EDPS 648 Learning Task Two

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**Introduction**

Teachers must be prepared for meeting the needs of a wide and diverse variety of students in the classrooms. Providing support for students depends on understanding their strengths and limitations within an academic setting (Government of Alberta, 2018). The two most pressing concerns in education is meeting the needs of students with learning disabilities and mental health concerns. This paper will discuss the nature versus nurture debate in the two leading areas of stress for educators today. First of all, Alberta Education (2018) describes a student with a learning disability as someone who struggles to process information and experiences difficulties in the classroom. Some characteristics of a learning disability is having difficulties in the following areas; processing information, linking concepts, reasoning and understanding, communication and language, physical characteristics such as gross and fine motor skills, medical conditions, physical challenges such as visual and hearing loss along with difficulties with school and age-appropriate behaviours (Alberta Education, 2003). Finally, depression affects more than 350 million people worldwide and is currently the second leading cause of disability and disease burden throughout the world (Manincor, et al., 2016). Depressive symptoms include diminished interest in activities, weight loss or weight gain, fatigue and loss of energy, feelings of worthlessness, inability to think or concentrate and recurrent thoughts of death (American Psychiatric Association, 2013). The following sections review learning disabilities and depression within the classroom regarding nature versus nurture.

**Learning Disabilities and Genetics**

Learning disabilities can be understood as part of the cognitive development architecture where much of the early research on learning disabilities, genetics and environment began in the 1970s (Kovas et al. 2007). Learning disability research has tried to explain whether nature or nurture has a more significant influence on learning disabilities.

Several major studies such as the Institute for Behavioural Genetics and the Colorado reading study were long term, large scale studies that focused on dyslexia and other learning disabilities and its connection to genetic and environmental influences (Olson, 2002). Early studies measured the reading and cognitive abilities of students, siblings and families with dyslexia which provided a basic understanding that learning disabilities can be passed through inheritance. However, such studies were limited as family members share both similar genetics and environments (Olson, 2002).

**Twin Research**

Learning disability research has used long range studies which focused on identification, characterization and validation of reading, writing disabilities along, ADHD and executive functions (Colorado Learning Disabilities Research Center, 2018). Twin research has been used in which test batteries measure cognitive and academic abilities in a sample of identical and fraternal twins (Colorado Learning Disabilities Research Center, 2018). Data obtained by research will assess the etiologies of learning disabilities in reading, writing, ADHD along with phonological awareness, decoding, orthographic coding, vocabulary, comprehension, processing speed and executive functions (Colorado Learning Disabilities Research Center, 2018). DNA samples have also been used alongside LD research to study possible gene mutations (Colorado Learning Disabilities Research Center, 2018).

Research with genes associated with learning disabilities such as language impairments, reading and math disabilities have linked the same genes found in learning abilities, that there are genetic links within and between types of learning disabilities (Plomin & Kovas, 2005). Findings explained that learning disability genes are not generalist but researchers do not ignore the importance of environmental factors or the discussion on nature vs nurture (Plomin & Kovas, 2005). Learning disabilities have origins in multiple genes which differs from other disabilities which are linked to a single gene such as down’s syndrome (Plomin & Kovas, 2005). Plomin & Kovas (2005) explain that genes affect both learning disabilities and abilities and that there may be no distinct etiology found in quantitative genetic research as learning disabilities may be considered the low end of normal distributions of ability.

This identification of a learning disability in each twin in research is known as concordance where if one twin is affected then the other is also affected (Stromswold, 2001). Twin data on learning disabilities is understood as twin concordances and twin correlations which are based on whether the characteristics or phenotype is presented as an affected vs unaffected dichotomy (Plomin & Kovas, 2005). If identical twins (MZ) have stronger disability concordances than fraternal twins (DZ), then genetic influence is suggested, while high correlations of ability between twins also suggest a genetic influence (Plomin & Kovas, 2005). Therefore, heritability and genetic effect size can be found in both twin concordance for disability and twin correlation for ability (Plomin & Kovas, 2005). Stromswold (2001) reports the likelihood of a learning disability being identified in another twin is at 75% for identical (MZ) twins and 43% for fraternal (DZ) twins.

