The Association of BMI and Blood Pressure Measurements with Depressive and Quality of Life Scores in University Students

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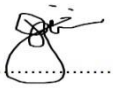
Declaration

I declare that *The Association of BMI and Blood Pressure Measurements with Depressive and Quality of Life Scores in University Students* is my own work, that it has not been submitted before for any degree or examination at any other academic institution, and that all the sources I have used or quoted have been indicated and acknowledged as completereferences.

Ammaarah Gamieldien January 2022

Signed.....  .....

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Abstract

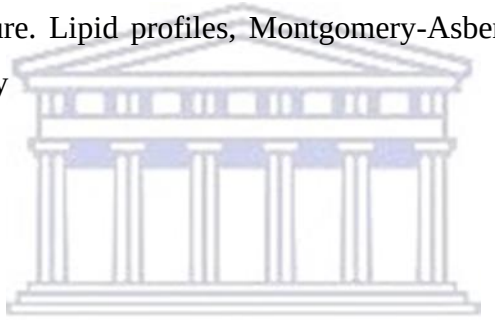
Depression is a very common and serious mental illness. Studies have shown that depression has a significant impact on both social and economic aspects of sufferers worldwide as well as in South Africa. Research has shown that university students in South Africa have a higher prevalence than the general South African population. Major Depressive Disorder (MDD) is the most common and incapacitating form of depression. MDD is characterized as at least two weeks of a low mood that is apparentafter most situations. There are several contributing factors to depression as well as comorbidities which may lead to depression. When a patient suffers from depression along with another illness, it is described as the comorbidity of depression. Individuals who suffer from chronic diseases, mental illnesses, chronic pain and other physical conditions have an increased chance of experiencing depression, compared to those that are healthy. This study aimed to investigate the association between body mass index (BMI), blood pressure and lipid profiles with depression in university students. The other purpose was to determine secondary objectives which included the associations between the variables (BMI, blood pressure and lipids) with themselves as they are key factors in cardiometabolic disease.

Sixty-three (63) students participated in the study. Thirty-two (32) were assigned to the control group (minimal-mild depressive symptoms), while 31 were assigned to the depressivegroup (moderate to severe depressive symptoms). Montgomery-Asberg Depression Rating Scale (MADRS) and Beck Depression Inventory (BDI) were used to assess depressive scores. Anthropometric measurements such as weight (kg), height (m) waist circumference (WC) and hip circumference was measured. Body mass index (BMI) and ratios such as waist to hip ratio (WHR) and waist to height ratio (WtHR) was also calculated. Blood pressure was measured using an automated AfriMedics blood pressure machine, while lipids was measured using a CardioChek plus analyser machine. Statistics were analysed via SPSS statistics program.

There were no significant associations between anthropometric measurements and depressive scores ($p > 0.05$). There were no significant correlations between lipid profiles and depression when running a Spearman's rhocorrelation ($P > 0.05$). However, total cholesterol and LDL-C were negatively associated with depression and triglycerides were positively associated with depression, after running a point-biserial correlation ($P < 0.05$). Overall, there were no significant associations between blood pressure measurements and depression ($P > 0.05$).

However, there was a significant moderate positive correlation between systolic blood pressure and MADRS scores in males ($P < 0.05$). Depressive scores positively and strongly correlated to how long it takes participants to fall asleep. There were also significant associations with regards to the secondary objectives. This study indicates the importance of determining the prevalence of depression among university students in South Africa and around the world as well as determining the factors influencing and associating with depression. If the prevalence and factors associated with depression are addressed, depressive symptoms in university students may be improved.

Keywords: Depression, Major Depressive Disorder, University of the Western Cape, Body Mass Index, Blood pressure. Lipid profiles, Montgomery-Asberg Depression Rating Scale, Beck Depression Inventory



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Abbreviations

MDD	Major Depressive Disorder
SSRI's	Selective Serotonin Re-uptake Inhibitors
NDRI's	Norepinephrine and Dopamine Re-uptake Inhibitors
MADRS	Montgomery-Asberg Depression Rating Scale
BDI	Beck Depression Inventory
WC	Waist circumference
WHR	Waist to Hip Ratio
WtHR	Waist to Height Ratio
WHO	World Health Organization
TCAs	Tricyclic Antidepressants
MAIOs	Monoamine Oxidase Inhibitors
LDL-C	Low-Density Lipoprotein Cholesterol
HDL-C	High-Density Lipoprotein Cholesterol
BMI	Body Mass Index
SP	Systolic Blood Pressure
DP	Diastolic Blood Pressure
MAP	Mean Arterial Pressure
HR	Heart Rate

