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The Link Between Genetics and Depression
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Introduction
• The intention of this poster is to discuss the link between genetics and depression.
• Depression is known to be related to nature and nurture.
• Parents worry about the safety and happiness of their children, and no parents want their child to suffer from mental illness, especially if the parent himself suffers from it.
• This topic was chosen to gain insight on the possibility of psychological disorders such as depression being passed on to offspring.
• Depression can be seen in multiple generations, but it can also affect those with no family history.

Signs and Symptoms of Depression.
• Persistent sadness and loss of interest, poor memory, thoughts of suicide and death (Fakhoury, 2015).
• Extreme sadness, worthlessness, or hopelessness, changes in habits, persistent fatigue, mood swings, difficulty concentrating, wanting to be alone (Harvard Health Letter, 2018).

Pathophysiology – Environment.
• Depression is mostly associated with environmental factors such as stress and trauma, but can be caused by genetic, biological, and psychological factors (Fakhoury, 2018).
• Individuals under a large amount of stress for a prolonged period of time can develop hypercortisolism which can lead to symptoms of depression (Sharpley et al., 2018).
• Stress also causes a reduction in the production of the brain-derived neurotropic factor (BDNF) protein that contributes to an individual’s ability to think clearly, which is another symptom of depression (Sharpley et al., 2018).

Pathophysiology – Genetics.
• A study by Pettersson et al. (2019) examined the genetic link between siblings and psychological disorders. It was determined that common genetics variants play a large role in the development of major depressive disorder (MDD) and other psychiatric disorders.
• While environment may increase the risk of an individual being clinically depressed, genetics contribute greatly as far predisposition to being influenced by environment.
• Personality is linked to a person’s vulnerability as well (Navrady et al., 2018).
• Scientists have discovered a linkage between the 12q23 region of the human genome and the risk of developing MDD (Fakhoury, 2018).
• Those with MDD often have an imbalance of multiple neurotropic factors. These neurotropic factors can be seen in Table 1.
• Each neurotropic factor gene has a different mechanism of action. If the chromosome that the gene resides on is mutated, then the gene may be altered, thus altering its function and increasing the risk of the individual exhibiting depressive symptoms (see Table 1).
• MDD is characterized by an altered level of serotonin (S-HT), and genes that control the synthesis or inhibition have the potential to influence the severity of the disorder. S-HT levels can be altered by the serotonin transporter protein (S-HTT), S-HT receptors (S-HTR2A), and tryptophan hydroxylase 2 (TPH2) (Fakhoury, 2018).
• Additionally, individuals with one or two copies of the short allele of the S-HTT polymorphism are more likely to be affected by stressful life events and show more depressive symptoms than those homozygous for the long allele (Fakhoury, 2018).
• Changes in gene expression have also been linked with MDD. For example, “DNA methylation of BDNF is linked to the pathophysiology of depression due to reduced neural levels of BDNF in the brain” (Fakhoury, 2018, p.173). This can happen as a response to environmental influences such as diet and drug use. In sum, environmental factors can alter genetic expression causing depressive symptoms.

Table 1 Candidate genes involved in the pathophysiology of MDD.

<table>
<thead>
<tr>
<th>Gene</th>
<th>Location</th>
<th>Name</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>S-HTR2A</td>
<td>Chromosome 13</td>
<td>Hydrotroplamine receptor 2A (HTTR2A)</td>
<td>G-protein coupled serotonin receptor that can activate signal transduction inside the cell</td>
</tr>
<tr>
<td>S-HTT</td>
<td>Chromosome 17</td>
<td>Serotonin transporter protein (S-HTT)</td>
<td>Transports serotonin from synapses to presynaptic neurons</td>
</tr>
<tr>
<td>BDNF</td>
<td>Chromosome 11</td>
<td>Brain-derived neurotropic factor (BDNF)</td>
<td>Promote the survival of neurons by preventing apoptosis</td>
</tr>
<tr>
<td>FGF</td>
<td>Chromosome 6</td>
<td>Fibroblast growth factor (FGF)</td>
<td>Important growth factor that ameliorates hippocampal neurogenesis</td>
</tr>
<tr>
<td>IGF</td>
<td>Chromosome 12</td>
<td>FK506-binding protein 5 (FKBPs)</td>
<td>Involved in immunoregulation and in the modulation of stress and anxiety</td>
</tr>
<tr>
<td>TPH2</td>
<td>Chromosome 12</td>
<td>Tryptophan hydroxylase 2 (TPH2)</td>
<td>Involved in serotonin synthesis increases endothelial cell number and promotes neurogenesis, neuronal cell survival and synaptic plasticity</td>
</tr>
<tr>
<td>VEGF</td>
<td>Chromosome 6</td>
<td>Vascular endothelial growth factor (VEGF)</td>
<td>Promote the survival of neurons by preventing apoptosis</td>
</tr>
</tbody>
</table>

Significance.
• The genetic risk for MDD that presents in adulthood is higher than that for MDD that presents in childhood (Mistry et al., 2018). This is significant because it is more likely that it will be unknown if the child will develop MDD until he or she reaches adulthood thus possibly delaying interventions to reduce the severity of the disorder.
• Additionally, a family history of psychotic illness increases the chance of the individual having a more severe form of MDD (Mistry et al., 2018). If this is well known, earlier treatment options may be available as well as the drive for more advanced screening tools. These resources could decrease the risk for suicide, need for antidepressants, and increase the individual’s productivity level.
• Knowing exactly which genes control depressive disorders is essential in the development of medications for treatment. For example, SNRIs inhibit the reuptake of serotonin and dopamine, and serotonin levels are influenced by specific genes.

Conclusion.
• In conclusion, depressive disorders are the product of environmental, biological, and psychological factors. Understanding how genetics plays a role in the development and progression of depression will facilitate the improvement of treatment plans and medication. It is known that environmental factors can alter genetic factors, therefore it is important for nurses and providers to deliver proper education to patients, especially expecting parents, on the affects of certain life choices on mental health.

Nursing Implications.
• Nurses could be trained in genetic counseling to help expecting parents determine the risk of their child developing a depressive disorder.
• Better understanding of the cause of depression will lead to better understanding of effective treatment and medications.
• If a nurse understands that the patient isn’t just “crazy” or “emotional”, then treatment will be conducted similarly to that of physical disorders instead of as if it is all in the patient’s head.
• Nurses will be able to better educate patient’s about depression and what has caused the patients individual case.

References
• Fakhoury, 2018