FROM SADNESS TO HEART DISEASE: A COMPREHENSIVE REVIEW ON THE CORRELATION BETWEEN DEPRESSION AND CARDIOVASCULAR DISEASE

ABSTRACT

Depression has been underlined as one of the key diagnosis among patients with cardiovascular disease. Academic literature indicates an increased link between depression and cardiovascular disease. Although studies has been done in relation to the pharmacological and cognitive-behavioral therapy, ts impact to the understanding of the association between depression and cardiovascular disease remains a debate. This comprehensive review provides an in-depth understanding of depression, cardiovascular diseases, the epidemiology and pathophysiological mechanism impacting cardiovascular disease with depression, activation of platelet, the effects of anti-depressant treatment, lifestyle of depression with cardio disease patients as well as its relation to sub-Saharan Africa.

Keywords: Depression, Cardiovascular disease, Patients, Sadness, Heart Disease.

INTRODUCTION

Existing data reveals a bidirectional association between depression and the morbidity and mortality rates of cardiovascular disease **(1)**. This places it the forefront of the field of mental illnesses. The first definitive link between cardiovascular disease and depression was discovered by Frasure-Smith and colleagues in 1993. It found that 35.3% of Turkish people who had been diagnosed with cardiac disorders also showed signs of depression **(2)**.

In 2009, Sitar-Taut and colleagues study emphasized the possible detrimental effects of this disorder on an individual's health and quality of life **(3)**.

There is more to the variety of symptoms and experiences that make up depression than just an overwhelming sense of sadness. Having symptoms in both the mental and physical realms, the aforementioned illness is systemic in nature. Particularly, it interferes with the functioning of the immunological and neurological systems, which in turn affects mental and emotional processes. Unlike transient emotional sensations of melancholy, It is considered a pathological illness when it emerges as a persistent impairment of an individual's daily functioning. In contrast to the repetitive patterns associated with more traditional mourning processes, the observed phenomena is steadfast and profound. Clinically depressed people differ from individuals going through normal grieving, they do not place blame people for their emotional pain on external events or others **(4)**.

Every year, more than 300 million people around or 4.4% of the world's population suffer from severely debilitating depression **(5)**. In 2013, Kesseler and Bromet found that this happens to about 10% of the general population, which makes it reasonable to assume that more people may experience it in the future **(6)**. Females have a twofold higher risk of having this mental health disorder as compared to males **(7)**.

According to the aforementioned research by Ferrari et al. in 2010, 3% of all disability-adjusted life years are caused by this disorder worldwide **(8)**.

Chronically depressed people are more likely to develop co-morbid diseases and die at an early age **(9)**. According to a study done by Coryell and Young in 2005, the annual global suicide death toll is somewhere about 800,000 **(10)**. Healthcare service cost rises because people with the disorder use more medical services **(11)**. According to the research of David et al. in 2014, the incidence of this complication is higher among those who are also dealing with cardiovascular disease **(12)**.

Researchers have found that the incidence of depression increases thrice in those with cardiovascular disease (CVD) compared to the general population. Rudisch and Nemeroff observed in their 2003 study that between 17 and 27 percent of people with CVD also have significant depressive illness **(13)**. More than half of these individuals show some degree of depression, with estimates ranging from 45% to 55%. Several studies' findings point to a link between clinical depression and an elevated risk of cardiovascular diseases' occurrence in the patient population. A statistically significant link between depression and an increased susceptibility to developing coronary artery disease was shown in a meta-analysis of 21 prospective trials, including a large cohort of 124,509 adults diagnosed with depression. According to the results, those with depression have an increased risk of this cardiovascular illness of 1.81 times. Individuals experiencing a melancholic mood had a 1.5-fold greater risk of having CVD, whereas those with a severe depressive condition had a 2.7-fold increased risk of acquiring CVD **(14)**.

By 2030, depression is expected to become a major factor in shortened life expectancy in high-income countries **(15)**. Major economic losses due to tardiness and lateness are the primary reason for this forecast. According to a large-scale study on

mental health conducted in 2003 by the highly regarded World Health Organization, the cost of providing healthcare to people with depression is roughly 4.2 times higher than the cost of providing healthcare to people without it **(16)**. In 2000, the United States economy suffered a significant loss of $83.1 billion due to the Great Depression **(17)**.

Depression's negative effects on cardiovascular health can be traced back to the disorder's pathophysiological manifestations, which include elevated platelet receptor expression, increased platelet signaling, and heightened platelet degranulation **(18)**. Platelet distribution width (PDW) has been proposed as a potential shared biological marker between major depressive disorder and cardiovascular disease. The examination of platelet activity markers as a measure of depression severity has the potential to be of great benefit in predicting and guiding treatment interventions. Both depression and cardiovascular disease are strongly linked to behavioral and lifestyle factors, as shown in this research. Precarious behaviors linked to obesity and insulin resistance have been linked to the occurrence of depression. Reduced physical activity, increased consumption of processed foods, weight gain, and tobacco use are all included here.

A higher score on the Dietary Inflammatory Index (DII) has been repeatedly linked in numerous studies to an increased risk of developing a wide range of health problems, such as but not limited to depression, anxiety disorders, atherosclerosis, and stroke. Individuals who followed a pro-inflammatory dietary pattern were shown to be more likely to develop cardiovascular diseases than those who followed an anti-inflammatory dietary pattern. Amadio et al.'s (2020) research confirms that factors that positively affect cardiovascular health include eating a diet rich in fruits and vegetables, drinking alcohol in moderation, not smoking, and regularly participating

in physical activity. Moreover, our research confirms that genetic variables contribute significantly to the existing literature between depression and CVDs. Increased levels of glucocorticoids are connected with a decrease in the production of microRNA-132, a protein linked to the onset of mental and neurological diseases. Increased levels of PCSK9, a mediator of low-density lipoprotein (LDL) cholesterol, have been linked to the onset of insulin resistance, according to a recent study. This result lends credence to the hypothesis that insulin resistance may be linked to obesity. Miller et al. in 2005 found that people who were both overweight and depressed had a higher level of PCSK9, which was positively correlated with an increased vulnerability to cardiovascular disease **(19)**.

