The diagnostic histories of autism, dyslexia, and hyperlexia are complex. Because these conditions share both convergent and divergent properties, it is important to understand these relationships, especially in the context of research and how we interpret bodies of work which span decades of fluctuating criteria. It is also important to synthesize what we already know about the morphology of these conditions and pinpoint what we still don’t. Autism and dyslexia, for instance, share antipodal cerebral morphologies, such as minicolumnar density, neuropil width, cell size, corpus callosal volume, gyral complexity, gyral window size, and cerebral volume, while hyperlexia has not been studied in this fashion, although it shares much in common with autism. Meanwhile, the fluctuation in criteria of dyslexia...
Although strikingly similar clusters of behavior were noted prior to the 1940s (1, 2), it was not until 1943 and 1944 that Leo Kanner and Hans Asperger published their respective monographs on the condition which we now know as autism. While Kanner focused on moderately affected children and Asperger on higher-functioning individuals, they both hit upon fundamental traits which are still considered the core of the conditions: 1) social and communicative deficiencies, and 2) repetitive or restrictive mannerisms, routines, or interests.

To review the history of developmental dyslexia, however, one must trace the literature back to the 19th century. Alexia, or acquired word blindness, has been recognized since the 1870s. While Sir William Broadbent originally claimed “word blindness” could not exist without other comorbid conditions such as verbal aphasia or amnesia, Adolph Kussmaul showed that it could in fact occur as an isolated condition (3). Later it was recognized that congenital forms of the condition existed, spanning a range of
тешко да се подели историјата на таквите вродени и стекнати нарушувања во читање, тоа поради тоа што раните научници ја замењали зборовната агнозија со денес пошироко познатото збор дислексија. Денешните дефиниции на развојната дислексија се движецат од предизвици до нејасни, како што примарниот критериум се движи од дефицит на фонално декодирање на зборови, до генерално нарушување при читањето. Како што можеме да се замисли, второто критериум опфаќа поголема хетерогеност на индивиди за разлика од претходниот.

Додека развојната дислексија е поврзана со нормална или натпросечна интелигенција, хиперлексијата е начело поврзана со когнитивно нарушување, и се користи за опишуване на деца со развојно задоцнување кои имаат напредни вештини за препознавање на зборови, но нарушено разбиране (4). Иако многу од децата во литературата сега би биле дијагностицирани како аутистични, во минатото опсегот на поврзаните развојни состојби вклучуваше аутизам, ментална ретардација и различни детски психотични нарушувања, како што е детската хипофенија (5). Моментално, нашето разбиране се простири и ги вклучува: 1) нормалните или надарените лица кои читаат рано, 2) лицата кои пројавуваат симптоми слични на аутизам, иако не е целосно изразена состојбата и хиперлексија и 3) луѓе со двете состојби и аутизам и хиперлексија (6). Според Treffert, овие две категории на хиперлексија покажуваат споредбено послабо разбиране при читањето и понекогаш недостаток на разбирано читање, карактеристики кои се поврзуваат со оригиналната дефиниција на терминот. Во продолжение, истржувачите пронајдат дека хиперлексијата придружена со основните услови различни од аутизмот е относително невообичаена, предизвикани споделени врскви во етиологијата на аутизмот и хиперлексијата (7). И покрај дивергенцијата во нивните критериуми, и двете, хиперлексијата и дислексијата може да се придружуваат аутизмот, иако првата со поголема фреквенција (6). Кај некои од претходните истржувачи, ние предложуваме дека аутизмот и хиперлексијата споделуваат заедничка етиологија, severity. It is, however, difficult to percolate the history of such acquired and congenital reading disorders because early scientists collapsed the word agnosia with the dyslexia more commonly known today. Today’s definitions of developmental dyslexia span from precise to vague, such that the primary criterion ranges from a deficit in phonological word decoding to that of a general reading disorder. As one might imagine, the latter criterion subsumes a greater heterogeneity of individuals than the former.

While developmental dyslexia has been associated with normal or above average intelligence, hyperlexia was initially associated with cognitive impairment, used to describe children with developmental delay who had advanced word recognition skills but impaired comprehension (4). Although many of the children in the literature would have probably now been diagnosed as autistic, in the past the range of associated developmental conditions included autism, mental retardation, and various childhood psychotic disorders such as childhood schizophrenia (5). Currently, our understanding has expanded to include 1) normal or gifted individuals who read early, 2) individuals who express autistic-like symptoms, although not the full-blown condition, and hyperlexia, and 3) people with both, autism and hyperlexia (6).

