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Analyzing Genetic Causes of Dyslexia to Provide Implications for Early Identification

Abstract

Dyslexia is a severe learning disorder that has always attracted various researchers who have debated on the causes and consequences of this particular disability from different angles. Numerous scientific works and articles have been written on this issue. Even though modern technologies provide scientists and researchers with specific tools that allow them to perform accurate analyses, the subject of Dyslexia still relies on hypotheses and assumptions rather than actual scientific evidence. That can explain the fact why the received findings are somewhat contradictory. Thus, a detailed analysis is needed to evaluate the results of the conducted research. The results of the studies should provide information as to better treatment of this disorder. This paper aims two-fold, to analyze genetic causes of Dyslexia, apply specific research evidence, and provide implications for early identification.

Keywords: Dyslexia, Genetic Causes, Early Identification.

Introduction

Dyslexia influences people's reading abilities, although it is wrong to consider them abnormal because they usually succeed in other activities. Some talented people, such as Sir Winston Churchill, Hans Christian Andersen, Thomas Edison, Albert Einstein, had Dyslexia, experiencing abstract thinking, concentration, coordination, observation, and recollection. Thus, this disorder changes the brain processing of the provided information, influencing not only reading skills but also the ways of communication. The person's brain mainly develops in the fetal period; during this formation, many brain cells move throughout the brain, passing via different connections that unite one neuron. This process occurs in the genes. Thus, if something influences development accuracy, some genes are affected, resulting in certain diseases like Dyslexia and other learning disorders. People with such disabilities can learn. However, they perceive information differently because dyslexic individuals' neurobiology differs from people without Dyslexia.

According to Miles and Miles (1999), 10% of the whole population in the world experience certain kinds of Dyslexia that decrease their learning abilities. However, the contradictory nature of this disorder prevents scientists from providing accurate findings regarding this learning disorder. Thomson (2009) opines that Dyslexia is a prolonged difficulty that undergoes various changes. In particular, young children experience complex reading and phonological skills, but as time passes, they start facing issues with the rapidity of writing and organizational activities. Davis (2009) considers that it is necessary to understand that learning disabilities are not a reflection of a student's intelligence, physical health, cultural background, or socioeconomic status.

Nowadays, researchers identify two principal causes for the phenomenon of Dyslexia: genetic and neurobiological causes. Neurobiological research conducted with the help of SPECT (single-photon emission computed tomography), MRI (magnetic

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resonance imaging), or PET (positron emission tomography) scans point at biological causes; in particular, they reveal a specific language area. The planum temporale is equal in left and right sides in a person with Dyslexia, while usually, the left side should be more significant. Some biologists consider this difference the cause of problems with some areas of learning in children and adults. Recent medical research indicates that Dyslexia and other learning difficulties possibly appear due to the wrong brain activity. Much genetic analysis explains these Neurological differences. According to these investigations and studies, Dyslexia is transferred from relatives to a dyslexic person through certain genetic links. Hornsby (2011) points out that more than 80% of dyslexic individuals have a close relative with similar learning disorders. Thus, if a mother or a father has Dyslexia, their child will also likely experience this disorder.

Materials and Methods

Systematic Reviews of Observational Studies guided the review. Articles based on observational studies on genetic causes of Dyslexia and early identification of the disorder were considered eligible for inclusion. Articles were excluded from the study if they did not meet the inclusion criteria.

This research made use of data from three databases. The initial search was performed on September 26, 2020, and encompassed all original articles published in English. It was restricted to "dyslexia" terms to minimize the risk of missing potentially relevant articles. In October 2020, the screening process started. After reviewing all titles and abstracts, all remaining articles underwent full-text screening. A thorough search of the reference list on October 28, 2020, produced other research works that the initial search missed.