HEART DISEASE AND DEPRESSION: ANALYZING THE EPIDEMIOLOGICAL CONNECTION

It is notable that both depression and cardiovascular illness are so pervasive in today's medical discourse. In 2012, researchers Beaulieu et al. found that depression had a 16.6% lifetime prevalence rate **(20)**. This major conclusion was reached by an examination of data from the United States National Comorbidity Survey. Sub-clinical depression is more common in primary care settings and is more common overall, especially among women. The combined mortality risk from cardiovascular events in people with no known cardiovascular risk factors is between 4.7% and 6.4%. However, within the group of people who exhibit two or more known risk factors for cardiovascular disease, the risk ranges from 20.5% to 29.6%. Numerous studies have looked into the effects of lifestyle choices on modifiable risk factors for CVD. Numerous factors have been revealed to play a significant impact in the initiation and

progression of CVD, including those that exhibit potential for modification or control through a variety of therapeutic approaches. Significant modifiable risk factors include diabetes, smoking, uncontrolled hypertension, high levels of low-density lipoprotein-C, and an increased waist-to-hip ratio **(21)**. It is beyond dispute that the aforementioned elements have a crucial role in shaping the underlying mechanisms that cause CVD. This is why the need for carefully considering their implications and take concrete measures to offset their negative effects through targeted therapeutic procedures. Depression, as shown by the statistics, is the second leading cause of work disability, right behind cardiovascular disease. According to a large meta-analysis performed by Nemeroff and Goldschmidt-Clermont (2012), people with anxiety disorders are 26% more likely to develop CVD **(22)**.

Holt et al. (2013) and Iosifescu et al. (2005) focused their in-depth research on those with decompensated heart failure. According to this study's findings, depression is approximately 15%-20% prevalent among the population with coronary artery disease (CAD). It is possible that inadequate or poor therapeutic approaches contribute to the maintenance of sub-clinical depressive symptoms in persons with CVD despite treatment. Non-adherence emerges as a major etiological factor contributing to damage within this group **(23, 24)**.

Holt et al. (2013) carefully analyzed a large dataset of 2,832 people who had never been diagnosed with CVD. There was a marked increase in the incidence of fatal and nonfatal ischemia episodes in this group compared to the general population. The highest susceptibility was observed in people who already had a melancholy disposition at the time of diagnosis **(23)**.

When Taylor et al. (2009) took attrition bias and other relevant risk factors into consideration, they found that people with depression had a significantly increased

risk of CAD development **(25)**. According to follow-up studies with a total sample size of 2,299 people, a sizeable percentage of both men and women (3.7% and 4.6%) had symptoms consistent with depression due to cardiovascular disease. Results from a 2012 study by Nemeroff and GoldSchmidt-Clermont showed that men had an odds ratio for CVD of 1.162 (95% CI 1.096-1.231, p 0.001), while women had an odds ratio of 1.107 (95% CI 1.038-1.181, p = 0.002) **(22)**. Depression in male medical students has been linked to an increased risk of CAD and myocardial infarction (MI), according to the "Precursors Study" done at Johns Hopkins University. In this investigation, researchers calculated a relative risk (RR) for coronary artery disease (CAD) of 2.12, with a 95% CI of 1.24 to 3.63. In addition, the p-value of 0.01 indicates that there is indeed a correlation. With a 95% confidence range (CI) from 1.11 to 4.06, the computed relative risk (RR) for myocardial infarction (MI) was 2.12. The p-value for this analysis was also calculated to be 0.01.

Eighteen thousand two hundred and sixty-one men and eleven thousand three hundred and eighty-eight women in the EPIC-Norfolk United Kingdom Prospective Cohort Study showed no signs of cardiovascular disease. According to research performed by Sutees et al. (2008), depressive people had a 2.7-fold higher risk of dying from a cardiovascular incident than their non-depressed counterparts **(26)**. This conclusion was reached after an exhaustive and extended monitoring period of 8 years. No conclusive evidence was found linking the presence of extra cardiovascular risk factors to the effect of interest. The Danish Psychiatric Central Research Registry mirrored the pattern as well. Kendler et al. (2009) found a strong correlation between clinical depression and the occurrence of myocardial infarction (MI) **(27)**.

According to the available data, patients diagnosed with heart failure and atrial fibrillation have an increased risk of developing depression **(28)**.

Curiously, studies show that people with both Acute Coronary Syndrome (ACS) and Chronic Coronary Syndrome (CCS) have poorer outcomes if they also suffer from depression. Taylor et al.'s 2009 landmark study found that screening for depression had significant benefits for people with a history of coronary artery disease or suspected CVD **(25)**. As a result, the American Heart Association actively promotes the widespread adoption and use of such tests. This advice stresses the importance of recognizing and treating depression in people with cardiovascular disease as soon as possible.

PATHOPHYSIOLOGICAL MECHANISMS BETWEEN DEPRESSION AND CARDIOVASCULAR DISEASE

Since the central nervous system and numerous physiological systems are intricately intertwined via neuroendocrine mechanisms , the potential impact of depression goes far beyond coronary artery disease (29). Studies examining the link between CVD and depression have shed light on the complex dynamics at play, including predisposition at the genetic level, biochemical mechanisms at work, and environmental factors that all play a role in the observable pathological processes. However, a single, uncomplicated etiological explanation that perfectly captures the essence of this medical illness is highly unlikely to be discovered. Heart rate variability has been well acknowledged for its role in pathophysiology. According to a comprehensive study by Erbas et al. in 2023, people with depression had less natural variation in their heart rates than those who are not depressed (30). This was found in a group of people who had no outward signs of cardiovascular disease.

Myocardial infarction (MI) patients with depression have significantly lower heart rate variability compared to MI patients without depression **(31)**. It is unclear how much irregular heart rates contribute to poor outcomes for people who already have heart problems. Researchers found that those with depression had significantly higher heart rates than a control group of healthy people **(32)**. Patients who had been treated for myocardial infarction and released from the hospital were the primary focus of the research. Holter recordings were used to capture heart rates continuously for 24 hours **(32)**.

Wariach et al. (2022) performed an analysis to determine if there was a correlation between left ventricular ejection fraction and mortality rates over a two-year period **(33)**. Heart rate was shown to be the most important prognostic predictor among the many indicators considered, with the highest predictive efficacy in terms of cardiac mortality and sudden death. Increased sympathetic discharge, insufficient parasympathetic stimulation, or a synergistic interplay between these two have all been linked to decreased heart rate variability. Researchers have known for a long time that heart rate variability (HRV) is correlated with autonomic nervous system health and function. It is speculated that the complex effects of depression on the hypothalamic-pituitary-adrenal axis (HPAA) may be at the root of the disorder. The HPAA is dysfunctional in around half of those with a major depressive disorder diagnosis. According to Warriach et al. in 2022, this discovery holds true whether or not the individual is suffering from cardiovascular disease **(33)**.

Deuschle et al.'s important study from 1998 elucidated the phenomena of hyperactivation, which increases the perceptible influence by increasing plasma cortisol concentrations **(34)**. People who suffer from depression and are also under constant psychological stress have cortisol levels that are significantly higher than

average. Increases in both cholesterol and triglycerides have been linked to increases in cortisol. Cortisol's ability to increase fluid volume via promotion of salt retention and concomitant elevation of peripheral vascular resistance contributes to its hypertensive effects. It has been proven that increased cortisol levels coincide with decreases in total and intramyocardial potassium levels. It has also been found that this increase lowers the threshold for ventricular arrhythmias and hastens the development of atherogenesis. The risk of cardiovascular disease is greatly increased by the aforementioned circumstances.

