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The role of intelligence in the definition of dyslexia

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Abstract

The disputable role of intelligence in the concept and definition of developmental dyslexia is critically evaluated. The implicit assumptions of the discrepancy concept connected with the underachievement conceptualization are explicated. Further it is shown that the regression-based operationalisation of a discrepancy is logically inconsistent with the underlying concept of underachievement. Finally, an attempt is made to explain the specificity paradox: the requirement that dyslexia be defined as a deficit that is reasonably specific to the reading task, while cognitive differences are ubiquitous between reading disabled and nondisabled children.

Since the very beginning of the research into dyslexia a hundred years ago, developmental dyslexia has been defined as a specific reading problem which is ‘unexpected’ considering the normal aptitudes of the affected individuals. This specificity requirement has been traditionally been operationalized as a discrepancy between intellectual functioning and academic achievement (e.g., reading performance). Despite the recent critiques formulated against the discrepancy concept of learning disabilities (Aaron, 1997; Stanovich, 1991), most contemporary definitions of specific learning disabilities include aptitude-achievement discrepancy as an essential criterion. For instance, according to the ICD-10 Classification of Mental and Behavioural Disorders (World Health Organization, 1993), and to the Diagnostic and Statistical Manual of Mental Disorders-IV [DSM-IV] (American Psychiatric Association, 1994), the diagnosis of a specific reading disorder requires that reading achievement is substantially below the level expected on the basis of the person’s chronological age and general intelligence score. Moreover, in practice, the identification of students with learning disabilities is usually based on one or the other operationalization of the discrepancy concept (Frankenberger & Fronzaglio, 1991; Mercer, Jordan, Allsopp, & Mercer, 1996). In this contribution I critically evaluate the role of intelligence and intelligence test scores in the concept and definition of developmental dyslexia.

Unexpectedness, underachievement and the discrepancy formula

The reading problems of children with dyslexia are “unexpected” because they cannot readily be ascribed to obvious causal factors. This is the reason why poor reading after a period of school absence (e.g. due to an illness) or as the result of serious sensorial, mental or emotional disorders is not diagnosed as dyslexia. Therefore, most definitions include some exclusionary criteria. Whereas the traditional definitions (e.g. World Federation of Neurology, 1968, cited in Critchley, 1970) stipulate that children should not have sensory impairments, detrimental instructional circumstances or very low IQ’s (e.g. under 70), contemporary definitions allow for these conditions but require that the reading problems are not their consequences (e.g. DSM IV, 1994). For example, the reading ability of children with hearing problems can be compared meaningfully with the reading level of a relevant norm-referenced group, that is other children with hearing difficulties. Consequently, when the reading difficulties are in excess of those usually associated with

a sensory or mental deficit, the diagnosis of dyslexia is still conceivable. Although the unexpectedness of the reading problems forms the “raison d’être” of the concept of dyslexia, it would be a fundamental mistake (e.g. in Dumont, 1976) to include “unexpectedness” per se as a necessary condition in the definition of dyslexia. Because human knowledge is not static, such a definition would destroy the very concept of dyslexia once the causes of dyslexia are revealed. In that case, nothing happens against expectations, turning dyslexia into an empty concept.

The discrepancy concept

For a clear understanding of the discrepancy concept of dyslexia, we need a small historical detour. According to Hammill (1993; see also Wiederholt, 1974) the roots of the present conceptual core of dyslexia are situated at the beginning of the nineteenth century. At that time observations were made that adults who had sustained head injuries, had lost the capacity to express themselves through speech, while preserving their intellectual abilities (a condition known as aphasia). In 1892 Dejerine reported a case of acquired dyslexia in an adult with an affection of the left gyrus angularis, while retaining his language abilities. When reading problems can be the result of brain injuries, then developmental reading difficulties without intellectual impairment, could be the outcome of minor congenital brain defects: this was the hypothesis expressed by Hinshelwood (1917). This line of reasoning gave birth to the idea of a discrepancy between intact intellectual functioning and specific academic disabilities. It is important to note that according to this historical tradition, exclusionary IQ criteria were imposed primarily as a means of ruling out confounding variables in order to enable researchers to discover more specific causal antecedents (Taylor, 1984; Taylor & Schatschneider, 1992).

