimally not rely on guessing and the task should remain the same in both unconscious and conscious processing measurements. Redundant target effect (RTE) (sometimes referred to as redundant signals effect or bilateral gain) is a phenomenon in which a stimulus that is not needed to complete the task affects the performance. More precisely, the task is to respond as quickly as possible when a stimulus or stimuli are presented and people respond faster to two stimuli than to a single stimulus (Kinchla, 1974; Miller, 1982). RTE can be easily employed to measure unconscious processing. The participant is instructed to respond to any number of stimuli and report how many stimuli he or she saw. The conscious perception of one of the two stimuli is suppressed and then the effect of that unconscious stimulus can be measured. If the participant reports seeing only one stimulus when two stimuli are actually presented, whether the unconscious redundant target speeds up reaction times can be measured. In practice, this can be performed by collecting trials in which only one stimulus is presented and the participant correctly sees them. Then, the reaction times to those single-stimulus trials are compared with the reaction times to the trials in which an unconscious redundant target was presented in addition to the conscious stimulus. If the reaction times are on average faster in the unconscious redundant target trials than in the single-stimulus trials, the unconscious redundant target had a measurable effect on behavior.

Why does the redundant target speed up reaction times? There are two hypotheses that explain the (conscious) RTE: race model and neural coactivation model (Miller, 1982). The race model explains the RTE in pure probabilistic terms. It assumes that both the stimuli are capable of producing the response and that there is an internal

competition, “a horse race,” between the signals elicited by the two stimuli. The signal that pushes over the response threshold faster produces the response. The faster of the two signals always wins and therefore faster reaction times are more probable. However, in some cases, the race model fails to explain the speed-up and the only possible explanation is that the neural coactivation elicits the faster reaction times, which means that both signals are contributing to the response. This neural coactivation can occur at perceptual, cognitive, or motor levels (Iacoboni & Zaidel, 2003). Miller (1982) proposed the race model test to determine whether the race model hypothesis can explain RTE. He found that the race model could not explain the speed-up in his experiment using multimodal (auditory and visual) signals. Therefore, the neural coactivation model was the only possible explanation for RTE. The race model test is in principle very simple: the reaction-time cumulative distribution functions (CDF) in one-stimulus trials are compared with the reaction-time CDFs in two-stimulus trials. If the cumulative probability of the fast reaction times is higher in the two-stimulus trials than in the one-stimulus trials, the race model has been violated. This means that the race model cannot explain the RTE and that the reaction times in the two-stimulus trials are faster than those that the two competing signals could produce. The whole is greater than the sum of its parts, so to say. One limitation of the race model test is that if the race model is not violated, the explanation can be either the race model or the neural coactivation model. One cannot exclude the neural coactivation model just because the race model *could* explain the RTE.

Then, what about unconscious RTE? RTE can be found even when the redundant target is unconscious (Savazzi & Marzi, 2002; Tamietto et al., 2010). It is safe to assume that the unconscious single target does not typically elicit a response. One does not react to a stimulus that is not consciously perceived. Thus, it is impossible to draw the CDF to the unconscious one-stimulus trials and therefore the typical race model test cannot be performed on unconscious RTE data because there is no race. Only conscious stimulus elicits the response, but the unconscious redundant target speeds up the reaction time of the response. The explanation to unconscious RTE is always neural coactivation, unless there is an unconscious single-stimulus trial that can elicit the response. One possible way to collect unconscious single-stimulus trials could be to measure eye movements because they are more automatic than manual responses. The RTE has clear advantages over forced-choice as a measure of unconscious processing; therefore, across all three studies, we employed the RTE as a measure of unconscious processing.

### 1.1.3 Unconscious visual processing in the brain

What happens in the brain during unconscious visual processing? Here I discuss the two different explanations to answer this question: the anatomical explanation and the physiological explanation. By physiological explanation, I mean that the same visual processing areas contribute to both conscious and unconscious processes but the timing and connectivity within time-windows might differ. By anatomical explanation, I mean that the unconscious information is processed in different brain areas or that the information is carried through different pathways from the conscious information.

Anatomical models explain unconscious processing based on the differentiated pathways for unconscious and conscious processes. Figure 1 schematizes these pathwaysFigure 2. The primary visual pathway has been extensively studied and it goes from the retina to the parvocellular and magnocellular layers of the lateral geniculate nucleus (LGN) and then connects to V1. This visual pathway is critical to visual consciousness, and a lesion of V1 (Holmes, 1918) or this pathway (Marzi, Mancini, Metitieri, & Savazzi, 2006) causes blindness. V1 is organized such that a specific part of a visual field is processed in a specific part of V1. A retinotopic map (Figure 2) represents the mapping between the spatial visual input and the corresponding part of the visual cortex processing it. It is acquired using functional magnetic resonance imaging (fMRI) (Henriksson, Karvonen, Salminen-Vaparanta, Railo, & Vanni, 2012; Vanni, Henriksson, & James, 2005). V1 is located in the calcarine sulcus that is in occipital cortex (Hinds et al., 2008). The left hemisphere processes the right visual field and vice versa. The upper bank of the calcarine sulcus

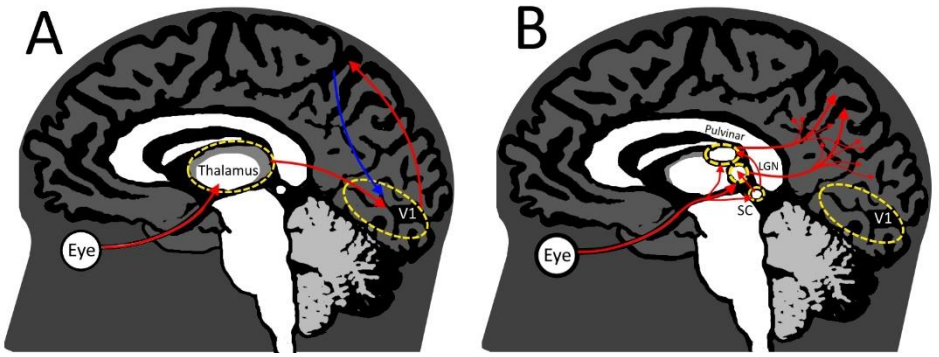


*Figure 1.* Schematic of the geniculo-striate pathway and the subcortical pathways that bypass V1. A) Geniculo-striate pathway. Red arrows represent the feedforward sweep from the eye via the lateral geniculate nucleus of the thalamus to V1. From V1, the feedforward sweep activates higher visual processing areas and recurrent activation takes place (blue arrow). B) V1-bypassing pathways from the superior colliculus, LGN, and pulvinar to the extrastriate and parietal areas.

processes the lower visual field and the lower bank processes the upper visual field. Therefore, for example, the lower left visual field is processed in the upper bank of the calcarine sulcus in the right hemisphere. The information is passed from V1 to the adjacent visual processing areas V2, V3 and V3a and to V4, which processes mostly chromatic signals, and to V5, which processes mostly motion. Rapidly (and partially simultaneously) after the feedforward sweep, the recurrent circuits activate. The exact mechanism of how this information through the geniculo-striate pathway gives rise to the conscious experience is debated (Dehaene & Naccache, 2001; Lamme, 2010; Lau & Rosenthal, 2011). However, the importance of V1 and the geniculo-striate pathway in conscious visual perception is not denied.

