

Visual masking & schizophrenia

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ABSTRACT

Visual masking is a frequently used tool in schizophrenia research. Visual masking has a very high sensitivity and specificity and masking paradigms have been proven to be endophenotypes. Whereas masking is a powerful technique to study schizophrenia, the underlying mechanisms are discussed controversially. For example, for more than 25 years, masking deficits of schizophrenia patients were mainly attributed to a deficient magno-cellular system (M-system). Here, we show that there is very little evidence that masking deficits are magno-cellular deficits. We will discuss the magno-cellular and other approaches in detail and highlight their pros and cons.

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1. Introduction

The obvious symptoms of schizophrenia are personality and thought disorders. However, already Kraepelin (1893) noted that visual information processing is strongly deteriorated in schizophrenia too (called dementia praecox). For this reason, sensory deficits were often even proposed to be the primary causes of schizophrenia (e.g., Braff, 1981; McGhie and Chapman, 1961; Saccuzzo and Braff, 1981, 1986; Saccuzzo et al., 1974; Schwartz and Winstead, 1982; Slaghuis, 1998; Venables, 1964; Yates, 1966).

One of the most popular paradigms to investigate visual information processing is visual backward masking (monograph: Breitmeyer and Ögmen, 2006). In visual backward masking, a target is followed by a mask which deteriorates the visibility of the target and, hence, performance (Fig. 1A). Bachmann (1994; p. 11) estimated that masking is used as a tool in 14% of all articles in vision research and psychology. Enns and Di Lollo (2000) came to a similar conclusion.

Visual masking is a powerful tool in schizophrenia research. First, schizophrenia patients show clear and reproducible performance deficits compared to healthy controls in all studies on visual masking (except Luber et al., 2007). Second, masking deficits are potential endophenotypes of schizophrenia (e.g. Bredgaard and Glenthøj, 2000; Chkonia et al., 2010; Green and Nuechterlein, 1999; Keri et al., 2001a; Nuechterlein et al., 1994; Rund et al., 1993), i.e., deficits are stable over time (Chkonia et al., 2010; Lee et al., 2008; Rund et al., 1993),

relatively independent of medication (e.g. Butler et al., 1996, 2002; medication improves performance: Brody et al., 1980; Butler et al., 1996; a trend for deterioration: Cadenhead et al., 1997), present in adolescents with psychosis, i.e., right from the beginning of the disease (Holzer et al., 2009; Perez et al., 2012; Rund et al., 1996; Saccuzzo and Schubert, 1981; Ueland et al., 2004; but see Lieb et al., 1996), present in healthy students scoring high on schizotypy (Cappe et al., 2012; Merritt et al., 1986) and, most importantly, present in first order relatives of patients (Chkonia et al., 2010; Green et al., 1997, 2006; Keri et al., 2001a). Indeed, genetic correlates have been reported (Bakanidze et al., 2013; Goghari and Sponheim, 2008). Third, masking deficits are not contaminated by differential aging effects (Green et al., 2003a), unaffected by learning (Rassovsky et al., 2004; Suslow and Arolt, 1998), independent of cognitive deficits such as working memory (Keri et al., 2001b), premorbid IQ, fluid IQ and intellectual decline (Koelkebeck et al., 2005), and personality aspects (Bogren and Bogren, 1999), and only slightly modulated by cognitive/emotional aspects such as reward (Rassovsky et al., 2005b). However, there is a correlation with social perception (Sergi and Green, 2002). Fourth, masking paradigms have a better specificity and sensitivity than cognitive tests such as the CPT (Chkonia et al., 2010).

Masking is sensitive to psychopathology showing strongest masking deficits for negative symptoms patients (Green and Walker, 1984, 1986; Slaghuis, 1998, 2004; Slaghuis and Curran, 1999; Weiner et al., 1990) and chronic schizophrenia patients (Rund, 1993).¹

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¹ However, empirical evidence is mixed, possibly, depending on the method of assessing psychopathology (e.g. Chkonia et al., 2010; Rund et al., 2004).

Here, we will show that masking is a powerful experimental tool for schizophrenia research but explanations about the underlying mechanisms need to be handled with care.

1.1. Definitions

In visual masking, a target is either preceded (forward masking) or followed by a mask (backward masking; Fig. 1A). If a mask does not spatially overlap with the target, it is called a paracontrast (in forward masking) or metacontrast (in backward masking) mask (Fig. 1C). If the mask spatially overlaps with the target, the mask is called a pattern mask (Fig. 1A).

