ELSEVIER

Neuroscience Letters 336 (2003) 151–154

Neuroscience Letters

www.elsevier.com/locate/neulet

Crossmodal temporal processing acuity impairment aggravates with age in developmental dyslexia

Veijo Virsu*, Pekka Lahti-Nuuttila, Marja Laasonen

Department of Psychology, P.O. Box 9, FIN-00014 University of Helsinki, Helsinki, Finland

Received 10 June 2002; received in revised form 19 September 2002; accepted 22 October 2002

Abstract

Temporal processing has been found to be impaired in developmental dyslexia. We investigated how aging affects crossmodal temporal processing impairment with 39 dyslexic and 40 fluent 20–59-year-old readers. Cognitive temporal acuity was measured at millisecond levels in six tasks. They consisted of order judgments of two brief non-speech stimulus pulses, the stimuli being audiotactile, visuotactile and audiovisual, and of simultaneity/nonsimultaneity detection of the pulses in two parallel three-pulse trains. Temporal acuity declined with age in both reading groups and its impairment was observed in developmental dyslexia. A new finding was that the crossmodal temporal impairment, directly relevant to reading, increased with age. The age-related exacerbation suggests a developmental neuronal deficit, possibly related to magnocells, which exists before dyslexia and is its ontogenetic cause.

Keywords: Dyslexia; Magnocellular deficit; Temporal processing; Age; Crossmodal; Vision; Audition; Tactile somatosensation

Developmentally dyslexic readers appear to suffer from a deficit of processing rapidly changing stimulus sequences, affecting unimodal perception (visual, auditory and tactile) [9,15] and crossmodal perception requiring two senses [10,15]. It has repeatedly been suggested that this pansensory temporal impairment is caused by a deficit in magnocellular system [5,7,14], as magnocells are specialized in signaling rapid input changes. However, the causal role of a neuronal deficit has been inferred from correlation only.

Information processing speed is known to also decelerate with normal aging [2,8,13]. Several reports indicate that if a neuronal system is deficient due to a developmental weakness or brain insult, aging deteriorates the processes apparently related to the early deficit abnormally fast [3,4,12]. We showed in an earlier report [8] that the impairment of temporal processing in developmental dyslexia aggravates with adult age as if it were based on a developmental neuronal deficit. However, only unimodal processing was investigated in that study. Reading is an inherently multimodal process that requires accurate synchronization of vision, eye and head movements, phonemes, and many other memory representations. The effect of aging on crossmodal temporal impairment would clearly be more relevant to reading and the cognitive processes it requires. Therefore, if it is a magnocellular or another neuronal deficit that is the origin of developmental dyslexia, dyslexic readers' crossmodal temporal impairment should aggravate with age in the same way as was found to be the case with unimodal temporal acuity. If, alternatively, a corresponding exacerbation does not occur in crossmodal temporal processing, then a developmental neuronal deficit affecting processing speed can hardly underlie developmental dyslexia.

However, because aging also deteriorates other functions than temporal acuity [13], the decline in temporal processing as such does not necessarily indicate a special role to the magnocellular or other rate-limiting neural system. The possible role of a neuronal developmental deficit in dyslexia was studied here by investigating the effects of age on reading and temporal processing separately. The effects of age on reading capacity are confounded by, for example, education, practice and compensation, but it is possible to control their effects and estimate temporal processing with methods that are novel to the observer.

Crossmodal temporal acuity at different ages was measured in six different tasks that were comparable with the earlier measurements with unimodal stimuli [8]. The participants were the same 39 developmentally dyslexic and 40 fluent readers as in the previous study. Their ages

* Corresponding author. Tel.: + 358-9-1912-9530; fax: +358-9-1912-9516.
E-mail address: veijo.virsu@helsinki.fi (V. Virsu).

