

CLINICAL DEPRESSION LESSON PLAN
DEPARTMENT OF NEUROPSYCHIATRIC DISORDERS

Week: 148 **Course:** Advanced Integrated Physiological / Psychological Experience **Periods:** 1, 2, 3, 4, 5 **Developed In Collaboration With:** Empirical Reality **Subject:** _____

Monday	Tuesday	Wednesday	Thursday	Friday
<p>Introduction:</p> <p>Let's explore the various sociological / environmental factors of clinical depression (i.e. loss of a loved one, physical abuse, neglect, divorce, financial instability etc.).</p>	<p>Introduction:</p> <p>Let's look at the role genetics plays in clinical depression! Studies have identified a region on the short arm of chromosome 3 (3p25-26) linked to recurrent episodes of clinical depression.</p>	<p>Introduction:</p> <p>Let's use our noggins and dig on into the ol' grey matter to see just how clinical depression can lead to structural changes in the brain both in terms of size and functionality.</p>	<p>Introduction:</p> <p>Circle time! Let's all gather 'round and examine the monoamine hypothesis whereby the depletion of serotonin, dopamine and other neurotransmitters in the central nervous system is said to account for many of the symptoms associated with clinical depression.</p>	<p>Introduction:</p> <p>Let's take a journey together into the world of cognitive behavioral therapy, psychoanalysis, antidepressants, electroconvulsive therapy (ECT), transcranial magnetic stimulation (TMS), and all the other options for treatment of clinical depression!</p>
<p>Activity #1:</p> <p>Subject experiences separation from or death of a parent before the age of 11. This will likely prompt great emotional upheaval and pain/take away subject's sense of control.</p>	<p>Activity #1:</p> <p>Conduct genome-wide association study (GWAS) wherein blood samples are provided by subject and subject's family to determine a pattern of inheritance/susceptibility to clinical depression.</p>	<p>Activity #1:</p> <p>The hippocampus regulates cortisol. Excessive cortisol is released during depressive episodes resulting in diminished production of new brain cells, shrinkage of extant cells, memory loss etc. Allow this to happen to subject</p>	<p>Activity #1:</p> <p>Speaking of serotonin (5-HT) restrict its production within subject thereby hindering the relay of messages from one area of the brain to another. Results will be stored in subject's portfolio and evaluated.</p>	<p>Activity #1:</p> <p>Selective serotonin reuptake inhibitors (SSRIs) limit reabsorption of serotonin in presynaptic cells / increase serotonin available to bind to postsynaptic receptors. Let's observe the biochemical effects of this drug on subject.</p>
<p>Vocabulary:</p> <p>HOPELESSNESS, VULNERABILITY, CODEPENDENCY, DESPAIR</p>	<p>Vocabulary:</p> <p>GENOTYPE, NUCLEOTIDE, POLYMORPHISMS, PHENOTYPE</p>	<p>Vocabulary:</p> <p>ENDOCRINE, RAPHE NUCLEI, SUBGENUAL CINGULATE, NEURALPLASTICITY</p>	<p>Vocabulary:</p> <p>SYNAPSE, AXONAL MEMBRANE, METABOLITES, EPINEPHRINE, NONREPINEPHRINE</p>	<p>Vocabulary:</p> <p>SERTRALINE, VENLAFAXINE, DISCONTINUATION SYNDROME, ANORGASMIA, HEALTHY DIET</p>
<p>Activity #2:</p> <p>Sexual trauma. All external sources must be cited in order for subject to receive proper accreditation.</p>	<p>Activity #2:</p> <p>A new spin on an old favorite! Subject should bring in a parent and/or sibling to explain the details of their own recurrent depressive episodes. Pancake breakfast at 9am!</p>	<p>Activity #2:</p> <p>Same as Activity #1 but with subject's amygdala where excess cortisol results in hyperactivity. Compare / contrast abnormalities in this region with those in subject's hippocampus.</p>	<p>Activity #2:</p> <p>Dopamine exerts its effects by binding to and activating cell surface receptors. Prohibiting its synthesis will severely alter subject's reward-motivated behavior. Visual aids are suggested.</p>	<p>Activity #2:</p> <p>Psychotherapy. Like show and tell, but instead of a pet turtle or collection of gemstones subject shares negative thought patterns, counter-productive behavior, lack of coping skills etc.</p>
<p>Assessment:</p> <p>Did a stressful event such as those listed above trigger the subject's first or second depressive episode? The KINDLING-SENSITIZATION HYPOTHESIS theorizes initial depressive episodes spark changes in the brain's chemistry and limbic system that make the subject more prone to recurrent depressive episodes. Refer to attached flowchart for further info.</p>	<p>Assessment:</p> <p>Can clinical depression be inherited? The jury's still out, but studies suggest heritability is 40-50%. Wow! In fact, 214 genes in the region of 3p25-26 have been linked to recurrent episodes of clinical depression. Encoded within these very genes are the receptors of numerous proteins, amino acids and brain-signaling chemicals incl. 5-HTT, which regulates serotonin uptake.</p>	<p>Assessment:</p> <p>A real brain-teaser, huh? Take the prefrontal cortex—responsible for processing emotions and decision-making—which, like the hippocampus, atrophies due to excess cortisol and thereby inhibits normal cognitive functioning. The ventromedial and dorsolateral sectors of the prefrontal cortex are particularly effected and lead to the pathogenesis of still further symptoms of clinical depression.</p>	<p>Assessment:</p> <p>Deficient levels of serotonin result in impulsivity, aggressive behavior, suicidal ideation. Deficient levels of dopamine result in diminished feelings of pleasure, lack of motivation, decreased motor skills. If subject displays such symptoms he/she is likely experiencing the neurophysiological effects of clinical depression, though there is still debate regarding this theory. Please see attached worksheet.</p>	<p>Assessment:</p> <p>For those who've experienced 3+ depressive episodes it's advised they stay on medication indefinitely. Yet very little research supports the theory clinical depression results from chemical imbalances. The side effects of meds meant to offset chemical imbalances (insomnia, weight gain, anxiety, sexual dysfunction) are severe enough to lead to still further depressive episodes. Refer subject to some sort of graph.</p>
<p>Independent Study:</p> <p>Clinical depression should impact all aspects of subject's everyday life including eating, sleeping, working, relationships, and how subject thinks about himself/herself. Fun fact: Clinical depression affects approximately 19 million Americans!</p>	<p>Independent Study:</p> <p>Clinical depression should impact all aspects of subject's everyday life including eating, sleeping, working, relationships, and how subject thinks about himself/herself. Did you know? Women are two times more likely to suffer from clinical depression than men. Sorry ladies!</p>	<p>Independent Study:</p> <p>Clinical depression should impact all aspects of subject's everyday life including eating, sleeping, working, relationships, and how subject thinks about himself/herself. Here's another brain-teaser: Why are African-Americans more likely to develop clinical depression than white people?</p>	<p>Independent Study:</p> <p>Clinical depression should impact all aspects of subject's everyday life including eating, sleeping, working, relationships, and how subject thinks about himself/herself. Strange but true: 20% of people with clinical depression develop symptoms of psychosis incl. hallucinations, delusions and paranoia!</p>	<p>Independent Study:</p> <p>Clinical depression should impact all aspects of subject's everyday life including eating, sleeping, working, relationships, and how subject thinks about himself/herself. Believe it or not: Up to 30% of clinically-depressed people may be suffering from treatment-resistant depression!</p>