terms in Equation 1 (above) are all stochastic variables and therefore the grand mean of FXDUR must equate to the combination of the means of the various contributing components. How does this work out? Our analysis below ignores refixations but otherwise tries to follow through Reichle et al.'s model.

Section 3.1.1 discusses the variable $t(V)$, which takes values upwards from 90 msec. L_1 is defined in Equation 2 of the target article as a product of two factors. The first ranges from 110 to 228 msec dependent on word frequency, and the multiplier ranges from 0.5 to 1.0 dependent on word predictability. A plausible overall mean value might be 130 msec. M_1 and M_2 are clearly set out to have mean values of 187 msec and 53 msec respectively.

OV_V, OV_L, and OV_M are not defined explicitly and depend on what happens when the model runs. OV_V and OV_L represent savings on the visual and lexical stages through peripheral preview advantage. OV_M represents modifications when saccadic programming stages overlap. Fixation durations are *shortened* when the planning for a saccade is able to take advantage of preparation already made (as with the second fixations in 5D and 5E of Fig. 5). Fixation durations may also be *lengthened* when saccade skipping necessitates a reprogramming of the location-distance stage, as in the first fixation of 5C.

The sum of the means of the first four terms of Equation 1 above is 460 msec. Therefore, to obtain a plausible overall mean, it seems necessary for the *OV* components to be quite substantial. OV_M can, as far as we can see, only be positive when two conditions are satisfied. First, peripheral preview has allowed completion of the $t(V)$ and L_1 stages of word$_{n+1}$. Second, the triggering signal falls in the 53 msec non-labile stage of the previous saccade preparation or during the saccadic movement itself (25 msec). Therefore, OV_M cannot exceed 78 msec. Whenever this combination of circumstances occurs, OV_L must equal the full value of L_1 (50 msec–228 msec). This suggests that the OV_M component will usually be smaller than OV_L. Our estimates of plausible parameters are as follows: $t(V)$ 90 msec, L_1 130 msec, M_1 187 msec, M_2 53 msec, OV_V 90 msec, OV_L 60 msec, OV_M 30 msec, summing to a mean FXDUR of 280 msec. Of this figure, 70 msec is "visual-lexical" and 210 msec "oculomotor." This reasoning assigns a very considerable role to peripheral preview, and two predictions seem to follow. If preview is prevented, fixations should be considerably lengthened; consequently, we find the 26 msec preview benefit figure given in section 3.2 surprisingly small. Second, the very first fixation on a text should be substantially longer than subsequent ones.

A similar exercise can be carried out with the variance of FXDUR, which again must be predictable from the variances of the component distributions, taking into account any nonindependence of the terms. How does the variance divide among the various components of the sum, and in particular between the visual-lexical and the oculomotor components? The calculations above suggest that the oculomotor components contribute about 75% to the mean. Unfortunately, the variance of the gamma-distributions from which M_1 and M_2 are drawn are not given in the target article (we very much hope the authors will supply these in their response). However, our rough estimates suggest the oculomotor components must contribute a considerable amount.

If indeed this is the case, it must be reconciled with the fact that in studies of saccades in simple situations, distributions with standard deviations in the 25–30 msec band are often found (Carpenter & Williams 1995; Walker et al. 1995; Wenban-Smith & Findlay 1991). It is, of course, possible that oculomotor variability depends on the circumstances in which the system is used and is higher in reading than in the cases cited. However, it could also be that the serial assumptions of the model are the source of the problem.

Frontal lobe functions in reading: Evidence from dyslexic children performing nonreading saccade tasks

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Abstract: Reichle et al. show that saccades in reading are controlled by linguistic processing. The authors' Figure 13 shows the parietal and frontal eye fields as parts of a neural implementation. This commentary presents data from dyslexics performing nonreading saccade tasks. The dyslexics exhibit deficits in antisaccade control. Improvement of the deficits is achieved in 85% of the cases and results in advantages in learning how to read.

From many different pieces of converging experimental evidence (Fischer 1987) the main components of saccade control have been identified as: (i) fixation, which stabilizes the direction of gaze; (ii) an optomotor reflex, seen under certain conditions as express saccade, when fixation/attention is disengaged; and (iii) a voluntary component, challenged by the instruction to generate antisaccades, that is, saccades in the direction opposite to a visual stimulus (Hallett 1978). Fixation is supported mainly by parietal (Motter & Mountcastle 1980; Robinson et al. 1978) and tectal functions (Munoz & Wurtz 1992), and the reflexes are mediated by the superior colliculus (Schiller et al. 1987; Sommer & Schiller 1992). The voluntary component relies on frontal lobe functions, because successful performance of the antisaccade task is impaired in patients with unilateral frontal lobe lesions (Guitton et al.1985).

