Causes of depression pdf articles

Watson, Stephanie. Depression. Gail Health and Wellness Online Collection, Gale, 2018. Depression is a mood disorder that causes constant feelings of sadness, despair, and decreased energy. To be diagnosed with major depression, you must have symptoms that are constant for at least two weeks. But depression is more than just an misfortune. It can be serious enough to interfere with work, school, and other daily activities. Doctors also refer to this condition as a major depressive disorder or clinical depression. According to the American Psychiatric Association, one in six or nearly 17 percent of Americans expect to experience depression at some point in their lives. About 7% of Americans had at least one major depressive episode last year. Although symptoms can begin in any age, depression is likely to start during a person's teens or 20s. With major depression and depression, there are several different types of depression, characterized by their symptoms or causes: Dysthymia, or persistent depression disorder, a mild form of depression where symptoms persist for at least two years. Postpartum depression involves feelings of extreme sadness, fatigue, anxiety that begin after a woman gives birth. Disorder (PMDD) is severe depression, irritability and anxiety that occurs in a week or two weeks before the period in women. Seasonal affective disorder (SAD) is a depression that occurs during the winter months and is alleviated by seasonal changes. Bipolar disorder, formerly called mood swings, is characterized by alternating episodes of very low mood (depression) and energetic best (mania). Psychotic depression includes characteristics of depression and psychosis (hallucinations), such as having false beliefs (delusions) or seeing and listening to things that are not there. Depression comes from a combination of biological, environmental and psychological factors. People with depression often have family members with the condition, suggesting that genetics are related. If one biological twin has depression, the other twins also have a 70% chance of having the condition. Researchers have found differences in the brains of people with depression, in the ability of chemical messengers called neurotransmitters. Hormonal changes may also initiate depressive symptoms; For example, during a woman's menstrual cycle or after childbirth. Almost every day for at least two weeks of the following symptoms: persistent sad or empty mood feelings of despair, helplessness, emptiness, worthlessness, or guilt, low energy, fatigue irritability, anxiety, slow thoughts, speaking, or loss of interest in exercise once enjoyed difficulty, memory, or loss of appetite, or eating too much weight loss or too much loss of weight, abdominal pain, and other pain and depression that are not a clear cause of death or suicide. Others will only have a few. The severity of depression symptoms can vary seriously from light enough to affect a person's daily life. The diagnostic doctor begins the diagnostic process with physical examination and laboratory work to rule out possible physical causes of depression, such as thyroid disorders or vitamin deficiency. A psychologist or doctor can make a psychological assessment, ask questions and assess symptoms according to established criteria for identifying depression and getting to diagnosis. Treatments typical for depression include antidepressants or other medications, psychotherapy (story therapy), or a combination of two interventions. Personalizing treatment to individuals can increase your chances of success. Antidepressants are a class of drugs used to treat depression. They include the following types: Selective Serotonin Reuptake Inhibitors (SSRIs) are often the first drugs prescribed by doctors for depression. These drugs affect chemical messengers, Serotonin, and help regulate mood. Low Serotonin levels have been linked to depression. Serotonin- Norepinephrine reuptake inhibitor (SNRIs) works on two brain chemicals - Serotonin and Norepinephrine. Atypical antidepressants work in the brain in a different way than other antidepressants. These drugs may be an option for people who have not found relief from SSRIs or SNRIs. Trisagoic antidepressants are a previous class of antidepressants. They work on three brain chemicals: Serotonin, Norepinephrine, and Dopamine. Tricles are not as frequently used as they once because they are at higher risk for side effects than new antidepressants. Monoamine oxidase inhibitors (MAOIs) sometimes doctors will prescribe another type of medication, such as anti-anxiety drugs, antipsychotics or stimulants, along with antidepressants. Antidepressants can take up to four weeks to start work. It may take some attempts to find the best medications and dosage combinations to alleviate your depression. Story therapy programs like Cognitive Behavioral Therapy (CBT) help people with depression identify negative thoughts and behaviors caused by depression and replace them with more positive strategies for coping skills and psychological resilience. Treatment can be done one-on-one As a member of a group or with a partner or other family member. If such treatment does not work, electroconvulsive therapy (ECT) can be an option. ECT is performed while under general anesthesia. A small current passes through the brain to induce seizures. Research finds that ECT is often effective if antidepressants