In masking experiments usually the onset of the target versus the mask is varied. This onset difference is called the stimulus-onset-asynchrony (SOA). If strongest masking occurs for a simultaneous presentation of target and mask (SOA=0 ms), masking is said to be of A-type (not shown in Fig. 1). Interestingly, for some target-mask combinations, strongest backward masking occurs for SOAs greater than 0 ms. The non-monotonic masking function is called to be of B-type (Fig. 1B). Often the terms, integration and interruption masking are used synonymously for A- and B-type masking. However, these terms refer to potential mechanisms of masking and will not be used here. Often, instead of SOA, ISI is reported, which is the inter-stimulus interval between the target offset and mask onset.

An important issue in masking research is the energy ratio of target and mask. Stimulus energy is usually defined as stimulus luminance times duration (luminance is sometimes replaced by contrast). Often combinations of a high energy target with a low energy mask yield B-type masking (but see discussion). The spatial frequency of a masking grating (or Gabor) is the number of changes from black to white, usually

determined in cycles per degree. Low spatial frequencies are in the range from 1 to 4 c/deg, high spatial frequencies from 8 to 16 c/deg.

1.2. M- vs. P-system

Physiologically, there are two major pathways in the visual brain, called the magno-cellular (M-) and the parvo-cellular (P-) system. The physiological characteristics of these systems are rather complementary. The M-system is particularly sensitive to high temporal frequencies such as abrupt onsets of stimuli and to low spatial frequencies. The M-system is “color blind”. The P-system is sensitive to high spatial frequencies and color differences but it is less sensitive to high temporal frequencies. The M-system is often assumed to primarily process motion and localization information. The P-system is assumed to be related to the detailed processing of shape and color.

2. Mechanisms and models

2.1. Attention and iconic memory

Visual masking was introduced to schizophrenia research “as a measure of attention” (Saccuzzo et al., 1974). The basic idea is that a visual stimulus is stored in an iconic memory (Neisser, 1967). Targets are vulnerable to masking as long as they are in this buffer. Items are not erased when they are read out by attention into a more stable memory before the “mask arrives”. In schizophrenia patients, this read out process is slower (or otherwise disturbed) and, hence, performance is more strongly deteriorated in visual masking (Braff, 1981; Braff and Saccuzzo, 1981, 1985; Merritt and Balogh, 1984; Patterson et al., 1986; Saccuzzo and Miller, 1977; Saccuzzo and Braff, 1981, 1986; Saccuzzo and Schubert, 1981; Saccuzzo et al., 1974, 1984; Schwartz et al., 1983; for