Besides the geniculo-striate pathway, multiple other connections from subcortical areas to extrastriate cortex and parietal areas do not go through V1. An extremely connected area is the pulvinar, which is connected to the superior colliculus (SC) and visual cortical areas V1, V2, V3, V3a, V4, and V5 as well as parietal areas (Leh, Chakravarty, & Ptito, 2008). LGN also directly connects to other visual cortical areas besides V1, such as V2 (Fries, 1981) and V5 (Gaglianese et al., 2015). The most widely acknowledged of these pathways is probably the retinotectal pathway that connects the SC via the pulvinar to extrastriate areas. Unfortunately, the role of these pathways in unconscious processing is difficult to study in humans. The activation of these subcortical structures can be evaluated using fMRI, but the activation in these pathways can reflect either feedforward or feedback activation. Sumner, Adamjee, and Mollon (2002) proposed a simple behavioral task to examine the role of the SC: if an effect is not found using S-cone isolating short-wavelength stimuli, the tract from the superior colliculus explains the effect. This assumption is based on the primate studies that have suggested that SC does not activate in response to short-wavelength stimuli (de Monasterio, 1978; Marrocco & Li, 1977; Schiller & Malpeli, 1977). However, this method of studying collicular contribution has recently been

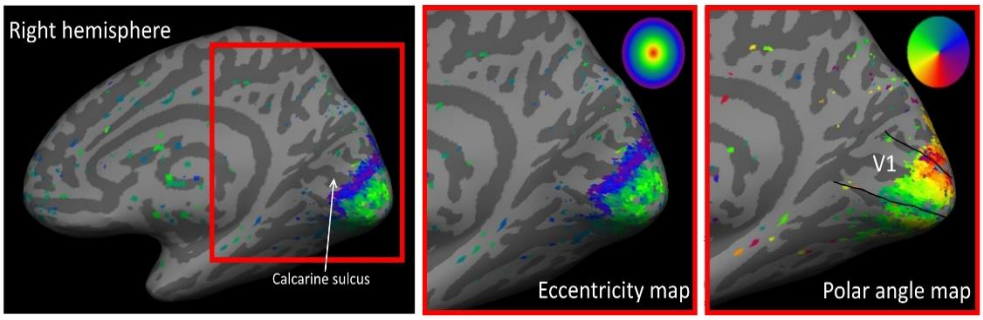


*Figure 2.* Functional magnetic resonance images of the visual cortex. Eccentricity map shows in same color the activation that is caused by stimuli that are presented at the same distance away from the fovea. There were no stimuli presented to the fovea in the sequence; therefore, the foveal activation is missing from the figure. Polar angle map shows the angle from the fovea at which the stimulus was presented.

challenged. S-cone isolating stimuli can elicit express saccades that are triggered by SC (Hall & Colby, 2016), and SC shows activation in response to short-wavelength stimuli (Hall & Colby, 2014). Despite these findings that challenge the validity of the short-wavelength stimuli as a way to study the function of the SC, interesting dissociations are found in unconscious processing between short- and long-wavelength stimuli (Savazzi & Marzi, 2004; Tamietto et al., 2010).

In contrast to the presented anatomical models, physiological models explain the differences between conscious and unconscious visual processing by stating that V1 is involved in both processes but within different temporal stages. An example of the physiological model explaining unconscious processing is Lamme's theory (Lamme, 2004, 2010; Lamme & Roelfsema, 2000). Lamme argues that information flowing from V1 to extrastriate areas is capable of producing unconscious processing. This unidirectional information flow is called the feedforward sweep, and rapidly after that, recurrent activation takes place. According to Lamme, the recurrent activation between extrastriate areas and V1 gives rise to conscious experience. This means that V1 serves as a relay station during the unconscious feedforward time-window and contributes to the conscious visual perception during the recurrent activation time-window. The global neuronal workspace model predicts similar outcomes for feedforward information (Dehaene, Changeux, Naccache, Sackur, & Sergent, 2006). Feedforward stimulus-driven activation could cause unconscious processing; however, if it is not connected to the parieto-frontal global workspace, it does not become conscious. Both of the physiological models presented here assume that the feedforward sweep is unconscious but can influence behavior. Therefore, conscious perception could be suppressed and unconscious processing could be observed as long as the feedforward activation is not disrupted.

Note that the anatomical and physiological models do not exclude one another. It is likely that some of the unconscious processes are caused by the subcortical pathways connecting to the extrastriatal areas that bypass V1 but other types of unconscious processes depend on the feedforward processing (and some even on recurrent processing) in V1. Also, the distinction that the information is not conscious is not sufficient to draw general conclusions about the processes in the brain. Multiple processing phases contribute to conscious vision; therefore, multiple processes can be disrupted to suppress conscious vision. One should figure out *why* the information is unconscious and during what processing phase was the processing disrupted. If the conscious vision is absent owing to damage in retina, we should anticipate different (unconscious) activations and processes than if the conscious vision was absent owing to V1 lesion.

## 1.2 Blindsight

Unconscious visual processing without the functioning of V1 has been demonstrated in the clinical cases of blindsight. Blindsight refers to the phenomenon in which a neurological patient who has a blind visual hemifield owing to V1 lesion can still unconsciously process stimuli presented in the blind visual field (Pöppel et al., 1973; Weiskrantz et al., 1974). Blindsight patients are able to, for example, discriminate colors (Brent, Kennard, & Ruddock, 1994; Stoerig & Cowey, 1989, 1992), speed up reaction times (Tamietto et al., 2010; Tomaiuolo, Ptito, Marzi, Paus, & Ptito, 1997), and discriminate the direction of the motion stimulus (Ffytce & Zeki, 1998) based on the stimuli that they report not being aware of. These findings suggest that visual information can affect the blindsight patients' behavior via pathways that do not depend on V1.

The term "blindsight" was coined by Lawrence Weiskrantz after he found the above chance discrimination performance in the blind visual field in some V1 lesioned patients (Weiskrantz et al., 1974). Even though "blindsight" is a catchy (and commonly used) expression, it is slightly misleading. To speak of these residual capacities as "sight" is very much an exaggeration. If a person with normal sight would, for example, discriminate vertical bars from horizontal ones at 70% accuracy, this would be a demonstration of difficulties in the task, whereas in blindsight, this would be phenomenal performance. A more accurate description of blindsight could be the residual unconscious visual capacities in the absence of V1.

