Backward Masking, Information Processing, and Schizophrenia

by John R. Schuck and Richard G. Lee

Abstract

Many researchers have used backward masking to examine information-processing speed in schizophrenic subjects. The validity of this approach rests upon two main assumptions. One is that the mask effectively limits the time a previously presented stimulus is available for processing. The other is that the components of the masking mechanism in schizophrenic subjects are comparable to those in control groups. It is argued that the masking procedures used in these studies fail to meet either assumption. Alternative interpretations of these investigations, however, suggest important hypotheses for further research in our quest to understand information-processing deficits associated with schizophrenia.

Led by the work of Saccuzzo and his colleagues, many researchers have used backward-masking methodologies to examine information-processing speed in patients with schizophrenia. This article calls attention to conceptual and methodological difficulties that raise important questions about the proper interpretation of these studies. Our purpose is not to discourage masking research with schizophrenic subjects nor to disparage the contribution of these investigations, when properly interpreted, to our understanding of perceptual deficits associated with schizophrenia.

A review of some basic technical terms will lay the proper foundation for the critique that follows.

Visual Masking Methodology and Terminology

Figure 1(a) illustrates the kind of masking typically used in schizophrenia research. The target stimulus is a letter. The masking stimulus spatially overlaps the target and is made up of elements with contours that structurally resemble those of the target. This is called masking by structure (Breitmeyer 1984) or pattern masking. In figure 1(b), the target is also a letter, but the mask consists of two adjacent bars that do not spatially overlap the target. This kind of masking is called paracontrast (when the mask temporally precedes the target) or metacontrast (when the mask temporally follows the target). Many other masking stimuli have been used, of course, but are not relevant to the purposes of this article.

Typically, masking performance is represented in one of two ways: (a) by plotting masking functions (accuracy curves) over a specified set of interstimulus intervals (ISIs) or stimulus onset asynchronies (SOAs), or (b) by determining a "critical" ISI. Critical ISIs are typically determined by delaying the onset of a backward mask until the subject reaches some specified accuracy criterion.

Given this summary of basic terms and methodology, we now turn to a review of some studies whose interpretation we consider to be open to question.

Representative Studies

Saccuzzo and Braff (1981) compared the performance of good and poor prognostic schizophrenic subjects with that of depressed, manic, and normal control groups on forward- and backward-masking tasks in...
which high-energy structural masks were used. After equating individual differences in initial input factors using a critical stimulus duration technique, Saccuzzo and Braff (1981) demonstrated that all groups performed equally in forward masking, but the poor prognosis schizophrenic subjects performed significantly more poorly in the backward-masking condition. These researchers argued that the equivalence across groups in forward masking implied that such factors as fatigue, attention, and medication were not contributing to the schizophrenic subject’s poor backward-masking performance. Additionally, since input variables were equated, the backward-masking deficit was attributed to impaired processing speed from icon to short-term memory.

In the same study, Saccuzzo and Braff (1981) also determined critical ISIs in the five groups previously noted. Their results indicated no significant differences in critical ISIs among the normal, manic, and depressed control groups; however, the good and poor prognosis schizophrenic subjects had significantly longer critical ISIs than controls. In another study, Saccuzzo and Miller (1977) reported significantly longer critical ISIs in delusional schizophrenic patients than in a normal control group. Both of these studies were interpreted as supporting the notion that backward-masking performance is a direct reflection of information-processing speed.

To wit:

[the] critical interstimulus interval... provides a measure of the minimum time required to extract a specific amount of information from iconic storage. [Saccuzzo and Miller 1977, p. 262]

Saccuzzo, his colleagues, and others have conducted numerous other backward-masking studies attempting to evaluate speed of information processing in individuals within the schizophrenia spectrum. These studies include Saccuzzo et al. (1974, 1982, 1984), Steronko and Woods (1978), Miller et al. (1979), Brody et al. (1980), Braff and Saccuzzo (1981), Saccuzzo and Schubert (1981), Schwartz et al. (1983), and Green and Walker (1984).