The role of intelligence in the assessment of specific reading disabilities (and learning disabilities in general) increased tremendously by the introduction of the concept of underachievement (Burt, 1950). Advocates of this concept argued that purely interindividual norm-referenced comparisons should be supplemented or even replaced by an ipsative measurement model which defines the disorder largely by the individuality of the student (Reynolds, 1992; Rutter & Yule, 1973; Thorndike, 1963). In this model the child’s score on the achievement measure is compared with the score of the same child on an aptitude measure reflecting the child’s potential for learning. In practice, the concept of underachievement has been translated into a discrepancy score between measured reading

ability and general intelligence measured by an IQ-test (Frankenberger & Harper, 1987; Reynolds, 1992). An important implication of this new conceptualization is that poor readers with below average IQ's are often excluded from the dyslexic population (when not discrepant), whereas average readers with high IQ's can be labeled as dyslexic when satisfying the discrepancy criterion. Despite the critiques leveled against the operationalization of the discrepancy concept, most recent definitions are still based on some form of this concept. For that reason, it is useful to explicate the implicit assumptions underlying the discrepancy notion.

The crucial premise of the underachievement conceptualization is the idea that intellectual capacity normally determines reading achievement. Only in a minority of children some specific disturbing factors are responsible for a reading level far below the level one would have expected considering the normal intellectual disposition (cf. Aaron, 1997; Stanovich, 1996). Notably, the assumption of a causal relationship between intelligence and reading performance surpasses largely the assumption that only very low IQ's impede reading development (cf. the exclusionary criteria).

The regression-based discrepancy formula

In a little but influential book Thorndike (1963) criticized the simple standard score discrepancy formula, that is $IQ - AS$ (Achievement Score), for its failure to recognize the regression effect (see also Cone & Wilson, 1981; McLeod, 1979; Reynolds, 1985, 1992). Generally, regression toward the mean occurs whenever two standardized variables are not perfectly correlated. In the case of a positive but imperfect correlation, subjects who are high on one of the variables will also tend to be high on the other variable, but relatively less so, thus "they regress toward the mean". Similarly, subjects who are low on one of the variables will also tend to be low on the other variable, but relatively less so (e.g. Pedhazur-Schmelkin, 1991; Rogosa & Willet, 1985). Because of the imperfect correlation between IQ and reading scores, children with above average IQ's tend to have reading scores that are lower than their IQ-scores, resulting in statistically expected discrepancies which "should" be considered normal. Conversely, children with below average IQ-scores tend to have reading scores that are higher than their IQ-scores, resulting in an underestimation of discrepancies (Shaywitz, Fletcher, Holahan & Shaywitz, 1992). In order to avoid the overidentification of dyslexic children with high IQ's, and the underidentification of dyslexic children with low IQ's, Thorndike proposed a regression-

based discrepancy formula that accounts for the imperfect relationship between IQ and achievement. According to this formula, which is generally considered as the proper one since Thorndike, a difference score is calculated between the predicted reading score (based on the correlation with IQ) and the actual reading achievement score. When a child's regression-based discrepancy score exceeds a predetermined number of standard deviations of the discrepancy distribution (mostly 1.5 or 2 standard deviations), then the child could be characterized as "reading disabled" or "dyslexic".

It has gone unnoticed, however, that the adapted discrepancy formula is inconsistent with the basic premise of the underachievement concept (Van den Broeck, 2002_a). Recall that according to the concept of underachievement, intellectual potential normally determines reading achievement. A discrepancy score is then an indication that the normal course of things has been perturbed by some disrupting factors. Now, taking into account the imperfect relationship between IQ and achievement, which itself is the consequence of the empirical fact that other factors than IQ determine reading achievement, neutralizes the goal of the discrepancy method. Clearly, a measure of the extent that specific causal factors depress the achievement score normally determined by the intellectual potential, should not be corrected for the influence of the same causal factors. The description of a difference (discrepancy) should not be influenced by the explanation of the difference. In short, what is wrong with the simple uncorrected discrepancy formula is not its mathematical form, but its underlying theory about the role of intelligence, which is empirically falsified by the far from perfect correlation between IQ and reading scores. In a review of the literature on the relationship between IQ and reading, Stanovich (1988) calculated that for beginning readers the median correlation between reading ability and intelligence measures was .34 (11.5 % explained variance).

To elaborate this critique on the regression-based discrepancy formula two important consequences of the regression method should be emphasized. First, because the regression-based discrepancy scores actually reflect reading achievement scores partialled out for the influence of IQ, these discrepancy scores tend to be more determined by the actual reading scores the lower the correlation between IQ and reading is. In other words, the similarity between the regression-based discrepancy model and the interindividual norm-referenced approach of reading disability increases with a decreasing correlation between IQ and reading. Second, since the correlation between regression-based discrepancy scores and IQ-scores is exactly zero (a consequence of partialling out the

influence of IQ), discrepant readers and non-discrepant readers have the same mean IQ-score. This last point illustrates the logical inconsistency of the regression-based approach connected with the concept of underachievement of which it pretends to be the operationalization. Logically, there should be more “under-achievers” with high IQ’s than with low IQ’s, a requirement that is fulfilled with the simple standard score discrepancy formula.