Results

1. Genetic Role to Identify the Dyslexia

Earlier genetic studies had revealed certain inaccuracies due to the lack of appropriate analysis tools; recent research provides more accurate findings (Brkanac, Chapman, Matsushita, Chun, Nielsen, Cochrane, & Raskind, 2007). Twin studies (conducted in identical and fraternal pairs), family pedigree analysis, and sibling research are the most successful methods for identifying genetic causes of Dyslexia and similar learning disorders.

It concerns the orthographic or phonological skills of dyslexic individuals. To define these skills, scholars have applied the following measures towards the analysis: nonword reading to identify phonological processing (Stevenson, 2012). Nonword reading proposed by researchers and educators to use with the help of phonemes and morphemes. Pseudo homophone exercise aims at recognizing the correct word from two almost similar orthographic models (for example, "pane' and "pain"). A combination of both measures contributes to a more profound analysis of various aspects of Dyslexia from this point on. Stevenson (2012) suggests dividing orthographic coding into two exercises: the correct reading of unusual words (i.e., irregular verbs) and the recognition of words that have a similar sound form (for example, "buy" and "by"). Studies carried out in London and Colorado on phonological processing confirm the theoretical implications of these exercises. The results of the London study have shown that the reading of unusual words (that is, orthographic measures) has not revealed a considerable inherited level. In contrast, the distinction of words with similar sound forms (that relate to orthography) has pointed at a high level of heritability in the participants in the study.

Therefore, the research has suggested that genetic factors greatly influence phonological coding. Moreover, orthographic processing is different. According to Stevenson (2012), contrary to the London study, later Colorado findings reveal a higher level of inherited features in orthographic measures. That may be explained by the increased volume of samples or by another proband criterion. However, applying these findings to other received results (e.g., the twin's research) shows that the environment intensifies phonological and orthographic measures in a person's life, although this cannot be considered the principal reason for a learning disorder.

The Colorado research compared the similarity in behavior of same-sex and identical twins. It tested the DNA of dyslexic children and their parents or relatives influenced by both environmental and genetic factors. It is crucial to realize that identical twins are born from one sperm and egg. Thus, they use all their genes. Fraternal twins are born from two separate sperm and egg, sharing just some genes. As children take an active part in creating their surroundings (both in and beyond the bounds of the family), then non-similar genes of fraternal twins may contribute to more considerable environmental distinctions than genes of identical twins. For instance, one of two fraternal twins may have normal genes that control reading and spelling skills, while the other may possess dyslexia genes. Indeed, the latter twin will experience problems with reading and spelling; their results in school will be wrong compared to the first twin, whose genes tend to

be average or even excellent. This point can hardly be considered disputable, based on specific scientific evidence (Stevenson, 2012). Therefore, examining fraternal and identical twin pairs in the Colorado study has shown that genes significantly impact the lack of phoneme knowledge in dyslexic groups of individuals. With the help of twin studies, scientists can explore the genetic causes of dyslexia-related phenotypes.

Besides, a special two-dimensional examination elaborated for twin research may help to understand to what extent specific spelling and reading abilities connect to the same genes. If one of the twins lacks phoneme knowledge, it will almost definitely influence the other, particularly with identical twins. The situation with fraternal twins is slightly different because here, the second twin's phonological coding deficit is much lower than in their identical counterpart. It may explain the fact that fraternal twins have only 50% of similar genes. Two-dimensional research shows that the influence of genes on the knowledge of phonemes takes around 70% of all measures, and about 30% belongs to the environment (Gayán, Willcutt, Fisher, Francks, Cardon, Olson, & DeFries, 2005). The effect of genes on the phonological deficit in groups is about 70%, so the researchers assume similar genes influence nonword reading and phoneme knowledge.

Earlier research shows that dyslexia transfers throughout three generations of individuals where autosomal transfer acquires a dominant position (Sharma, Kohli& Sankhyan, 2021). In addition, reading disorders may be polygenic (that is, multi-factor). Polygenic means caused by more than one gene and includes environmental factors. It can also be monogenic (with only one gene) in nature. For instance, the polygenic model can apply when the level of Dyslexia in children depends on whether it is a mother or a father exposed to this disorder (Scerri, & Schulte-Körne, 2010).