According to Treffert, these latter two categories of hyperlexia exhibit comparatively poorer reading comprehension and sometimes expressive language deficits, traits which harken back to the original definition of the term. In addition, researchers have found that hyperlexia comorbid with primary conditions other than autism may be relatively uncommon, suggesting shared links in autism’s and hyperlexia’s etiologies (7). Despite the divergence in their criteria, both hyperlexia and dyslexia may accompany autism, although the former with greater frequency (6). In some of our earlier work, we proposed that autism and hyperlexia share a common etiology, while autism and
In order to understand the etiology of these conditions and how they converge and diverge behaviorally, studying the underlying morphology is imperative. From the postmortem examinations performed on those with autism, it is clear that a significant portion of cases display vestiges of aberrant neurogenesis. Casanova et al. (10) have found that the neocortex of autistic individuals exhibits increased numbers of minicolumns and that the cells within these minicolumns are smaller and less compact than their control counterparts. The neuropil space which lies between adjacent columns is also reduced in size. Overall, these attributes suggest a larger total population of cells, the result of either overproliferation of the founder population during the neurogenic stage or reduced apoptosis (11). Increased neuron number have been noted in other areas such as the hippocampus, the amygdala, and other portions of the limbic system, while it is suspected that the well-replicated finding of reduction in the Purkinje’s cell number within the cerebellum is an acquired artifact (12, 13). This is evidenced by the reactive gliosis frequently present in the cerebellum at the time of death, and by the fact that Purkinje’s cells are a particularly vulnerable population
The current theory presently leans towards the overproliferation model in lieu of autism’s association with tumor suppressor genes such as Pten and TSC1/2 and other pro-proliferative mutations, while postmortem studies investigating apoptotically-associated molecular pathways have displayed considerable confounds and remain inconclusive (17–21). For example, the Araghi-Niknam and Fatemi (22) and Sheikh et al. (21) studies, both of which found reduction in Bcl-2 and increased expression of p53 in the postmortem brain tissue of autistic children, make no mention of how their subjects died. And yet, dependent upon the mode of death numerous artifacts can be created in postmortem tissue samples. To illustrate this point, the three common forms of premature death in autistic subjects are seizures, suffocation, and drowning, each of which can promote hypoxia or anoxia in the brain (23).

Interestingly, hypoxia has been shown to trigger suppression of Bcl-2 through an NFkB-dependent manner and accumulation of p53—findings which are unaccounted for in the results of the Araghi-Niknam and Fatemi (22) and Sheikh et al. (21) studies (17, 24).

In addition to the aberrant proliferation, abnormalities in differentiation and migration are also common in autism as evidenced by telencephalic heterotopias and dysplasias. Rather than occurring homogenously across the developing cortical plate, these deviations occur heterogeneously throughout the brain.
(25). So, looking at the symptomatic cortical anomaly, heterotopia, and synaptic one, it may be inferred that the cause for the high rate of epilepsy is the heterotopia, the heterotopias, and the seizures, it is possible that their occurrences in autism are the cause of the high rate of epilepsy (26–28). Given the relationship between the cortical dysplasias, the heterotopias, and the seizures, it is possible that their occurrences in autism are the cause of the high rate of epilepsy (26–28). Significantly, while most researchers have envisioned epileptiform activity arising from the heterotopic and dysplastic tissues themselves, animal models on dysplasia-induced polymicrogyria instead suggests that normal tissues adjacent to the malformation are in fact the seat of epileptogenesis (29).