All of these investigators concluded that their schizophrenic or schizotypal subjects were slow information processors. In their review, Nuechterlein and Dawson (1984) pointed out that the usual interpretation of these findings was that schizophrenic subjects were slow in transferring information from sensory (iconic) memory to short-term memory.

In all of the studies cited above, investigators used backward masking by structure to control the time the target stimulus remained available for processing. However, the success of this endeavor rests on two important assumptions. The first was stated quite explicitly by Saccuzzo et al. (1974):

... a high energy pattern mask will effectively limit the amount of time a previously presented stimulus is available for processing, regardless of how or why the mask operates. [p. 513, italics added]

The second assumption underlying these particular studies is that components of the neurophysiological or behavioral mechanisms underlying masking performance in schizophrenic subjects are comparable to those in normal or psychiatric controls. In this sense, backward-masking studies provide unequivocal evidence about information-processing deficits associated with schizophrenia only if the researchers can assume their results are not confounded by group differences in certain key aspects of
the masking processes or mechanisms themselves.

The remainder of this article is devoted to a discussion of the tenability of these two assumptions, as well as some other difficulties inherent in this particular use of backward masking. Eriksen (1980) has spelled out a number of such difficulties, and a summary of his critique is presented below.

**Eriksen’s Critique**

Eriksen (1980) considered two general mechanisms of visual masking: interruption and integration. Kahneman’s (1968) review presents a good traditional summary of these two kinds of masking mechanisms.

Interruption theories share the common assumption that presenting a mask after a target stimulus diverts processing mechanisms and thus disrupts further processing of the target; that is, processing of the target is said to be an all-or-none affair.

On the other hand, integration theories are based upon the well-known fact that two successive visual stimuli, when separated by a short temporal interval, are summed together into a composite or montage that may be likened to the double exposure of a photographic film. In contrast with interruption, masking by integration is not inherently all or none.

For these reasons, Eriksen (1980) argues that only interruption masking is “reasonably compatible with the assumption that a mask, following stimulation, effectively limits the time available for stimulus processing” (p. 89).

Although individual theories may stress interruptive or integrative mechanisms, many theoretical accounts of masking (Spencer and Shuntich 1970; Turvey 1973; Hellige et al. 1979) suggest that backward masking involves both, with integration predominating at very brief SOAs and interruption predominating at longer SOAs. A notable exception is the theory espoused by Felsten and Wasserman (1980). Evidence that many researchers consider a reflection of interruptive mechanisms, Felsten and Wasserman explain as actually reflecting integrative mechanisms at the neuronal level.

Despite this theoretical divergence, Eriksen (1980) argues that neither integrative nor interruptive visual masking is clearly an all-or-none affair. Assume for the moment that at shorter masking intervals integrative mechanisms result in degradation of the target. Obviously, this process can result in varying degrees of degradation. Since the extent of stimulus degradation may or may not be sufficient to stop processing, the assumption that input processing is halted at the onset of the mask is questionable.

A similar argument applies to interruptive mechanisms. Consider the following situation: the researcher is using a backward mask at an interval long enough to suggest that interruption is the major masking mechanism. Can the researcher now make the plausible assumption that masking effectively controls processing time? Not according to Eriksen (1980), who points out that this very same researcher could vary target-to-mask energy ratios to obtain any level of recognition performance desired from chance to 100 percent correct. How can that be explained by interruption theory? One possibility is that the interruption on any one trial is not complete; that is, processing is only partially impaired. Another is that the mask effectively and totally terminated processing but on an unknown proportion of the trials.

In our opinion, Eriksen’s (1980) critique essentially invalidates the first assumption cited in this article: the assumption that a high-energy pattern mask will effectively control processing time. At very brief intervals, a high-energy mask will degrade the target stimulus, but without explicit operations to demonstrate otherwise, it remains unknown whether such degradation is sufficient to stop processing. At longer intervals, interruption occurs. But, once again, without explicit control procedures, it is impossible to decide whether interruption has stopped information processing. Either case presents serious difficulties for the researcher attempting to use backward masking to control processing time.