The gradual decline of the discrepancy concept

Since more than two decades the discrepancy concept has been the subject of some critical inquiries. Although the field of learning disabilities seemed to have survived the first critical attacks which were primarily theoretical and methodological (Algozzine & Ysseldyke, 1983; Schlee, 1976), more recent critiques on the role of the IQ-score, and empirical comparisons between discrepant and non-discrepant poor readers accelerated the fall of the discrepancy concept.

Intelligence as “unlocked potential”

One of the many conceptualizations of intelligence is the view that intelligence reflects the general capacity for learning, included academic learning (Dearborn, as cited in Sternberg, 1985_a, p. 324). The concept of underachievement has often implied the assumption that children are not performing up to their innate potential (Burt, 1950). According to this view the intelligence test score is a measure of the child’s intellectual capacity, setting the maximum level of performance of which the child is capable. These assumptions, however, fared poorly under scrutiny. First, although genetic factors are unanimously accepted as important determinants of intellectual functioning, most recent theories incorporate environmental and contextual elements in their conceptualizations of intelligence (Ceci, 1990; Sternberg, 1985_a; but see Vernon, 1987). Moreover, as Stanovich (1991) pointed out, IQ-scores are at their best gross measures of current cognitive functioning (see also Detterman, 1982), and Siegel (1989) has made it clear that typical IQ-tests, as the Wechsler Intelligence Scale for Children-Revised (Wechsler, 1974), do not measure “potential” or basic reasoning skills but depend more on expressive language

skills, memory, fine motor abilities, and specific factual knowledge. Second, it has been shown that many children do achieve at a level well above their intellectual capacity, an anomalous outcome under the hypothesis of intelligence as potential for learning (Yule, Rutter, Berger & Thompson, 1974).

In an attempt to do justice to dyslexic children, some scholars have argued against the use of verbal intelligence tests, favoring instead performance IQ measures which are deemed to be “fairer” measures of the children’s potential (Birnbaum, 1990; Thomson, 1982; see also Miles, 1996). When the IQ-score is affected by the underlying dyslexic deficit, so the argument goes, the “real potential” of the dyslexic child is underestimated. Stanovich (1991) countered this argument persuasively. He questioned the tacit assumption of the fairness argument that performance tests would provide the best measure of the subject’s potential to comprehend verbal material. On the contrary, verbally loaded measures are arguably better estimates of future reading achievement (Hessler, 1987).

From an a-theoretical standpoint, intelligence test scores are achievement scores just as much as are reading scores, because an intelligence test assesses the child’s performance on a variety of tasks (Rutter & Yule, 1975). By implication, it would be just as valid to consider reading ability as a cause of intelligence as vice versa. As a matter of fact, there is sufficient empirical evidence that reading ability is a moderate determinant of vocabulary growth, verbal intelligence, and general comprehension ability (Hayes, 1988; Hayes & Ahrens, 1988; Juel, 1988; Share, McGee & Silva, 1989; Share & Silva, 1987; Stanovich, 1986_a; Stanovich & West, 1989; van den Bos, 1989). Stanovich (1986) places these causal effects of reading on verbal cognition in the broader developmental framework of “rich-get-richer and poor-get-poorer effects” and organism-environment interactions.¹ According to Stanovich, initial differences in reading skill grow larger as a consequence of the reciprocal or “bootstrapping” relationships with print exposure, vocabulary growth and motivational influences. Typically, a poor-reading child is not inclined to engage in reading activities, by which the delay in vocabulary and decoding ability grows larger. This framework offers an elegant explanation of the observed increasing correlation with age between reading ability and verbal competence (Bisshop & Butterworth, 1980; Brainerd, Kingma & Howe, 1986; Cohen, 1982; Stanovich, Cunningham & Feeman, 1984). Apparently, the vindication of the practice to exclude poor

reading children with low IQ's from the definition of dyslexia becomes exceedingly dubious when their low IQ's are at least partly the consequence of their reading disability.

To conclude, there is reasonable doubt that an intelligence test is an appropriate measure of the concept of learning potential, understood as an invariable disposition.