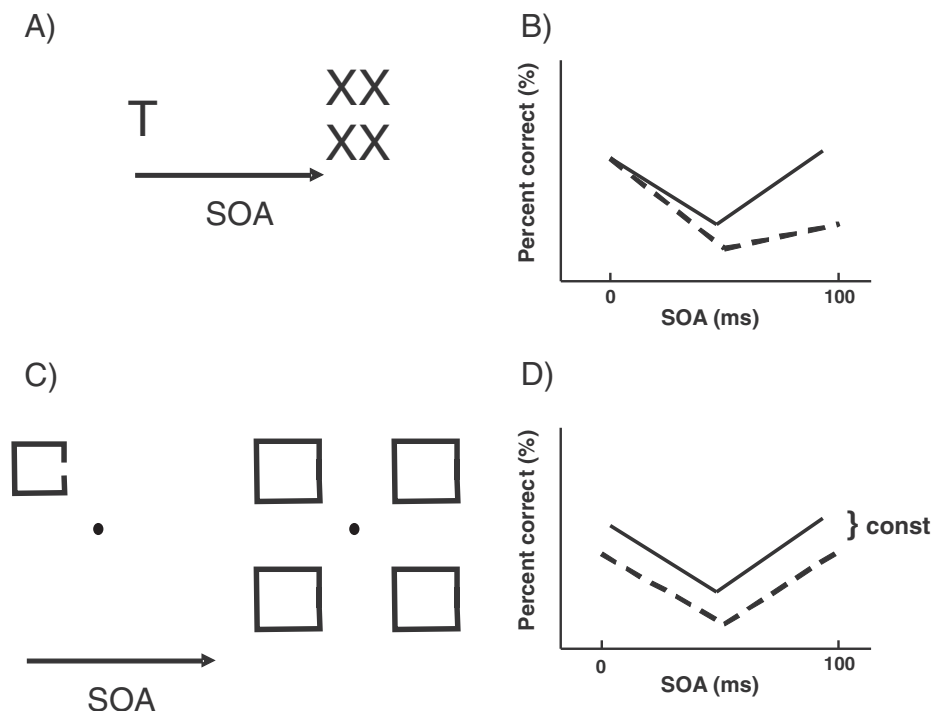


Fig. 1. **A)** Pattern masking. A target letter, e.g., a T, is followed by a mask, e.g., comprised of Xs after a variable SOA (backward masking). Pattern masks often produce A-type masking. **B)** B-type masking. Particularly, when the mask is a metacontrast mask or a pattern mask of weaker energy than the target, B-type masking occurs: good performance occurs for an SOA of 0 ms, i.e., simultaneous presentation of target and mask. Performance for medium SOAs, e.g., 50 ms, is worse than for shorter and longer SOAs. The solid line shows a typical B-type masking function for healthy controls, the dashed line a masking function for schizophrenia patients (e.g., Green et al., 1994a, 1994b). B-type masking is stronger in the patients in accordance with a hypothetical hyper-active M-system of schizophrenia patients. **C)** Localization task. A target square is presented randomly at one out of four positions. After the square, four larger squares follow at all four potential target positions. Observers indicate the position of the target square. In addition, an identification task is performed where observers indicate which of the four sides of the target square contains a gap (here, a gap on the right side is shown). The masking squares are metacontrast masks because they do not spatially overlap with the target square. **D)** With these stimuli, B-type masking occurs in both patients and healthy controls. Performance of controls (solid line) is higher than for patients by a constant factor (dashed line) for all SOAs (e.g. Rassovsky et al., 2004).

a review and criticism: Schuck and Lee, 1989; see also McClure, 2001). Research was concerned for about a decade with this hypothesis until the dual channel approach became the predominant view and research changed gears.

2.2. The dual channel model

To explain B-type masking, Breitmeyer and Ganz (1976) proposed a dual channel model, in which each stimulus is processed in a slower sustained and a faster transient channel (Fig. 2). The transient channel is usually identified with the M-system and the sustained channel is identified with the P-system. B-type masking occurs when the mask signals in the faster M-system catch up with and inhibit the slower P-system signals of the target (Fig. 2; monograph: Breitmeyer and Ögmen, 2006). A-type masking occurs mainly by intra-channel inhibition in both the M- and P-system.

In the next subsection, we show that there are fundamental problems when applying the dual channel model in schizophrenia research.

2.2.1. A mis-understanding

Research on schizophrenia, based on the dual channel model, started with a gross mis-understanding and can be summarized by the following syllogism.

1. The M-system is the crucial component in visual masking.
2. Schizophrenia patients show strong masking deficits.

Hence, the M-system is deficient in the patients.

However, the M-system (in more detail: interactions between the M- and P-system) is only crucial to explain B-type but not A-type masking. A type masking is thought to occur within channel (M–M or P–P interactions). In most studies on masking and schizophrenia, particularly at the beginning of research, A-type masking was found (see also Skottun and Skoyles, 2009). The few B-type studies, conducted later on, found absolutely no evidence for M-system deficits in schizophrenia patients as we show next.

2.2.2. B-type & forward masking

If the M-system is hyper-active, B-type masking should be more pronounced in patients than controls because the M-system inhibits the P-system more strongly. Indeed, Green et al. (1994a), using a low energy pattern mask, showed evidence for this proposal (Fig. 1B; range of SOAs tested: 0–70 ms; evidence from structural equation modeling: Rassovsky et al., 2005a).²

However, there is strong counter-evidence. Rassovsky et al. (2004) investigated 103 schizophrenia patients and 49 controls in a paradigm, where B-type masking occurred. Performance of the patients was deteriorated by almost the same amount for all SOAs (Fig. 1D; see also Butler et al., 2002, Fig. 3A; Green et al., 2003a).³

This study also investigated forward masking and found that patients performance was deteriorated about the same factor as for B-type masking (Fig. 1D; see also Green et al., 2003a). Forward masking deficits cannot be explained by a deficient M-system since the M-signal related to the mask cannot interfere with both the M- and P-signals elicited by the target since the mask precedes the target. Very similar effects were found in Brittain et al. (2010). Skottun and Skoyles (2009) found that only 11 out of 67 studies on masking and schizophrenia have

