EVIDENCE FOR IMPAIRED INTEGRATION-SEGMENTATION PROCESSES AND SLOWED SYNCHRONY-CODING IN DYSLEXICS

Cordula Becker[†‡], Thomas Lachmann[†§], Mark A. Elliott[†‡]

[†]Universität Leipzig, Germany, [‡]Ludwig-Maximilians-Universität München, Germany,
[§]Riken Brain Science Institute Wako-Shi, Japan
cbecker@psy.uni-muenchen.de

Using a primed figure-detection task we were able to reveal the existence of two distinct groups of dyslexics. One group is characterized by a significant impairment of visual integration-segmentation processes, resulting in slowed performance for the detection of a illusory Kanizsa-type figure in a matrix of distractor elements. These deficits appear to be produced by inefficient synchrony coding mechanisms. The second group of dyslexics shows slightly impaired integration-segmentation abilities. The results suggest a visual processing deficit apparent in a subgroup of dyslexics which is possibly based on slow magnocellular processing. The reported results are in agreement with previous findings reporting the existence of a distinct subgroup of dyslexics, which shows spatio-temporal processing deficits based on impairments of the magnocellular pathway in the visual system.

Dyslexia is a pronounced difficulty or inability in learning to read and/or to spell, despite otherwise normal intellectual functions. It has been proposed that reading requires the coordination of many functions and processes, such as visual and semantic decoding. Reading disability is supposed to be based on the failure of this coordination due to single or multiple impairments in functions involved in the reading process (Lachmann, 2001). This assumption explains why reading disabled persons do not show comparable impairments in everyday life. Specific deficient functions (e.g. visual decoding of written language) are only expressed phenotypically if they have to interact with other reading specific functions (e.g. semantic decoding).

Psychophysical and anatomical investigations (Livingstone, Rosen, Drislane and Galaburda, 1991; Lovegrove, 1980; Slaghuis and Ryan, 1999) have shown impairments of the magnocellular system (which is selective for low spatial and high temporal frequencies) in dyslexics, resulting in specific deficits in visual perception, such as longer duration of visible persistence and impaired contrast sensitivity. Smaller magnocells in dyslexics were specifically found in the lateral geniculate nucleus (LGN) of the thalamus (Livingstone et al., 1991). Despite these findings of a magnocellular disorder not all dyslexics seem to suffer from a magnocellular deficit. Following the dyslexia classification of Boder, magnocellular impairments were only apparent in the so-called dysphonetics and dysphoneidetics (Slaghuis and Ryan, 1999; Borsting, Ridder, Dudeck, Kelley, Matsui and Motoyama, 1996) who suffer from a deficit in word analysis-synthesis skills.

A changing retinal image due to environmental changes or to eye movements requires a fast disengagement of resources from one perceptual unit for correct perception of the following unit. The magnocellular pathway may be responsible for the disruption-by-inhibition of the parvocellular sustained activity following the presentation of a stimulus

(Breitmeyer, 1980) and is therefore crucial for the correct segmentation and integration of visual-scene elements. Inhibitory connections in the thalamus are also known to be responsible for the generation of oscillatory neuronal activity serving as a carrier for neuronal synchronization. Such synchronization of neurons oscillating with gamma-band frequencies is supposed to be a mechanism underlying the so-called binding, i.e. integration and segmentation of object features (see Engel, König, Kreiter, Schillen and Singer, 1992, and Whittington, Traub and Jefferys, 1995, for physiological in-vivo and in-vitro investigations and Tallon-Baudry and Bertrand, 1999, for electro-encephalographic investigations in humans).