0304-3940/02/$ - see front matter © 2002 Elsevier Science Ireland Ltd. All rights reserved.
doi:10.1016/S0304-3940(02)01253-3

varied from 20 to 59 years. There was no statistically significant difference between the dyslexic and fluent readers in age (37 ± 11.66 and 36 ± 11.93 years respectively; $t(77) = 0.36$, $P < 0.73$), level of education (14.2 ± 2.8 and 14.9 ± 3.1 years; $t(77) = 1.08$, $P < 0.28$), handedness (right/left/ambidextrous: 30/4/5 and 37/1/2; $\chi^2(2) = 3.81$, $P < 0.15$), sex (male/female: 10/29 and 14/26; $\chi^2(1) = 0.82$, $P < 0.37$), or Wechsler Adult Intelligence Scale Revised [17] Verbal Intelligence Quotient (IQ) (106.49 ± 7.95 and 110.45 ± 11.14; $t(70.63) = 1.82$, $P < 0.08$). In Performance IQ (PIQ), the groups did differ (107.31 ± 11.54 and 115.03 ± 11.16; $t(77) = 3.02$, $P < 0.004$), and therefore we controlled this, in addition to sex and handedness, in all analyses. All the participants' Full IQ was at least at the level of average ability. None of the demographic variables above correlated significantly with age. History of neurological disease was an additional exclusion criterion.

All the dyslexic readers consistently reported a childhood history of reading difficulties, 30 had a formal diagnosis made by a psychologist or speech therapist. The dyslexic readers' age corrected reading performance was compared to that of the fluent readers. Dyslexia verification required that performance was at least one standard deviation poorer than the norm in at least three reading related tasks. The tasks were the same as in our previous studies [8,9]: auditive discrimination of pseudoword pairs (accuracy), phonological synthesis of sequentially presented phonemes (accuracy), pseudoword span length, Wechsler Memory Scale Revised: associative learning [18], rapid alternating stimulus (RAS) naming speed [20], reading speed (number of words read in 1 min), lexical decision (reaction time), conjoint word segmentation (speed), reading comprehension with texts including fiction and non-fiction (accuracy), and letter rotation (reaction time). All the dyslexic readers had difficulties in phonological processing. Seventeen of them had difficulties in phonological awareness (auditive discrimination, phonological synthesis), 26 in phonological coding (pseudoword span), 23 accessed phonological information slowly and 19 erroneously (RAS), and 30 were slow and 17 erroneous readers.

Temporal processing was assessed by measuring the cognitive temporal acuity thresholds in audiotactile, visuotactile and audiovisual tasks with 8-ms stimulus pulses as demonstrated on http://www.helsinki.fi/hum/ylpsy/neuropsy.html. In temporal order judgment (TOJ) method, the participant judged which one of two stimulus pulses was presented first. Each pulse represented a different modality (tactile, auditory or visual). In temporal processing acuity (TPA) method, the simultaneity/nonsimultaneity of pulses in two parallel cross-modal trains of three pulses was estimated. Stimulus onset asynchrony (SOA) was varied adaptively in a four-up/one-down threshold search [19] without any ceiling or floor limitations to estimate the SOA at which the participant made a correct yes/no assessment at the probability of 0.84.

In the audiotactile experiments, the tactile stimuli were indentations of the left hand index finger produced by the blunt tip of solenoid axis (driving pulse 8 ms at 20 V, maximum mass lifted 92 g, maximum amplitude 2 mm). The participant wore headphones, and a masking solenoid acted in counterphase to attenuate the sound cues further. The auditory stimuli were square waves at 4 kHz without phase locking or smoothing, presented binaurally through headphones at 60 dB SPL. In the visuotactile experiments, the visual stimuli were flashes of a light emitting diode seen in central vision (565 nm, flash luminance 4 cd/m^2, 8 mm in diameter corresponding to 0.5 degrees in visual angle, background luminance 1.5 cd/m^2). The tactile stimuli were as described above. In the audiovisual experiments, the visual stimuli were the same as described above, but the auditory stimuli were now presented through a loudspeaker at 65 dB SPL.