ANOVA	Analysis of Variance
SADAG	South African Depression and Anxiety Group
HIV	Human Immuno-deficiency Virus

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Chapter 1
Introduction

1.0 Background

1.1 Depression

Depression (also known as unipolar disorder or major depressive disorder (MDD)) is a disorder which is characterized by having a constant low mood or a loss of interest in activities which were once enjoyed (Parekh, 2017). Depression is unlike natural mood fluctuations and emotional responses caused by "everyday" challenges. It may become a severe health concern if it is on-going and untreated. Depressive symptoms are known to vary from mild to severe and may include having a loss of energy or constantfatigue, feelings of worthlessness, difficulty thinking or making decisions as well as feeling sad. The worst result of depression as a result of no treatment or treatment failure is suicide. Annually, approximately 800 000 individuals pass away due to suicide. Suicide is known to be the second leading cause of mortality in 15–29-year-olds (World Health Organization, 2021). According to the World Health Organization (WHO), 50% of all the mental disorders begin at the age of 14, however most cases are untreated. In addition, it is said that women are more likely to experience depression compared to men (Pietrangelo, 2019).

When looking at the type or symptoms of depression, a clear division is made between individuals who have either had or have not experienced manic episodes. A manic episode is described as a period (at least one week) where an individual is in a state of amplified overall stimulation with enhanced expression and unpredictability (Berrios, 2004). Each of these cases may be chronic and may include relapses (World Health Orgnization, 2018). A manic mood may be described as feeling highly elated (euphoria) and feeling as if anything can be accomplished. Mania in a severe state may cause hallucinations or delusional states (Semple, 2005). Occasionally the manic episode can be classified as more irritable than elated. The manic feelings experienced should be adequate to cause impairment in their capability to function atwork or any other environment. Symptoms of mania cannot be the product of substance useor abuse or triggered by ill health (Bressert, 2019). Mania has been associated with creativity as well as artistic aptitude, thus, it is not always clear that the manic person needs medical help. These individuals often preserve adequate self-control to function normally or

are oblivious to that manic state. Manic persons often can be mistaken for being under the influence of drugs (Jamison, 1996).

Repeated depressive episodes are known as recurrent depressive disorder. At which point the person loses interest in every aspect of their day-to-day life as mentioned previously (Parker, 2014). Other symptoms experienced include anxiety, lack of sleep, decreased appetite and poor concentration. A person suffering from mild depression will often have trouble in completing everyday work as well as social activities, but will not entirely cease to function (World Health Organization, 2018).

Diagnosis of depression is based on behavioural reports of a patient by family members or friends, self-reports, as well as a professional examination and judgement. Depression is only diagnosed when these symptoms have been observed for at least two weeks (World Health Organization, 1992).

There are no current laboratory tests for depression, although doctors usually test for illnesses or ailments which may cause comparable symptoms (Dabrowski, 2005). This is important as some conditions may mimic the symptoms of depression, for example, thyroid imbalances, vitamin deficiency or brain tumours (Tartakovsky, 2018). If depression is not diagnosed in its initial stage, it could cause slow recovery in a patient's general health. The common onset age is between 20-30 years, with a peak between ages 30-40. However, when dividing the population according to the onset of depression according to age, there are five groups: Younger than 12 years of age - childhood onset, ages 12–17 - adolescent onset, ages 18–44 - early adult onset, ages 45–59 - middle adult onset, age 60 and older - late adult onset (Jaffe, 2002). Depressed individuals have a shorter life-expectancy than thosewithout depression, partially due to their increased vulnerability to illnesses and suicide.

Depression, according to the World Health Organization (WHO), significantly affects the disease burden as well as disability on a global level (Whiteford, et al., 2013). Depression affects 350 million people around the world (5% of the population), and one in 15adults is estimated to be affected (World Health Organization, 2011). Depression results in increased medical expenses and dysfunction in all areas of life which ultimately leads to a poor quality of life (World Health Organization, 2011). A 2013/2014 report indicates that the overall benefits per beneficiary per month (on average) paid to psychiatrists increased by almost 35% from 2011-2013 (Ismail A,

2017). The overall remuneration paid to psychologists increased by 26% and the overall benefits waged to mental institutions increased by approximately 58%.

1.2 Depression in South Africa

South Africa's Discovery Health's (Medical Aid Scheme) data shows that MDD contributes to > 40% of the mental disease burden. In-hospital mental costs amount to R773 million over a five-year period compared to R1.283m for out-of-hospital costs. In-house costs have increased by 113% from 2011-2016. This is due to the increased number of psychiatric beds available in hospitals (Ismail A, 2017).