An rise in cortisol levels is hypothesized to play a role in the development of metabolic syndrome, which includes diabetes, high cholesterol, high triglycerides, high blood pressure, and obesity. Risk factors for cardiovascular disease include the aforementioned conditions **(35)**. The stimulation of the sympathetic nervous system causes an increase in plasma catecholamine levels via activation of the HPAA. Vasoconstriction, platelet activation, and tachycardia are only some of the negative effects on the cardiovascular system that have been linked to elevated levels of catecholamines in the blood **(36)**.

Medical geneticists have done extensive study to clarify the complex link between genes and a wide range of characteristics. Scherrer et al.'s 2003 research showed that there is a genetic predisposition to both depression and cardiovascular disease, showing the complex interplay between these illnesses (37). Taskiran et al. (2019) did an extensive analysis of monozygotic and dizygotic twins to determine the presence or absence of correlations between cardiovascular disease, hypertension, and a number of depressive symptoms (38). This research uncovered substantial links between the aforementioned health issues, shedding insight on the complex interplay between cardiovascular wellness, blood pressure regulation, and the varying

manifestations of mood disorders. The study's findings showed a strong association between the aforementioned medical disorders, raising the possibility that depression and CVD share a same genetic basis.

Researchers are now looking into the serotonergic system, with an emphasis on discovering the genetic factors that affect its operation. As a key neurotransmitter, serotonin is involved in a wide range of mental activities, including the regulation of appetite, mood, anxiety, consciousness, and wakefulness. Important vascular physiological processes that this phenomenon is studied include the control of platelet aggregation and the modulation of smooth muscle contraction. Reduced serotonergic activity in the central nervous system has been linked to an increased risk of CVD and mortality, most likely as a result of its effect on the HPAA's stress response (39).

Researchers have shed light on the complex relationship between depression and inflammation, revealing a significant link buried deep inside the immune system's inner workings. Interleukin-6 (IL-6) and C-reactive protein (CRP) increases have garnered a lot of attention from the medical community. The 2004 seminal study by Libby and Ridker is particularly noteworthy since it highlights the importance of inflammation in the context of atherosclerosis. The IL-6 gene polymorphism has received a lot of interest from scientists because of its practical implications for ongoing studies. Males who carry the -174C variant, caused by a G/C conversion in the gene's promoter region, have been shown to have higher than average plasma concentrations of CRP and IL-6, as well as higher blood pressure and an increased susceptibility to CVD (40).

It has also been discovered that psychological and social aspects play a role in pathogenesis. However, no definitive hypothesis for causality has been identified in the form of as-yet-undiscovered behavioral mechanisms. Biobehavioral pathways,

which include a wide range of risky health behaviors, have been linked between CVD and depression. There is a reciprocal relationship between the above characteristics and the development and progression of CVD and its associated negative outcomes. Psychological and social stressors have an immediate impact on the stress response system, which may initiate maladaptive behavioral changes that further increase vulnerability to CVD. Several of these factors have been linked to high-risk health behaviors and lifestyle selections that are known to exacerbate CVD. Increased smoking, poor food choices, less exercise, and a lack of compliance with medical recommendations have all been linked to the development of depressed symptoms **(41)**.

Decreased optimism is a hallmark of depression and has been related repeatedly to premature death in studies both theoretical and empirical. Research involving both human individuals and animal models maintains the same association. When asked whether they had experienced negative feelings like sadness, disappointment, or hopelessness in the previous month, participants who answered yes showed a twofold increase in the risk of developing CVD, according to a large study. Two well-known risk factors for CVD smoking and high cholesterol have been linked in the literature to a combination of personality structure and stress **(42).**

A person is engaged in physical activity whenever their body is in a state of motion and their metabolism is being taxed. Frequent exercise is recommended as a preventative measure against CVD. However, it is important to note that this intervention reduces death rates overall by 25%, including mortality primarily due to cardiovascular problems. It is important to recognize, however, that people with depressive symptoms may face barriers that make it difficult for them to engage in physical activity and get the advantages of doing so **(35)**.

Multiple processes contribute to stress's effect on catecholamines. Changes in cholesterol and free fatty acid concentrations have been found to occur simultaneously with elevations in blood pressure. Consistent with increased oxygen consumption and myocardial contractility, a faster heart rate is also detected. Platelet adhesion has been shown to be on the rise, suggesting that this physiological phenomenon may continue to improve. There is also evidence that the threshold for ventricular fibrillation, a potentially fatal heart arrhythmia, has lowered. In 2001, Humphries et al. discovered that stress increases the likelihood of having high cholesterol. Low-cholesterol diets were associated with an increase in aggressive behavior in primates **(40)**. Furthermore, this food intervention was associated with a significant decrease in central nervous system serotonergic activity.

A significant dilatation of blood vessels within the human body is triggered by the physiological response known as stress, as was recently clarified by scientific research. About 15% of patients with hypertension are resistant to antihypertensive medication, according to the results of a study conducted on those diagnosed with treatment-resistant hypertension. There was a correlation between the resistance level and the intravenous volume load. In 2003, Scherrer et al. found that pharmaceutical therapies meant to reduce arterial plaque were much less effective when persistent stress and unfavorable interpersonal interactions were present **(37)**.

Smoking cessation is associated with a dramatic decrease in the mortality rate from myocardial infarction (MI) in those who have CVD. Miller and Blackwell's in 2006 research revealed a strong link between depression and an extreme uptick in cigarette smoking **(42)**. The presence of depressive symptoms has been shown to have a dampening effect on motivation, making it harder for people to follow their treatment plan. This plan often consists of a number of different steps, such as taking

medication, altering one's diet, and exercising regularly. Some people do have a tendency toward self-destruction, as shown by their regular use of tobacco products, excessive intake of alcoholic beverages, and preference for high-calorie, low-nutrient diets high in cholesterol. Major depressive disorder patients usually demonstrate a regular cognitive pattern characterized by self-inquiry, in which they consider the justification for putting themselves through additional hardship in the event of their own imagined death **(43)**.

ACTIVATION OF PLATELET

By efficiently attaching to endothelial stem cells, platelets serve a vital role in facilitating arterial wall repair and maintaining adequate blood flow. Atherosclerotic plaque development is facilitated by the recruitment of inflammatory cells to the vascular intima. Since platelet activity decreases with age and in the context of chronic inflammation, Mause et al. in 2010 point out the importance of this procedure **(44)**. The emergence of arterial injury triggers a series of reactions within the organism's ecosystem. In particular, platelets get activated, setting off a cascade of complex biological responses. In turn, this activation helps bring in chemokine-dependent macrophages for cleanup duty. The death of intimal cells is the final step in this well crafted process, which perpetuates the degenerative effects of the arterial lesion.