Figure 1 shows the basic optomotor cycle consisting of series of periods of fixation (Stop) and saccades (Go). The cycling must not work on its own. It must be controlled by voluntary and/or cognitive processes that make each saccade a meaningful event within the process of active vision. Neurons in the frontal eye fields are activated before purposive saccades – not before any saccade (Bruce & Goldberg 1985).

How can one get more inside, into the relationships between the cognitive processing and the neural systems for saccade control? One possibility is to look at saccade control in nonreading tasks and to compare the corresponding data obtained from subjects who read normally with those of subjects who have reading problems; for example, dyslexics.

Deficits in the acquisition of reading skills may be (and have been) attributed to deficits of a number of different subfunctions within the reading process. One possibility is a deficit in saccade

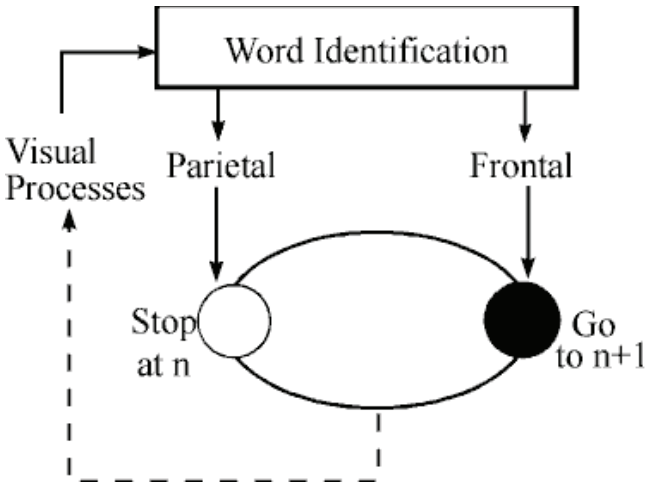

Figure 1 (Fischer). Schematic drawing of the optomotor Stop-and-Go cycle and its control by parietal (Stop) and frontal (Go) functions.

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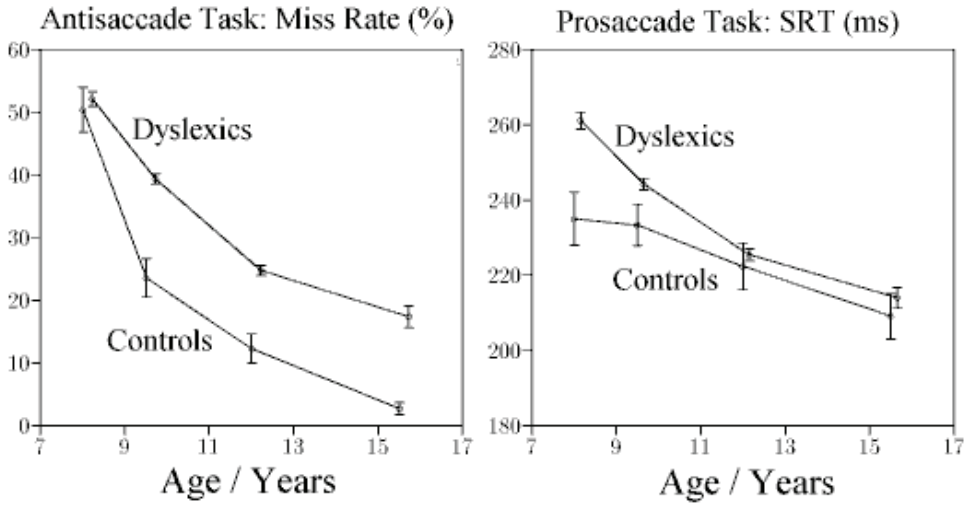

Figure 2 (Fischer). Pairs of age curves obtained from dyslexics (N=1,849) and controls (N=117). Vertical bars represent the confidence intervals.

control. Controversial results have been accomplished with interpretations ranging from "eye movements hold the key of dyslexia" (Pavlidis 1981) to "saccade control is normal in dyslexia" (Olson et al. 1983). Figure 1 reconsiders the problem and makes the following predictions:

1. If only linguistic processing (e.g., word identification) is impaired, saccade control will be affected during reading, but not during nonreading tasks.
2. If the frontal system is impaired, saccade control will be affected during reading and also during those nonreading saccade tasks, which challenge the frontal control component. However, other saccade tasks, for example prosaccade tasks, may be performed normally.
3. If fixation is unstable, reading may become more or less difficult even with intact linguistic processing.