In contrast to autism, the fluctuation in criteria of dyslexia over the last several decades has made it extremely difficult to adequately assess neurogenic anomalies across research studies. A number of recent studies restrict the diagnosis to those who fulfill the stringent criterion of a phonological deficit, while older studies including the most highly cited of postmortem studies report either vague diagnostic inclusion criteria or base diagnosis on a broader reading deficit (30–33). Therefore, the dyslexia we know today which has roots in faulty speech processing is not the same as the general reading deficit studied in earlier research, making numerous studies incomparable. Ricketts (34), for instance, has noted that children with poor comprehension skills, as opposed to the poor word recognition which typifies dyslexia, are approximately two times more common than dyslexics. This provides a heterogeneous group of individuals who ultimately may be subsumed under the broad category of Reading Disorder. For those studies which have utilized this broader definition, the potential heterogeneity of their subject samples leads to considerable confounds. Nevertheless, we can summarize the findings of some of the more stringent studies and review those studies which utilized more lenient criteria with due caution. Utilizing stringent diagnostic criteria, our own studies have noted differences in the minicolumnar
гркупата со аутизм, примарната дислексия се
манифестира преку зголемена миниколум-
нарна ширина, средна пространост на клетки-
те и неуоришена ширина, во споредба со кон-
тролната група (10). Исто така беше откривана
намалена гирилна сложност, зголемен прос-
тор межу гирисите и зголемен волумен на
корпус колозум кај дислексијата, споредено
со двете групи, контролната група и аутисти-
те (9, 14, 35). Во продолжение, дислексијата
има намалено фокусирање на волумените кај
сивата и белата маса како во левата средина и
инфернорното слепооче гирус, така и во
arcuate fasciculus, невообичаена симетрија
номеру левата и десната plana temporal и
вкупна редукција во мозочинот волумен (36–
39). Овие пронајдци се придружени од кори-
kолналата хетеротопија и дисплазија забеле-
жани во состојбата, особено оние во состав на
левата хемисфера (31, 32). Како што може да
се забележи, епилепсијата е значително повр-
зана со оваа состојба и може да има слични
корени со појавата на хетеротопија и диспл-
азија (40).
Кај дислексијата, значителен дел на овие ек-
tопични кластери се јавува во состав на моле-
kуларниот слој на неокортексот спротивно на
аутизмот кој има склоност кон первентрику-
ларна и субкортikalна екторпија (25). Можно
е овие различни типови на хетеропии да настанат од различни вродени грешки на
кортиногенезата која може да помогне да се
одвои и нагласи нивното соодветно потекло.
Поради тоа што времето на одржување на
пролиферацијата, миграцијата и смртта на
клетките се мешавина од автономната и
неавтономната на клетките, различните
хетерокронии во аналогните ткаива може да ре-
зултираат во различни типови на хетеропии
и дисплази, како оние што се забележани кај
аутизмот и дислексијата (41).
Во состав на таламусот се откривени абнор-
малности на магноцеоларната патека, визу-
ellна патека која процесира брзи нискоон-
трасни визуелни информацији, за разлика од
бравата високоонтрасна патека, каде се најдени неколку различни (42).
Поради овие пронајдци, во комбинација со
други истражувања кои покажаа дека дислек-
tичарите имаат тенденција лошо да ги из-
вршуваат задачите на брзото визуелно про-
цесирање, Livingstone и сор. (42) констати-
рале дека основниот дефицит во дислексијата
morphometry in primary dyslexia as
compared to controls. In stark contrast to our
autism group, primary dyslexia displays
increased minicolumnar width, mean cell
spacing, and neuropil width as compared to
controls (10). We have also found reduced
gyr shape complexity, increased gyral window
size, and increased corpus callosal volume in
dyslexia, compared to both controls and
autistics (9, 14, 35). In addition, dyslexics
appear to have focal reductions in gray and
white matter volumes such as within the left
middle and inferior temporal gyri and arcuate
fasciculus; an unusual symmetry between the
left and right plana temporale; and an overall
reduction in the total brain volume (36–39).
These findings are complemented by the
cortical heterotopias and dysplasias noted in
the condition, particularly those within the
left hemisphere (31, 32). As is also seen in
autism, epilepsy is significantly associated
with the condition and may have similar roots
in the occurrence of heterotopias and
dysplasias (40).
In dyslexia, a significant portion of these
ectopic clusters occurs within the molecular
layer of the neocortex in contrast to autism
which has a predisposition for periventricular
and subcortical ectopias (25). It is possible
that these different types of heterotopias arise
from different inborn errors of corticogenesis
which could help differentiate and pinpoint
their respective etiologies. Because the timing
of proliferation, migration and cell death are a
mix of cell autonomous and non-autonomous
processes, different heterochronies within
analogous tissues may result in different
types of heterotopias and dysplasias such as
those seen between autism and dyslexia (41).
Within the thalamus, abnormalities have been
found in the magnocellular pathway, a visual
pathway which processes rapid low-contrast
visual information, whereas there are few
differences noted in the slow high-contrast
parvocellular pathway (42). Because of these
findings, in combination with other works which
have shown that dyslexics tend to
perform poorly on tasks of rapid visual
processing, Livingstone et al. (42) have proposed that the fundamental underlying deficit in dyslexia lies not within a given sensory modality but with the rapid processing of information which bears its roots within the thalamus. However, Galaburda (43) has shown in a promising animal model that the neocortical anomalies (e.g.: ectopias, microgyri) appear to promote the thalamic ones in a top-down model of thalamic pruning. Ramus (44) likewise argues that while other symptoms may be secondary to the condition, such as abnormalities in visual and sensory-motor processing, the literature suggests that deficits in phonological decoding are the primary cause in the reading impairment which defines the condition.