² An extended version of the dual channel model (Purushothaman et al., 2000) predicts that M–P-systems interactions can produce, under certain conditions, oscillating masking functions exceeding the simple B-type masking function. Interestingly, whereas controls showed oscillating masking functions fluctuating in the γ range, patients did not (Green et al., 1999, 2003b). This result is in agreement with corresponding EEG recordings showing reduced γ activity in the EEG of patients compared to controls (Green et al., 2003b; Wynn et al., 2005).

³ Only the schizophrenia studies by Rassovsky et al. (2004, 2005a, 2005b) and Green et al. (2011a, 2011b) used metacontrast masks (Merritt et al., 1986, for schizotypal observers).

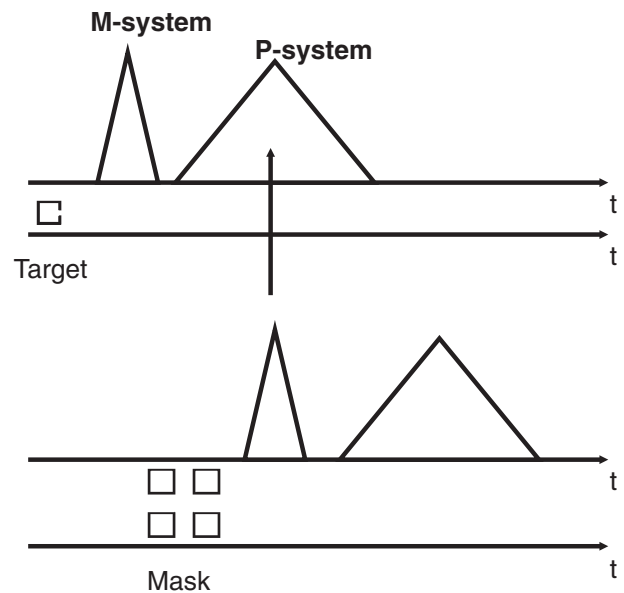


Fig. 2. Dual channel approach. Any stimulus is processed in both the fast M-system, sensitive to location and motion information, and in the slower P-system, sensitive to detailed form and color information. The target is presented (upper part of figure). First, a fast M-system response is elicited (narrow triangle), followed by the sustained P-system response (broad triangle). If the mask is presented at an intermediate SOA (lower part of figure), e.g., at 50 ms, the M-system response to the mask inhibits the P-system response to the target (dashed arrow). As a consequence, B-type masking occurs. If the M-system is hyper-active in schizophrenia patients, the P-system response is even stronger inhibited. B-type is more pronounced.

investigated forward masking. In the majority of these 11 studies, forward masking deficits were evident in the patients compared to controls.

It should be mentioned that some studies found differences in performance between patients and controls in backward but not in forward masking (Saccuzzo and Braff, 1981; Saccuzzo et al., 1996; Slaghuys and Bakker, 1995; Green et al., 2006, for siblings of patients; critical review: Skottun and Skoyles, 2009). However, the study of Saccuzzo and Braff (1981) must be considered a pilot experiment. It should be mentioned that in the study of Saccuzzo et al. (1996) forward masking in the patients was *selectively* deteriorated only after some data manipulation (subtraction of unmasked performance). In the study of Slaghuys and Bakker (1995), backward masking was only deteriorated in negative symptoms patients.

These results show that there is an overall performance deficit in the patients, independent of SOA. Hence, forward and B-type masking studies provide no evidence for an M-dysfunction deficit. Quite to the contrary, these studies show a main effect of Group (patients vs. controls) but no interaction of group by SOA that should have occurred when the M-system had been deficient. Such a deficit can be caused by many deficits and may not even be related to masking or even visual processing. For example, deficits may come from various types of attentional deficits.

Sometimes, it is proposed that both the M- and the P-systems are deficient (e.g. Brittain et al., 2010; Rassovsky et al., 2004; Slaghuys, 1998, 2004). However, this is not a tenable move because, again, many general deficits may explain the data, such as attentional or cognitive dysfunctions. Hence, to show M-system deficits it needs to be shown that the M-system is deficient and the P-system is intact (and task difficulty is similar).