As we have noted, data showing schizophrenic subjects to be abnormally vulnerable to backward masking have led some authors to conclude that schizophrenic subjects are slow information processors on this task. At a purely descriptive level, that conclusion is, of course, true. But does “slow information processing” offer a viable explanation of the schizophrenic subject’s deficit in backward masking? We believe not.

Eriksen’s (1980) critique clearly suggests a number of equally plausible and testable interpretations of this deficit. For example, Nuechterlein and Dawson (1984) point out that backward masking by structure degrades the target through integration and that schizophrenic subjects may be less able than controls to process the resulting montage when a mask is superimposed upon the target. Another possible explanation is that schizophrenic subjects are less able than controls to remain on task
when the target stimulus is only partially interrupted by the backward mask. This latter interpretation is based on well-established evidence that schizophrenic subjects are more distractible than controls (e.g., McGhie et al. 1965), and the mask may well act as a distractor.

Most of the masking theories cited by Eriksen (1980) suffer from serious limitations. The terms integration and interruption are not theoretically well defined. The theories do not specify with any precision how particular stimulus patterns will affect the components of underlying masking mechanisms. Consequently, the selection of stimulus targets and masks is often quite arbitrary.

Breitmeyer and Ganz (1976) and Breitmeyer (1984) have proposed a very general theory of masking that overcomes many of these limitations. Their model, which is based on a broad range of neuropsychological and psychophysical data, has important implications for the topic of this review.

The Breitmeyer Theory of Masking

Breitmeyer (1984) compiled an extensive review of research relating to the retinal and brain processes involved in masking phenomena and attempted to tie masking performance in humans to underlying neurophysiological processes. A brief examination of this literature is necessary for an adequate understanding of the discussion to follow.

Neural Channels and Visual Masking. Studies of the mammalian visual system have provided electrophysiological support for the existence of two different classes of neural cells in the visual system: transient and sustained (e.g., Enroth-Cugell and Robson 1966). Additionally, psychophysical studies have provided evidence for such channels in humans (e.g., Breitmeyer 1984; Green 1984).

Although transient and sustained channels do not represent a clear-cut dichotomy (Green 1984), the evidence does indicate that transient channels are optimally sensitive to stimuli with low spatial and high temporal frequency. Such stimuli are characterized by rapid motion (Tolhurst 1973), flicker (Kulikowski and Tolhurst 1973), or abrupt stimulus onset and offset (Breitmeyer and Julesz 1975). The transient channels, which have been characterized as an alerting mechanism (Breitmeyer and Ganz 1976), have very short firing latencies, brief durations, and relatively high conduction velocities.

Sustained channels, on the other hand, are most sensitive to stimuli having high spatial and low temporal frequency. These channels respond optimally to slow-moving or stationary stimuli (Kulikowski and Tolhurst 1973). Sustained cells, which are thought to be primarily involved in stimulus resolution, exhibit relatively long response latencies and firing durations as well as relatively slow conduction velocities.

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The top part of figure 2(a) schematically illustrates the fast-latency, brief transient response and the slow-latency, long-duration sustained response to a target. The remaining parts of figure 2 are used later to illustrate the theoretical roles transient and sustained activity play in various kinds of masking.

In addition to having different response latencies and durations, transient and sustained channels also reciprocally inhibit the activity of one another (Singer and Bedworth 1973; Breitmeyer 1978). In light of this research, Breitmeyer and Ganz (1976) and Breitmeyer (1984) have contended that the best way to understand visual masking is by the interactions among these two quite different channels in the visual system, and they have offered an explicit and detailed theoretical account of different visual masking phenomena.

In forward masking by structure, the sustained activity elicited by the forward mask and the sustained activity elicited by the target stimulus are competing for the same neural pathways. This is called intrachannel (sustained-sustained) integration. Intrachannel integration is like the double exposure of a photographic film where one exposure interferes with the clear representation of the other.