Discrepancy as a means to ensure specificity

The aforementioned arguments were directed primarily against the concept and measurement of intelligence in the definition of dyslexia. However, the discrepancy concept itself was not really questioned (e.g. Lyon, 1995; Stanovich, 1993). On the contrary, it was felt that abandoning the discrepancy concept would undermine the very foundation of dyslexia, namely the assumption of specificity. In the words of Stanovich (1988):

this assumption ... is the idea that a dyslexic child has a brain/cognitive deficit that is reasonably specific to the reading task. That is, the concept of a specific reading disability requires that the deficit displayed by the disabled reader not extend too far into other domains of cognitive functioning. Were this the case, there would already exist educational designations for such children (e.g., underachiever, slow learner, low intelligence), and the concept of reading disability or dyslexia would be superfluous. That is, if the deficits displayed by such children extended too far into other domains of cognitive functioning, this would depress the constellation of abilities we call intelligence, reduce the reading /intelligence discrepancy, and the child would no longer be dyslexic! His reading problem would become predictable from his problems in a range of other cognitive domains and no other explanation would be necessary. (p. 155)

Stanovich' formulation of a brain/cognitive deficit hypothesizes specificity at the causal, explanatory level: this is "causal specificity". Although causal specificity is a viable hypothesis when a disorder is characterized by phenotypic specificity, the common idea that causal specificity is essential for the concept of dyslexia leads to a number of problems in the diagnosis and research of dyslexia.

Pertaining to diagnostic issues, the requirement of a specific underlying deficit divides the group of poor readers theoretically into two subgroups: those whose poor reading is explained by the deficit (the dyslexics), and those whose poor reading is the

consequence of other non-specific factors (e.g. low intelligence), often called the “garden-variety” poor readers (Gough & Tunmer, 1986). In order to substantiate this theoretical subdivision in the assessment of an individual’s reading disability, the causal specificity requirement is often translated into the discrepancy criterion. To be sure that a child has a specific deficit, its reading level should be below a measure of reading potential. To avoid the disadvantages of the intelligence test, an alternative test procedure was proposed: the discrepancy between reading ability and listening comprehension (Aaron, 1989; Spring & French, 1990; Stanovich, 1991, 1993). As Stanovich explains: “Presumably, their listening comprehension [of discrepant readers] exceeds their reading comprehension because their word recognition processes are inefficient, causing a ‘bottleneck’ that impedes comprehension.” (Stanovich, 1991, p. 20; see also Perfetti, 1985). Completely in line with the underachievement premise of different causation of dyslexic versus other readers, children who are simultaneously low in reading and listening comprehension are said to have no “unexplained” or “unexpected” reading problem (Hoover & Gough, 1990; Stanovich, 1991). Importantly, a measure of listening comprehension is considered as superior to verbal IQ for its capacity to isolate a specific, “modular” deficit. Since listening comprehension correlates higher with reading than verbal IQ does, the deficit displayed by discrepant readers will be more likely a very specific one.

The fundamental problem with any discrepancy measurement procedure is that it contradicts the basic observation that reading problems are largely independent from any readily identifiable cognitive ability. It was exactly this independence that gave rise to the concept of dyslexia. In other words, the relative domain-specificity of word recognition (dis)ability is an observable fact, not a theoretical assumption or principle that should be substantiated in every particular case. The demand to observe a discrepancy in every dyslexic child actually implies a dependence relationship between reading and general cognition: whether poor reading will be characterized as dyslexia depends on the height of the IQ or listening comprehension score. But to the extent that reading ability is really domain-specific and modular, an IQ or listening comprehension score is irrelevant for the nature or explanation of the reading problem. The idea that the reading problems of children with low IQ or listening comprehension scores are explained by their weak general cognitive ability, contradicts the domain-specificity of reading. For instance, a child that is poor in reading, arithmetic and has a low IQ still has an unexplained specific reading problem by virtue of the domain-specificity of reading ability. Comparably, to get

the diagnosis of the flu, it is not necessary to be otherwise completely healthy (Van den Bos, 1998; see also Stanovich, 1996 for a comparable example). It seems that the deeply entrenched experimental logic of ruling out confounding variables was unperceived but erroneously translated into a diagnostic procedure. In explanatory research designed to discover the causal factors of dyslexia, to rule out confounding variables (e.g. IQ) in trying to exemplify the causal status of a hypothesized factor is utterly justified (Torgesen, 1989).