Contrary to the polygenic model, the monogenic model is the actual location of a particular gene and its isolation. It is essential as the lack of expression of the definite gene in persons with the clear manifestation of the interconnection of mutations in the gene shows evidence that a particular gene is responsible for this disease. In this case, such gene isolation may result in substituting the defected gene with an identical copy, which will operate correctly and positively affect a person. However, some scientists argue about the monogenic nature of diseases as they claim. Those monogenic disorders tend to be more polygenic. In some cases, to define the severity and seriousness of illnesses, one needs to involve other genes rather than just one gene.

Therefore, twin monozygotic and dizygotic studies reveal the significance of genes in various learning disorders and depict the differences between genders. Besides, inventions in molecular genetics provide scientists with additional opportunities to recognize the particular genes responsible for Dyslexia. Molecular genetic research introduced in 1983 confirm heritable phenotypes (Asherson, & Curran, 2013). The results of these studies indicate that learning disorders, in particular Dyslexia, can be attributed to a combination of genes (either oligogenic or polygenic) rather than to one gene, as some scientists had previously suggested (Pennington, McGrath, & Peterson, 2019).

2. Chromosomes Responsible for Dyslexia

The successful analysis of complex disorders such as Dyslexia with the help of the so-called L.D. (linkage disequilibrium) mapping strategies (Fisher, 2006) provided more accurate results in contrast to previous studies. Contrary to standard linkage mapping, L.D. Mapping depends on segregation variety in natural populations, and thus samples received from this method comprise much more important meioses than in traditional mapping samples. Of course, no doubt that the successful consequence of L.D. Mapping also relies on other essential factors: the number of examined persons, analytical techniques applied, population structure, and other additional factors.

Testing dyslexic DNA has provided evidence that genetic effects on Dyslexia directly connect to chromosomes 1,2, 6, 15, and 18 (Grigerenko, 2003). Gayán, Willcutt, Fisher, Francks, Cardon, Olson, & DeFries (2003) proved this hypothesis. Grigorenko, for instance, applied information, which he and his colleagues received from extended families with reading disorders and revealed that there is quite a strong linkage between phoneme deficits to similar HLA on chromosome 6. Conversely, there is a weaker linkage to the said chromosome for word recognition, and it tends more to chromosome 15. Further study of additional genetic markers will prove whether definite genetic links bear responsibility for each person's different skills.

Nowadays, several different viewpoints exist on what exact gene or even a sequence of genes causes Dyslexia. The studies of a few scientific groups have shown that there are at least three genes that are responsible for causing this disorder, that is, DYX1 (on chromosome 15), DYX2 (on chromosome 6), and DYX 3 (on chromosome 2) (Scerri, & Schulte-Körne, 2010). According to Grigorenko (2003), six phenotypes influence the development of dyslexia-related cognitive

processes: phonemic awareness, phonological decoding, rapid naming, single-word reading, vocabulary, and spelling. Lately, a group of Finnish researchers identified the gene that bears the principal responsibility for dyslexia onset and its further aggravation (Proceedings of the National Academy of Sciences, 2003).

Earlier Genetic studies for Dyslexia found that chromosome 15 affects Dyslexia (Smith, Kimberling, & Pennington, 1991) more than chromosome 6 as seen in recent studies (Fisher & DeFries, 2002). Chromosome 6 seems to be the main chromosome affecting Dyslexia. Grigorenko (2003) found that the linkage of phonological awareness phenotype to 6p21.3 influences dyslexia and called DYX2 on chromosome 6- and single-word reading ascribed to chromosome 15. However, (Nöthen, Schulte-Koerne, Grimm, Cichon, Vogt, Müller-Myhsok, Remschmidt (1999) suggested that spelling and reading difficulty link to chromosome 15, but they found no strong confirmation for linking spelling difficulty with chromosome 6.