In paracontrast masking, however, there is no spatial overlap of target and mask. Thus, there is no competition for common sustained neural pathways (i.e., no “double exposure”). Instead, the sustained activity elicited by the mask interferes with the sustained activity elicited by the target through lateral inhibition. This is called intrachannel inhibition. The double-headed arrow in figure 2(a) indicates intrachannel integration when masking is by structure and lateral intrachannel inhibition when the procedure involves paracontrast. Note that in forward masking, the transient activity elicited by the mask temporally precedes the target and thus plays no masking role.

Theoretically, transient channels do play a crucial role, however, in metaccontrast and backward masking by structure. Remember that transient responses are typically characterized by a brief latency and
a short duration while sustained responses have a longer latency and also a longer duration. This implies that the transient activity of a backward mask can interfere with the sustained activity of the target in neural channels leading from the eye to the brain or in the visual cortex itself. This kind of interference (called transient-on-sustained inhibition) is indicated in figure 2(b) by the single-headed arrow pointing from the mask's transient activity toward the sustained activity of the target.

In summary, Breitmeyer (1984) presents a theoretically explicit picture of the kinds of neurophysiological mechanisms underlying integration and interference masking. Moreover, the model implies that transient activity plays a crucial role in backward masking, that basic masking mechanisms depend on the kind of mask used, and that the spatial frequency and energy level of both the target stimulus and the mask are crucial variables affecting the process.

The foregoing discussion speaks only briefly and superficially to the complexity of the neurophysiological processes theoretically involved...
in masking phenomena. It does, however, highlight the critical importance of understanding the specific nature of the masking deficit observed in schizophrenic subjects. From this theoretical perspective, there is considerable evidence to suggest that crucial components of the masking mechanisms outlined above are not comparable across schizophrenic and non-schizophrenic groups. We consider this comparability problem in detail in a later section.

**Alternative Explanations of Schizophrenic Deficit**

In this section, we consider and evaluate a number of different explanations of the schizophrenic subject’s deficit on backward masking tasks.

**Stimulus Classification Explanations.** Green and Walker (1984) suggested that masking interrupts stimulus classification processes and that the masking deficit is observed in schizophrenic subjects because of their slowness in classifying stimuli. A similar explanation has been offered by Knight (1984), who proposed that the greater interference schizophrenic subjects suffer with pattern masks may reflect their inability to reject the mask elements as irrelevant.

**Stimulus Degradation Explanations.** As mentioned previously, one suggestion offered by Nuechterlein and Dawson (1984) is that schizophrenic subjects are less able than controls to process the degraded montage resulting from the integration of mask and target.

**Metacontrast and Paracontrast Studies.** At the time of Nuechterlein and Dawson’s (1984) review, pattern masking or masking by structure was the only kind of masking that had been used with schizophrenic subjects. Since then, Lee (1985) used targets and masks like those illustrated in figure 1(b) to obtain paracontrast forward-masking functions and metacontrast backward-masking functions in schizophrenic and control groups. The results were comparable to those found with pattern masks. Schizophrenic subjects exhibited a deficit on backward- but not on forward-masking tasks. Merritt et al. (1986) also used paracontrast and metacontrast masking of target letters with schizotypal college students; their results were comparable to Lee’s (1985).

These results are important since paracontrast and metacontrast masks interfere with the target through intrachannel or interchannel inhibition rather than integration. Hence, a deficit in processing the integrated montage of target and mask does not account for these data.

Moreover, in masking by structure, the contours of the mask structurally resemble those of the target, while in paracontrast and metacontrast, the elements of the mask are distinctively different from those of the target (see figure 1(a, b)). Thus, the results obtained by Lee (1985) and Merritt et al. (1986) are not easily explained by deficits in stimulus classification processes.