Dankert and Rosetti (2005) highlighted the differences of these residual abilities and the respective neural pathways enabling these abilities in their review on blindsight studies. They created taxonomy of three types of blindsight: action-blindsight, attention-blindsight, and agnosopsia. *Action-blindsight* refers to the ability to guide motor responses based on the stimuli presented in the blind field. The enabling residual pathway is connected from SC to pulvinar and from there to posterior parietal cortex. *Attention blindsight* is manifested when unconscious stimulus captures patient's attention or elicits a sensation or feeling of a stimulus that lacks visual properties. This might sound self-contradictory; what does a visual sensation that lacks visual properties look like? One example of this is Riddoch's syndrome, in which a patient can perceive the motion in his or her blind field but not the visual stimulus itself (Riddoch, 1917). One patient with Riddoch's syndrome described the experience as "a black shadow moving on a black background" and that "shadow is the nearest I can get to putting it into words so that people can understand" (Ffytce & Zeki, 1998). These not fully unaware unconscious blindsight processes are sometimes referred to as Type 2 blindsight, whereas the in-absence-of-awareness blindsight is called Type 1 blindsight. The Type 2 blindsight, or attention blindsight as Dankert and Rosetti call it, is assumed to be caused by the

information from the SC–pulvinar–extrastriate cortex pathway. The last category in Dankert and Rosetti's taxonomy is agnosopsia, the term coined by Zeki and Ffytche (1998) that means literally "to not know what one sees." This form of blindsight is most likely in action when making a correct forced-choice guess about the properties of the stimulus presented in the blind visual field. This kind of processing is attributed to the pathway connecting the interlaminal layers of the lateral geniculate nucleus (LGN) to the extrastriate areas. This three-fold taxonomy is a great reminder that even though blindsight might seem like a single phenomenon, there might be multiple underlying explanations to its residual capacities. Therefore, the researchers who claim in their article that they have found an explanation for blindsight in general (e.g., Schmid et al., 2010) may have in fact found an explanation for just one type of blindsight.

This thesis is focused on the motor responses affected by the unconscious visual processing that does not depend on V1; therefore, the most relevant blindsight findings are those of action blindsight. Unconscious RTE, which is a great example of unconscious action, has been employed in the study of blindsight since the early years of blindsight studies (Marzi, Tassinari, Aglioti, & Lutzemberger, 1986). Some, but not all, of the patients showed RTE when the redundant target was presented in their blind visual field. This finding was later replicated in two blindsight patients (Tomaiuolo et al., 1997). Conclusive evidence for the neural mechanism of this speed-up has not been found. Savazzi and Marzi (2004) found in neurologically healthy participants that (conscious) RTE with red, gray, and white stimuli violated the race model, but RTE in purple stimuli could be explained by the race model. They concluded that the activation of SC is needed for interhemispheric summation, that is, in Miller's terms, neural coactivation. Tamietto et al. (2010) hypothesized that the most probable explanation for unconscious RTE after V1 lesion is the information carried by the retinotectal tract; therefore, they employed the short-wavelength stimulus to study the contribution of the SC. They presented one stimulus to the normal visual field and sometimes a redundant stimulus to the blind visual field. Blindsight measured as RTE was found with gray and red stimuli but not with purple stimuli. They also found fMRI activation in the SC in response to gray stimuli but not to purple stimuli. Unfortunately, they did not measure activation in response to red stimuli so that the difference in fMRI might be explained by chromatic versus achromatic information processing. However, two hemispherectomized patients who have no other intact ipsilateral subcortical visual structures than the SC showed RTE as well (Georgy, Celeghin, Marzi, Tamietto, & Ptito, 2016). Therefore, the contribution of SC seems to be the most prominent explanation of blindsight measured as RTE.

The problem with blindsight studies is that the findings are not straightforwardly generalizable to healthy population. Neural plasticity can play a crucial role in

explaining the residual abilities in blindsight patients. After V1 lesion, new connections are formed and the existing connections change their functions in the patients' brain (Leh, Johansen-Berg, & Ptito, 2006; Mikellidou et al., 2017; Payne & Lomber, 2001). Neurological patients with blindsight (measured as RTE) have functional connections from the V1-lesion-sided SC to the ipsilateral and contralateral extrastriatal areas (Leh et al., 2006) and from LGN to V5 (Ajina, Pestilli, Rokem, Kennard, & Bridge, 2015), whereas patients with no blindsight do not have these connections.

## 1.3 Methods for suppressing conscious vision in neurologically healthy people

To study unconscious processing in normal brain, it is necessary to suppress the conscious perception of the target stimulus. Here I present two methods for suppressing conscious vision, both used in the original studies: metacontrast masking and TMS. In addition to these, there are other methods to interfere with conscious perception, but they fall outside the topic of this thesis and therefore are not discussed here.

### 1.3.1 Metacontrast masking

Visual masking paradigms are powerful methods to suppress the conscious perception of the stimulus. Metacontrast masking refers to a specific type of visual masking, and it has been proven to be effective way to interfere with conscious perception (Breitmeyer, Ogmen, & Chen, 2004; Breitmeyer, Ro, Öğmen, & Todd, 2007). Metacontrast mask is an additional visual stimulus that is presented after the target stimulus, and the mask stimulus does not overlap with the target stimulus. The masking effect as a function stimulus onset asynchrony (SOA) typically shows a U-shaped function. Stimulus is not suppressed with very short or long SOAs; however, with intermediate SOAs, the masking effect peaks. The problem with visual masking is that it is difficult to evaluate the mechanisms that enable the mask to suppress conscious perception. One interpretation is that the mask interferes with the recurrent processing of the target stimulus and therefore conscious perception is suppressed (Lamme, Zipser, & Spekreijse, 2002), whereas others claim that the mask interferes with the feedforward processes (Macknik & Martinez-Conde, 2007).

Unconscious processing of visual information is possible when the conscious perception of the visual stimulus is suppressed using metacontrast masking

(Breitmeyer et al., 2004, 2007; Railo & Koivisto, 2012). This implies that methods to selectively interfere with the conscious processing while leaving unconscious processing intact exist. In our Studies II and III, we used metacontrast masking to demonstrate that unconscious processing is possible with the selected stimuli.