Kovas et al. (2007) explain that identical (MZ) twins are genetically the same so they must share more similarities however they may also share the same environment, and therefore treated similar within their environments which might skew results. Another benefit from using fraternal twins (DZ) in research is that they solving the problem of being treated the same as they will often look different and that half of all fraternal twins may be the opposite sex (Kovas et.al, 2007).

Another major twin sample and study was conducted by the Colorado Learning Disability Research Center study which consisted of 2690 individuals, with a mean age of 12 years, within 27 school districts (Olson, 2002). Results of the Peabody Individual Achievement Test provided evidence for genetic influences on dyslexia was found in areas such as word reading, spelling and comprehension (Olson, 2002). The genetic influences from this study linked a possible connection between dyslexia with the eye and how it processes visual stimuli (Stein, 2000), while another connected to a child’s attention span and how it can impair decoding skills (Olson, 2002).

Results from the National Merit Scholarship Qualifying test in the US had also found correlations for identical (MZ) twins at .70 and for fraternal (DZ) twins at .50 demonstrating heritability at around 40% and environmental at 30% (Kovas et al. 2007). While a European study was conducted from the results of the Dutch national test of educational achievement for 12 year olds and reported heritability for twins at 60% and shared environmental influences at 30% (Bartels, Rietveld, van Ball, & Boomsma, 2002).

**Nonverbal learning disabilities**

Nonverbal disabilities can be regarded as a subtype where a child may have average to above intelligence but struggle with visual, motor, social and academic difficulties (Dhanalakshmi, 2015). Early research from Austrian neurologist Gerstmann characterized the disability but left right confusion, finger agnosia (the ability to transmit information through your fingers), dysgraphia and dyscalculia (Dhanalakshmi, 2015). In the 1970s Rourke et al. focused his research on deficits such as visual and tactile tasks, psychomotor coordination, social competencies, academic performance, emotional functioning (Dhanalakshmi, 2015). Nonverbal learning disabilities may have origins in both genetic and environmental influences but research is inconclusive (Dhanalakshmi, 2015). Genetically, Nonverbal learning disabilities has similarities with Turner and Fragile X syndromes as the role of the cerebellum and the levels of dopamine and norepinephrine may be involved (Dhanalakshmi, 2015). Premature birth, radiation, chemotherapy and HIV are possible environmental conditions that may have reduced white matter or myelin-covered axons involved with processing found within the brain which can lead to several learning disabilities (Dhanalakshmi, 2015).

**Learning Disabilities and Environment**

There has been ongoing research in education and psychology on how an environment may affect a child’s learning (Kovas, Haworth, Dale, & Plomin, 2007). Environmental factors such as schools, teachers, facilities, education, poverty, crime, pollution and family systems are at work and have been a major source of research (Kovas et al.2007). Many of the nurture factors can be explained through Bronfenbrenner’s bioecological models (Berk, 2013). The environment was once limited to events and conditions surrounding the child, such as interactions with parents (Berk, 2013). Bronfenbrenner’s Bioecological expanded the environment into several structures that extend beyond the reach of parents (Berk, 2013). In teaching educators can use Bronfenbrenner’s models to understand factors around student success.

Kavale (1988) found a strong relationship between learning disabilities and low socioeconomic status. The prevalence and rate of children with learning disabilities in areas of low socioeconomic backgrounds is higher than the rate from those not found in low socioeconomic backgrounds (Blair & Scott, 2002). Blair and Scott (2002) found that 30% of male students and 39% of female students of LD placements can be attributed to low socioeconomic markers. A question for research would be whether the environment increases the likelihood of learning disabilities or whether the environment is a cause of learning disabilities (Blair & Scott, 2002).