**Defective Sustained Mechanisms.** Using a two-pulse temporal resolution task, Schwartz and Winstead (1982) found evidence for longer iconic persistence in schizophrenic subjects than in controls. The researchers concluded that abnormally long visible persistence may prevent new information from efficiently superseding the old, leading to congestion of visual processing.

Subsequently, Schwartz et al. (1983) suggested that schizophrenic subjects’ backward-masking deficit can be explained by abnormally long visible persistence of target signals (i.e., abnormally persistent activity in sustained channels).

There are problems with this explanation. In any masking task, the designation of which stimulus is the “target” and which is the “mask” is arbitrarily determined by the researcher and his or her research goals. In other words, if the target persists longer in backward masking, then the mask should also persist longer in forward masking. Thus, this hypothesis predicts that schizophrenic subjects should suffer a deficit in both forward and backward masking. As we have seen, however, no such deficit has been demonstrated in forward masking tasks with schizophrenic subjects. Since sustained mechanisms play a crucial role in both forward and backward masking, it is difficult to see how defective sustained mechanisms could selectively interfere only with backward masking.

**Abnormal Transient Activity.** Theoretically, transient activity elicited by the mask plays a crucial role in backward but not in forward masking. Consequently, certain kinds of abnormalities in transient channels do provide tenable accounts of the schizophrenic subject’s deficit in backward masking.

Consider these three examples. Lee (1985) raised the possibility that transient discharges of abnormally high amplitude are associated with schizophrenia. Since transient channels serve an alerting function, this hypothesis is compatible with the evidence that schizophrenic patients suffer a deficit in both selective
attention (Stilson and Kopell 1964; McGie et al. 1965; Stilson et al. 1966; Venables 1984) and sustained attention (Garvey 1978; Nuechterlein and Dawson 1984).

Another proposal offered by Schwartz and Winstead (1982) was that schizophrenic subjects have more random transient discharges. Although not discussed by these authors, this notion is compatible with the hypothesis that transient units in schizophrenic subjects have abnormally low thresholds.

Schwartz et al. (1988) also cited abnormal transient activity as a possible explanation of the performance of schizophrenic subjects on a task designed to measure visible persistence. The researchers suggested an abnormally long persistence in transient activity in their schizophrenic subjects. (Note that abnormally long persistence in transient channels has far different implications than abnormal persistence in sustained channels.)

Most schizophrenia researchers have failed to consider the theoretical implications of evidence suggesting that schizophrenic subjects suffer from abnormal activity in transient channels.

To illustrate these implications schematically, we arbitrarily selected for discussion Lee’s (1985) proposal that schizophrenic subjects have transient responses characterized by abnormally high amplitudes. After considering this discussion, the reader may intuit that theoretically equivalent consequences should result from abnormally persistent transient discharges or from transient units with abnormally low thresholds.

Consider two hypothetical observers, one with “normal” transient activity and another with abnormally large transient responses. As previously explained, figure 2(a,b) portrays forward- and backward-masking mechanisms for the normal observer. Figure 2(c,d) illustrates the comparable mechanisms for an observer with transient responses of excessively high amplitude.

In figures 2(c,d), the bold single-headed arrows symbolize the greater transient interference associated with excessive transient signals. Note that in forward masking, excessive transient activity is not involved and thus does not increase masking. In backward masking, however, larger transients should cause greater masking because of the greater interchannel (transient-on-sustained) interference.

Compare figure 2(b) with figure 2(d). The temporal delays between presentations of the target and mask are identical. In other words, the operationally defined information-processing time is constant in both situations. However, the observer represented by figure 2(d) should suffer much more transient channel interference with the sustained channel’s neural representation of the target’s form than the observer represented by figure 2(b). Descriptively, the one observer would appear to process information more slowly than the other but, as the preceding argument suggests, slow processing would constitute neither a proper nor an adequate explanation of these data.

Hereafter, we use the phrase abnormal transient activity to refer to any of these kinds of deviant transient activity (i.e., those that within the framework of Breitmeyer’s model would produce a backward-masking deficit).