Accumulation of free radicals in the presence of apoptosis within arterial smooth muscle cells initiates intimal smooth muscle migration. When atheroma protrudes into the arterial wall and positive remodeling occurs as a compensatory response, it causes an increase in smooth muscle cell proliferation within the intima, leading to arterial dilatation **(45).** Depression, platelet thrombosis, and the activation of smooth muscle proliferation all interact in complex ways that make for a fascinating area of study.

A significant difference in the percentage of p-selectin-positive platelets was found between those with a diagnosis of depression and those without the disorder. In 2009, Morel-Kopp et al. observed that the participants under study had considerably higher levels of platelet-leukocyte aggregates compared to the control group (p 0.001) **(46)**. Platelet reactivity to adenosine diphosphate is increased in people with depression, according to an in vitro study by Morel-Kopp et al., 2009. This finding adds weight to the theory that activated platelets are a typical symptom of depression **(46)**.

When activated, platelets produce a pair of proteins known as beta-thromboglobulin and platelet factor 4. Supporting the foregoing anecdotes is data from a large-scale study of elderly people (n = 21) with a clinical diagnosis of depressive disorder who were given aspirin as a preventative measure **(47)**.

However, in the large Heart and Soul Study including 104 people with CAD and concomitant depression, no noticeable rise in beta-thromboglobulin or platelet factor 4 was identified **(48)**.

DIAGNOSING FOR DEPRESSION IN CARDIOVASCULAR DISEASE PATIENTS

Given the well-established link between CVD and depression, experts have spent considerable time debating whether or not everyone with a cardiac condition should undergo depression screening and what the best methods for collecting relevant data might be. Examining people with cardiac issues for concomitant depression has not led to conclusive results, despite the approval of prevailing recommendations **(49)**. Due to the absence of evidence linking the screening for depression in cardiac patients to an improved prognosis, it may be regarded unnecessary to screen for depression. It's also important to note that some antidepressants may increase the danger of dying from heart disease if they're taken regularly. In 2015, Erbas et al. Study Individuals

presenting with severe atherosclerotic disease should be evaluated for concomitant anxiety and depression **(50)**.

Thombs et al. (2008) conducted a meta-analysis of 11 papers on the topic of evaluating depression screening in people with cardiovascular disease, and they also looked into six studies on the efficacy of depression therapy **(51)**. They found no evidence that screening for depression in people with cardiovascular disease leads to an improvement in depressive symptoms or outcomes related to cardiovascular disease. If a patient exhibits more symptoms of anxiety and depression than can be determined through a standard screening, it may be wise to have them evaluated by a mental health professional. Since they can be used alone or in addition to other interventions, psychosocial therapies show great versatility within the field of cardiac rehabilitation **(51)**.

CARDIOVASCULAR EFFECTS ON ANTIDEPRESSANT TREATMENT

A study was done to determine if treating cardiovascular disease (CVD) could help alleviate depression, as there appears to be a correlation between the two conditions. One of the earliest studies on this topic was undertaken by Glassman et al. (2002), who gave the antidepressant sertraline to people suffering from clinical depression and followed them for 24 weeks **(52)**. After being diagnosed with myocardial infarction and unstable angina within the preceding month, these individuals were closely observed in a clinical setting. After a heart attack, it was unrealistic to assume that sertraline would be more effective than a placebo in reducing the risk of further cardiovascular problems. The current state of research does not give strong evidence that treating depression in people with cardiac problems improves their prognosis.

Although it was once thought that antidepressants were a safe and effective treatment for depression, recent studies have shown otherwise. There is a strong correlation

between the presence of depressive symptoms and the occurrence of fatal coronary events in the cohort of women without a diagnosis of existing coronary artery disease. It is important to highlight, however, that antidepressant medication in this population has been linked to an increased risk of sudden cardiac mortality **(53)**. Antidepressant use has been linked to an increased risk of sudden cardiac death, however the underlying cause of this correlation is unknown. Empirical studies, however, have revealed that the severity and length of depression in patients receiving antidepressant treatment may have a contributing role.

In 2010, Davidson et al.'s research is an important addition to the literature since it shows that treating depression can be beneficial for people with heart problems **(54)**.

High-liposolubility beta-blockers have been linked to depression and other central effects, according to a 2011 study by Luijendijk et al **(55)**. Patients with cardiovascular disease who take their statins as prescribed have less depressive symptoms and a lower risk of developing depression, according to research by Stafford and Berk in 2011 **(56)**.

LIFESTYLE OF CARDIOVASCULAR DISEASE AND DEPRESSION PATIENTS

Risk factors for gaining weight and being obese include smoking, drinking alcohol, not taking medications as recommended, and not getting enough exercise **(57)**. It has been found that people with depression are more likely to engage in these actions. The likelihood that these people will show motivation to make positive lifestyle changes is lower. Paganini-Hill found no statistically significant variation in the frequency of cardiovascular disease across sexes in a cross-sectional study **(58)**. Interestingly, low levels of physical activity were linked to the presence of depressive symptoms.

Future studies must place a premium on lifestyle therapies for people experiencing depression because of the gender gaps and the high prevalence of CVD. Kim et al. (2020) recommend focusing on increasing physical activity levels in addition to managing depression symptoms **(59)**. It is worth noting in the context of an elderly cohort that people who did not smoke, drank alcohol in moderation, ate a healthy diet, were physically active on a regular basis, and kept their body mass index within the normal range had better health outcomes. According to research by Melin et al. the severity of depressive symptoms is positively correlated with later worsening of cardiovascular outcomes **(60)**.

Recent studies have found that depressed people of both sexes may be at a higher risk for developing CAD. However, there may be an increased risk of depression in women who have been diagnosed with CAD. The effects of risk factors for CVD such hypertension, weight increase, and raised cholesterol levels are similar for men and women. Finnell and Wood's 2016 research highlights, however, that long-term smoking has a considerably higher negative effect on women than men **(61)**. Ghazizadeh et al. in 2020 found that severe sadness and other psychiatric problems were linked to dietary inflammation **(62)**. Consuming a diet high in energy drinks, saturated fat, and simple carbs has been linked to an increase in peripheral inflammatory markers **(63)**. However, it has been shown that these indicators can be reduced by eating a diet high in fruits, vegetables, and fiber.