The researchers found out that the gene DYX1 (situated on chromosome 15) is either destroyed in people with Dyslexia or is in the wrong place, causing cells' production of a shorter protein version of DYX1 (Nöthen et al., 1999). Further, the scientists suggested that a break occurs in chromosome 15, and a significant amount of albumen is collected there (Nöthen et al., 1999). The scholars checked gene DYX1 in more than one hundred affected people and about two hundred non-affected children and grown-ups and got the following result.

At the first level of the Finnish research, the break of gene DYX1 occurred in about nine percent of affected people and less than three percent non-affected (Nöthen et al., 1999). At the second level, the break of the gene appeared in twelve percent of those with Dyslexia and about five percent of non-affected individuals (Bruce Bower, 2003). Moreover, the conducted analysis has shown that only definite (and not all) cells answer for the DYX1 protein; that is, there is still no information about the neural function of the protein.

In this regard, the molecular structure of human beings' DYX1 protein dramatically differs from the similar proteins in monkeys. As a result, several scholars consider that the detailed study of gene DYX1 may also help to find the differences between the human brains system and the brains of apes. Another group of scientists considers gene DYX3 (that belongs to chromosome 2) responsible for difficulties with reading and spelling (Fagerheim, Raeymaekers, Tønnessen, Pedersen, Tranebjærg, & Lubs, 1999). Of course, they suppose that it may not be the only gene that influences the onset of Dyslexia, but it confirms the genetic nature of this disease.

Doctor Toril Fagerheim states that she and her colleagues from Antwerp University in Belgium and Florida University in Miami managed to find the gene while examining a large Norwegian family with a long dyslexia pedigree (Nöthen et al., 1999). All the family representatives have passed nine tests to identify the disorder (including blood analysis). In the process of research, it was revealed that eleven out of thirty-six individuals have Dyslexia. Conducting the genetic analysis of all affected family members, the scientists have identified the gene link with chromosome 2.

Finding a short sequence of genetic substances between D2S2352 and D2S1337 markers suggests that this particular gene causes inherited Dyslexia in most infected individuals. Doctor Fagerheim considers that cloning the gene DYX3 will contribute to the easier detection of other genes that influence reading and spelling processes because external factors or the environment may cause Dyslexia.

Scientists from Sheffield University insist on the biological cause of Dyslexia. Nicolson, Fawcett and, Dean (2001) have put forward a hypothesis that Dyslexia may result from abnormalities in the cerebellum – the brain's part responsible for coordination, pose, and equilibrium. Using scanners and specially developed testing, they have found that cerebellum activity is 10% lower in affected people than in ordinary people. Thus, they aim their further research at evaluating how genes DYX1, and possibly some others, influence a person's brain and cause various disorders and how the albumen is responsible for other aspects that connect with the brain's activity, for instance, problems with speech and hyperactivity. It is necessary to examine more children and grown-ups with Dyslexia to confirm the results and define how the gene and its protein operate.

After a certain period, the number of families was increased by ten more pedigrees, and all families underwent research using parametric and nonparametric means (Smith, Kimberling & Pennington, 1991). Parametric and nonparametric measures have become very popular nowadays for determining complex diseases and disorders. It is an obligatory condition for parametric analysis to specify exactly the information about the suggested inheritance mode. They concluded that statistical maintenance for a locus (located near the analyzed markers) was responsible for causing diseases. To get accurate results, precise and robust statistical techniques for identifying complex disorders are necessary because there is an excellent possibility of the wrong specification of characteristics for the genetic

model, like gene frequency, penetrance, genetic heterogeneity.

Recently, two more research types have confirmed Smith's hypothesis regarding the locus connection to chromosome 15. Grigorenko (2003) initiated the first research. Moreover, the received outcome of the research group has shown that five different dyslexia constituents - reading of separate words, phonological knowledge, the deciphering of phonology, rapid naming, and discrepancy of composite words from the vocabulary – relate to chromosome 15.

Table 1.