2.2.3. Modeling

Hence, masking research to date has shown that, if the dual channel approach is true, the M-system would be intact. However, the dual

channel approach is just one model, which is far from being an accepted model of masking (as any other model of masking as well).⁴

For example, two channels are not necessary to explain B-type. It was shown that “one channel” is sufficient to explain B-type masking when recurrent processing is used (Anbar and Anbar, 1982; Bridgeman, 1971, 1978; Francis, 1997, 2000; Hermens et al., 2008; Herzog et al., 2003).

2.3. Stimuli biased towards the M- and P-system

2.3.1. Stimuli biased towards the M- and P-system: localization

The dual channel approach triggered a series of studies to investigate M-system deficits by using stimuli biased towards the M-system.

2.3.1.1. Empirical findings. One approach to isolate the M-system is the use of localization tasks where, for example, a target is presented randomly in *one* out of four possible locations (Fig. 1C). After the target, four metacontrast masks are presented, one at each location. Observers are asked to indicate the target location. Because of the peripheral presentation and the localization task, it is assumed that the task is primarily based on signals of the M-system. In addition to the localization task, an identification task is carried out and observers identify a feature of the target, e.g., gap location within the target square (Fig. 1C). The identification task is assumed to be processed by the P-system. In these experiments, schizophrenia patients are worse in the localization task compared to controls but rather unaffected in the identification task (Cadenhead et al., 1998; Koelkebeck et al., 2005). Similar results were found for the siblings of schizophrenia patients (Green et al., 2006). Because only the localization task is assumed to rely on the M-system, these results are taken as evidence for a hyper-active M-system. However, this argument is in this form not tenable.⁵

2.3.1.2. Shortcomings with the localization approach. First, there is simply no physiological evidence that localization tasks are processed *exclusively* by the M-system. The P-system is likely involved too. Otherwise, humans could never localize a red square on an iso-luminant green background (which people with normal color vision easily can).

Second, most approaches using stimuli *biased* towards the M- or P-system are conceptually flawed. Biased stimuli are not sufficient. When target and/or mask are *only* biased towards the M-system, but do not exclusively trigger it, the remaining P-system deficits can still explain masking deficits (particularly when effect size is small). For valid conclusions, it is necessary that stimuli are exclusively processed within the M-system. It must be shown that the P-system is intact, in addition, to the deficient M-system. Performance deficits, which occur when both M- and P-stimuli are used, can otherwise not be distinguished from non-specific deficits, such as attention deficits, which may affect both systems. We do not know of any study which successfully isolated both systems and showed deficits only related to the M-system.

Third, if target localization were primarily based on M-signals, a hyper-active M-system would yield better performance of patients compared to controls: the M-signals of patients would be faster or stronger than those of the controls and, hence, lead to better

performance. However, just the opposite result was found (e.g. Koelkebeck et al., 2005; Rassovsky et al., 2004; see also Weiss et al., 1992).

2.3.2. Stimuli biased towards the M- and P-system: spatial frequency (SF) and color

Because the M- and P-system are differently sensitive to spatial frequencies, several studies use stimuli with high or low spatial frequencies. Other approaches exploit the fact that the M-, but not the P-system, is color blind and, hence, unable to detect iso-luminant stimuli. Whereas localization approaches usually try to isolate the M-system related to target processing, the SF and color approaches focus on isolating the M-system related to both target and mask processing.

2.3.2.1. Empirical evidence. In a typical experiment, target letters or gratings are presented, followed by a grating mask. The rationale of the experiments is that a low spatial frequency (LSF) grating selectively triggers the transient M-system whereas a high spatial frequency (HSF) grating triggers the sustained P-system. With this set-up, schizotypic college students (Merritt and Balogh, 1989, 1990) showed deteriorated performance with the LSF masking grating but not with the HSF grating. This result is in accordance with a hyper-active M-system in schizophrenia: the LSF mask triggers primarily the M-system which is hyper-active and, hence, inhibits the P-system more strongly. Performance deteriorates more strongly compared to controls (Fig. 2). Slaghuys and Curran (1999) showed that a target grating of 3 c/deg was strongly masked by a LSF grating (1 c/deg) but not by a HSF target grating of 11 c/deg in negative symptoms patients (but not positive symptoms patients).⁶ However, with a very similar set-up, schizophrenia patients (not only negative symptoms patients) showed deteriorated performance for both LSF and HSF masks (Butler et al., 2002).⁷