The cognitive temporal acuity thresholds of dyslexic and fluent readers, assessed with the two methods (TOJ and TPA), are presented in Fig. 1 for each age group for clarity's

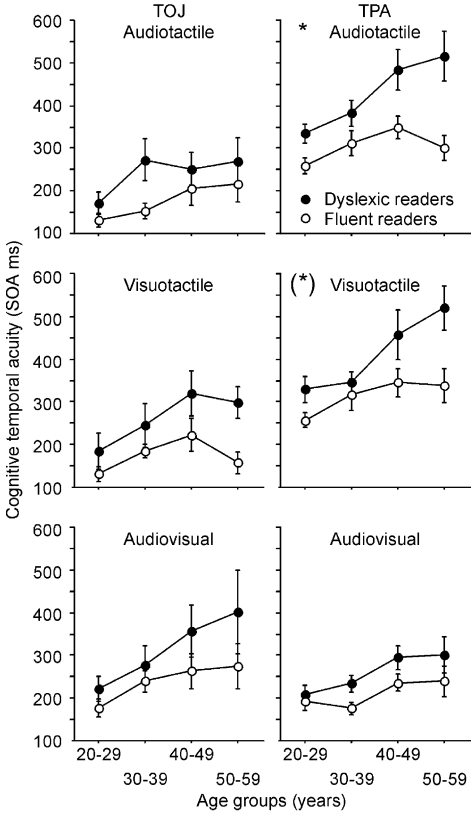

Fig. 1. Cognitive temporal acuity of the dyslexic (filled-in symbols) and fluent readers (open symbols) estimated with order (TOJ) and simultaneity/nonsimultaneity (TPA) methods as a function of age. The values indicate the age group mean threshold in each task. The lower the score, the better the temporal acuity is. Line segments represent SEM. Asterisks refer to $P < 0.05$ in the age x group interaction (in parenthesis when without outliers). Group sizes were (dyslexic/fluent readers): 12/15 of those 20–29 years, 11/9 of those 30–39 years, 9/8 of those 40–49 years, and 7/8 of those 50–59 years.

sake, but general linear model (GLM) analyses were conducted for continuous age variables. As expected on the basis of our earlier results [8–10], dyslexic readers showed an overall crossmodal temporal processing impairment. An anticipated result was also the decline of temporal processing ability with age in both reading groups. The critical new finding was that the interactions between crossmodal temporal impairment and age were qualitatively similar to those found in the unimodal study [8]. The dyslexic readers' crossmodal temporal processing impairment aggravated with age disproportionately in the TPA tasks but, as in the unimodal study, the exacerbation was not statistically significant in the TOJ tasks.

Group differences were analyzed for continuous age variables using a GLM with hierarchical decomposition of the sum-of-squares separately for the TOJ and TPA tasks. In this model, the terms were adjusted for those preceding them. We first entered the variables that we wanted to control (sex, handedness, PIQ), and then age, reading group, and reading group x age interaction.

The analyses for the TOJ showed a significant effect of age ($F(1,72) = 13.87$, $P < 0.001$) and reading group ($F(1,72) = 5.04$, $P < 0.03$), but the interaction reading group x age was not significant ($F(1,72) = 0.93, P < 0.34$). In the case of TPA, both the effects of age ($F(1,72) = 23.72$, $P < 0.001$), reading group ($F(1,72) = 11.81$, $P < 0.002$), and the interaction reading group x age ($F(1,72) = 4.61$, $P < 0.04$) were statistically significant. In single TPA tasks, however, the interaction reading group x age reached significance in the audiotactile combination only ($F(1,72) = 4.10$, $P < 0.05$); the result for the visuotactile interaction was $F(1,72) = 1.98$, $P < 0.17$ and for the audiovisual one $F(1,72) = 1.91$, $P < 0.18$. When we discarded poorly performing outliers whose responses were clearly biased at low presentation rates, the reading group x age interaction remained essentially the same in the audiotactile task ($F(1,70) = 4.31, P < 0.05$), but now the interaction reached significance also in the visuotactile task ($F(1,68) = 4.14$, $P < 0.05$). In the audiovisual task the interaction remained nonsignificant ($F(1,71) = 1.23, P < 0.28$).