Comparing Recent Linkage Studies of Dyslexia

Study	Materials and Methods	Phenotype	Chromosomal Regions	Results
Smith et al. (1991)	18 multiplex families Tests; LOD-Score Analysis, sib-pair analysis (QTL)	Reading disability	6p21,15cen -15qter	Evidence for linkage to 6p21.3, 15cen, and 15q15-qter; evid. for heterogeneity
Schulte-Körne, Deimel, Müller, Gutenbrunner, & Remschmidt, (1996)	Seven multiplex families, Tests; LOD-Score and nonparametric analysis	Spelling disability	6, 15	Evidence for linkage to 15q21
Fagerheim et al. (1999)	One multiple family with36 members; history and tests, LOD-Score and nonparametric analysis	Test results in two of 6 tests of a test battery: word reading with and without time constraint, phonological awareness (phenol. blending with words and nonwords), phonological decoding, spelling	2p16-p15	Evidence for linkage to 2p16-p15.
Morris et al. (2000)	178 parent-proband trios, association study	Reading disorder	15q15-q21	Highly significant association in both samples with a 3-marker haplotype
Fisher & DeFries, (2002)	82 families, 181 sib pairs, Tests, sib-pair analyses (QTL)	Four different phenotypes: word reading, IQ-reading discrepancy, orthographic coding, nonword reading	6p25-21.3	Evidence for a QTL in 6p21.3(reading of irregular words and nonwords)
Grigorenko (2003)	Eight multiplex families; Tests; LOD-Score and nonparametric analysis	Six different phenotypes: phonemic awareness, phonological decoding, rapid automized naming (RAN), single word reading, vocabulary, spelling	6p22.3-6p 21.3	Evidence for linkage of single-word reading, vocabulary, and spelling to 6p21.3
Gayán et al. (2005)	79 families, 126 sib pairs, Tests, sib-pair analyses (QTL)	Four different phenotypes: word reading, orthographic coding, nonword reading, phonological awareness	6p22. 3-p 21.1	Evidence for linkage of phonological and orthographic skills to 6p22.3-p21.3

In another study conducted by Morris, Robinson, Turic, Duke, Webb, Milham, & Williams (2000), the results revealed a considerable linkage between dyslexia and three-maker haplotype, particularly D15S146, D15S214, D15S994. These studies have suggested that the locus responsible for reading and spelling disorders directly connects to chromosome 15; however, the researchers consider that this area may include many genes, each of which may pass through correspondent mutation. Therefore, it is essential to narrow the chromosomal area, using corresponding molecular genetic methods and including candidate genes in further study. According to Doyle (2008), such methods used with specific remedial-teaching recommendations (unique learning approaches, educators specialized in Dyslexia) contribute to the treatment of Dyslexia, primarily if it identifies at an early stage. This aspect is discussed in detail in the next section.

5. Implication for Early Identification

Based on the research, scientists have suggested that Dyslexia can be successfully overcome if identified early and treated correctly. However, this success also depends on the complexity of Dyslexia. If this disorder is relatively mild, specific structural reading programs with particular stress on phonics and phonological aspects can improve the reading abilities of a dyslexic person approximately in a period of one year. In this regard, their problems connect with the incapacity to distinguish sounds. The situation will aggravate if dyslexic persons experience more severe issues such as speech disorder and memory deficit. In these cases, specialists need more time and more sophisticated programs (primarily based on advanced computer technologies) to cope with Dyslexia in individuals.

In addition, computers and other automated machines can provide an opportunity to conduct proper diagnostics of definite reading disabilities within groups at an early stage and suggest unusual environmental aid to improve a person's reading skills. Unquestionably, this way is especially crucial because Dyslexia often possesses various symptoms. It is sometimes difficult to distinguish this disorder from other learning difficulties that people (especially children) face. Smith, Kimberling, & Pennington (1991) used a few successful methods for identifying Dyslexia to find out the connection of reading disorder to Chromosome 15.