### 1.3.2 Transcranial magnetic stimulation of V1

The causal role of a targeted brain region in a task can be studied using TMS. A transcranial magnetic stimulator comprises a stimulator unit that stores and delivers the electric current and a coil that is placed over the targeted brain area. When electric current is conducted through the coil, a magnetic field is generated. This magnetic field easily passes through the skull and induces electric current in the brain (Steven & Pascual-Leone, 2006). The induced electric current is the strongest in the brain area that is directly under the coil, but the nearby areas are also affected, albeit to a lesser degree (Jahanshahi & Rothwell, 2000). How focal the stimulation is depends on the shape of the stimulation coil. The coil that is most frequently used in early TMS studies is a circular coil, which has no focal point. The induced electric current is the same along the coil. A circular coil is typically used when the goal is to stimulate a relative large area, a few square centimeters, in the brain. When more focal stimulation is needed, the figure-of-eight (also known as a butterfly coil) is the correct choice. The strongest electric field in the figure-of-eight coil is induced in the few-millimeter focal point that is in the middle of the coil, where the two circular coil sections intersect. The figure-of-eight coil has a clear advantage over the circular coil: the ability to selectively stimulate a very small area in the brain.

TMS of the early visual cortex (V1 and surrounding areas) can suppress the conscious perception of the stimulus (Amassian et al., 1988; de Graaf, Koivisto, Jacobs, & Sack, 2014). The classical (referring to the early findings of Amassian et al. (1988)) and most systematic suppressive effect is found when TMS is applied around 100 ms after the stimulus onset; however, there is a wide range of occipital TMS masking studies reporting suppressive effects from 30 ms before the stimulus onset to 200 ms after the stimulus onset (de Graaf et al., 2014). These findings index the same that lesion studies have found earlier: V1 plays an important role in conscious visual processing.

## 1.4 TMS-induced blindsight

TMS-induced blindsight refers to a phenomenon in which TMS of V1 suppresses conscious perception but the participant has some residual visual capacities, just like

the blindsight patients. After the ability to interfere with V1 activity using TMS was found, it was a natural step to try to replicate blindsight (V1-independent unconscious processing) in neurologically healthy observers. For the most part, studies have not found V1-independent unconscious processing in the neurologically healthy using various stimulus types, but some studies report TMS-induced blindsight. Because our hypotheses are largely based on these limited number of studies (besides those on blindsight), a detailed overview of the studies is presented here. First, I will present the studies that have found unconscious processing when the conscious perception of the stimulus was suppressed using TMS of V1. Next, I will present the studies that have concluded that both conscious and unconscious processing depend on V1.

Ro, Shelton, Lee, and Chang (2004) were the first to examine TMS-induced blindsight. They found that TMS-suppressed unconscious foveal distractor stimulus affected the response times in a task, wherein participants indicated whether the target stimulus was presented to the left or right visual field. Interestingly, the distractor effect was found only when the task was to perform a saccadic eye movement toward the stimulus and not when the task was performed by pressing a button. Ro (2008) later found that using a similar distractor paradigm, it was possible to affect reaction times in a reaching task as well. Moreover, Christensen, Kristiansen, Rowe, and Nielsen (2008) conducted an experiment in which the participant was instructed to reach towards a stimulus. They found that the participants corrected their reaching motion toward a stimulus because of an unconscious TMS-suppressed distractor stimulus. These examples are manifestations of *TMS-induced action blindsight*, but TMS-induced blindsight has been found even with more complex stimulus types. V1-independent unconscious processing of location, orientation, and color discrimination has been reported in few studies. Boyer, Harrison, and Ro (2005) found unconscious processing of color and orientation in a forced-choice task. Allen, Sumner, and Chambers (2014) used arrow stimuli pointing either to the right or left and the participants could unconsciously discriminate them even when V1 was stimulated. They found the effect using both S-cone isolating and luminance stimuli. Railo and Koivisto (2012) reported that participants could unconsciously discriminate the location of the TMS-suppressed stimulus that was presented either to the left or right visual field. Besides these more fundamental stimulus types, there are two studies reporting *TMS-induced affective blindsight*, which is the unconscious processing of the emotional content of the TMS-suppressed stimulus. Jolij and Lamme (2005) found that the accuracy in determining the location of an emoticon with non-neutral expression was affected by TMS of V1, but the accuracy was not affected to the same degree when the participants reported the emotional content. Filmer and Monsell (2013) reported that the classification

accuracy of emotional body postures were less affected by TMS of V1 than by similar neutral postures.

Even though the abovementioned studies have found V1-independent unconscious processing, many studies have concluded that both conscious and unconscious processing depend on V1. Some of these studies have concluded that at some SOAs, it is possible to selectively interfere with conscious processing and reveal unconscious processing. This, however, does not constitute as TMS-induced blindsight because unconscious processing was disrupted at some other SOA. Unconscious visual processing in that case depends on V1 but not in same time-windows as conscious processing does. Sack, van der Mark Schuhmann, Schwarzbach, and Goebel (2009) found that when the accuracy of detecting the prime stimulus was lower owing to TMS of V1, the priming effect was also impaired. Koivisto, Mäntylä, and Silvanto (2010) studied the role of V1 in motion processing. They used a set of dots coherently moving either to the right or to the left. TMS of V1 impaired both the forced-choice accuracy and the awareness of the motion. Koivisto, Railo, and Salminen-Vaparanta (2011) studied the processing of the orientation of a bar and pointing arrows and found that both conscious and unconscious processing depended on V1. When the participants reported not being aware of the stimulus, they performed at chance level in the forced-choice task. Koivisto, Henriksson, Revonsuo, and Railo (2012) studied the role of V1 in unconscious priming. The prime was an arrow pointing to the left or to the right, and the target stimulus, which also served as a visual mask, was an arrow-shaped contour. The priming effect was found even though the participants saw only the target arrow. However, TMS of V1 impaired this unconscious priming effect. Persuh and Ro (2013) also studied unconscious priming. They found that unconscious priming depended on V1 at SOAs ranging from 5 to 25 ms and 65 to 125 ms and found unconscious priming at 45 ms SOA. They concluded that unconscious priming depends on V1 in specific temporal phases of processing. Koivisto, Lähteenmäki, Kaasinen, Parkkola, and Railo (2014) reported that TMS of V1 impaired conscious and unconscious shape discrimination. Railo, Salminen-Vaparanta, Henriksson, Revonsuo, and Koivisto (2012) studied the unconscious processing of color using the unconscious priming paradigm. They found that unconscious color priming was impaired by TMS of V1. Railo, Andersson, Kaasinen, Laine, and Koivisto (2014) studied the role of V1 in chromatic RTE. TMS of V1 suppressed the perception of the redundant chromatic stimulus and eliminated RTE.