Sometimes the older age groups performed slightly better than the younger ones on average, but this was evidently a chance effect because no relevant difference was statistically significant in *t*-tests. Error variance was large, probably because the participants were not experienced in psychophysical experiments. Therefore, we avoided conclusions based on comparisons between single perceptual systems. The reading group x age interaction was the smallest in the audiovisual TPA. This may be caused by its massive every-day rehearsal. Practice improves TPA considerably, but, as was found earlier [16], the same amount of practice improves audiovisual TPA much less than the audiotactile or visuotactile TPA, possibly because the neuronal systems involved in the audiovisual TPA had been practiced extensively prior to the experiment. The reading groups did not differ in response strategies, overall accuracy, attentional control, or response latency [8].

TPA was probably more clearly affected by the age-related temporal impairment aggravation than TOJ because the TPA stimuli are periodic and tap rate-limiting neural processes more exclusively. The stimuli in the TOJ tasks occur only once per spatial location and the task requires order estimation of the memory representations of stimulus pulses because the pulses are not repeated. The TPA stimulus pulses are repeated at the same spatial location causing less cognitive memory load in the synchronism estimation.

Age-related changes in other cognitive functions (reading, intelligence, and short term or working memory), or demographic differences between the groups were investigated. They did not explain the dyslexic readers' accelerated decline in crossmodal TPA and its statistical absence in the case of TOJ. The age-normalized Full IQ did not change with age in either group. None of the WAIS-R subtests or years of education indicated different age-related changes in the two reading groups, either. The dyslexic readers did not show age-related exacerbation or improvement in any reading-related task. In every analysis we controlled the sex, handedness (dextral vs. nondextral) and Performance IQ. When the WAIS-R Span Forward or Backward were additionally controlled in the statistical analyses the results did not change.

The higher-order interactions for the earlier unimodal [8] and the present crossmodal results were computed and were found nonsignificant for the reading group x age interaction. Thus, a statistically significant difference was not found in the age-related exacerbation of temporal impairment between crossmodal and unimodal perception. Therefore, our results agree either with the claim that magnocellular functions are impaired in dyslexia because this system is not functionally limited to single modalities but has also been associated with brain areas linked to crossmodal processing (parietal lobe and cerebellum) [1,7,14], or alternatively, with the claim that it may be some other early developmental neural deficit that impairs temporal processing of rapid stimulus sequences [9]. Our results also agree with the notion of high heritance of dyslexia [6] and with the finding that the brains of newborns having a high familial risk for developmental dyslexia may process auditory stimuli at a slower rate than control infants [11].

The same participants were used both in the unimodal [8] and the crossmodal study. However, this does not negate our inference based on their similarity. Firstly, crossmodal processing is generally slower than its unimodal components suggest and cannot be derived from its unimodal components [10]. Secondly, correlations between unimodal and crossmodal temporal acuity thresholds do not suggest a relevant common determination. Positive correlations are expected between any temporal tasks due to the necessarily common components, but if the tasks measure the same timing processes, the correlations should be more substantial. For the 40 normal readers (Ref. [8] and this study), the

nine correlation coefficients between unimodal and cross-modal TPA tasks varied from 0.09 to 0.58, averaging 0.26, and in the TOJ tasks, from 0.16 to 0.64, averaging 0.42 (Bonferroni corrected significance level for nine comparisons, $P < 0.006$ requires $r > 0.43$). For the 39 dyslexic readers the dependencies were somewhat larger. The corresponding numbers were 0.26–0.61/0.48 (TPA) and 0.34–0.77/0.54 (TOJ), respectively.

In sum, assuming that an originally, developmentally weak neuronal system deteriorates with age more than normally expected, developmentally dyslexic readers appear to have, on average, a weaker than normal neuronal system responsible for processing rapidly changing temporal sequences. The affected neural system can be the magnocellular system, or the deficit may be related to some other features of neural network that impair rapid temporal processing, for example a disorganized connectivity [5,9]. The existing, possibly inborn neural disadvantage compels the individual to develop compensatory strategies when learning to read. Some of these psychological and neuronal strategies may tap the rate-limiting neural system and thus cause the temporal impairment, but this causal relation is only partial. Therefore, temporal impairment or its concomitant neural deficits are not sufficient to predict dyslexia, particularly in the case of adults.