Case number 2 is supported by experimental evidence: Large proportions of dyslexics have a specific problem with the voluntary saccade component, while only a minority exhibit deficits also in prosaccade generation during nonreading tasks (Biscaldi et al. 2000). A preponderance of intrusive saccades (Fischer & Hartnegg 2000b) and/or binocular instability (Stein & Fowler 1993) has been reported in dyslexia as well (case number 3), but will not be discussed here in detail.

Children between the age of 7 and 17 years were categorized as dyslexic or control using the diagnostic tests described earlier (Biscaldi et al. 2000). Two nonreading saccade tasks were administered: a prosaccade task with overlap conditions and an antisaccade task with gap (200 msec) conditions. From the prosaccade task we determined the reaction time, from the antisaccade task we measured the percent number of errors and the percent number of corrective saccades. The methods are described elsewhere (Fischer et al. 1997b).

The normal development of the different components of saccade control from the age of 7 to the age of 85 years has been assessed earlier (Fischer et al. 1997a; Klein et al. 2000). A comparison with the data of dyslexic subjects was described (Biscaldi et al. 2000). Here we present an updated analysis of the data.

Figure 2 shows a pair of age curves of the reaction times of prosaccades (overlap condition) and another pair of age curves of the percentage of those error trials, in which the errors were not corrected. These trials are called *misses.*

The curves show that the initiation of prosaccades is affected only for the youngest group. However, the generation of antisaccades exhibits systematic deficits increasing with age. Counting the percent number of dyslexics, who performed the antisaccade task with miss rates above the mean of the controls plus one standard deviation, reveals that the percentage of affected dyslexics increases with age from about 30% to 55%.

In these dyslexics the reading problem is caused partly by an insufficient frontal control of saccade generation, not by a general impairment of saccade control.

Earlier experiments have shown that daily practice can change saccade control (Fischer & Ramsperger 1986). Three visual tasks were designed for training: One requires fixation, one prosaccades, and another antisaccades (Fischer & Hartnegg 2000a; Fischer et al. 2000). The miss rates of eye movements of 148 dyslexic subjects were measured before and after the training (Fig. 3). About 85% of the subjects improved their antisaccade performance. The training improved only those aspects of saccade con-

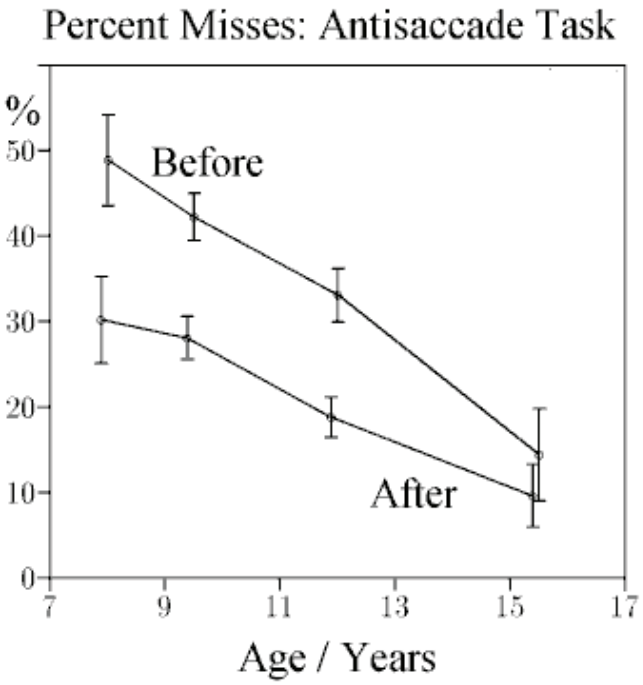

Figure 3 (Fischer). Percent misses in the antisaccade task before and after the training (N=148).

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trol that were part of the training program (Fischer & Hartnegg 2000a).

Finally, a group of 20 dyslexics with deficits in saccade control was divided into a test and a control group. Only the test group was given the antisaccade training. Then both groups were recombined and received six weeks of reading instruction. The test group reduced their reading error rate by about 49%, the control group by only 19% ($p=0.01$). The improvement of saccade control facilitates the learning process but does not replace it.

Among the executive functions of the frontal lobe is the execution of saccadic eye movements during reading. An impairment of this function does not imply that reading is completely impossible, only that the chances of reading errors due to inappropriate saccades are increased. It is suggested that a neural implementation of the E-Z Reader model does indeed include the frontal lobe, and that the model could also serve as a model of dyslexia.

Dimensionality and explanatory power of reading models

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Abstract: The authors' review of alternative models for reading is of great value in identifying issues and progress in the field. More emphasis should be given to distinguishing between models that offer an explanation for behavior and those that merely simulate experimental data. An analysis of a model's discrete structure can allow for comparisons of models based upon their inherent dimensionality and explanatory power.