As mentioned at the beginning of this chapter, the study of TMS-induced blindsight seems to be bipartite. There are research groups that are convinced that unconscious processing does not depend on V1, whereas some think that both conscious and unconscious processing depend on V1. The studies that have found unconscious processing have either used two-point awareness scales or long TMS

SOAs. A two-point awareness (seen/unseen) scale can lead to false positive findings as participants are not necessary fully unaware of the stimulus when they report not seeing using the two-point scale. Long TMS SOAs, on the contrary, might allow the feedforward sweep to pass, disrupting only recurrent activation and conscious processing. Therefore, the unconscious processing that has been found in these studies would not be independent of V1, but the processing can be explained by the early feedforward activation from V1. The studies that have concluded that unconscious processing depends on V1 have their limitations. As seen with blindsight patients, the stimulus and task characteristics play a critical role in unconscious processing without V1. Thus, one should examine multiple visual processes with different stimuli before concluding that unconscious processing in general depends on V1.

## 1.5 Aims

The main aim of the present thesis was to examine whether unconscious processing measured as RTE is possible without the intact functioning of V1 (i.e., whether the information carried via the subcortical pathways is sufficient for unconscious processing). The secondary aim was to investigate whether unconscious processing would depend on V1 in a specific time-window whereas conscious processing could be suppressed with multiple SOAs. The reasoning behind this is that because the feedforward sweep could produce unconscious processing, TMS applied at long SOAs could selectively disrupt conscious processing while allowing unconscious processing to remain unaffected. In Studies I, II, and III, we used different stimuli types to measure different visual processes and to unveil the residual visual capacities after TMS was used to interfere with the functioning of V1. The stimuli were achromatic (Study I), motion (Study II), and chromatic (Study III). These stimulus types were selected because their processing reflects the residual capacities found in blindsight patients.

# 2 Overview of the studies

The studies were designed to explore "TMS-induced action blindsight" using three different stimuli types and therefore examine three different visual processes. We wanted to ensure that the differences between the unconscious processing of the stimulus types were not attributable to the experiments themselves. Therefore, we used similar paradigm in all three studies. Figure 3 schematizes the experimental trial used in the experiments. All studies employed RTE as a measure of unconscious processing. In other words, unconscious processing was operationalized as the speeding up of the reaction times in the trials wherein an unconscious redundant target was presented. The participants were instructed to press a gamepad button

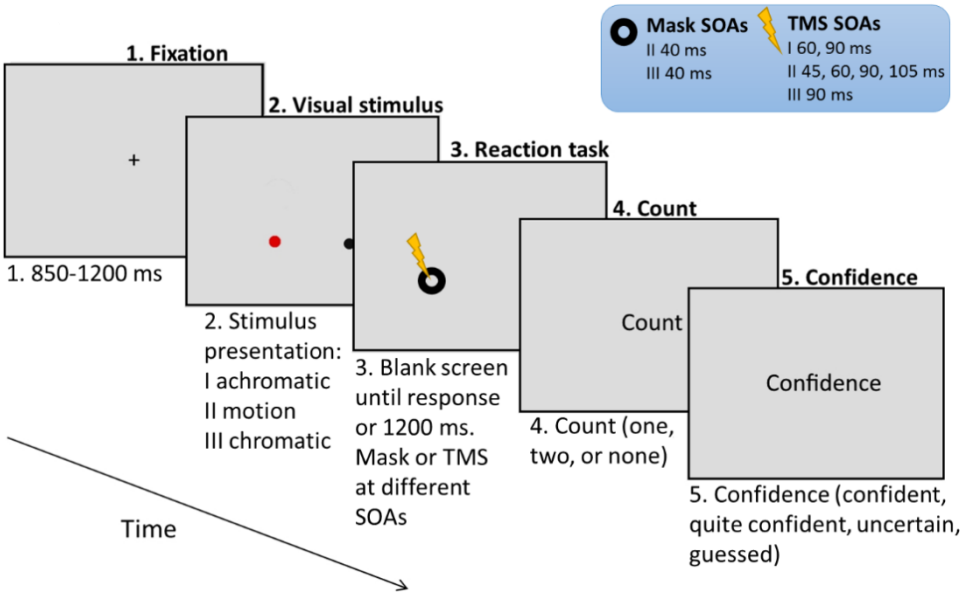


*Figure 3.* Schematic of an experimental trial used in all three studies. The stimuli and the method to manipulate conscious vision varied across studies. Roman numerals represent the studies in order of publication.

immediately when they saw an achromatic stimulus (Study I), a motion (Study II), or a chromatic stimulus (Study III). After the reaction task, they were asked how many stimuli (Studies I and II) or chromatic stimuli (Study III) they saw and how confident they were about that number response. Across studies, we used TMS of V1 to suppress the conscious perception of one of the stimuli. In Studies II and III, we also conducted a control experiment in which conscious perception was suppressed using metacontrast masking.

Some of the effects of TMS are not related to neuronal modulation and need to be controlled. These effects are loud clicking noise and tactile feedback on the scalp. These additional response cues can influence the behavior of the participant; therefore, we needed an active control condition. Thus, we had to include trials in which participants were presented with one stimulus and received TMS that did not affect their conscious perception. Typically, vertex stimulation has been used as a control condition; however, with vertex simulation, the participant can easily identify the experimental and control conditions. In our experiments, TMS was always targeted to the visual cortex. This allowed us to include the experimental (two stimuli, one of them unconscious) and control trials (one conscious stimulus) within the same blocks. In Studies I and II, we were able to include conscious baseline (two conscious stimuli) trials within the same block as the experimental and control trials because the stimuli were sometimes presented to the unaffected upper visual field. Because this option was unavailable in Study III (see section 2.3, Study III), in which

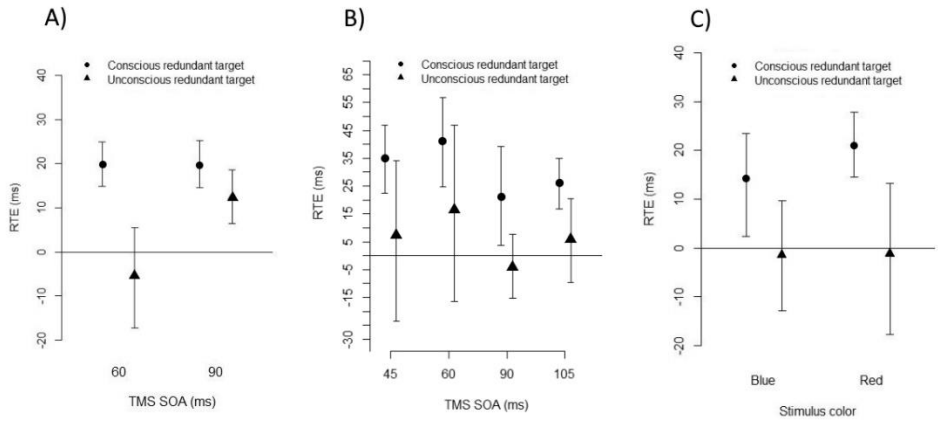


*Figure 4. Main findings of the studies. Dots represent the effect of the conscious redundant stimulus, and triangles represent the effect of an unconscious redundant target on reaction times (i.e., RTE). Error bars represent bootstrapped 95% confidence intervals. A) In Study I, we found RTE in conscious baseline conditions but unconscious conditions only at 90-ms TMS SOA. B) In Study II, we found conscious RTE with motion stimuli at all SOAs but did not observe any unconscious RTE. C) In Study III, we found conscious RTE with both colors but did not observe unconscious RTE.*

we always had bilateral stimuli in the lower visual field, conscious baseline RTE was acquired while participants received TMS that affected their upper visual field.