Schechter et al. (2003) triggered the M- vs. P-system by using high and low contrast isoluminant stimuli. They found stronger masking for patients only when a P-system biased target letter was followed by an M-biased mask (P-M combination). All other combinations of target and mask were not significantly different.^{8,9}

2.3.2.2. Shortcomings with the SF and color approach. First, as outlined in the previous section, “biased stimulus approaches” need to show that the M-system is deficient and the P-system is intact. If performance is deteriorated for both stimulus types, deficits may be caused by many other factors than a dysfunction of both the M- and P-system. None of the above mentioned studies tested for an intact P-system.

Second, recent articles have seriously questioned, on physiological grounds, whether current approaches were successful to isolate the M-system (Skottun and Skoyles, 2007a,b; reply by Keri, 2008).

⁶ We would like to mention that there were obvious, but not statistically significant, performance deficits also in the positive symptoms patients and statistical power was small (sample size: 10 observers in each group).

⁷ In some experiments, particularly when letters are used as targets, patients show masking deficits for ISIs longer than 500 ms (Butler et al., 1996). This is definitely outside the range where the transient M-system can make an impact, and likely outside the range of early visual processing in general (see also Skottun and Skoyles, 2011). Possibly more cognitive aspects are involved as it was also shown in experiments with pupillometry revealing deficient masking of patients with SOAs of up to 317 ms and related to an increase of cognitive load (Granhölm and Verney, 2004).

⁸ However, there was also a trend when both target and mask were biased towards the M-system (M-M combination).

⁹ Bedwell et al. (2003) presented target letters to relatives of schizophrenia patients followed by an X mask on either a red background, assumed to suppress the M-system, or an isoluminant gray background, which does not suppress the M-system. The two backgrounds affected performance differently in siblings of patients but not in controls. However, we like, first, to mention that this effect was significant only for one out of four ISIs. Second, it was not determined whether the white color of the target and mask was isoluminant to the red and gray background. Third, there was no condition showing that unmasked letter identification was comparable across the two populations. Fourth, target durations were not adjusted individually (see also Bedwell et al., 2006).

⁴ It must be mentioned that there is not one dual channel model but various versions (Bachmann, 1994; Ögmen, 1993). Slight variations between models or of parameters even within one model can change the outcome strongly, particularly, when non-linear interactions are involved. Without computer simulations, it is usually almost impossible to know how the intra-channel interactions and the inter-channel interactions influence each other. Interestingly, none of the studies on masking and schizophrenia has used, for example, the RECOD model to test predictions (Ögmen, 1993). Moreover, it is not always clear what is meant by hyper-active. Is the M-system just more noisy, non-specifically increasing the P-system inhibition, or are stimuli, biased towards the M-system, processed more efficiently, as proposed in the localization approach?

⁵ Even though we do not doubt the potential involvement of the M-system in the localization tasks because of the abrupt onset of stimuli.

Moreover, there is also counter-evidence for a deficient M-system in general, be it hyper- or hypo-active. Keri et al. (2000a) selected patients with intact contrast detection as tested with a very low spatial frequency grating of 0.5 c/deg which selectively triggers the M-system. Hence, the M-system was intact. Still, there was a performance deficit in a localization backward masking task, which is thought to be carried out by the M-system (see previous subsection). Based on this result, Keri et al. (2000a) postulated that central mechanisms are deficient rather than the M-system.¹⁰ Herzog et al. (2004) masked a vernier target with a very high spatial frequency grating (12 c/deg) which is rather selective for the P-system. Performance of schizophrenia patients was strongly deteriorated in this task indicating that also a P-system biased mask can strongly deteriorate performance (see also Chkonia et al., 2010; Roinishvili et al., 2008; Schütze et al., 2007).

2.3.3. Stimuli biased towards the M- and P-system: Related non-masking studies

Biased stimulus approaches were also used in studies on early visual processing, not using masking, of which we like to mention only some (review: Butler et al., 2008). For example, studies used checker boards and other stimuli, of which the parameters were proposed to selectively trigger either the M- or the P-system. Only for M-system biased stimuli, schizophrenia patients showed deteriorated performance but not for P-system biased ones (e.g. Butler and Javitt, 2005; Butler et al., 2001, 2007; Lalor et al., 2008). However, some of these studies were strongly criticized because stimuli do not as selectively trigger the M-system as proposed. The controversy is similar as with masking studies (Skottun and Skoyles, 2007a).