Elliott and Müller (2000) showed that internal feature-binding mechanisms such as the synchronization of gamma-band oscillating neuronal assemblies may be entrained by appropriate external stimulation: the detection of a target Kanizsa-square within a matrix of distractor corner junction elements is facilitated if the target display is preceded by a flickering premask which contains a specific information about the later target location (synchronous premask condition; see figure 1 for an example display-configuration). The

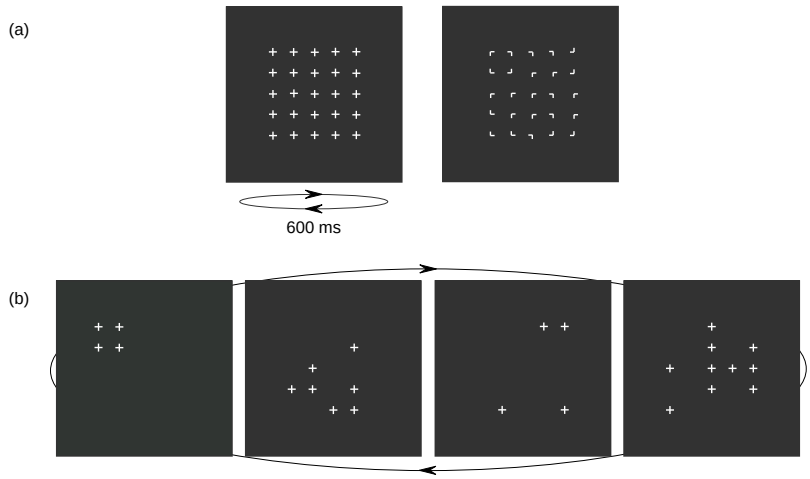

Figure 1: (a) The premask display (left) and the target display with the target in the upper left (right). (b) A possible configuration for a synchronous premask display with four crosses presented synchronously in the first premask frame.

premask consists of four frames of crosses which are recycled during premask presentation resulting in a presentation frequency of 40 Hz and the subjective impression of a flickering premask. One of the presented frames may consist of four crosses in a square arrangement at the target location (synchronous premask condition) or otherwise of four elements presented in pseudo-random arrangement (random premask condition). At a presentation frequency of 40 Hz subjects are not aware of the prime and the prime acts in a target-specific way, i.e. responses are only facilitated if the synchronous prime is followed by a target-present display.

The Kanizsa-target detection task

We used the 40-Hz flicker paradigm of Elliott and Müller in order to investigate integration and segmentation processes in dyslexics and the influence of oscillatory synchrony induced by the premask on these processes in dyslexics (Becker, Lachmann and Elliott, 2001). Two stimulus configurations differing in saliency were generated by changing the inducer length of the corner junctions, a more salient one (60% inducer specification) and a less salient, i.e. more difficult one (40% inducer specification).

A cluster analysis of the RT scores revealed the dyslexic subjects to be differentiable into two subgroups (dyslexics-1, seven participants; dyslexics-2, four participants) which were subsequently analyzed separately and in comparison to a control group of normal readers (13 participants). Subject groups did not differ significantly in age or intelligence level.

The unprimed Kanizsa-target detection task

The Kanizsa-square detection task without presentation of a flickering premask was used for a preliminary investigation of integration and segmentation processes in dyslexics. The detection of an illusory Kanizsa-square requires the integration of the square elements and the segmentation of these elements from distractor elements. We find that dyslexics-1 had a pronounced difficulty to process the presented stimuli resulting in significantly slower reaction times (RTs) in comparison to the control group (see figure 2). Furthermore,

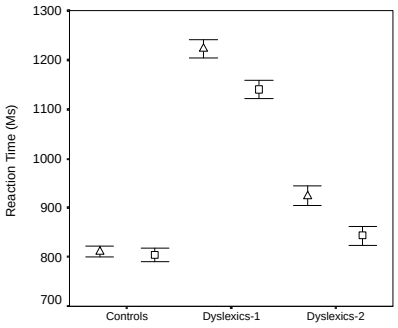

Figure 2: Mean RTs and standard errors for the inducer specification conditions as a function of the subject status. Triangle and square symbols represent 40% and 60% inducer specification conditions, respectively.