South Africa has a 4.60% prevalence of depression, which accounts for approximately 2 400 000 people (World Health Organization, 2017). According to the South African Depression and Anxiety Group (SADAG), 1 in 6 South Africans suffer from substance abuse, anxiety ordepression (South African Depression and Anxiety Group, 2019). According to research conducted by the Department of Psychiatry and Mental Health at the University of cape Town, 1 in 3 women from low-income or informal settlements surrounding Cape Town, suffer from postnatal depression (Cooper et al., 1999). Similar research conducted in KwaZulu-Natal indicates that 41% of women that are pregnant are depressed (Rochat et al., 2006). This statistic is three times greater than the percentage of depressed pregnant women in developed countries. The operations director of SADAG, Cassey Chambers, stated that the statistics gathered are not a true reflection of the depression rate as people don't know where to get help or are too scared to ask for assistance. SADAG states that fewer than 16% of depressed patients receive treatment and over 85% of mentally ill sufferers are dependent on the public health-care services. Only 27% of severely depressed patients are treated, so ¾ of sufferers don't receive any treatment. There are only 18 beds for every 100 000 persons and only 1% are set aside for children and adolescents. Since the public sector of health care cannot cope with disease burden, the mental healthcare responsibility falls on the community.This includes community leaders, support groups, clinics, and counsellors (South African Depression and Anxiety Group, 2019).

Additionally, stigmas surrounding mental health illnesses cause major issues when it comes to disease treatment. For example, In the Zulu culture, there is no word for "depression", so it is not even seen as an illness that exists. Depression sufferers are therefore afraid of being disowned or afraid of discrimination from other members of the community. People who suffer

from mental illnesses are still seen as being “crazy”, “weak” or dangerous. Since depression often has no physical symptoms, it is seen as a figment of imagination. Experts suggests that dealing with the metal health issue in South Africa requires a combination of traditional healing and western medicine (Sorsdahl et al., 2009). This is because cultural beliefs playa major role in helping patients and their families and traditional healers may assist in this.

South Africa is a country with great levels of poverty and inequality (Posel, 2021). Over half of the South African population live below the poverty line, with countless living ininformal settlements, such as shacks, with large families (Statistics South Africa, 2017). The rate of domestic violence is high, and this could be linked to high levels of food insecurityand poverty (Gibbs et al., 2018). In addition, high unemployment statistics worsens these conditions. These socio-economic factors play a major role in depression and disease progression (Dartnell, 2020). The Western Cape Burden of Disease Reduction report stated that mental illness has a much higher prevalence in Western Cape than any other province. Western Cape had the highest 1-year and lifetime mental illness prevalence in South Africa, accounting for 39% of cases. To fight the disease burden, numerous policies have been set-up, such as the Mental Health Policy Working Group and the Integrated Counselling Strategy to combat the shortage of mental health workers.

1.3 Depression in University students

The mental health status of South African University Students is a serious health concern. For example, in the last five years, there has been an exponential increase in the number of suicides in university students across South Africa (van Zyl et al., 2017). The exponential rise in mental health cases has led experts to believe that South Africa is dealing with a mental health epidemic with regards to university students. There is a shortage of comprehensivedata on the prevalence of depression and suicide in university students of South Africa. This gap in knowledge is surprising considering that most mental health concerns begin in adolescence or early adulthood (Kesseler et al., 2007). Some research conducted investigating the prevalence of depression in university students (Bantjies et al., 2019; Ebert et al., 2019 and Pillay et al., 2021) have found that both depression and suicide is common and that university students have a higher rate of depression than the general population (Ibrahim et al., 2013). The increase in the number of mental health concerns among university students demands urgent attention. A study conducted by Rousseau et al., found that depression and suicide increased over a four-year period, 2016-2019 (Rousseau et al., 2021). The study also found that first-year students

had a higher depressive rate than other years. A study conducted by Stellenbosch University included 1337 students, found that 12% of students experienced moderate depression, while 15% experienced moderate to severe depression. This percentage is higher than the reported 9.1% average of the general South African population suffering from depression (Bantjes et al., 2016). Accessing mental healthcare is a great challenge in South Africa. Even though most universities and other tertiary institutions offer professional counselling or psychological help, students still struggle to access minimal care to support their mental well-being. Students who struggle to find adequate mental health care are at an increased risk of failing university and have a greater chance of dropping out of university. This would also exacerbate their already dilapidating condition. An e-intervention has been developed by Pharma Dynamics to address the issue of affordable and accessible mental healthcare. The electronic platform encourages mental health sufferers to share their concernsand struggles and addresses the barriers that prevent patients from seeking help, such as fear of discrimination or poor economic background.