Many studies, including controlled experiments and non-interventional observations, have contributed to the current body of knowledge **(64)**. Consumption of processed carbohydrates and energy drinks causes hyperglycemia and hyperinsulinemia, which can increase the generation of free radicals and pro-inflammatory cytokines and hence contribute to the inflammatory response. Researchers were able to establish a link

between the Dietary Inflammatory Index (DII) and several inflammatory markers during the Asklepios study. Using the food frequency questionnaire (FFQ), researchers compared participants' eating habits with their blood levels of several inflammatory indicators. These markers included interleukin-6 (IL-6), C-reactive protein, homocysteine, and fibrinogen. This in-depth evaluation allowed for the determination of the DII. However, a comprehensive multivariate study revealed notable positive relationships between the DII and inflammatory markers like IL-6 and homocysteine, and this was the case even after taking into account any confounding factors. In 2015, Shivappa et al. found that diet plays a crucial role in the body's ability to control inflammation **(65)**.

In 2020, Asadi et al. followed up on their initial Iranian study by examining the prevalence of depression and anxiety in the MASHAD cohort, which included people with diagnoses of stroke and coronary atherosclerotic disease **(63)**. The major purpose of this research was to investigate whether or not the DII is linked to the development of depression. Remembering the function of the HPA axis and the importance of glucocorticoids can help with grasping the idea at hand. Depressed people appear to have increased activity of the complex metabolic mechanism involved for the endogenous manufacture of glucose, known as gluconeogenesis. Increased gluconeogenic activity is associated with increased body fat and insulin resistance in this population. With the goal of assessing the likely inflammatory consequences associated with their eating patterns, the DII was generated within a cohort consisting of middle-aged adults of Iranian heritage, both male and female.

FFQ can be used to examine 45 different dietary characteristics and generate a DII score. It is possible that pro-inflammatory and anti-inflammatory values could coexist on the DII scale. Additionally, quartile analysis was performed, wherein the data was

broken down into four categories (Q1, Q2, Q3, and Q4) based on the scores. When it comes to the inflammatory response, Q1 acts as an inhibitor, while Q4 stimulates. After controlling for potential confounding factors, this study's findings suggest that women in the third and fourth quartiles of the DII are more likely to experience depression than those in the first quartile of the DII (the control group).

According to the results of this study, severe depression, as measured by the DII, is more common among women than men in this sample of Iranians. Modifications to an anti-inflammatory dietary program may, therefore, be useful within the context of therapeutic interventions and preventative measures for depressive symptoms. In 2019, Tolkien et al. conducted a study that found the DII score to be significantly predictive of atherosclerosis and other cardiac disease markers such as intimal thickness, plaque development, and cardiac output **(66)**. Given these results, it is clear that more research into the DII score's possible ramifications and significance in the context of cardiovascular health is warranted .

DEPRESSION AND CARDIOVASCULAR DISEASE AND RELATION TO SUB-SAHARAN AFRICA

There is a lack of information on the incidence of depression in the countries of Sub-Saharan Africa (SSA). Researchers frequently use point prevalence as a substitute for nationally representative data when national surveys are unavailable. There is a lack of depression epidemiology data from the African continent, and the data that does exist shows substantial methodological differences and heterogeneity across different countries. Extrapolating results or combining results from additional investigations is extremely difficult due to the situation's inherent complexity (67, 68).

There is a worrying lack of data about the global prevalence of depression, and this is a problem for the medical research community. Examples of African countries include South Africa, Nigeria, Cameroon, Ethiopia, Sudan, Kenya, Uganda, Ghana, the Democratic Republic of the Congo, Zambia, Botswana, and Zambia.

Cross-sectional and cohort studies conducted in hospitals, schools, and prisons account for the lion's share of data collected in these countries, increasing the incidence of missing data and potential bias.

Roughly 3.9% of Nigeria's overall population, or 7 million people, currently call the country home. An estimated 29.19 million Africans, or around 9 percent of the continent's total population, suffer from clinical depression. In 2016, Esan and Esan propose a range of estimates for the lifetime prevalence of major depressive disorders, which they place between 3.3% and 9.8% **(68)**. In 2017, Ojagbemi et al. found that almost a third of those who have had a stroke also show signs of clinical depression. After a stroke or other neurological event, Bernard et al. in 2017 found a correlation between the increased level of concordance they had seen and functional reliance and lower quality of life **(69)**.

People who are HIV-positive are more likely to experience depression than the general population. In 2016, Esan and Esan, in 2018, Opondo et al. (2018), in 2013, Assil and Zeidan and in 2008, Stein et al. cite values ranging from 11.5% in Uganda to approximately 30% in countries like South Africa, Zambia, Sudan, and Botswana (68, 70, 71, 72). Depending on the source, the estimated prevalence of depression in Ethiopia ranges from 11% to 38% (73; 74). In 2017, Ngasa et al. did a cross-sectional study and found that depression is common among medical students in Cameroon. The study also found that those with depression were more likely to have chronic health conditions and to have undergone major life transitions (75).

Among the sample of women who took part in the study, 36 percent met the criteria for a diagnosis of major depressive disorder (95% confidence interval [CI]: 22.8-36.7). According to research by Ngasa et al. in 2017, a sizeable fraction of the student population displays symptoms consistent with depression (75). The percentage of pupils exhibiting mild depressed symptoms ranged from 34.6% to 26.4%, while 3.4% reported symptoms of moderately severe depression and 0.8% indicated symptoms of severe depression.

According to the World Health Organization's most recent Global Health Estimate for Depression and Other Common Mental Disorders, 5.4% of the African population suffers from depression, while 3.2% of the population experiences anxiety. The current data may be an underestimation of the true prevalence of depression in African countries due to variables like the possibility of misdiagnosis, underdiagnosis, and a lack of recorded data.

CONCLUSION

Multiple studies have shown that people with depression are more likely to develop new cardiovascular problems. Having a history of depression has been linked to poorer results for people who have been diagnosed with cardiovascular disease. When depression and heart disease occur together, it has a devastating effect on both the patient's physical and mental well-being. Multiple molecular processes contribute to the establishment of this harmful reciprocal inhibition.

The American Heart Association and the European Society of Cardiology have both accepted that depression is a modifiable risk factor in people with CAD. Effective treatment measures may help reduce this threat. Accordingly, diagnosing and treating

depression should be a standard part of routine monitoring methods for people with cardiovascular problems.

The reduction of depressed symptoms has been linked to a considerable improvement in cardiovascular health, according to research. Regular physical activity has been linked to a reduction in depression symptoms and an increase in cardiorespiratory fitness, both of which are beneficial to cardiovascular health. Cognitive behavioral therapy (CBT) has been shown to be effective in several clinical settings. The use of pharmaceutical therapies, particularly antidepressant medication, has the potential to reduce the increased risk of CVD in these individuals.

Mental health issues, such as depression and cardiovascular disease, are a major public health concern in the countries of Sub-Saharan Africa, and there is a pressing need for in-depth study of their causes and effects.

CONFLICT OF INTEREST

The writer has stated that they have no stake in the outcome of this article's publishing.