and talk therapy fail. Some alternative remedies and supplements are used to treat depression, including acupuncture, meditation, guide images, and taepole chi. herbal supplements like St. John's wort and SAMe are inconclusive evidence of help for depression symptoms, and fda-approved treatments. Because these supplements can often cause side effects or interact with other medications you are taking, warn your doctor first if you want to try them. Prospect depression can be very treatable. Up to 90% of people will eventually be improved with medication, treatment, combination of the two or other treatment. However, it can take some trial and error to find the best treatment for you. From 1-800-662-HELP (4357) to the U.S. Substance Abuse and Mental Health Service Administration (SAMHSA) helpline, we can help people who are struggling with depression. Suicidal thoughts and actions can occur in people who are severely depressed. If you have any intention of hurting yourself, call a trusted healthcare provider immediately, contact a support friend or family member, or call the National Suicide Prevention Lifeline at 1-800-273-TALK (1-800-273-8255). If you are at risk of self-harm, call 911 or local emergency services. Resource website The National Institute of Depression Mental Health. February 2018. (accessed February 13, 2020). Depression (major depressive disorder). Mayo Clinic. February 3, 2018. (accessed February 13, 2020). What is depression? American Psychiatric Association. January 2017. (accessed February 13, 2020). Organized American Psychiatric Association Street 800 Main Avenue, S.W., Suite 900 Washington, D.C. 20024 Call (888) 357-7924 National Alliance for mental illness (NAMI) Street 3803 N. Fairfax Drive, Suite 100 Arlington VA 22203 Phone (800) 950-6264 National Mental Health Street 6001 Executive Boulevard, Room 6200 Bethesa MD 20892-9663 (866) 615-6464 this issue contains four numbers of articles on the main aspects of the main aspect. Three suggests that the effects we often think of may actually cause depression. Overall, the article points to a number of important questions worth of further research. Over the past few years, two main brain regions Implicated in major depression - the frontal cortex / anterior cingulate and hippocampus. The article in this issue points to anomalies that include one of these areas, including Frodl et al. and Lockwood et al. Report on major neuropsychological damage in depressive patients such as Lockwood, based on comparisons with healthy subjects. Elderly subjects demonstrated impairments in the performance of the work of the executive function (suggestive of the frontal lobe/anterior cingulate function), and in particular the disorder was seen in the elderly depressed subjects. These findings support a number of other observations involving the frontal lobe area in depression (1, 2). Comprehensively, these data point to the main consequences or correlations of depression at first glance, which means that cognitive dysfunction associated with prefrontal cortex/anterior cingulate, but also suggests that performance reductionin older people including this area may be a risk factor for being severely depressed. Frodl et al. The first episode explores the hippocampus volume in patients with major depression. Sheline et al. (3) and Bremner et al. (4) reported small hippocampus volume in more depressed patients in healthy subjects. Further, Sheline et al. (3) reports that small hippocampus depression is associated with a larger lifetime period and suggests that excessive glucocorticosteroid activity may be a factor in visible atrophy. These findings extend the days of rats and Sapolski (5). An article by Frodl Et. raises serious questions about the importance and cause of small hippocampus volumes. In fact, studies suggest that the facts involving first-episode patients, especially in men, are risk factors for rather than the consequences of major depression. This data fits well with the results from three other recent studies that point to a strong genetic impact on hippocampus volume. My colleague and I (6) used a model that included a paternity half-brother among squirrel monkeys to explore the early life stress and relative contribution of genetics to hippocampus size in young adulthood. Father genetics - but early stress - appeared to explain most of the differences in hippocampus size. Certain fathers showed up with small offspring of the hippocampus. These animals also showed greater cortisol reactions at the time of weaving, suggesting risk factors for depression. Two other studies of human twins (2001 personal communications and presentations) of human twins by Sullivan et al and R.K. Pitman (2001 personal communication and presentation at the Annual Meeting of the American College of Neuropsychiatry) showed that rather convincingly the hippocampus size is primarily genetically determined, as well as serves as hippocampus size in studies by Sullivan et al. In a study by Pitman Et al. small hippocampus appeared to be a risk factor for developing post-traumatic stress disorder. Still, reserve Studies are needed to determine whether depression and other psychiatric disorders may result in further reductions in hippocampus volume. Therefore, the small hippocampus may still reflect both the causes and effects of major depression. Articles such as Frodl and Lockwood highlight two different areas, and don't pay a little