In contrast to both autism and dyslexia, little work has been performed on the hyperlexic brain. No postmortem work has been performed to date, and the few functional imaging studies that exist, focus on extremely small numbers of patients. For instance, Turkeltaub et al. (45) studied a 9-year-old hyperlexic boy finding increased activation of the left inferior frontal, left superior temporal, and right inferior temporal gyri. From these results, the researchers concluded that reading precocity occurs via enhanced activation of both the left auditory and right visual systems. Tirosh & Canby (46) in their small study also found that two of the five hyperlexic autistic children in the experimental group were macrocephalic. One of those two children had a sibling with hyperlexia which was not included in the study, an occurrence suggesting potential heritability.

The majority of research on hyperlexia focuses on its comorbidity with autism. In fact, the enhanced word recognition and poverty of comprehension which typify many forms of hyperlexia are also the predominant trends in autism, although not all autistics exhibit such superlative word recognition capacity as to be labeled hyperlexic and likewise not all hyperlexics fulfill the criteria for autism (47). As Frith and Snowling (48)
Додека ниската конективност на долго влакнестите трактови кај аутизмот може да придонесе за слабо разбиравање на читањето кај хиперлексичните и типичните аутисти, просечната и натпросечната фонолошка вештината може да се јави поради локалната прекомерна конективност (8). Како поврзан пример, синестезијата е почеста кај аутизмот отколку кај генералната популација; од неодамна е утврдено дека капацитетот кој е основа на графемата на синестезијата во боја, е поврзан со поголема конективност во инфериорниот темпорален кортекс (49). Би очекувале слични откритија во релевантни области кај хиперлексичниот мозок, на пр. локална прекомерна конктивност. Се надеваме дека иден истражувања ќе бидат во содржба да ја истражуваат оваа претпоставка.

Врската помеѓу аутизмот и дислексијата, се поклопува и е дихотомна. Дислексијата може да биде споредна појава кај аутизмот, додека примарната дислексија покажува церебрална морфолошка антиподи за разлика од претходната. Во меѓувреме, хиперлексијата се манифестира преку силички сличности со аутизмот и многу често може да биде коморбидна со него. Иако хиперлексијата е често сметана како суперспособност, често се појавува заедно со некаква форма на потешкотија во читањето. И вкупност, нарушувањата во читањето често се наоѓаат и кај другите членови од семејството (5, 50). Вгнездувањето на овие содржби многу веројатно претставува момент кога општорифатениот однесување се поклопува со двете антиподни морфологи. И поради тоа, со цел да се определи дали тоа се случува, многу е важно нашите дефиниции во истражувањето да бидат прецизни и конзистентни.

Проблем и дефиниции

Frith и Happé (51) констатираат дека „чистите“ случаи на развојни нарушувања каде постои само една единствена состојба, се ретки. Наместо тоа, паралелната појава на нерво-развојните нарушувања е честа и е дел од поголема динамика на примарната состојба. Како што се дискутирало, аутизмот може да биде коморбид или со хиперлексијата или со дислексијата (7, 34).

Од карактеризацијата во 1940-тата на нава-

have suggested: dyslexics read for meaning, autistics read for sound.

While the under connectivity in long-range fiber tracts in autism may underlie poor reading comprehension in hyperlexic and typical autism, average-to-above-average phonological skill may be due to local over connectivity (8). As a related example, synaesthesia is more common in autistics than in the general population; it has recently been found that the capacity which underlies grapheme-color synaesthesia is related to greater connectivity in the inferior temporal cortex (49). We would expect similar findings in relevant areas within the hyperlexic brain, i.e., local over connectivity. Hopefully, future research will be able to explore this prediction.

The relationship between autism and dyslexia is both an overlapping and dichotomous one. Dyslexia can be secondary to autism, while primary dyslexia exhibits cerebral morphology antipodal to the former. Meanwhile, hyperlexia presents with striking similarities to autism and may frequently be comorbid with the same. Although hyperlexia is often regarded as a super ability, it usually co-occurs with some form of reading deficit. And in fact, reading disorders are often found also in close family members (5, 50). The imbrication with which these conditions present likely represents the point at which a common behavior overlaps two antipodal morphologies. Therefore, in order to determine whether this is the case, it is imperative that our definitions in the research are precise and consistent.