From the above literature, it is seen that there is not enough detailed and available data with regards to depression prevalence among university students. This study aims to investigate the associations between body mass index (BMI), lipid profiles and blood pressure measurements with depression in university students.

1.4 Depression in relation to BMI, lipid profiles and Blood pressure

Obesity is defined as an illness whose main symptom involves excessive body fat that increases the risk of health problems (Mayo Foundation for Medical Education and Research, 2021). Depression and obesity are serious illnesses that both create large public health concerns and repercussions (Mathers & Loncar, 2006). Since depression and obesity both cause a higher risk in the development of cardiovascular disease and because both illnesses have such high prevalence, many studies have observed the association between obesity and depression (Penninx et al., 2001). Hand-in hand with obesity is the risk factor of having a high body mass index (BMI). Higher BMIs are associated with having an increased risk of developing depression, with women having a higher odds of developing depression than men (Newman T, 2018).

Dislipidaemia can be explained as suffering from abnormally high or low levels of lipids (fats) in the blood (REF). Abnormal levels of lipids in the blood may cause long-term health issues

such as increasing the chance of developing clogged arteries (atherosclerosis), stroke, heart attacks and other health related issues. The association between dyslipidaemia and depression stems from the effects of dysregulation of lipids on serotonergic neuron membranes. Therefore, serotonin transmission and receptor function become impaired (Wu & Comings, 1999). Thus, the incorrect lipid levels result in decreased active serotonin, leading to a depressed mood. Dysregulation of lipids may also result in an altered inflammatory profile which is associated with depression (Myint & Kim, 2003).

Another health factor associated with depression, is blood pressure, however, the relationship between depression and blood pressure has been an ongoing investigation. Certain studies indicated that blood pressure increased with depression or even indicated hypertensive stages (Rutledge et al., 2002 and Scherrer et al., 2003). Otherresearch studies observed decreased blood pressure measurements within depressed patients (Hildrum et al., 2007). In addition, some studies found no association between blood pressure and depressive symptoms (Yan et al., 2003 and Shinn et al., 2001).

From the above literature, it is seen that BMI, irregular lipid levels and blood pressure are all factors which are associated with depression, and the dysregulation of these factors puts one at risk of developing other serious health issues, such as cardiovascular disease and cardiometabolic diseases.

1.5 Problem statement and rationale

Depression is a very common and serious mental illness. This condition negatively affects the way you think; how you feel as well as the way you behave (Parekh, 2017). Several studies have shown that depression has a significant impact on both social and economic aspects in South Africa. Major Depressive Disorder (MDD) is the most common and incapacitating forms of depression. It is indicated by the South African Stress and Health Study that one in 4South Africans will suffer from this illness at some stage in their lives (South African Stress and Health Study, 2018). MDD has devastating potential on a person as it not only affects the patient, but it also has negative effects on other aspects of their life. Thus, both occupational and social aspects of a patient's life are negatively affected.

A depressed patient is therefore less productive in the workplace (Rovner et al., 1990). If a depressed employee is present in the workplace, it is shown that they are less likely to be

productive than an employee who is not present at work due to the condition.

A study performed by HEXOR Pty (Ltd), showed the effect of depression on employees at work-50% of depressed employees made and increased number of mistakes at work, whereas 54% stated that they took a longer period of time to perform straightforward tasks (Stander & August Korb, 2015). Bateman 2015 stated that the average number of days taken off work by depressedemployees is 18. This was taken from a survey conducted by the South African Depressionand Anxiety Group (SADAG) in 2015 (South African Depression and Anxiety Group, 2015).

Depression is not only associated with feelings of sadness but consists of other negative effects as well. Medical morbidity, suicide as well as mortality are also negative effects of MDD (Lepine & Briley, 2011). Furthermore, those suffering from chronic diseases have a higher risk of the development of depression compared to those who are healthy. Chronic diseases include cardiovascular diseases, hypertension as well as diabetes (Harpole et al., 2005)

Another disturbing issue is the prevalence of depression among young adults. University students are among the worst hit in South Africa, as well as around the world. South African university students are suffering more than the average general population (Bantjes et al., 2016). University students have heavy burdens placed on them, such as performing academically, perhaps learning in a different language or being the first family member to graduate. There are many risk factors for depression and this study will investigate whether body mass index (BMI), blood pressure and lipid profiles are associated with depression in University students at the University of the Western Cape. Obesity has been linked toincrease the risk of depression (Luppino et al., 2010). In addition, low blood pressure has also been linked to depression (Hildrum B et al., 2007). Furthermore, dyslipidemia (imbalanced lipids) is also associated with increased risk of depression (van Reedt Dortland et al., 2013). These factors will be investigated during this study.