**Depression and Genetics**

It is estimated that 30-40% of the risk for depression is genetically determined (Heim & Binder, 2012). A research study by Warner et al., (1999) showed that 49% of grandchildren were diagnosed with depressed if both a parent and grandparent were depressed as well. There are many contributing genetic factors on the influence of depression. The following section will discuss the three main genetic findings through which depression is detected.

**5-HTTLPR Gene**

The alternation in the levels of one or more monoamines in the serotonin linked polymorphic region 5-HTTLPR gene has been linked to be a contributor in the cause of depression (Capsi et al., 2003; Dean and Keshavan, 2017). The short allele in the 5-HTTLPR is connected to lower transcriptional efficiency and associated with an increase vulnerability to depression compared with the long allele (Capsi, et al., 2003). Individuals with one or more copies of a short allele of the H-HTTLPR gene demonstrate more depressive symptoms, and suicidal tendencies in relation to stressful life events compared to those who have the homozygous long allele (Capsi, et al., 2003). Research by Capsi, et al., (2003) illustrated that the interaction between 5-HTTLPR and stressful life events, such as childhood maltreatment, showed higher levels of depressive symptoms by the age of 26 among individuals carrying the short allele but not among l homozygotes. Heim and Binder (2012) argue that children with a short allele who are exposed to stressful life events and early trauma are more vulnerable to mood and psychiatric disorders as adults and are more likely to suffer from alcoholism, substance abuse, and sleep disorders. Periods of heightened plasticity of brain regions during childhood and the gene-environment interaction combined being a carrier of the short allele are more likely to cause depression as an adult (Heim & Binder, 2012)

**Brain-Derived Neurotrophic Factor Val 66-Met Polymorphism (BDNF)**

Brain-Derived Neurotrophic Factor is a neurotrophin that promotes the existence of neurons and growth of new neutrons and synapse in the brain (Dean & Keshavan, 2017). The BDNF polymorphism has been connected with reduced hippocampal volume and cortical morphology which increases the risk of depression (Ménard, Hodes, & Russo, 2016). Research by Schmitt, et al., (2014) illustrate that chronic stress increases adrenal corticosterone levels which causes a reduction in the hippocampal BDNF levels and an altered expression of neuroplasticity. A study involving 780 Chinese adolescent twins demonstrated that stressful life events on adolescent depressive symptoms were moderated by the BDNF Polymorphism. Those twins who were carriers of the Val allele in BDNF were more than to suffer from depression when exposed to stress compared to twins who carry the Met allele in BDNF (Chen, Li & McGue, 2013). Studies have shown that chronic stress causes a decrease in serum levels of BDNF and hippocamal volume which likely leads to the pathophysiology of depression (Dean & Keshavan, 2017).

**Hypothalamus-Pituitary-Adrenal** (**HPA) Axis Dysregulation**

Abnormalities in the HPA axis has been linked to depression (Dean & Keshavan, 2017). A hypersensitive HPA axis results in elevated cortisol levels in response to chronic psychological stress (Dean & Keshavan, 2017). Several studies have explored the mechanism underlying HPA axis dysregulation. Early deprivation and early life stress can cause HPA axis hyperactivity and can cause biological sensitivity to later stressors in life (Dean & Keshavan, 2017). Heim and Binder (2012) found that depressed patients with a history of child abuse have a hypersensitive HPA axis due to an increase in coritisol levels from the stress of abuse. Furthermore, they also found that prenatal stress increases HPA axis dysfunction the increase of glucocorticoid hormones negatively affected the growth of the placenta (Heim & Binder, 2012). Post-mortem and imaging studies of depressed patients show that psychological stress causes HPA axis hyperactivity and an increase in corticotropin-releasing hormones (CRH) in the hypothalamus and enlarged pituitary and adrenal gland volumes (Smart, et al., 2015).

**Depression and Environment**

Environmental influences such as, early life stress, chronic stress, abuse, neglect, and adverse family situations are all risk factors that are likely to increase the development of depressive disorders (Heim & Binder, 2012).