Problem and Definitions

Frith and Happé (51) have suggested that “pure” cases of developmental disorders in which only a single condition exists are rare. Instead, co-occurring neurodevelopmental disorders are both common and part of the larger dynamic of the primary condition. As discussed, autism can be comorbid with either hyperlexia or dyslexia (7, 34).

Since its characterization in the 1940s, the
The definition of autism has not changed considerably. Although we now have a broader schema which includes higher-functioning and lower-functioning individuals than Kanner would have originally diagnosed, defining autism by its social and language deficits and its restricted or repetitive mannerisms has not evolved considerably. Science still struggles to identify a reliable biomarker or common macroscopic/microscopic correlates which are consistent across the heterogeneous conditions; to date, however, we are forced to rely on behavioral constructs. Our own studies on minicolumnar morphometry reveal that a significant portion of cases of autism exhibit reduced cell size, minicolumnar size, and neuropil width; however, this is a group analysis and reflects larger trends and can say little of individual cases.

Unfortunately, the history of dyslexia is plagued by greater problems—not solely because this neurodevelopmental condition is represented by a drastically simplified criteria list, but because that definition has changed over time and is still in a fluctuation state (see below for the APA’s current proposal for “Specific Learning Disorder”). Because many pathological behaviors rely on a continuum with normality, behavioral definitions tend to be relatively arbitrary and thus to alter them so fundamentally makes research and health care extraordinarily difficult, akin to hitting a moving target. Current research suggests that while dyslexia may reveal itself through its deficits in reading, at its core may be underlying differences in how dyslexics’ process speech sounds; this disruption may subsequently disturb the cognitive translation of visual word symbols into representative and meaningful sounds. As Snowling (52) suggests, this is due to “basic deficits in speech perception, speech production, or temporal processing [and that these people] have difficulties first in establishing, and later in accessing adequate phonological rep-
дека новородениците со ризик кои потекнуваат од мултиплекс дислексички семејства покажуваат аномални говорни процесирана дури и пред да научат да зборуваат (53–55). Во продолжение, во функционално прикажани студии за дислексијата, дислектичарите покажуваат споредбено намалена активација во областите од говорното процесиране во левата хемисфера, со дланово нарушување на мултиполарната интеграција за време на фонолошки задачи кои вклучуваат внатрешен говор (56). Истражувањата пропашле површен а сепак траен дефицит на фонолошко процесиране кај аутизенти и младинци кои еднаш ја привиле дијагнозата, но повеќе не се квалификовани (56, 57).

Хиперлексијата исто така имаше удел и во дилемата за дефиниции. Во еден момент таа се смета за недостаток поврзан со невроразвојните состојби. Со текот на времето таа не беше повеќе сметана за недостаток, туку супер или ненormalна способност. Сега се смета за комбинација од недостатоци (сиромашно разбиране во читањето) и способност (добро до одлично препознавање на зборовите) - или во случајот на „невротипичните” индивидуи, не постои воопшто недостаток (4, 6, 50). Во продолжение, постојат многу дебати дали хиперлексијата е сама по себе АСН, иако Treffert (6) го има нагласено тоа, предлагајќи дека додека два од три типови хиперлексија се поврзани со аутистичните однесувања, третата група не се смета во или близка до спектарот. Оваа последна група, сепак е често прецизна и светла, и ако се земе предвид врската, се појавува со зголемен волумен на сива и бела маса, овие невротипични деца може да делат аспекти на церебрална морфологија заедно со аутизмот (58).

Два проблема кои повлекуваат истражувања во областа на нарушување во читањето се: 1) недостаток на согласен договор при што се дефинираат различните нарушувања во читањето и 2) специфично сиромашни критериуми. Во однос на претходниот проблем, дефинициите на дислексија се менуваа од публикации до публикации. Додека многу истражувања ја дефинираа дислексијата како дефицит од фонолошко потекло, тековини реvizии на Прирачникот за статистика и дија-

resentations”. Numerous studies have found that at-risk infants from multiplex dyslexic families exhibit abnormal speech processing even prior to language acquisition (53–55). Additionally, in functional imaging studies dyslexics show comparatively decreased activation in speech processing areas in the left hemisphere, with a poignant dysregulation of multimodal integration during phonological tasks involving inner speech (56). Studies have also found subtle yet enduring deficits in phonological processing in adolescents and young adults who once received the diagnosis but were no longer qualified (56, 57).