In all studies, the reaction time data was analyzed using single-trial-based mixed-effects modeling instead of the more traditional ANOVAs that are based on aggregated data. In mixed-effects models, some variables are fixed, that is, the effect is a fixed constant across the data. Besides the fixed factors, random factors can be added, which allow the slope or the intercept of the model to vary between cases. In our studies, random factors were used to control participant-wise variation in reaction times. I will now present an overview of these studies in the order of their publication date. The main findings of the three studies are presented in Figure 4.

## 2.1 Study I

Hurme, M., Koivisto, M., Revonsuo, A., & Railo, H. (2017). Early processing in primary visual cortex is necessary for conscious and unconscious vision while late processing is necessary only for conscious vision in neurologically healthy humans. *NeuroImage, 150*, 230-238.

In Study I, we examined whether the simplest form of unconscious processing, the processing of an achromatic stimulus appearance, could be found without the contribution of V1. Tamietto et al. (2010) found unconscious RTE using achromatic stimuli in a blindsight patient. In light of this, we designed an experiment to study unconscious RTE caused by achromatic stimuli in neurologically healthy people. We used TMS of V1 to suppress conscious perception of the redundant stimulus. We wanted to also take into account Lamme's (2010) theory explaining unconscious processing. We assumed that the classical TMS suppressing effect around 100 ms SOA might actually have two separate processes within it: the feedforward activation from V1 and early recurrent activation. The 60 ms SOA was selected to represent the feedforward activation because that is approximately the time required for the visual input from the retina to reach V1 (Wilson, Babb, Halgren, & Crandall, 1983). The 90 ms SOA was selected to represent early recurrent activation because it has been effective in suppressing conscious vision (de Graaf et al., 2014) and recurrent activation begins rapidly after the feedforward activation (Boehler, Schoenfeld, Heinze, & Hopf, 2008).

In Study I, TMS at both 60 and 90 ms SOAs was capable of suppressing conscious perception of the redundant stimulus. However, we found unconscious RTE only at 90 ms SOA and not at 60 ms SOA. Our mixed-effect model showed that the unconscious redundant target did not speed up reaction times across the SOAs but at 90 ms SOA, the reaction times were on average approximately 9 ms faster than those

in the single-stimulus condition. This indicates that conscious and unconscious processing depend on the feedforward activity in V1, whereas TMS at 90ms selectively interferes with conscious processing. The results of Study I indicated that the subcortical tracts are not sufficient for unconscious processing in the neurologically healthy even when very simple stimuli were used. Unconscious RTE depended on the feedforward activity in V1. We concluded that because the simple stimuli could not produce unconscious processing, it is unlikely that more complex features such as shape or orientation could be processed unconsciously without the contribution of V1.

## 2.2 Study II

Hurme, M., Koivisto, M., Revonsuo, A., & Railo, H. (2019). V1 activity during feedforward and early feedback processing is necessary for both conscious and unconscious motion perception. *NeuroImage, 185*, 313-321.

The motivation for Study II was two-fold. First, unconscious motion processing is a phenomenon commonly found in blindsight patients and is therefore a promising way to demonstrate V1-independent unconscious processing. Motion could be unconsciously processed in V5 that receives direct inputs form subcortical structures (Ffytce & Zeki, 1998). Second, we wanted to revisit our finding in Study I regarding the dissociation between feedforward and early recurrent activations in conscious and unconscious processing and test whether this also applies to the unconscious processing of motion. In Study II, TMS was applied at four different SOAs. Two of the SOAs (45 and 60 ms) targeted feedforward activation. We again estimated that the 60 ms SOA would most likely reflect the feedforward activation, but we also included 45 ms SOA to ensure that we could suppress any earlier signals in V1. Recurrent activation (90 and 105 ms SOAs) was targeted again with 90 ms SOA, but 105 ms SOA was also added to interfere with slightly later recurrent activation and to minimize the effect on feedforward signals. The task in Study II was to respond as fast as possible when motion stimuli were presented but to not respond when stationary stimuli were presented.

Perception of the redundant motion stimulus was suppressed at all SOAs. When conscious perception of the redundant motion stimulus was suppressed, RTE was also absent, suggesting that both feedforward and recurrent time-window activations are needed for the unconscious processing of motion. However, we were uncertain whether our experimental paradigm was capable of revealing unconscious processing at all; therefore, we measured unconscious RTE when the redundant target was suppressed using a metacontrast mask. Masked unconscious redundant

target produced RTE. The mixed-effect model showed 18 ms faster reaction times when unconscious masked redundant target was presented compared with the trials in which only one stimulus was presented.

## 2.3 Study III

Hurme, M., Koivisto, M., Henriksson, L., & Railo, H. (in press). Neuronavigated TMS of early visual cortex eliminates unconscious processing of chromatic stimuli, *Neuropsychologia, 136,* 107266

In Study III, we examined the unconscious processing of chromatic information. The study was designed to fix the methodological issues in the study by Railo et al. (2014) and to revisit the idea that there might be differences in the unconscious processing of different wavelength stimuli. Railo et al. (2014) used rapidly flickering luminance masking to ensure that the participants could only rely on chromatic information in the RTE task. The participants were presented with a strong attention-capturing mask and then a weak TMS-suppressed chromatic stimulus. We assumed that the luminance mask could have interfered with the participants' ability to unconsciously process the chromatic targets. It is impossible to produce a long-lasting, unconscious luminance mask with TMS because the strength of TMS suppression is limited. The assumption that unconscious processing in general was not possible because of luminance masking is impossible to verify because luminance-masked stimulus cannot be suppressed using metacontrast masking. With this limitation in mind, we changed the paradigm in Study III to make the luminance mask unnecessary. Unlike in the traditional RTE paradigm, in Study III, the participants were always presented with bilateral luminance-matched stimuli, but they were instructed to respond only if they see a chromatic stimulus. We compared two stimulus colors: short wavelength (blue) and long wavelength (red). The two stimulus colors were selected to test whether retinotectal tract (pathway via SC) could explain unconscious processing. This comparison of the two colors as a way to unveil collicular contribution was based on the assumption that the retinotectal tract cannot transmit the signals elicited by short wavelength stimuli (Sumner et al., 2002). To control non-neuronal effects, we compared the trials in which two chromatic stimuli were presented, one of them suppressed, with the trials where one chromatic stimulus was presented and the perception of one achromatic stimulus was disturbed. The task was to respond as fast as possible when the participant saw color. We used two methods to suppress the conscious perception of the redundant chromatic stimulus in two different experiments: metacontrast masking and TMS of V1. In Experiment 1 of Study III, the redundant chromatic stimulus was suppressed with a metacontrast mask that was

presented 40 ms after stimulus onset. In Experiment 2 of Study III, the redundant stimulus was suppressed by TMS of V1 at 90 ms SOA.