FUNDING

The author did not receive a grant to conduct the study or write this article.

REFERENCES

1. Bobo, W. V., Ryu, E., Petterson, T. M., Lackore, K., Cheng, Y., Liu, H., Suarez, L., Preisig, M., Cooper, L. T., Roger, V. L., Pathak, J., & Chamberlain, A. M. Bi-directional association between depression and HF: An electronic health records-based Cohort Study. Journal of Comorbidity. 2020; 10. doi.org/10.1177/2235042x20984059
2. Frasure-Smith N. Depression following myocardial infarction. JAMA. 1993;270(15):1819. doi:10.1001/jama.1993.03510150053029
3. Dădârlat A, Sitar-Tăut AV, Zdrenghea D, Pop D, Buzoianu A. The profile of cardiovascular risk factors in heart failure obese patients hospitalised in a rehabilitation Romanian Hospital. Balneo Research Journal. 2018;9(1):28–33. doi:10.12680/balneo.2018.167
4. Muzi L, Tieghi L, Rugo M, Lingiardi V. Eating pathology and psychodynamic diagnostic manual (PDM-2) Diagnostic Assessment: Implications for Treatment Outcomes in a psychodynamic-oriented residential setting. Psychoanalytic Psychology. 2021;38(4):300–10. doi:10.1037/pap0000337
5. World Health Organization Depression and other common mental disorders. Geneva: World Health Organization, 2017.
6. De Aquino JP, Londono A, Carvalho AF. An update on the epidemiology of major depressive disorder across cultures. Understanding Depression. 2017;309–15. doi:10.1007/978-981-10-6580-4_25
7. Jaeschke K, Hanna F, Ali S, Chowdhary N, Dua T, Charlson F. Global estimates of service coverage for severe mental disorders: Findings from The Who Mental Health Atlas 2017. Global Mental Health. 2021;8. doi:10.1017/gmh.2021.19
8. Ferrari AJ, Erskine HE, Charlson FJ, Santomauro DF, Leung J, Whiteford HA. The global burden of mental and substance use disorders. Oxford Textbook of Public Mental Health. 2018;35–44. doi:10.1093/med/9780198792994.003.0004
9. Cabello M, Borges G, Lara E, Olaya B, Martín-Maria N, Moreno-Agostino D, et al. The relationship between all-cause mortality and depression in different gender and age groups of the Spanish population. Journal of Affective Disorders. 2020;266:424–8. doi:10.1016/j.jad.2020.01.162
10. Trivedi MH. Major depressive disorder in primary care. The Journal of Clinical Psychiatry. 2020;81(2). doi:10.4088/jcp.ut17042br1c
11. Hajebi A, Motevalian SA, Rahimi-Movaghar A, Sharifi V, Amin-Esmaeili M, Radgoodarzi R, et al. Major anxiety disorders in Iran: Prevalence, sociodemographic correlates and service utilization. BMC Psychiatry. 2018;18(1). doi:10.1186/s12888-018-1828-2
12. Medwell M, Toukhsati S, Hare D. Psychological resilience protects against depression one month following acute coronary syndrome. Heart, Lung and Circulation. 2021;30. doi:10.1016/j.hlc.2021.06.372
13. Vedamurthy D. How does psychological treatment compare with usual care for people with coronary artery disease and comorbid depression? Cochrane Clinical Answers. 2022; doi:10.1002/cca.4098
14. Cao H, Zhao H, Shen L. Depression increased risk of coronary heart disease: A meta-analysis of prospective cohort studies. Frontiers in Cardiovascular Medicine. 2022;9. doi:10.3389/fcvm.2022.913888
15. Minh A, Bültmann U, Reijneveld SA, van Zon SK, McLeod CB. Depressive symptom trajectories and early adult education and employment: Comparing

Longitudinal cohorts in Canada and the United States. International Journal of Environmental Research and Public Health. 2021;18(8):4279. doi:10.3390/ijerph18084279
16. Banatvala N, Akselrod S, Webb D, Sladden T, Hipgrave D, Schneidman M. Actions needed to prevent noncommunicable diseases and Improve Mental Health. Bulletin of the World Health Organization. 2019;97(2). doi:10.2471/blt.18.228700
17. Notenbomer A, van Rhenen W, Groothoff JW, Roelen CA. Predicting long-term sickness absence among employees with frequent sickness absence. International Archives of Occupational and Environmental Health. 2018;92(4):501–11. doi:10.1007/s00420-018-1384-6
18. Amadio P, Zarà M, Sandrini L, Ieraci A, Barbieri SS. Depression and cardiovascular disease: The viewpoint of platelets. International Journal of Molecular Sciences. 2020;21(20):7560. doi:10.3390/ijms21207560
19. Nowzari H, Wilder-Smith P. Effects of mouthwash on Oral Cytomegalovirus Epstein- Barr Virus Herpes Simplex Virus type-1. Advances In Clinical And Medical Research. 2022;3(4):01–9. doi:10.52793/acmr.2022.3(4)-44
20. Zarate CA. Commentary on the Canadian network for mood and anxiety treatments (CANMAT) Task Force recommendations for the use of racemic ketamine in adults with major depressive disorder. The Canadian Journal of Psychiatry. 2021;66(6):537–9. doi:10.1177/07067437211004023
21. Lee KH, Xu H, Wu B. Gender differences in quality of life among community-dwelling older adults in low- and middle-income countries: Results from the study on Global Ageing and Adult Health (SAGE). BMC Public Health. 2020;20(1). doi:10.1186/s12889-020-8212-0
22. Terentes-Printzios D, Ioakeimidis N, Rokkas K, Vlachopoulos C. Interactions between erectile dysfunction, cardiovascular disease and cardiovascular drugs. Nature Reviews Cardiology. 2021;19(1):59–74. doi:10.1038/s41569-021-00593-6
23. Bevilacqua G, Westbury LD, Bloom I, Zhang J, Ward KA, Cooper C, et al. Investigating the relationship between self-perception of fracture risk and prior fracture: Findings from the Hertfordshire Cohort Study. Aging Clinical and Experimental Research. 2022;35(3):599–606. doi:10.1007/s40520-022-02322-6
24. Greer TL, Joseph JK. Pharmacological and non-pharmacological treatment effects on functional outcomes in major depressive disorder. Major Depressive Disorder. 2020;131–46. doi:10.1016/b978-0-323-58131-8.00010-0
25. Suarez EC. The association between measures of inflammation and psychological factors associated with an increased risk of atherosclerotic cardiovascular disease: Hostility, anger and depressed mood and symptoms. The Oxford Handbook of Psychoneuroimmunology. 2012;170–94. doi:10.1093/oxfordhb/9780195394399.013.0010
26. Myint PK, Luben RN, Surtees PG, Wainwright NW, Wareham NJ, Khaw K-T. Physical functional health predicts the incidence of coronary heart disease in the European prospective investigation into cancer-norfolk prospective population-based study. International Journal of Epidemiology. 2010;39(4):996–1003. doi:10.1093/ije/dyq061
27. Kendler KS, Gardner CO. Dependent stressful life events and prior depressive episodes in the prediction of Major Depression. Archives of General Psychiatry. 2010;67(11):1120. doi:10.1001/archgenpsychiatry.2010.136