The authors are to be congratulated for their structural analysis and comparison of several widely varying models of eye-movement control during reading. The emphasis on the basic structures and assumptions of the models is welcome, as is the authors' recognition that the ability to simulate experimental results is not the only measure of a model.

This commentary presents a further evaluation of reading models in terms of their discrete structures and dimensionalities, which represent the inherent expressive power of the model. It is seldom necessary to consider the output of computer simulations, using specific formulae, to understand the range of phenomena that can be predicted. Moreover, a focus on structure distinguishes the facets of a model that offer explanatory relationships from those that only quantitatively simulate data.

We begin with an example of such a discrete, structural explanation with regard to the spillover effect, then consider two notions of the dimensionality for reading models.

Two lexical stages accommodate the spillover effect. The ability of the E-Z Reader model to correctly predict the "spillover effect," under which the difficulty of one word can lengthen the fixation on the next, has nothing to do with specific formulae or simulations but is inherent in the separation of lexical processing into two stages. In Morrison's earlier model (Morrison 1984), of which E-Z Reader is an elaboration, lexical processing is considered as a single unit, and the signal to generate a saccade originates only after this process is complete. This basic structural assumption implies that the difficulty of processing the current word can have no influence on the following fixation. Therefore, Morrison's model cannot possibly account for the spillover effect.

In contrast, the E-Z Reader model has a second stage of lexical processing following the signal to generate a saccade. This automatically gives the possibility of a spillover effect of various amplitudes, because the duration of this second stage can contribute to the *following* fixation. Therefore, the ability of E-Z Reader and the inability of Morrison's model to account for the spillover effect does not in any way depend upon the specific equations but is implicit in the models' structures.

If the model is fundamentally correct, the requirement of two degrees of freedom in the lexical processing system must be reflected in physiological processes. In this way, the dimensionality of a model of sensorimotor function has implications for physiological organization.

Dimension as the measure of the space determining average fixations or individual fixations. There are various ways of assessing the dimensionality of a model, depending on the facets of interest. Here we give two examples of ways to make dimensional assessments based on a model's ability to predict fixation durations.

One notion of dimensionality is the average number of input variables determining fixation durations in a sequence. In Morrison's model, for example, the sequence of fixation durations is determined by the sequence of lexical processing times for each word. This is an average of one variable per fixation, giving the model a dimension of one. In the E-Z Reader model, the sequence of fixation durations depends on the durations of both stages of lexical processing for each word, as well as the word lengths (which determine early processing rates). This is an average of three variables per fixation, so E-Z Reader has dimension three.

Alternatively, dimension can be determined as the potential number of variables affecting the duration of an individual fixation. In Morrison's model, the length of the fixation is determined by either the duration of lexical processing on the fixated word $L(0)$ or by this duration plus that of lexical processing on the next word $L(+1)$, in case the next word is skipped. A graph of the possible contributions of these variables to the duration of a single fixation is given in Figure 1A. This is a two-dimensional subset of real two-space, giving Morrison's model a dimension of two. A similar analysis shows that with the E-Z Reader model (excluding early processing), individual fixations are determined from the durations of L_2 on the preceding and fixated words and L_1 on the fixated and following words. A graph of possible contributions of $L_1(0)$, $L_2(0)$, and $L_1(+1)$ to a fixation duration is shown in Figure 1B. This three-dimensional graph gives E-Z Reader a dimension of four when the possible contribution of L_2 on the previous word is included.

Both estimates of dimensionality show E-Z Reader to be more complex than Morrison's model, as expected, but they do give different numbers. The reason is that, because of parallel processing of saccades, an individual fixation can involve more cognitive processes and more free variables than does the average fixation. Note that if some fixations have more than average freedom of determination, then others necessarily have less! This is reflected in Figure 1 by the lower-dimensional components of the graphs. The two-dimensional measures are not incompatible but emphasize different aspects of the models.

Conclusion. The essential complexity and expressive power of a model can be represented in a discrete, schematic way. A correctly designed discrete model indicates all of the variables influencing the system, and all the ways in which values of one parameter can constrain those of another. Given the discrete model, simulations can be generated by constructing formulae that provide the best "fit" to the data. However, a focus on simulations can obscure the fundamental properties of the model by presenting results in a form similar to experimental data.

We feel that it is of great importance to distinguish those parts of a model that offer an explanation for behavior and physiology from those parts that merely simulate data. E-Z Reader offers real explanations for how the brain controls saccade timing, while its handling of saccade lengths and refixations is explicitly constructed for purposes of quantitative fit. Other models show their strengths in other areas, as can be seen from the excellent analysis of the target article. Our understanding of reading would be best served by attention to the dimensionalities necessary to explain observed sensorimotor behaviors and their implications for physiological processes.

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