The requirement of causal specificity leads also to a conceptual paradox in the research on individual differences in the cognitive processes related to reading disability. This paradox was clearly pointed out by Stanovich (1986_b, 1988). He reported that cognitive differences between reading-disabled and nondisabled children were found virtually everywhere. It has been demonstrated that children with reading problems obtain lower achievement levels compared to normal children on syntactic awareness tasks (Bentin, Deutsch, & Liberman, 1990; Byrne, 1981; Siegel & Ryan, 1984; Vogel, 1974), on measures of general linguistic awareness (Johnson, 1993; Kotsonis & Patterson, 1980; Menyuk & Flood, 1981; Siegel & Ryan, 1984), on general rule learning tasks (Fletcher & Prior, 1990; Manis et al., 1987; Morrison, 1984, 1987), on short term memory and processing strategy tasks (Bauer, 1977, 1979; Brady, 1991; Newman & Hagen, 1981; Share, 1994; Siegel, 1994; Torgesen, 1978-1979), and on measures of metacognitive strategies (Baker, 1982; Foster & Gavelek, 1983; Wong, 1991). These observations are also in agreement with the one-half standard-deviation-IQ deficit displayed by children with learning disabilities (Stanovich, 1986_b). Although some of these cognitive processing liabilities can partly be attributed to reading or reading-related deficits (cf. reciprocal and Matthew effects), the generalized nature of the depressed cognitive abilities of dyslexic children threatens to undermine the assumption of causal specificity. To avoid the impending deadlock, Stanovich suggests that research into the underlying causal deficits of dyslexia should not aim at these general cognitive processes, but should be directed at more modular cognitive mechanisms, particularly phonological processing.

Explaining the specificity paradox

To reconcile the idea of a specific cognitive deficit with the finding of more general cognitive problems in poor readers, Stanovich formulated his “phonological-core variable-difference model” (Stanovich, 1988, 1991, 1993). In this model a deficit in

phonological processing is thought to be common to all poor readers, discrepant or non-discrepant, although the phonological deficit is hypothesized to be more severe in dyslexic (discrepant) readers. Indeed, a vast volume of evidence has established that poor readers exhibit deficits in various aspects of phonological processing. They have difficulty to detect, compare or manipulate phonemic segments in words, they are slower at naming objects and symbols, they show problems with short-term memory tasks (presumably due to inefficient phonological rehearsal), and they reveal subtle difficulties with speech perception and speech production tasks (Ackerman, Dykman & Gardner, 1990; Blachman, 1984; Bowers & Wolf, 1993; Brady, 1991; Brady, Mann & Schmidt, 1987; Catts, 1986; de Weirtdt, 1988; Kahmi & Catts, 1989; Katz, Shankweiler & Liberman, 1981; Pallay, 1986; Rapala & Brady, 1990; Snowling, Goulandris, Bowlby, & Howell, 1986; Wagner & Torgesen, 1987; Werker & Tees, 1987; Wimmer, Landerl, Linortner, & Hummer, 1991; Wolf, 1986, 1991). The term “variable difference” refers to the differences in cognitive skills between discrepant (dyslexic) and non-discrepant poor readers. These differences between dyslexic and non-discrepant poor readers magnify when one moves on the continuum from children with a discrepancy toward children without a discrepancy. Because the cognitive deficits of the garden-variety poor readers *do* extend into a variety of cognitive domains, the phonological difficulties of these children are considered in this model as epiphenomenal to their general cognitive deficits. Thus, a developmental lag model is thought to be an appropriate description of the cognitive status of the garden-variety poor reader. In other words, albeit the common phonological problem of discrepant and non-discrepant poor readers, the differing etiology of non-discrepant poor readers offers an explanation of the reported relationship between poor reading and general cognitive functioning. Again, the subdivision in two distinct etiological categories, reflecting the discrepancy concept, comes to the rescue to warrant specificity. In a recent study, Stanovich and Siegel (1994) could reject the prediction that the phonological core deficit would be more severe for discrepant poor readers than for non-discrepant poor readers. The authors explain this finding by referring to the “acquired modularity” of word recognition ability. However, by disconnecting the phonological problems of the garden-variety poor readers from their general cognitive deficits, the phonological-core variable-difference model no longer explains the moderate association between poor reading and general cognitive disability.