28. Cadrin-Tourigny J, Shohoudi A, Roy D, Talajic M, Tadros R, Mondésert B, et al. Decreased mortality with beta-blockers in patients with heart failure and coexisting atrial fibrillation. JACC: Heart Failure. 2017;5(2):99–106. doi:10.1016/j.jchf.2016.10.015
29. Poponina TM, Gunderina KI, Poponina YuS, Soldatenko MV. The effects of agomelatine on heart rate variability in patients with anxiety-depressive disorders who suffered acute coronary syndrome. Siberian Medical Journal. 2018;33(3):36–45. doi:10.29001/2073-8552-2018-33-3-36-45
30. Erbas O, Altuntaş İ, Nesil P, Sasani H, Odabaşı M. Neuroprotective properties of peptides. Translational Neuroprotection - The Way Ahead [Working Title]. 2023; doi:10.5772/intechopen.109967
31. Carney RM, Steinmeyer B, Freedland KE, Stein PK, Hayano J, Blumenthal JA, et al. Nocturnal patterns of heart rate and the risk of mortality after acute myocardial infarction. American Heart Journal. 2014;168(1):117–25. doi:10.1016/j.ahj.2014.04.012
32. Hayano J, Ueda N, Kisohara M, Yuda E, Carney RM, Blumenthal JA. Survival predictors of heart rate variability after myocardial infarction with and without low left ventricular ejection fraction. Frontiers in Neuroscience. 2021;15. doi:10.3389/fnins.2021.610955
33. Gachemba YM, Khan Z, Njau E, Wanyoike M. Vitamin D deficiency and its association with cardiovascular diseases among patients attending a private tertiary sector Cardiovascular Heart Clinic in Nairobi. Cureus. 2023; doi:10.7759/cureus.43012
34. Schilling C, Gilles M, Blum WF, Daseking E, Colla M, Weber-Hamann B, et al. Leptin plasma concentrations increase during antidepressant treatment with amitriptyline and mirtazapine, but not paroxetine and Venlafaxine. Journal of Clinical Psychopharmacology. 2013;33(1):99–103. doi:10.1097/jcp.0b013e31827cb179
35. Kuhlman KR. Early life stress and the pathogenesis of depression. Psychoneuroendocrinology. 2019;100. doi:10.1016/j.psyneuen.2018.12.174
36. Andersen S, Andersen A, de Man FS, Nielsen-Kudsk JE. Sympathetic nervous system activation and β-adrenoceptor blockade in right heart failure. European Journal of Heart Failure. 2015;17(4):358–66. doi:10.1002/ejhf.253
37. Honkalampi K, Lehto SM, Hintikka J, Koivumaa-Honkanen H, Niskanen L, Viinamäki H. Symptoms of depression and alexithymic burden in middle-aged men. Psychotherapy and Psychosomatics. 2010;79(4):259–61. doi:10.1159/000315132
38. Gürkan G, Atasoy Ö, Çini N, Sever İH, Özkul B, Yaprak G, et al. Reparative, neuroprotective and anti-neurodegenerative effects of granulocyte colony stimulating factor in radiation-induced brain injury model. Journal of Korean Neurosurgical Society. 2023; doi:10.3340/jkns.2023.0049
39. Williams MS, Ziegelstein RC, McCann UD, Gould NF, Ashvetiya T, Vaidya D. Platelet serotonin signaling in patients with cardiovascular disease and Comorbid Depression. Psychosomatic Medicine. 2019;81(4):352–62. doi:10.1097/psy.0000000000000689
40. Mariampillai JE, Prestgaard E, Kjeldsen SE, Liestøl K, Engeseth K, Erikssen J, et al. Exercise systolic blood pressure and quartile-based risk of coronary heart disease in healthy men during 28 years of follow-up. Journal of Hypertension. 2018;36(Supplement 1). doi:10.1097/01.hjh.0000538966.81513.41

41. Rozanski A. Psychosocial risk factors and cardiovascular disease: Epidemiology, screening, and treatment considerations. Cardiovascular Innovations and Applications. 2016;1(4). doi:10.15212/cvia.2016.0033
42. LeMoult J. From stress to depression: Bringing together cognitive and biological science. Current Directions in Psychological Science. 2020;29(6):592–8. doi:10.1177/0963721420964039
43. Hage FG, Szalai AJ. The role of C-reactive protein polymorphisms in inflammation and cardiovascular risk. Current Atherosclerosis Reports. 2009;11(2):124–30. doi:10.1007/s11883-009-0020-z
44. Mause SF, Ritzel E, Deck A, Vogt F, Liehn EA. Endothelial progenitor cells modulate the phenotype of smooth muscle cells and increase their neointimal accumulation following vascular injury. Thrombosis and Haemostasis. 2021;122(03):456–69. doi:10.1055/s-0041-1731663
45. Wang H, Liu L, Liu C, Wang L, Chen J, Wang H, et al. Induction of meiosis by embryonic gonadal somatic cells differentiated from pluripotent stem cells. Stem Cell Research & Therapy. 2021;12(1). doi:10.1186/s13287-021-02672-4
46. Milovanovic M. Major depression is associated with high platelet activity and reactivity. Austin Thrombosis research and Treatment. 2019;3(1). doi:10.26420/thrombhaemostres.2019.1019
47. Kuijpers PMJC, Hamulyak K, Strik JJMH, Wellens HJJ, Honig A. Beta-thromboglobulin and platelet factor 4 levels in post-myocardial infarction patients with major depression. Psychiatry Research. 2002;109(2):207–10. doi:10.1016/s0165-1781(02)00017-3
48. Lee C, Whooley MA. Networks of c- reactive protein and depression symptoms in patients with stable coronary heart disease: Findings from the heart and Soul study. International Journal of Methods in Psychiatric Research. 2023; doi:10.1002/mpr.1968
49. Kashif M, Hamid M, Raza A. Influence of preoperative anxiety level on postoperative pain after cardiac surgery. Cureus. 2022; doi:10.7759/cureus.22170
50. Salmasi M, Ashrafi F. Comparison of the effects of pegylated granulocyte-colony stimulating factor and granulocyte-colony stimulating factor on cytopenia induced by dose-dense chemotherapy in breast cancer patients. Journal of Research in Medical Sciences. 2018;23(1):73. doi:10.4103/jrms.jrms_463_17
51. Thombs BD, Markham S, Rice DB, Ziegelstein RC. Does depression screening in primary care improve mental health outcomes? BMJ. 2021; doi:10.1136/bmj.n1661
52. Carney RM, Freedland KE, Rubin EH, Rich MW, Steinmeyer BC, Harris WS. Omega-3 augmentation of sertraline in treatment of depression in patients with coronary heart disease. JAMA. 2009;302(15):1651. doi:10.1001/jama.2009.1487
53. Heianza Y, Wang X, Tiwari S, Sun Q, Rexrode KM, Watrous J, et al. Atherogenic gut microbial metabolite scores and risk of coronary heart disease among women in the Nurses' Health Study. Circulation. 2023;147(Suppl_1). doi:10.1161/circ.147.suppl_1.mp24
54. Ye S, Shaffer J, Rieckmann N, Schwartz J, Kronish IM, Ladapo JA, et al. Long-term outcomes of enhanced depression treatment in patients with acute coronary syndromes: The copes randomized controlled trial. Journal of the American College of Cardiology. 2013;61(10). doi:10.1016/s0735-1097(13)60201-6
55. Ham AC, Aarts N, Noordam R, Rivadeneira F, Ziere G, Zillikens MC, et al. Use of selective serotonin reuptake inhibitors and bone mineral density change.