[1] Calvert, G.A., Brammer, M.J. and Iversen, S., Crossmodal identification, Trends Cogn. Sci., 2 (1998) 247–253.
[2] Cerella, J. and Hale, S., The rise and fall in information processing rates over the life span, Acta Psychol. (Amsterdam), 86 (1994) 109–197.
[3] Corkin, S., Rosen, T.J., Sullivan, E.V. and Clegg, R.A., Penetrating head injury in young adulthood exacerbates cognitive decline in later years, J. Neurosci., 9 (1989) 3876–3883.
[4] Dalakas, M.C., Bartfeld, H. and Kurland, L.T., The Post-Polio Syndrome: Advances in the Pathogenesis and Treatment, , Ann. N.Y. Acad. Sci., Vol. 753, New York Academy of Sciences, New York, 1995.
[5] Galaburda, A.M., Rosen, G.D., Denenberg, V.H., Fitch, R.H., LoTurco, J.J. and Sherman, G.F., Models of temporal processing and language development, Clin. Neurosci. Res., 1 (2001) 230–237.
[6] Grigorenko, E.L., Developmental dyslexia: an update on genes, brains, and environments, J. Child Psychol. Psychiatry, 42 (2001) 91–125.
[7] Hari, R. and Renvall, H., Impaired processing of rapid stimulus sequences in dyslexia, Trends Cogn. Sci., 5 (2001) 525–532.
[8] Laasonen, M., Lahti-Nuuttila, P. and Virsu, V., Developmentally impaired processing speed decreases with age more than normal, NeuroReport, 13 (2002) 1111–1113.
[9] Laasonen, M., Service, E. and Virsu, V., Temporal order and processing acuity of visual, auditory, and tactile perception in developmentally dyslexic young adults, Cogn. Affect. Behav. Neurosci., 1 (2001) 394–410.
[10] Laasonen, M., Tomma-Halme, J., Lahti-Nuuttila, P., Service, E. and Virsu, V., Rate of information segregation in developmentally dyslexic children, Brain Lang., 75 (2000) 66–81.
[11] Leppänen, P.H.T., Pihko, E., Eklund, K.M. and Lyytinen, H., Cortical responses of infants with and without a genetic risk for dyslexia: II, group effects, NeuroReport, 10 (1999) 969–973.
[12] Rice, D. and Barone, S.J., Critical periods of vulnerability for the developing nervous system: evidence from humans and animal models, Environ. Health Perspect., 108 (2000) 511–533.
[13] Spear, P.D., Neural bases of visual deficits during aging, Vision Res., 33 (1993) 2589–2609.
[14] Stein, J.F., The magnocellular theory of developmental dyslexia, Dyslexia, 7 (2001) 12–36.
[15] Tallal, P., Merzenich, M.M., Miller, S. and Jenkins, W., Language learning impairments: integrating basic science, technology, and remediation, Exp. Brain Res., 123 (1998) 210–219.
[16] Virsu, V. and Aura, M., Implicit learning of temporal discriminations in perception, Perception, 26 (1997) 123.
[17] Wechsler, D., Wechsler Adult Intelligence Scale - Revised: Manual, Psykologien Kustannus Oy, Helsinki, 1992.
[18] Wechsler, D., Wechsler Memory Scale - Revised: Manual, Psykologien Kustannus Oy, Helsinki, 1997.
[19] Wetherill, G.B. and Levitt, H., Sequential estimation of points on a psychometric function, Br. J. Math. Stat. Psychol., 18 (1965) 1–10.
[20] Wolf, M., Rapid alternating stimulus naming in the developmental dyslexias, Brain Lang., 27 (1986) 360–379.