Before discussing an alternative proposal to explain the specificity paradox without relying on the discrepancy concept, a caveat about the specificity of explanations is in place. The idea that specific phenomena require specific causes, as an application of the “representativeness heuristic” (Nisbett & Ross, 1980), is consistent with the dominant habit in cognitive science to induce a cognitive structure from the observation of a behavioral effect: the effect = structure assumption (Lakoff, 1987; see also Van Orden, Jansen op de Haar & Bosman, 1997). Although the effect = structure heuristic is often an indispensable tool for deriving a first-order description of behavior (Bechtel & Richardson, 1993), it fails to reduce the complexity of the behavioral phenomena (Van Orden & Goldinger, 1994). Most scientific explanations reduce empirical phenomena as special cases of more comprehensive regularities. Clearly, explaining specific reading disability by a specific reading deficit doesn’t explain much. This is of course the reason for the qualification in Stanovich’ formulation that the deficit may extend into other domains of cognitive functioning, although not too far! Moreover, an entirely reading-specific brain/cognitive deficit as the cause of dyslexia, is theoretically implausible, if not impossible. Such a deficit would require an extremely fast Darwinian evolution, since “homo sapiens sapiens has been illiterate for more than 40,000 years, and universal education is only about 100 years old, much too short a time span for such a complex behavior to evolve” (McGuinness, 1997, p. 124). What might be heritable is one or the other “natural” underlying aptitude involved in learning to read, instead of the “unnatural” ability of reading that depends on instruction and requires a cultural evolution (Gough, 1996; Gough & Hillinger, 1980; Liberman & Liberman, 1990). Also, a phonological deficit, although rather specific and therefore an excellent candidate cause, is clearly not entirely specific to reading. The hypothesis of a phonological deficit predicts phonological processing difficulties outside the reading domain (e.g. in dyslexic children’s preliterate language development).

Stanovich’ recommendation not to search for causes in the domain of general cognitive processes, lest the concept of dyslexia would become superfluous, is justified as concerning the central cognitive processes. Central processes like metacognitive and strategic functioning are surely too allied to intelligence to be good places to look for the cognitive locus of reading disability. They are bad candidates, not because they violate a theoretical principle of causal specificity, but because they are incompatible with the empirical fact showing a low correlation between intelligence and word recognition

ability. Not all general cognitive processes are necessarily central processes. To name a few examples of general non-central cognitive processes that were proposed as candidate causes for dyslexia: a general language disorder (Catts & Kamhi, 1999; Metsala & Walley, 1998), a skill automatization deficit (Nicolson & Fawcett, 1990, 1994, 1995), and a deficit in temporal processing speed (Tallal, 1980; Tallal, Miller & Fitch, 1993). Naturally, these hypotheses should explain why the proposed deficits manifest themselves primarily in the process of reading development, and less so in other cognitive tasks. In short, they should explain phenotypic specificity. As a common scheme in hypotheses assuming a general cognitive deficit, phenotypic specificity is explained by referral to an interaction of the underlying deficit with task specific characteristics. For example, to explain why dyslexic children do not exhibit serious problems in the automatization of all cognitive skills, the “dyslexic automatization deficit” hypothesis assumes that the reading task is extremely vulnerable because of its specific resource-intensity (Nicolson & Fawcett, 1990). Notably, the demand to explain phenotypic specificity is not confined to these general cognitive hypotheses. As already argued, every viable causal hypothesis is to a certain extent non-specific, included the more specific ones. In a similar vein, the phonological deficit hypothesis has to explain why the phonological processing deficit doesn't manifest itself, or at least much less strikingly, outside the reading process. One would indeed expect that the extremely complex phonological processes in speech perception and production are even more vulnerable than the phonological processes involved in reading relatively simple words at the start of reading development. How is it possible that children who prove to be experts in speech phonology (by producing and understanding the most complex utterances) still have a subtle phonological deficit explaining their poor reading? (for attempts to answer these questions, see Fowler, 1991; Liberman, 1971; Liberman, Shankweiler, Liberman, Fowler, & Fisher, 1977; Metsala & Walley, 1998).

The fundamental point is that phenotypic specificity is a necessary and sufficient condition for the meaningfulness of the dyslexia concept. Causal specificity, on the other hand, is not a requirement for the tenability of the dyslexia concept, but is a possibility or hypothesis that has to be established empirically (Stanovich, personal communication, March 24, 2000). A priori imposing causal specificity on a theoretical basis endangers the concept of dyslexia (instead of saving it), when no single specific cause can be discovered. To strengthen the case for phenotypic specificity, it may be pointed out that this proposal

is entirely compatible with medical practice. Many medical diagnoses are based exclusively on phenotypic specificity (symptom patterns). Furthermore, many diseases are known for which definite causes are not yet been found. In medical science causal factors are only included in the diagnosis if the cause of the disease is empirically established as a fact by the scientific community.