Journal of Clinical Psychopharmacology. 2017;37(5):524–30. doi:10.1097/jcp.0000000000000756
56. Larsen SV, Mikkelsen AP, Lidegaard Ø, Frokjaer VG. Depression associated with hormonal contraceptive use as a risk indicator for postpartum depression. JAMA Psychiatry. 2023;80(7):682. doi:10.1001/jamapsychiatry.2023.0807
57. Cheng Y, Mou L, Li Z. Trends in adherence to recommended physical activity and its association with cardiovascular risk factors in US adults with cardiovascular disease: A cross-sectional study. BMC Cardiovascular Disorders. 2022;22(1). doi:10.1186/s12872-022-02854-9
58. Paganini-Hill A, Corrada MM, Kawas CH. Increased longevity in older users of postmenopausal estrogen therapy: The Leisure World Cohort Study. Menopause. 2018;25(11):1256–61. doi:10.1097/gme.0000000000001227
59. Kim JA, Choi S, Choi D, Park SM. Pre-existing depression among newly diagnosed dyslipidemia patients and cardiovascular disease risk. Diabetes & Metabolism Journal. 2020;44(2):307. doi:10.4093/dmj.2019.0002
60. Melin EO, Wanby P, Neumark T, Holmberg S, Neumark A-SN, Johansson K, et al. Depression was associated with younger age, female sex, obesity, smoking, and physical inactivity, in 1027 patients with newly diagnosed type 2 diabetes: A Swedish multicentre cross-sectional study. BMC Endocrine Disorders. 2022;22(1). doi:10.1186/s12902-022-01184-3
61. Wood SK. The role of inflammation and oxidative stress in depression and cardiovascular disease. Cardiovascular Implications of Stress and Depression. 2020;175–209. doi:10.1016/b978-0-12-815015-3.00008-8
62. Li X, Liu H, Zhang L, Yang X. Association between dietary theobromine with depression: A population-based study. BMC Psychiatry. 2022;22(1). doi:10.1186/s12888-022-04415-y
63. Vallée A. Association between socio-economic status and estimated atherosclerotic cardiovascular disease risk: Results from a middle-aged population-based study. Public Health. 2023;221:1–9. doi:10.1016/j.puhe.2023.05.014
64. Isaacs A, Beauchamp A, Sutton K, Kocaali N. Care coordination can reduce unmet needs of persons with severe and persistent mental illness. Frontiers in Psychiatry. 2019;10. doi:10.3389/fpsyt.2019.00563
65. Moludi J, Shivappa N, Alisgharzadeh S, Hébert JR, Alizadeh M. Dietary inflammatory index is related to heart failure risk and cardiac function: A case–control study in heart failure patients. Frontiers in Nutrition. 2021;8. doi:10.3389/fnut.2021.605396
66. McBean L, O'Reilly S. Diet quality interventions to prevent neurocognitive decline: A systematic review and meta-analysis. European Journal of Clinical Nutrition. 2021;76(8):1060–72. doi:10.1038/s41430-021-01032-y
67. Moini J, Koenitzer J, LoGalbo A. Global Epidemiology of Mental Disorders. Global Emergency of Mental Disorders. 2021;57–67. doi:10.1016/b978-0-323-85837-3.00004-2
68. Esan O, Fela-Thomas A. The significance of sleep quality in EUTHYMIC bipolar patients from Nigeria. South African Journal of Psychiatry. 2022;28. doi:10.4102/sajpsychiatry.v28i0.1739
69. Yan X, Du J, Ji G. Prevalence and factors associated with fertility desire among people living with HIV: A systematic review and meta-analysis. PLOS ONE. 2021;16(3). doi:10.1371/journal.pone.0248872

70. Gudisa B, Gemechis B. Prevalence of, and factors influencing first line antiretroviral treatment failure among adult HIV patients at Antiretroviral Treatment Clinic of mettu karl referral hospital, South Western, Ethiopia: A prospective cross sectional study, 2021. International Journal of Virology and AIDS. 2021;8(1). doi:10.23937/2469-567x/1510075
71. Mabyoue MO. Prevalence of post-partum depression among Sudanese women using Edinburgh Postnatal Depression Scale (EPDS) in two major delivery hospitals in Khartoum State. Sudan Journal of Medical Sciences. 2020; doi:10.18502/sjms.v15i4.8158
72. Anic A, Robertson LJ. Prevalence and clinical correlates of substance use amongst acute psychiatric inpatients in Gauteng, South Africa. South African Journal of Psychiatry. 2020;26. doi:10.4102/sajpsychiatry.v26i0.1526
73. Dereje Zena, Berhanu Elfu, Kebadnew Mulatu. Prevalence and associated factors of precancerous cervical lesions among women in Ethiopia: A systematic review and meta-analysis. Ethiopian Journal of Health Sciences. 2021;31(1). doi:10.4314/ejhs.v31i1.20
74. Tiki T, Taye K, Duko B. Prevalence and factors associated with depression among pregnant mothers in the West Shoa Zone, Ethiopia: A community-based cross-sectional study. Annals of General Psychiatry. 2020;19(1). doi:10.1186/s12991-020-00275-6
75. Dingana TN, Ngasa SN, Ngasa NC, Tchouda LA, Abanda C, Niba J, et al. Prevalence and factors associated with post-partum depression in a remote area of Cameroon: A cross-sectional study. 2022; doi:10.1101/2022.04.12.22273774