**Childhood Maltreatment**

Childhood maltreatment has shown a significant impact on mental health disorders in adults. Child abuse, including, physical, sexual and verbal abuse are more likely to report depression symptoms as adults than those from non-abusive families (Li, D’Arcy, Meng, 2016). Children with a history of abuse are two to three times more likely to suffer from depression compared to those without abusive pasts (Li, D’Arcy, Meng, 2016). In addition, childhood abuse is connected with reduced hippocampal volume, amygdale hyperactivity and an increased risk for suicidal behaviours as adults (Schmitt et al., 2014). Adolescents and adults are up to 5 times more likely to develop a mood disorder if they were neglected, traumatized, lost a parent or abused as children (Heim & Binder, 2012). Childhood is a sensitive time for development and the relationship between any maltreatment and abuse increases the disk for depression later in adulthood.

**Chronic Stress**

Chronic stress from issues such as sickness, work, family and relationship issues, past history of abuse or neglect can all are risk factors for developing a mood disorder. Schmitt et al., (2014) illustrated that chronic stress results in the degeneration of hippocampal neurons, thereby resulting in a loss of hippocampal volume, which increases the risk for mood disorders and psychic symptoms. Chronic stress from sicknesses such as autoimmune, metabolic, and cardiovascular disorder has also shown to increase depression (Teicher & Samson, 2013). Inflammatory diseases, cancer, and infections all require energy for treatment, coupled with added responsibility; such children, spouses/ partners, finances, and other activities can all lead to chronic stress, deterioration in health, and depression (Dean & Keshavan, 2017). Finally, stress from family environments such as, poor parental and partner relationships is associated with an increase risk for depression. Individuals in abusive relationships are at an increased risk for not only mood disorders, but post traumatic stress disorder (PTSD), substance abuse, sleep distorters, and are more likely to attempt suicide (Heim & Binder, 2012).

**Counselling implications**

**Learning Disabilities**

Students with access to counselling will improve their chances of academic success and graduation (Sicoli, 2006). Sicoli (2006) illustrates that only 60% of college students graduate and that a learning disability increases the odds of dropping out.  Pressure and stresses of academics on students demonstrate a need for additional supports such as counselling for students with a learning disability (Sicoli, 2006). Supports provided by counselling could include a positive therapeutic alliance which provides a safe environment and emotional support (Sicoli, 2006). Counselling could also complete proper assessments such as WISC and WIAT testing which will identify strengths and areas of improvement in an academic setting (Pearson, 2017). Counsellors can advocate for accommodations found within a student’s IEP/IPP (individual program plan) including extra time, reading software, scribes, tests on audio (Alberta Education, 2019). Counselling strategies such as CBT, positive psychology and narrative therapy can be used to handle issues associated with learning disabilities such as anxiety and depression (Sicoli, 2006).

**Depression**

Students suffering from depression are also more likely to also suffer from anxiety, eating disorders, and attention deficit disorders (Kumara & Kumar, 2016). Left untreated, students face numerous risks such as, poorer academic results, avoidance of social activities, and substance abuse problems (Kumara & Kumar, 2016).  Treatment for depression such as, pharmaceutical medications and/or therapeutic interventions is necessary as it may reduce the impact and persistence into adulthood (Connolly, Suarez, & Sylvester, 2011). Counsellors in the school system are trained in various areas around mental health to best fit the needs of each student (Alberta Education, 2019). In addition, counsellors can advocate for accommodations including, extra time, scribes, and scaffolding assignments (Alberta Education, 2019).  Counselling strategies such as CBT, ACT, positive psychology, and mindfulness- based reduction strategies are the most common forms of therapeutic interventions for those suffering from depression (Corey, 2011; Galla, et al., 2015).