attention to each article in different areas. It is, however, both areas are involved and those activities in both of them work between them and the connections between them help to explain both the somatic cells and cognitive symptoms of the disorder. One common thread that affects both areas is excessive glucocorticoid activity. The administration of cortisol in healthy subjects results in dysfunction in both regions (8, 9). Although high cortisol levels in rats can reduce hippocampus size relatively quickly, excessive glucocorticococoy probably does not explain their hippocampus discovery in first-time patients. Based on the relevant period, this seems reasonable. However, they also cite works other than Sanchez (10) which type II glucocorticocoid receptors were not significantly found in the hippocampus of the temporal monkeys, although they were found heavily in the prefrontal cortex, for another reason not to call glucocorticoids. Human glucocorticoid receptor probes were used in their studies. In contrast, Patel et al. (11) used specific probes for glucocorticoid receptors in squirrel monkeys to detect glucocorticoid receptors in the hippocampus and prefrontal cortex. Thus, glucocorticide could play an important role in the results of both studies. Smaller hippocampuses can be associated with higher glucocorticoid levels for many hours of stress, which can lead to cognitive dysfunction associated with the effects of prefrontal/anterior cingulate and hippocampus. In turn, excessive glucocorticide can lead to atrophy in certain brain regions. This area is worth further investigation and requires vertical research. My colleagues and I have been studying prefrontal cortex/anterior cingulate and hippocampus function or activity in psychotic major depression, hypothalamus - pituitary gland - subtype characterized by excessive activity of the adrenal axis. We have reported that patients with 12 psychotic main depression show impaired performance on both the color word part of the Stroop color word test and the short-circuit recall test (verbal memory). This data suggests damage to two areas of interest. However, the performance deficit in the short-circuit recall test was not maintained, suggesting that attention, response suppression, and encoding could account for a significant portion of the deficit. In another study (13), we reported that patients with psychosis major depression proved errors in commissions but were not missing out on word list tests, depending on the issue of focus and encoding rather than memory/se per recall. We are currently This functional magnetic resonance image is using a specific antagonist for the low-affinity II glucocorticoid receptor separating these functions and brain regions to access whether cortisol blocking improves performance. Besides Takeshita, the article is another visible effect article and follows several recent articles pointing to the link between depression and a high rate of mortality. The data is cold: Takeshita et al. Reports that depressive symptoms are associated with higher mortality rates in otherwise healthy elderly subjects. The findings suggest that some aspects of the biology of depression are associated with early mortality. But what are the factors? This document does not provide an explanation, and further research is needed to rule out the cause of premature death. A change in blood clotting and a high risk for arrhythmias is a possible culprit that can lead to dissonance. But they will pave the way for innovative interventions. For example, if depression is associated with high levels of circulatory catecholamine and arrhythmias, certain treatments could aim to prevent early death. However, it may be premature to conclude that depression leads to an early mortality rate. As you might think, the biological process may be in play with a number of physical symptoms, some of which may include subtle cardiovascular dysfunction that produces casting, such as depression or depressive in nature. In addition to Kendler' research, the study tested the hypothesis previously put forth by this group (14) on the multilateral causes of depression. Using twin cohorts, they elegantly dissect roles such as early and later episodes of depression (e.g. childhood abuse), genetics, and substance abuse. The factors studied are those that we see in general by clinicians when dealing with depressed patients. This elegant pathway analysis may be enhanced if we have better candidate genes to add to the schematic, as well as baseline hippocampus volume. Biology could seemingly play an important role in psychosocial factors. Comprehensively, this article points to possible cause factors as well as adverse consequences or correlations of major depression. The use of cross-sectional technology can truly obfusify causes and effects easily. Still, these studies give us important clues, and vertical research can ultimately give us a better sense of the causes and effects of troublesome disorders in modern society. Dr. Schatzberg, Department of Psychiatry, Stanford University Medical School, 3rd Floor, 401 Quarry Rd., Stanford, address reprinted requests at CA 94305-5717; [Email Protection]d.stanford.edu (email). See 1. Liotti M, Maiberg HS: The role of functional neuroimaging in neuropsychology of depression. J Clean XF Neuropsychiatric 2001; 123:121-136Crossref, Google Scholar2. 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