In addition, whereas most of the above studies are in favor of a hypo-active M-system, other studies found rather mixed results or even evidence for a hyper-active system. For example, Slaghuys (1998) biased contrast detection towards the M- and P-system by using high and low spatial frequency gratings. Negative symptoms patients performed worse for all frequencies whereas positive symptoms patients performed worse only for higher frequencies. Hence, the negative patients seem to have M- and P-system deficits whereas the positive symptom patients have only a P-system deficit. However, in the masking study of Slaghuys and Curran (1999), the pattern of results was almost the opposite: strong masking occurred only for LSF masks, particularly in the negative symptoms patients, indicating an M-system deficit. Keri and Benedek (2007) even found evidence for a hyper-active M-system for prodromal patients.¹¹ Moreover, it seems, again, that performance is rather constantly reduced in most of the above studies as shown in a recent review article (Skottun and Skoyles, 2007b). Also, Delord et al. (2006) did not find a selective M-system deficit but rather constantly deteriorated performance. In addition, Lalor et al. (2012) found no evidence for a magno-cellular deficit in an EEG study using stimuli biased towards the M-system. They attributed deficits to extrastriatal cortical areas.

Keri et al. (2004) used a biased stimulus approach for *unmasked* vernier stimuli isoluminant to the background and, therefore, invisible to the M-system. In addition, luminance defined verniers, visible to the color-blind M-system, were used.¹² With a sample of 22 patients, significant performance differences between patients and controls were reached only for the M-biased stimuli. For the other stimuli, there was no significant difference. However, the difference became significant with a larger sample in a subsequent study (Keri et al., 2005). Hence, the M-system turned out *not* to be selectively deficient because significant results were reached for both biased stimulus types.

Finally, two studies linked visible persistence to backward masking. Also, here, results are mixed. Slaghuys (2004) found a strong correlation between reduced contrast sensitivity and backward masking (in negative symptoms patients) which he attributed to a prolonged visible persistence. However, Grimsen et al. (2013) found that visible persistence cannot explain backward masking deficits of patients.

2.4. The object substitution model (OSM)

As mentioned, the dual channel model was the most influential model for two decades. In the last decade new models were proposed such as object substitution masking (OSM; e.g., Di Lollo et al., 2000). In OSM, the target is, first, thought to be processed in a feedforward manner. During subsequent reentrant processing, the target signals are replaced by mask signals, which lead to a reduction of target visibility.

Recent studies on schizophrenia proposed that “masking by object substitution is thought to rely solely on re-entrant processing” and hence masking deficits can be caused by deficits of recurrent processing only (Green et al., 2011b). However, this proposal is strongly simplified, reminiscent of the gross mis-understanding of the dual channel approach. For example in OSM, there is a feedforward processing stage and there are many other factors involved, such as attention, which could all be deficient leading to masking deficits.¹³ In addition, the OSM model is just another model of masking and is as heavily debated as all other models (Francis and Hermens, 2002; Pöder, 2013).

2.5. Neuromodulation and attention

We proposed that masking deficits are not genuinely visual deficits, such as M-system deficits, but are an instantiation of a general deficit of neuromodulation (Herzog et al., 2013). Whenever there is a task-relevant fragile element, such as a briefly presented faint target followed by a high luminance mask, the human brain needs to amplify this information, e.g., by neuromodulators or attention. Otherwise the element goes unnoticed, which is the default when the element is of no task relevance. For example, it was shown that attention can increase response of neurons in many visual areas in humans (e.g., Gandhi et al., 1999) and monkeys (e.g., Treue and Maunsell, 1996), in particular, when stimuli are weak. Based on a genetic study, we suggested that the cholinergic nicotinic system may be deficient in schizophrenia (Bakanidze et al., 2013). Our results are in agreement with studies on monkey physiology, showing that acetylcholine release increases responses to weak, low contrast stimuli already at the level of the primary visual cortex (e.g., Disney et al., 2007). These findings are in line with our EEG studies showing that neural responses to a target are strongly diminished even before the mask arrives in schizophrenia patients compared to controls (Plomp et al., 2013; see also Patterson et al., 1987). Interestingly, EEG amplitudes were only slightly different when only a strong mask was presented (Plomp et al., 2013). We attribute the diminished EEG amplitudes related to the target to a diminished enhancement of neural activity. However, these considerations are just speculations and are far from ground truth.