Researchers use parametric and nonparametric methods for such identification. The parametric linkage test requires exact and reliable data that are sometimes difficult to collect in several generations, negatively influencing the analysis. However, nonparametric methods can give accurate results even when parameters are unknown to researchers. Therefore, nonparametric methods can operate without information concerning inheritability. Moreover, penetration rates can determine genes susceptible to various disorders, including Dyslexia. The researchers have developed specific tests that can identify Dyslexia quite early. According to Reynolds & Caravolas (2016), the Bangor test of Dyslexia is relatively successful for recognizing disorders in verbal memory processes.

The Lucid Cognitive Profiling System and the Dyslexia Screening test depend on the literacy development processes, evaluating verbal and visual skills of children. The ACID profile test was also considered an essential tool for identifying certain learning disorders. However, Reid (2016) showed that this test did not recognize literacy problems in children, especially in dyslexic individuals. Researchers have proposed tests to analyze phonological abilities, such as the Phonological Abilities Test and the PhAB (Phonological Assessment Battery). Several recent advances in technology provide more exact data for the early identification of Dyslexia. For instance, C.T. (computed tomography) scanning allows for reconstructing a part of the brain and recognizing certain learning disorders, such as Dyslexia. The MRI (magnetic resonance imaging) provides an opportunity to reveal the differences in the parietal lobe of dyslexic and non-dyslexic persons. In particular, with this method's help, scientists have managed to identify that in dyslexic people, the angular gyms, specific segments of the parietal lobe are similar, contrary to normal individuals. Future research on Dyslexia will utilize these findings.

The PET (positron emission tomography) scan, the SPECT (single-photon emission computed tomography), and the rCBF (regional cerebral blood flow) are appropriate methods for evaluating those segments of the brain that respond to the process of reading. The AEP (averaged evoked potential) and the ERP (event-related potentials) techniques reveal the activity of the person's brain when it associates with a specific stimulus, for instance, a word. Generally, MRI (magnetic resonance imaging) and PET (Positron Emission Tomography) analyze normal incidence. MRI and PET research parents and children with dyslexia disorder, paying particular attention to genetic factors. However, "familiality" (or family relations) is not always a reliable confirmation of genetic heritability.

Based on the proposed methods, it is clear that the clinical environment, with the help of specific medical tools, is needed for early identification of Dyslexia analysis; however, further treatment can be conducted, applying to educational means (Reynolds & Caravolas, 2016). Such an approach contrasts the conventional methods for treating Dyslexia and similar learning disabilities, producing better results in most dyslexic people.

Discussion

The analysis in this paper has discussed the research on genetic causes of Dyslexia and its implication for early identification. Some studies have found that a few phenotypes directly link with spelling and reading disorders; however, casual linkages between these phenotypes require additional research. As genes influence the areas of the person's brain, scientists have to combine the findings of the studies in neuroscience and molecular neuroscience. This step is crucial because the high level of heritability does not always show that genetic factors fully affect spelling and reading disorders and are the only reason for the appearance of Dyslexia. There are many other environmental factors identified in twin studies that can contribute to the formation of Dyslexia. However, several studies have shown that genes are indirectly responsible for Dyslexia and other similar learning disorders.

The findings of various scientific studies have shown that the first symptoms of Dyslexia appear between the ages of three and four. Moreover, the considerable and crucial genetic interconnection between word definition and sound processing gives necessary proof that phonological coding directly depends on a genetically based perceptive process that affects phonological and orthographic abilities (Boets, Wouters, Van Wieringen & Ghesquiere, 2007). Providing persons with Dyslexia the appropriate information about this disorder and its treatment methods can help them cope with the disease in a faster and more effective way. Modern scientists should pay more attention to the

problem of Dyslexia and other learning disabilities, finding the location of dyslexia genes, and with the help of molecular research detecting dyslexia symptoms. This research will positively influence timely intervention and faster prevention or treatment and provide definite answers to the fundamental questions of neurobiology.

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