Let us return now to the specificity paradox. Although the correlation between IQ and reading ability is very modest, it is reliably larger than zero. For one thing, this means that the major determinants of reading (dis)ability are not to be found in the central processes: the explained variance of reading being only 10 à 15 %. At the other hand, the relationship has to be explained. Besides the already mentioned reciprocal relationship between reading and IQ, there appears to be some additional sources to explain the correlation. First, because an IQ-test is no pure measure of central processes, it is plausible that an IQ-score is partly determined by lower-order processes like for example, phonological processing in digit span, associative learning in digit symbol (WISC-R). If this assumption is reasonable, then the observed correlation between IQ and reading is even an overestimation of the real relationship between reading ability and comprehension or thought processes. Second, it is conceivable that central processes have no direct effect on the reading process itself, but exercise an indirect influence on reading ability in two ways. (a) Less intelligent children, who have weak attentional capabilities, are probably less inclined to engage in sustained reading sessions. (b) Engendered by organism-environment interactions, these children get probably less stimulation from their environment to read, resulting in a lag of print exposure (cf. Stanovich, 1986_a). The detrimental entailments of low print exposure on reading ability are well-documented (Allington, 1984; Biemiller, 1977-1978; Cunningham & Stanovich, 1991; McBride-Chang, Manis, Seidenberg, Custodio & Doi, 1993; Nagy & Anderson, 1984; Stanovich & West, 1989). Finally, in all existing models of word recognition it is assumed that vocabulary knowledge facilitates the process of word recognition directly. In sum, there may be a multitude of factors determining reading (dis)ability, some of which are direct and others indirect, and some of which are intra-individual and others external (e.g. the influence of instruction methods). After all, learning to read is a learning process and any learning process can go awry for a multitude of reasons. Therefore, it will usually prove very difficult to pin down which determinant or which complex of determinants caused the reading problem of a reading disabled individual. Whether the child's reading problem

is the result of a specific cognitive weakness, say a phonological deficit, exacerbated by low motivation and stimulation and a mediocre instruction method, or any other combination, is very difficult to tell. Therefore, although most reading problems are probably determined partly by intrinsic specific factors, a realistic definition of dyslexia should leave open the possibility for multidimensional causality.

Are intelligent poor readers different from unintelligent poor readers?

The demonstration of the independence of word recognition from higher order central processes is reason enough to discard the discrepancy notion. However, it was not until direct comparisons between discrepant and non-discrepant poor readers revealed no qualitative differences in their reading-related processes, that most scholars were prepared to abandon the discrepancy concept.

Remember that the basic premise of the underachievement-discrepancy concept is the thesis of different etiology of the reading problems of unexpected (smart) poor readers and expected (dull) poor readers. By implication, it was predicted that the cognitive makeup, the nature of reading processes, the educational prognosis and the sensitivity for remedial interventions would be different for both groups (for an extensive review of these issues, see Aaron, 1997). Empirical studies have failed to show significant differences in psychometric profiles (e.g. WISC profiles) of discrepant and non-discrepant poor readers, despite the overall lower cognitive level of the last group (e.g. Fletcher, Francis, Rourke, Shaywitz & Shaywitz, 1992; Kavale & Forness, 1994; Ysseldyke, Algozzine, Shinn & McGue, 1982). A series of studies, using diverse measurements of phonological processing abilities in reading, couldn't reveal any substantial differences between discrepant and non-discrepant poor readers. Both groups performed equally on pseudoword (nonexistent pronounceable words) reading (Badian, 1994; Ellis & Large, 1987; Ellis, McDougall & Monk, 1996_a; Felton & Wood, 1992; Fredman & Stevenson, 1988; Jorm, Share, Maclean & Matthews, 1986; Share, 1996; Siegel, 1988, 1989; Stanovich & Siegel, 1994) and on other phonological skills (Ellis et al., 1996_a; Johnston, Rugg, & Scott, 1987; Siegel, 1992; Stanovich & Siegel, 1994). Nor have differences been found between both groups in the reading of regular words and exception words, indicating a failure to find a differing regularity effect (Fredman & Stevenson, 1988;

Share, Jorm & Maclean, 1988; Siegel, 1992; Stanovich & Siegel, 1994; see Metsala, Stanovich & Brown, 1998 for a meta-analysis).