If masking deficits reflect general deficits such as dysfunctions of neuromodulation, then, visual masking would be one of the most sensitive tools to investigate these topics. In addition, results may generalize to other deficits and symptoms of schizophrenia since neuromodulation and attention are crucial in almost all cognitive tasks and for behavior.

It is important to note that we do not attribute masking deficits to slips of attention such as missed stimuli but to a diminished gain of attention and/or neuromodulation. This proposal is in accordance with

¹⁰ However, effects and sample size were rather small; only 12 patients and controls each.

¹¹ This is a very interesting study because patients performed consistently better than controls, a very rare finding.

¹² It should be mentioned that the isoluminant blue–yellow stimuli possibly activated not only the P-system but also the koniocellular system, a third visual pathway.

¹³ There are other problems with this study. For example, results could not be replicated in a subsequent study by the same research group (Lee et al., 2014). The authors now concluded that recurrent processing is intact. This claim is as unsubstantiated as their previous one. For example, recurrent processing may not be involved in this experiment. In addition, statistical power was too low to “prove” a null result.

other studies on masking and attention (Lalanne et al., 2012) and studies pointing to attentional dysfunctions (Green et al., 2011a; Rassovsky et al., 2005b; Wynn et al., 2013), or short term memory (Wynn et al., 2006; see also Knight et al., 1985 for poor premorbid schizophrenia patients).

3. Summary

Visual masking is one of the most powerful tools in schizophrenia research with very reproducible results and excellent effect sizes, outperforming many other, for example cognitive, paradigms. Additionally, backward masking is an endophenotype of schizophrenia. However, explanations about the mechanisms of masking must be heeded with caution. For example, most studies over the past 25 years have related masking deficits to a dysfunctional magno-cellular system. Some research groups have proposed that the M-system is hyper-active whereas other groups have favored a hypo-active system. Interestingly, there are no studies, which have tried to resolve this controversy. Moreover, the involvement of the M-system in B-type masking itself is questionable. In particular, many masking studies have shown that performance of patients is deteriorated by roughly the same amount for all SOAs, pointing to an overall deficit rather than a specific M-system deficit.

One of the most important questions, hence, is where in the human brain abnormal functions contribute to the visual deficits. Because of the M-system deficit most studies have focused rather on early visual processing stages but other masking studies have localized masking deficits to higher cortical stages (Del Cul et al., 2006; Delord et al., 2006; Keri et al., 2000; Lalor et al., 2012), such as the LOC (Green et al., 2009; Harvey et al., 2011).

Importantly, masking deficits are neither sufficient nor necessary for schizophrenia. Quite to the contrary, there are patients without masking deficits and healthy controls with masking deficits. Moreover, there are likely many factors causing masking deficits, of which only some are related to schizophrenia. A related question is whether masking deficits are indicative for sub-populations, for example, patients with stronger negative symptoms (Green and Walker, 1984, 1986; Slaghuis, 1998, 2004; Slaghuis and Curran, 1999; Weiner et al., 1990). In addition, the question is whether different types of masks or masking paradigms lead to stronger deficits for specific types of schizophrenia as for example shown by Knight et al. (1985).

We would like to finish with a general comment. Often, it is proposed that the visual system is well understood in both physiological and computational terms, which make tests of visual deficits particularly valuable tools in schizophrenia research (Green et al., 2011a; Sponheim et al., 2013). Whereas it is, indeed, true that the visual system has been studied in much more detail than most other systems, such as audition or cognition, our knowledge of the visual system is still strikingly limited. There is almost no circuit in the visual brain that is fully understood and, potentially, many circuits are waiting to be discovered. Consequently, all models of vision, including masking models, must be considered to be preliminary. The very same is true for cognitive models and for other research areas (we would like to mention that there are also many common but unsubstantiated claims in cognition, such as that the continuous performance test (CPT) is an endophenotype of schizophrenia, see Chkonia et al., 2010). In summary, visual masking is very sensitive and versatile to investigate schizophrenia and is, for example, more and more used to search for the underlying genetic causes (e.g., Bakanidze et al., 2013). In addition, a better understanding of the exact physiological and computational mechanisms of visual masking in the healthy population will lead to new and promising avenues in schizophrenia research.

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