In Experiment 1, we found unconscious RTE in both stimulus colors. Therefore, metacontrast masking selectively interfered with the conscious perception of the redundant stimulus and unconscious processing was observed. In Experiment 2, the TMS of V1 eliminated both conscious and unconscious processing of the chromatic stimulus. In both experiments, we found no dissociation between short and long wavelength colors. This supports our conclusion that the retinotectal tract is not sufficient for the unconscious processing of visual stimuli in intact brain.

# 3 Discussion

In this thesis, I studied the role of V1 in unconscious visual processing in neurologically healthy observers. In this chapter, I will discuss the findings of the three studies presented in this thesis and how they relate to the previous studies on this topic. I will provide also my view on the future directions for research on unconscious visual processing and TMS-induced blindsight as well as some ideas for blindsight experiments.

## 3.1 Unconscious visual processing depends on V1

The main finding in the three presented studies is that the unconscious processing of visual stimuli depends on V1. We further investigated the classical TMS suppression "dip" around 100 ms after stimulus onset. We found dissociation between early and late V1 processing only in Study I. In Study I, early TMS at 60 ms SOA interfered with conscious and unconscious processing but late TMS at 90 ms SOA only interfered with conscious processing, as revealed by unconscious RTE. In Study II, both early (45 and 60 ms) and late (90 and 105 ms) TMS interfered with both the conscious and unconscious processing of motion. In Study III, wherein we investigated the unconscious processing of chromatic stimuli, we used only late TMS (90 ms). The SOA was selected because it had been the most effective in suppressing conscious processing in Studies I and II. TMS at 90 ms SOA interfered with both conscious and unconscious processing of chromatic stimuli.

Why unconscious processing was found when late processing was suppressed using TMS of V1 in Study I but not in Studies II and III? In Study I, we used the simplest stimulus and task possible. The stimulus was an achromatic dot and the task was to press a button when the stimulus appeared. In Studies II and III, we used a modified version of the RTE task that included inhibition to press the button when task-irrelevant stimulus appeared. In Study II, participants responded only to motion stimuli but were instructed to not press the button when stationary stimulus was presented. In Study III, participants were always presented with two luminance-matched stimuli but were instructed to press the button only when they saw

chromatic stimulus. The tasks used in the studies, despite all being RTE tasks, were therefore quite different from each other. Unconscious processing of a simple luminance stimulus appearance seems to be possible without late processing in V1 whereas unconscious processing of motion and chromaticity depends on V1 in both early and late time-windows.

## 3.2 What explains blindsight?

Based on our studies, I conclude that unconscious visual processing depends on V1 in the neurologically healthy, but blindsight patients can sometimes process visual stimuli without V1. Blindsight seems to be the solid evidence that unconscious processing does not rely on V1. However, it is important to note that not all patients with V1 lesion show blindsight; in fact, it is a rather rare phenomenon (Marzi et al., 1986). So, the question is why these rare cases are able to process visual stimuli despite V1 lesion when other patients are not. There are three major explanations for this dissociation. First, blindsight can be explained by neural plasticity after lesion. New connections form and existing connections change their functions in the patients' brain after V1 lesion (Ajina et al., 2015; Leh et al., 2006; Mikellidou et al., 2017; Payne & Lomber, 2001). Second, the patients lack geniculo-striate input and therefore might become more sensitive to V1-bypassing signals. Because the only input from the blind field comes from the subcortical pathways, such as the retinotectal tract, the blindsight patients have *learned* to attend to this information and to make the most of the residual visual signals. Third, TMS of V1 might have disturbed the activation of subsequent visual areas (V2 or even V3) and those areas might be involved in blindsight. However, most of the studies that explicitly express the extrastriate areas that are assumed to be part of the pathways behind blindsight do not mention V2 or V3. The most common hypothesis is that the connections go from the LGN (Ajina & Bridge, 2018, 2019; Ajina et al., 2015) or SC (Barbur, Watson, Frackowiak, & Zeki, 1993; Tamietto et al., 2010) to V5, an area that is not affected by the TMS of early visual cortex.

In my view, the findings of blindsight studies do not contradict my conclusions. If unconscious processing was truly V1 independent, we should find blindsight in nearly all patients with specific V1 lesion. However, this is not the case. Blindsight has been a great inspiration to study V1-independent unconscious processing; however, it is likely that blindsight patients' brains work very differently in unconscious processing than neurologically heathy participants' brains. I would assume that the processing is more similar between neurologically healthy participants and patients without blindsight than between neurologically healthy participants and blindsight patients.

## 3.3 Explaining the studies that have found TMS-induced blindsight

There are the studies that have found unconscious processing when the stimulus was suppressed using TMS of V1. How can this seemingly contradicting evidence be interpreted? The one clear difference between these studies and the three studies presented in this thesis is the definition of unconscious processing. Ro and his lab (Koenig & Ro, 2018; Persuh & Ro, 2013; Ro, 2008; Ro et al., 2004) use a two-point scale (saw/did not see) for visual awareness and their awareness question is often ambiguous, for example, "did you see the line" when the task is to report the orientation of the line. Participants can interpret the question as "did you see the line well enough that you can tell the orientation quite confidently" or "did you see anything at all" or anything between those options. In Boyer, Harrison, and Ro's (2005) study, the participants were even explicitly asked whether they saw the orientation of the line; thus, the participant might have some kind of visual perception but they could not identify the task-relevant features. The suppression was also incomplete in the studies that found *affective blindsight* (Filmer & Monsell, 2013; Jolij & Lamme, 2005). The studies interfered only with the task-relevant features of the stimuli, but the participants were not unaware of the stimuli. The studies that have two-point awareness rating typically overestimate the number of unconscious trials, if the participants have conservative response criterion in the seen-unseen evaluation (Lloyd, Abrahamyan, & Harris, 2013). In our Studies I and II, we suppressed the perception of the stimulus and denoted only the trials in which participants were confident that no stimulus was presented as "unconscious." In Study III, we asked participants to report only the number of chromatic stimuli; therefore, we cannot be sure if they saw something in the suppressed visual field but just not the chromatic stimulus. Most likely, the perception of the entire stimulus was suppressed in Study III as well and even if there was some residual vision, the participants were certain that there was no redundant *chromatic* stimulus. One might say that we and Ro's lab are measuring two different things. They are measuring something that is more like TMS-induced Type 2 blindsight and we are measuring TMS-induced Type 1 blindsight. Both camps seem pretty confident that their approach is the correct one, and there is likely some truth in both approaches. This discussion reminds me about Tim Minchin's (2013) speech at the graduation ceremony of the University of Western Australia: "We tend to generate false dichotomies and then try to argue one point using two entirely different sets of assumptions. Like two tennis players trying to win a match by hitting beautifully executed shots from either end of separate tennis courts." Of course, I argue that our approach better reflects the phenomenon. In my opinion, you are not *blind* as in

blindsight if you have a visual perception but just cannot detect the task-relevant feature.