**Conclusion**

The nature versus nurture debate has been a heavily discussed issue in many areas of research. Do genetic abnormalities cause disabilities and disorders or environmental influences?  Whether learning disabilities develop from the influence of genetics or from our environment has been an area of research for decades. In regards to learning disabilities Mizen & Cooper (2012) state that there may be thousands of causes for learning disabilities and that it would be quite difficult to understand any of these without comprehensive assessments. Plomin (2004) however, explains that current research points to both nature and nurture contributing to a child’s learning development. Furthermore, in regards to depressive disorders, the answer found throughout research is that genetic dispositions and environmental stressors interact with each other which cause depression. Dean and Keshavan (2017) stated, “It is a combination of stressors and a pre-existing vulnerability, of nature and nurture that causes disorder” (p. 104). Improving knowledge and beliefs surrounding mental health concerns and learning disabilities will help teachers become educated on how to best serve the needs of students suffering.

**References**

Alberta Education. (2003). *The Learning Team A handbook for parents of children with special needs.* Retrieved from Alberta Education: https://education.alberta.ca/media/3531893/learning-team-handbook-for-parents.pdf

Alberta Education. (2019). *IPP/ISP Overview.* Retrieved from Alberta Education: https://education.alberta.ca/instructional-supports/individualized-program-plan-ipp/?searchMode=3

American Psychiatric Association, (2013). *Diagnostic and statistical manual of mental*

*disorders.* (5th ed.) Arlington, VA: Author.

Berk, L. (1997). *Child development, fourth edition.* Boston: Pearson.

Berk, L. (2013). *Child Development 9th Edition.* Boston: Pearson.

Blair, C., & Scott, K. (2002). Proportion of ld placements associated with sow socioeconomic status: evidence for a gradient? . *The Journal of Special Education, 36, 1*, 14-22.

doi: 10.1177/00224669020360010201

Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., … Poulton, R. (2003). Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science,* *301*(5631), 386-389. doi:10.1126/science.1083968

Chen, J., Li, X., & McGue, M. (2013). The interacting effect of the BDNF Val66Met polymorphism and stressful life events on adolescent depression is not an artifact of gene-environment correlation: Evidence from a longitudinal twin study. *Journal of Child Psychology and Psychiatry,* *54*(10), 1066-1073. doi:10.1111/jcpp.12099

Colorado Learning Disabilities Research Center. (2018). *Colorado Learning Disabilities Research Center*. Retrieved from University of Colorado Boulder: http://ibgwww.colorado.edu/cldrc/

Connolly, S., Suarez, L., Sylvester, C. (2011). Assessment and treatment of anxiety disorders in

children and adolescents. *Current Psychiatry Reports, 13*(2), 99-110. DOI:

10.1007/s11920-010-0173-z.

Corey, G. (2011). *Theory and Practice of Counselling & Psychotherapy*. Toronto, ON: Nelson

Education, Ltd.

# Dean, J., & Keshavan, M. (2017). The neurobiology of depression: An integrated view. *Asian Journal of Psychiatry, 27*, 101-111. doi:10.1016/j.ajp.2017.01.025

Dhanalakshmi, D. (2015). Nonverbal learning disabilities. *Indian Association of Health, Research and Welfare, 6*(1), 109-113.

Dingle, S. (2010). Relationship between test performance of students with learning disabilities and the poverty levels of their schools. *A thesis presented to the faculty of the department of curriculum & instruction east carolina university*.

Galla, B. M., O'Reilly, G. A., Kitil, J. M., Smalley, S.L., & Black, D. S. (2015) Community-

based mindfulness program for disease prevention and health promotion: Targeting stress

reduction. American Journal of Health Promotion, 30(1), 36-41.