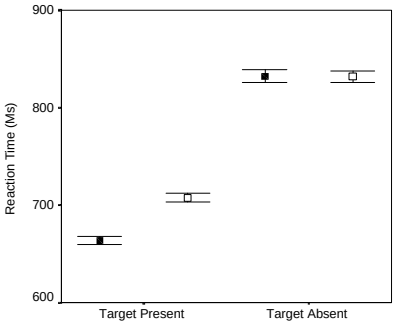

Figure 3: Controls: Mean RTs and standard errors for the premask-synchrony conditions as a function of the target presence. Filled and unfilled symbols represent synchronous and random premask conditions, respectively.

this integration-segmentation processing difficulty was expressed by significantly slowed responses for the less salient, i.e. more difficult, 40% inducer specification condition for dyslexics-1. However, even RTs of dyslexics-2 were slowed for the less salient stimulus condition. Against the background of otherwise unaffected integration-segmentation processes in this subgroup the presented result is likely to indicate a response strategy specifically adopted to the processing of less salient stimuli.

The primed Kanizsa-target detection task

Consistent with previously reported results we obtained the target-specific synchrony enhancement effect for the control group. This effect is characterized by significantly faster RTs following the presentation of a synchronous premask on target-present and not on target-absent trials (see figure 3). RTs of dyslexics-2 did not differ significantly from RTs of controls indicating that dyslexics-2 may benefit of the premask presentation serving as a prime to the later target presentation. This hypothesis is confirmed by the finding of a target-specific synchrony enhancement effect for dyslexics-2 (see figure 4). In comparison, dyslexics-1 not only produced significantly longer RTs than controls and

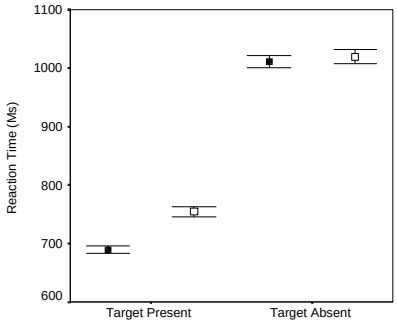

Figure 4: Dyslexics-2: Mean RTs and standard errors for the premask-synchrony conditions as a function of the target presence. Filled and unfilled symbols represent synchronous and random premask conditions, respectively.

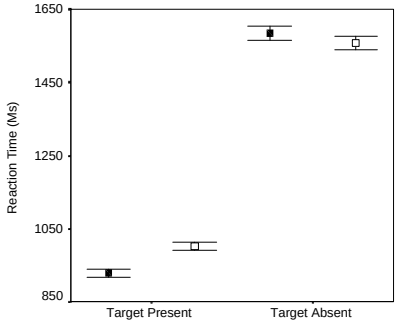

Figure 5: Dyslexics-1: Mean RTs and standard errors for the premask-synchrony conditions as a function of the target presence. Filled and unfilled symbols represent synchronous and random premask conditions, respectively.

dyslexics-2, but in addition no target-specific synchrony enhancement effect was found for this subgroup of dyslexics (see figure 5). The synchrony-coded prime facilitated reactions to a present target; but if presented preceding a target-absent display it slowed down responses.

Conclusion

Our results indicate the existence of two groups of dyslexics. Dyslexics-2 did not differ significantly from control subjects with respect to overall RT latencies and showed a target-specific synchrony enhancement. By contrast, the dyslexic-1 subgroup showed both substantially slower RT latencies and no specific synchrony enhancement. On this basis it can be concluded that the two dyslexic subgroups differ with respect to visual processing performance related to the integration and segmentation of visual information. Further, on the basis of the existing taxonomy, the performance of the dyslexic-1 subgroup might equate with that of 'dyseidetic' dyslexics, who have been shown to exhibit visual processing deficits (Slaghuis and Ryan, 1999; Borsting et al., 1996). A combination of physiological and behavioral studies (including the study detailed here) tends to favor the idea that poor performance of dyslexics in visual processing tasks may be related

to reduced or impaired neural inhibition within the magnocellular pathways: Inhibitory connections are thought to maintain the temporal fidelity of the magnocellular response while crucially mediate generation of neural synchrony. Given that the synchronous firing of neurons is considered to be the basic mechanism for operations related to perceptual organization, it seems reasonable to assume that the impaired integration and segmentation processes in the dyslexic-1 group may result from disruption of the mechanisms by which neural synchrony becomes established.