Hyperlexia has also had its share of definitional dilemmas. At one point it was considered a disability associated with neurodevelopmental conditions; eventually it was no longer a disability but a super- or savant ability; and now it is considered a combination of disability (poor reading comprehension) and ability (good-to-excellent word recognition)—or in the case of “neurotypical” individuals, no disability at all (4, 6, 50). In addition, there has been much debate as to whether hyperlexia is by itself an ASC, although Treffert (6) has addressed this, suggesting that while two of the three types of hyperlexia are associated with autistic or autistic-like behaviors, the third group is not considered on or near the spectrum. This final group, however, is often precocious and bright, and given the relationship intelligence appears to have with increased gray and white matter volumes, these neuro-typical children may share aspects of cerebral morphology in common with autism (58).

Two problems which have triggered a reading disorder research are: 1) lack of consistent agreement in what defines the various reading disorders and 2) poor criteria specificity. In regards to the former issue, definitions of dyslexia have varied from publication to publication. While many researchers have defined dyslexia as a deficit of phonological origin, current revisions for the Diagnostic and Statistical Manual of Mention Disorders
Specific learning disorder (DSM) will be collapsing the term “Reading Disorder” into the larger category of “Specific Learning Disability” which will simultaneously include other reading disorders (59). While we understand the desire for simplicity, the over-inclusive definition of “Specific Learning Disorder” utilized will subsume what many researchers and clinicians consider a variety of reading deficits, ranging from difficulties in phonological decoding (lower order) to general reading comprehension (higher order), making treatment of these conditions more difficult. A child, for instance, with a deficit in general reading comprehension may not benefit from the same intervention as one with classic dyslexia. In addition, research suggests that deficits in general reading comprehension are more common in school age children than is classic dyslexia (60, Error! Reference source not found.). Nevertheless, according to the proposed DSM-5 criteria, criterion A of Specific Learning Disorder will now be defined as (59):

<table>
<thead>
<tr>
<th>History or current presentation of persistent difficulties in the acquisition of reading, writing, arithmetic, or mathematical reasoning skills during the formal years of schooling (i.e., during the developmental period). The individual must have at least one of the following:</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Inaccurate or slow and effortful word reading;</td>
</tr>
<tr>
<td>2. Difficulty understanding the meaning of what is read (e.g., may read text accurately but not understand the sequence, relationships, inferences, or deeper meanings of what is read);</td>
</tr>
<tr>
<td>3. Poor spelling (e.g., may add, omit, or substitute vowels or consonants);</td>
</tr>
<tr>
<td>4. Poor written expression (e.g., makes multiple grammatical or punctuation errors within sentences, written expression of ideas lack clarity, poor paragraph organization, or excessively poor handwriting);</td>
</tr>
<tr>
<td>5. Difficulties remembering number facts;</td>
</tr>
<tr>
<td>6. Inaccurate or slow arithmetic calculation</td>
</tr>
<tr>
<td>7. Ineffective or inaccurate mathematical reasoning;</td>
</tr>
</tbody>
</table>
8. Avoidance of activities requiring reading, spelling, writing, or arithmetics. We commend the APA for including poor reading comprehension within the new criteria list, which will hopefully lend greater specificity than the previous criteria of Reading Disorder. However, the inclusion of several etiologically different reading disorders under a single heading may continue to promote similar confusions as before. Though the criteria are now separated, this new definition still blurs the line between a disorder of reading comprehension and classic dyslexia by subsuming them under the same diagnosis. Unfortunately, not only will diagnosis and treatment remain complex, it may continue to prove a challenge in studying etiological mechanisms of this heterogeneous group of conditions. Because structural and postmortem research already suggests divergent morphologies of the various reading disorders as our review of the postmortem and neuro-imaging literature illustrates, we would propose their separation rather than lump them under a single umbrella category. In this way, different reading disorders will be studied separately, the co-occurrence of secondary dyslexia with autism would be separated from primary dyslexia, and treatment modalities would be better suited to deal with the underlying deficits, e.g., reading comprehension versus word recognition. Thus, we would be better equipped to help deficient individuals improve their reading skills with precisely tailored programs, and research will not confound the study of etiology by including excessive heterogeneity within their samples.

Ресеряенци/ References


44. Ramus F. Neurobiology of dyslexia: A reinterpretation of the data. TRENDS in Neurosciences 2004; 27:720–726.