Government of Alberta. (2018). *Diverse Learning Needs*. Retrieved from Alberta Education: https://education.alberta.ca/diverse-learning-needs/learning-disabilities/?searchMode=3

Heim, C., & Binder, E. B. (2012). Current research trends in early life stress and depression: review of human studies on sensitive periods, gene-environment interactions, and epigenetics. *Experimental Neurology, 233*(1), 102-111. doi:10.1016/j.expneurol.2011. 10.032

Integrated Learning Strategies. (2018). *Learning Corner*. Retrieved from Nature versus Nurture: Why My Child's Learning Development is a Product of the Environment I Create: https://ilslearningcorner.com/2016-07-nature-versus-nurture-why-my-childs-learning-development-is-a-product-of-the-environment-i-create/

Jensen, E. (2005). *Teaching with the Brain in Mind, Revised 2nd Edition.* ASCD.

Kovas, Y., Haworth, C. M., Dale, P. S., & Plomin, R. (2007). The genetic and environmental origins of learning disabilities and disabilities in the early school years. *Mongraphs of the Society for Research in Child Development*.

Kumara, H., & Kumar, V. (2016). Impact of cognitive behavioural therapy on anxiety and

depression in adolescent students. *Journal of Psychosocial Research, 11*(1), 77-85.

Li, M., D'Arcy, C., & Meng, X. (2016). Maltreatment in childhood substantially increases the risk of adult depression and anxiety in prospective cohort studies: systematic review, meta-analysis, and proportional attributable fractions. *Psychological Medicine, 46*, 717–30. doi:10.1017/S0033291715002743

Manincor, M. , Bensoussan, A. , Smith, C. A., Barr, K. , Schweickle, M. , Donoghoe, L. , Bourchier, S. & Fahey, P. (2016). Individualized yoga for reducing depression and anxiety, and improving well- 100being: A randomized controlled trial. Depression & Anxiety, 33, 816-828. doi:10.1002/da.22502

Mizen, L., & Cooper, S.-A. (2012). Learning disabilities. *Psychiatric Disorders*.

doi: 10.1016/j.mpmed.2012.08.001

Olson, R. K. (2002). Dyslexia: nature and nurture. *DYSLEXIA, 8*, 143-159.

doi: 10.1002/dys.228

Pearson. (2017). *Using the WISC-V and WIAT-III to Diagnose Learning Disorders .* Retrieved from Using the WISC-V and WIAT-III: http://www.pearsonclinical.com.au/filemanager/uploads/Other%20Files%20to%20share/Using%20the%20WISC-V%20&%20WIAT-III%20to%20diagnose%20Learning%20Disorders%20-%20handouts.pdf

Plomin, R., & Kovas, Y. (2005). Generalist genes and learning disabilties. *Psychological Bulletin, 131*(4), 592-617.

doi: 10.1037/0033-2909.131.4.592

Sicoli, M. (2006). Counseling strategies for college students with learning disabilities. *Journal of Reading, Writing, and Learning Disabilities International*.

doi: 10.1080/0748763860020406

Schmitt, A., Malchow, B., Hasan, A., & Falkai, P. (2014). The impact of environmental factors in severe psychiatric disorders. *Frontiers in Neuroscience, 8*(19), 1-10. doi:10.3389/ fnins.2014.00019

Smart, C., Strathdee, G., Watson, S., Murgatroyd, C., & McAllister-Williams, R. H. (2015). Early life trauma, depression and the glucocorticoid receptor gene—an epigenetic perspective. *Psychological Medicine, 45*(16), 3393–3410. doi:10.1017/ S0033291715001555

Ménard, C., Hodes, G. E., & Russo, S. J. (2016). Pathogenesis of depression: Insights from human and rodent studies. *Neuroscience,* *321*, 138-162. doi:10.1016/j.neuroscience.2015. 05.053

Stromswold, K. (2001). The heritability of language: a review and metanalysis of twin, adoption and linkage studies. *Linguistic Society of America*, 647-723.

doi: 10.1353/lan.2001.0247

Warner, V., Weissman, M. M., Mufson, L., & Wickramaratne, P. J. (1999). Grandparents, parents, and grandchildren at high risk for depression: a three-generation study. *Journal of the American Academy of Child & Adolescent Psychiatry, 38*(3), 289–296. doi:10.1097/00004583-199903000-00016