Another way of misinterpreting unconscious visual processing to be independent of V1 contribution is to make the task so difficult that even baseline (without TMS) detection probability accuracy is low. That is the case in Allen, Sumner, and Chamber's (2014) study, wherein they concluded that V1 interferes with conscious perception but not with unconscious processing. Their task was calibrated such that in baseline, the participants detected the stimulus in half of the trials; therefore, there were many unconscious trials even without TMS of V1. Accuracy slightly decreased when V1 was stimulated, but it had little effect on unconscious processing, which they interpreted as TMS-induced blindsight. The problem here is that TMS of V1 did not cause suppression in most of the trials; in fact, it might be that TMS did not have any effect in most of the trials. Most of their trials therefore likely reflect near-threshold vision rather than TMS-induced blindsight.

## 3.4 Critical remarks

Even though our studies are methodologically sound and the results support the conclusions, some aspects might affect the generalizability of the conclusions. A common fact across the studies is the variance in the strength of TMS suppression. There were participants whose conscious perception was not much disturbed by TMS of V1. Some of this can be explained by anatomical differences: if V1 is located further away from the skull, stimulating it is more difficult. More interestingly, the participants have different SOAs in which the suppression is most effective. Some participants' conscious perception can be easily suppressed using 60 ms SOA but not very effectively with 90 ms SOA and vice versa. There could be individual differences in the timing of the feedforward sweep; therefore, the timing of TMS SOAs may be off. This issue can be resolved by obtaining the event-related potentials (ERP) of the visual stimulus and determining the feedforward time-window based on the earliest signals that reach the visual cortex. If the timing of the feedforward sweep is not the explaining factor, then the question is why feedforward TMS suppresses some participants' perception more effectively than feedback TMS and vice versa for other participants' perception.

In Study I, my greatest critique is that the results are at odds with the assumption that feedforward activation was disturbed at 60 ms SOA: overall, the strength of the suppression of conscious vision was not as high at 60 ms SOA as it was at 90 ms SOA. If the TMS of V1 at 60 ms SOA suppresses the feedforward sweep, we should observe more effective suppression at 60 ms SOA. Unconscious RTE varied

considerably between participants at 60 ms SOA whereas at 90 ms SOA, we observed consistent positive unconscious RTE across almost all participants.

Study II was quite challenging to execute because of technical limitations. The suppressive effect of TMS is brief; therefore, the stimulus duration should be minimized. The visual field that TMS of V1 using figure-of-eight coil disrupts is also spatially quite small; thus, the stimulus must be small and its motion is limited. In blindsight studies, the stimuli can be large and the motion can be extensive because the patients have larger scotomas in their visual field. We could not find the unconscious processing of motion without V1 probably owing to the small size or slow speed of the stimuli, whereas with larger stimuli or faster motion, unconscious processing might be found.

In Study III, the timing of TMS could have played a critical role. The processing of the color might take longer time than processing of achromatic stimuli in V1 and therefore we could have actually suppressed feedforward information at 90 ms SOA. We could have included longer SOAs to test this possibility. However, the study demonstrated that the unconscious processing of color is not independent of V1. At least some processing phase in V1 is needed for the unconscious processing of color.

## 3.5 Conclusions and future directions

Based on our findings and previous studies, I argue that unconscious visual processing depends on V1 in the neurologically healthy. By unconscious, I mean the stimuli that are completely suppressed by TMS of V1. In cases wherein the participant sees something but not the task-relevant features of the stimulus, unconscious processing might occur. But in these cases, a stronger TMS pulse could have eliminated the conscious perception of the whole stimulus and also eliminated unconscious processing. Therefore, the conclusion that unconscious processing does not depend on V1 is misleading. There might be some features of conscious perception that can be suppressed by TMS of V1 whilst unconscious processing is left intact. However, this does not mean that unconscious processing does not depend on V1, but instead it implies that conscious processing is easier to suppress than unconscious processing. In most cases, V1 contributes to unconscious processing in feedforward and early recurrent time-windows, but the results of Study I showed that very elementary stimuli can be unconsciously processed if the feedforward activation from V1 is not disturbed.

In future studies, a few areas in this field need further investigation. One difference between our studies and those by Ro's lab (they have reported TMS-induced blindsight) that might be worth further investigation is the role of the stimulus location in the visual field. While Ro's lab has used mostly foveal stimuli,

we have been studying peripheral vision. It would be interesting to study V1-independent unconscious processing with stimuli presented into fovea, in contrast to peripheral vision, using a four-point perceptual awareness scale. In other words, the TMS-induced blindsight studies would be replicated such that participants can choose awareness ratings that better reflect their subjective experience. Simultaneously, whether the difference is in fact attributable to the location of the stimulus in the visual field would be tested. Another possible future direction is to concentrate on the tasks that are even more action-based or include emotional content. In our studies, we have used speed response to reflect action to stimulus, but more direct and concrete methods are available. Pointing, reaching, and touching actions as well as saccadic eye movements could be used to indicate the location of the stimulus. Perhaps these visuomotor systems are even more automatic and can reveal unconscious processing more easily than our paradigm. Finally, I would suggest that instead of single pulses, studies should employ burst TMS, which includes a few consecutive pulses within a certain interval. Using burst, it would be possible to disturb activation in the brain for a longer interval and therefore interfere with both feedforward and feedback activity in a single trial. This would be a more analogous situation to blindsight because all the processing in V1 would be interfered with.

# Abbreviations

| | |
|---|---|
| fMRI | Functional magnetic resonance imaging |
| GNW | Global neuronal workspace model |
| LGN | Lateral geniculate nucleus |
| RTE | Redundant target effect |
| SC | Superior colliculus |
| TMS | Transcranial magnetic stimulation |
| V1 | Primary visual cortex |

# List of references


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