Concerning the issue of differential etiology, in a twin study Olson, Rack, Connors, DeFries & Fulker (1991) couldn't find a statistically significant difference in the heritability of both groups' reading deficit. In the Colorado Reading Project, Pennington, Gilger, Olson & DeFries (1992) compared an IQ-regression based discrepant group with a group of backward readers and concluded that "The heritability analyses are primarily consistent with the hypothesis that the same genes influence each diagnostic phenotype" (p. 570). In another study, Olson, Forsberg, Gayan, and DeFries (1999) did find a higher heritability-score for high IQ poor readers versus low IQ poor readers. The authors suggest that a poor home and educational environment could be determinants for both the poor reading and low IQ, an interpretation that is consistent with the idea, expressed in the previous section, of a moderate indirect influence of IQ on reading disability. In their classic study Rutter and Yule (1975) reported that the educational prognosis for the discrepant poor readers was better than that for the non-discrepant poor readers. However, this finding could not be replicated in subsequent studies using longitudinal growth curve analyses of reading development (Francis, Shaywitz, Stuebing, Shaywitz & Fletcher, 1994; Share, McGee, McKenzie, Williams & Silva, 1987). Finally, concerning the differential sensitivity for remedial interventions, Aaron concludes from his review that "no overall, compelling evidence of educational gains has been obtained to warrant the continuation of the policy of classifying poor readers into learning-disabled and non-learning-disabled categories" [discrepant vs. non-discrepant] (Aaron, 1997, p. 475).

Respecting the methodology to compare discrepant and non-discrepant poor readers, an important remark about these studies should be made. As was already explained, when using regression-based discrepancy scores the mean IQ-score of discrepant and non-discrepant readers is the same. So, the natural meaning of discrepant as "smart but poor reader" vanishes because these readers are not smarter than non-discrepant readers are. Moreover, taking the median correlation between IQ and reading (.34) as a reference, the correlation between the reading scores and the regression based discrepancy scores is -.94. This means that most (regression-based) discrepant readers are also poor readers, and vice versa. Therefore, it is not easy to find a group of (regression-based) non-discrepant readers that are also poor readers. This is probably the reason why in the reported studies all kind of groups were compared with each other: groups with

regression based discrepancies, groups with simple standard score discrepancies and groups of low reading achievement with or without discrepancies. Nevertheless, despite the methodological and conceptual ambiguity of these studies, the overall conclusion that intelligence is quite irrelevant for the characterization of reading disability remains uncontested and is consistent with the empirical documentation of the modularity of reading. There is however some irony in the fact that the field has been persuaded most about the irrelevance of the discrepancy concept by studies which were only partly capable of disconfirming the discrepancy concept. To the extent that these studies made use of the generally commended but invalid regression based method, a priori implying the irrelevance of intelligence by statistically controlling for IQ, the discrepancy concept was not really challenged. Fortunately, the simple observation of a low correlation between IQ and reading is a sufficient reason for the definitive falsification of the discrepancy notion. With respect to the history of reading disabilities research, one can only endorse Stanovich' admonition that:

One might have thought that researchers would have begun with the broadest and most theoretically neutral definition of reading disability – reading performance below some specified level on some well-known and psychometrically sound test – and then proceeded to investigate whether there were poor readers with differing cognitive profiles within this broader group. Unfortunately, the history of reading disabilities research does not resemble this logical sequence. Instead, early definitions of reading disability assumed knowledge of differential cognitive profile (and causation) within the larger sample of poor readers and defined the condition of reading disability in a way that actually served to preclude empirical investigation of the unproven theoretical assumptions that guided the formulation of these definitions! (Stanovich, 1994, p.16).

Are intelligence tests obsolete in the research and clinical diagnosis of dyslexia?

Although we have come to the conclusion that intelligence is essentially irrelevant for the concept of dyslexia and is therefore not needed in the diagnosis of dyslexia, a more modest role of intelligence testing can be defended. First, in research it remains a perfectly legitimate question to ask what the exact role of intelligence is in specific learning disabilities; after all, the aptitude-achievement correlation is always

larger than zero. This kind of research requires a design in which groups of individuals with learning disabilities with high IQ scores (i.e., discrepant) and low IQ scores (i.e., nondiscrepant) who are matched on all possible confounding variables are compared. Secondly, in a clinical diagnosis it may be informative to segregate the role of intelligence-related and intelligence-unrelated determinants of an individual reading achievement score. This can be achieved by comparing the simple discrepancy score (IQ minus achievement score) with the regression-based discrepancy score (IQ-expected achievement score minus achievement score) (see Van den Broeck, 2002_b).

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Footnotes

¹ The “rich-get-richer and poor-get-poorer” effects are dubbed by Stanovich (1986) “Matthew effects” following Walberg (Walberg & Tsai, 1983) after the Gospel according to Matthew: “For unto every one that hath shall be given, and he shall have abundance: but from him that hath not shall be taken away even that which he hath” (XXV:29).