The hypothesis that schizophrenic subjects suffer from abnormal transient activity also leads to the prediction that schizophrenic subjects will be superior to controls on certain kinds of forward-masking tasks. This prediction is based on the fact that the designation of which stimulus is the “target” and which is the “mask” is an arbitrary one determined by the researcher. Even though the researcher designates the situation as forward masking, that specification does not preclude the target from exerting backward-masking effects on the mask.

Our hypothesis predicts that masking of the mask (and thus reduced forward-masking effects) should be maximized by the use of (1) a paracontrast procedure to minimize intrachannel integration, (2) a target stimulus with very low spatial frequency components for optimal transient response, and (3) a target-to-mask energy ratio greater than unity. If these conditions are met and if schizophrenic subjects do have abnormal transient channel activity, then they should be less vulnerable to forward-masking effects than controls.

However, the crucial implication of Breitmeyer’s (1984) model is this: if schizophrenic subjects do have abnormal transient channels, then comparing backward-masking performance between schizophrenic and control subjects does not provide any unambiguous evidence about relative “speed of information processing” (except in a purely descriptive sense), since the crucial transient component of the masking mechanism differs across the very groups being compared.

Conclusion

There is little dispute that deficient backward-masking performance is associated with schizophrenia. But what is the proper interpretation of this well-established finding?
Suggestions for Future Research

The model of visual masking presented in figure 2 has important implications for masking research with schizophrenic subjects. Within the framework of this model, information about crucial components of the masking process is obscured when attention is not paid to the spatial and temporal properties of target and masking stimuli. The kinds of traditional targets and masks pictured in figure 1 do not give the researcher adequate control over the elicitation of the transient and sustained activity that is so crucial to the theoretical model.

These arguments lead to an obvious suggestion: Whenever feasible, schizophrenia researchers should vary experimentally the spatial and temporal frequencies of stimuli used in masking or in other kinds of visual tasks. In this way, researchers can attempt to compare the functioning of transient and sustained visual channels across schizophrenic and control groups.

In schizophrenia research, a series of studies by Schwartz and his colleagues (Schwartz and Winstead 1982, 1985; Schwartz et al. 1987, 1988) demonstrates this approach for studying contrast sensitivity and visible persistence in schizophrenia. As mentioned previously, these studies have strongly suggested that schizophrenic subjects suffer from abnormal transient activity.

Using normal subjects, Mitov et al. (1981) have demonstrated how sine-wave gratings can be used as both target stimuli and masks. Comparable procedures should be fruitful in masking studies with schizophrenic subjects.

Another quite different area of research, that dealing with dopaminergic retinal cells, is relevant to this discussion, (for a brief review, see Ehinger [1977]). There are two kinds of neurons, horizontal cells and amacrine cells, providing lateral interconnections in the retina. Considerable evidence has accumulated that amacrine but not horizontal cells receive innervation from dopamine pathways in the brain (Malmfors 1963; Ehinger 1966; Ehinger and Falk 1969). Moreover, amacrine cells provide the lateral interconnections for retinal transient cells.

The evidence linking schizophrenia with disruption of dopamine systems in the brain is well known (Davis 1978; Snyder 1978). It should not be surprising, then, for that disruption to affect the activity of transient neural channels. At present, the research evidence is not sufficient to spell out the precise nature of neural activity in dopamine-amacrine-transient pathways in the retina. However, future research in this area may provide invaluable help toward our understanding of early visual processing in schizophrenia.

These two kinds of studies—those stressing a biochemical, psychopharmacological orientation and those where the researcher has manipulated both the spatial and temporal frequencies of stimulus patterns—are examples of how widely discrepant areas of research may converge to provide better explanations of the schizophrenic subject's deficit in backward masking and ultimately contribute to our understanding of the perceptual and cognitive deficits associated with schizophrenia.

References


Saccuzzo, D.P., and Schubert, D.L. Backward masking as a measure of slow processing in schizophrenia


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