In the context of the experimental paradigm used here, magnocells might be expected to be responsible for locally signaling the rapid succession of premask-frame onsets (i.e. at 10 Hz) and thereby play a role in generation of an induced oscillation across the prime that has been found to match the global rhythm incurred by premask-display presentations with 4 local frames (i.e. at 40 Hz, see Elliott and Müller, 2000, for details). Thus, the prime is thought to represent the segmentation both in phase and space of the synchronous-premask frame relative to the remaining premask-display elements and, although other evidence (Elliott and Müller, 2001) suggests that the magnocellular system may not be uniquely responsible for prime generation and maintenance, the prime is nevertheless maintained with the precise temporal frequency of stimulus presentation. On this basis, poor temporal resolution in the magnocellular pathways might be expected to adversely affect the generation of the prime given that the prime requires the proper segmentation in time of the phase of premask display presentation.

The relationship between the onset of the target and concurrent activity within the prime is complex, but on current evidence appears to favor a point of perfect phase interaction between the local, premask frame and global premask-display rhythms (see Kompass & Elliott, in this volume). The locus of this interaction is suggested to be in early visual cortex (Elliott and Müller, 2000), which suggests that prime-target convergence may occur within early magnocellular pathways responding to target-display onset. If, as is suggested, synchrony generation is slowed or impaired by virtue of a magnocellular deficit in dyslexics, this may affect both the quality of the temporal responses to target and the convergence dynamics of target with prime. There is some evidence of a time cost for target displays following premask display presentations (unpublished results), which might suggest that the onset of the target display induces magnocellular activity that disrupts the synchronous prime activation (perhaps for the purpose of generating new synchronizations to code the target display). When the target display contains a target at the location of the prime activity the target overlaps with the concurrently active prime thereby enhancing synchronization (across both prime and target) relative to candidate synchronizations at other display locations. This would then result in the faster integration and deployment of attentional resources to facilitate detection of the target. Conversely, when no target is presented the magnocellular response to target-display presentation would simply disrupt activity across the prime effectively canceling any subsequent influence the prime might have upon mechanisms of integration and attentional deployment and, as is observed for controls and dyslexic-2 subjects, result in an absence of any particular priming effects.

As has been noted above, where magnocellular deficits are pronounced (as might be the case for the dyslexic-1 subjects), they might be expected to influence the temporal regularity and thereby reduce the fidelity of synchronization across the prime. However, the effects of a badly conditioned prime are likely to be compensated because the disruption caused by the general response target-display onset is also likely to be weakened. This then explains the maintained positive effect of priming for target-present trials de-

spite potentially weak priming. The inverted target-absent priming effect found for the dyslexic-1 subjects may be explained by the same logic. In this instance, the small time cost for synchronous vs. random trials may be based upon some persistent prime activity, which is not sufficiently canceled by target-absent display onset. In this instance the prime might encourage the deployment of attention to a non-target location necessitating subsequent disengagement (and a related time cost relative to random target-absent trials) such that other display locations might be searched for the target.

If reading is supposed to require integration and segmentation abilities, the presented results may explain reading disabilities in a subgroup of dyslexics in terms of slowed magnocellular activity resulting in slowed synchrony generation in response to visual stimulations. Although this deficit is not apparent in everyday-life performances which are not related to the reading process, the coordination of the different reading-specific functions may be impaired, resulting in a reading-specific phenotypical manifestation of this deficit.

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