By investigating the relationship of obesity (BMI), lipids and blood pressure with depression, we will be able to advise students on their current health status. Since students are under high pressure, they are likely to suffer from ill behaviours such as sedentary lifestyles or bad eating habits, which may increase BMI and lead to obesity. Others may suffer from high blood pressure or irregular lipids. By identifying students with these risk factors, who may or may

not suffer from depression, we may help educate them about their health risk factors and advise on how to improve their condition, should anything be found. This study also provides a gap in knowledge as it is very difficult to find the prevalence of depression among university students.

1.6 Research Question

Are BMI, blood pressure and lipid profiles associated with depression in university students?

1.7 Research Hypotheses

- High BMI scores will associate positively with high depressive (Montgomery-Asberg Rating Scale (MADRS) and Beck Depression Index (BDI)) scores
- High blood pressure levels will associate positively with high depressive scores
- High triglycerides will associate positively with depressive scores
- High levels of low-density lipoprotein cholesterol (LDL-C) will associate positively with high levels of depression
- Low levels of high-density lipoprotein cholesterol (HDL-C) will associate negatively with depressive scores
- High levels of cholesterol will associate positively with depressive scores

1.8 Research Aims and Objectives

1.8.1 Aim

This study aimed to determine the association of BMI, blood pressure and lipid profiles with Depressive Scores in university Students.

1.8.2 Objectives of the study

- To use standardized questionnaires to measure the severity of depression. These include the Montgomery-Asberg Depression Rating Scale (MADRS) questionnaire and the Beck Depression Inventory (BDI) questionnaire.
- To determine the association between BMI and depression in students.
- To determine the association between blood pressure and depression in students.
- To determine the association between lipid profiles and depressive scores in students.
- To obtain BMI scores by acquiring anthropometric measurements such as height (m) and weight (kg).
- To obtain waist circumference (WC), waist to hip ratio (WHR) and waist to height

ratio (WtHR) measurements.

- To obtain blood pressure measurements by taking blood pressure measurements of the participants using an automated blood pressure machine.
- To obtain lipid profiles of the students, including total cholesterol, triglycerides, high-density lipoproteins (HDL-C), and low-density lipoproteins (LDL-C) using the finger-prick method and lipid testing strips.
- To calculate the total cholesterol/HDL-C ratio after obtaining lipid scores via finger prick.
- To obtain habits such as food intake and sleeping habits.
- These habits and feelings will be obtained via an extended questionnaire to the already standardized MADRS and BDI questionnaires.
- To determine whether there are associations between depressive scores and food intake.
- To determine if there are associations between depressive scores and sleeping habits.
- To determine the prevalence of depression using the Depressive scores.

1.8.3 Secondary Objectives

1.8.3.1 To determine the relationship/associations between the variables and their relationshipto cardiometabolic disease

- For example, the association between blood pressure and BMI, since both blood pressure and BMI are risk factors for cardiometabolic disease.

1.9 Relevance of Research

There is research suggesting that there is an exponential increase in depression and suicide among university students in South Africa (van Zyl et al., 2017). However, there is notenough data and information to make definitive conclusions. This study aims to add to this gap in knowledge, particularly to the associations between BMI, blood pressure and lipid profiles with depression. All these factors have discrepancies within themselves where research is concerned. For example, some research has found that obesity is a risk factor and is associated with depression (Akinyemi et al., 2020), while others have found a U-shaped association (de Wit et al., 2009). This study will also provide insight to the secondary associations – associations between the variables, for example, lipid profiles vs. blood pressure. These variables are modifiable risk factors for cardiometabolic complications/disease which is also linked to comorbidities of depression.

1.10 Thesis Outline

This thesis is comprised of five chapters: Chapter one provides background knowledge on depression and the prevalence of depression in South Africa and Universities in South Africa. It also provides the research question, aims and objectives of the study as well as the relevance of research. The second chapter includes the literature review, exploring the treatment of depression as well as the relationships of each variable (BMI, blood pressure and lipid profiles) with depression. It also includes co-morbidities and risk factors associated with depression. Chapter three defines the methodology as well as research design of the study. The fourth chapter includes the results and statistical analysis, while the final chapter containsthe discussion of results in relation to other literature and our own findings. It also concludes the study and mentions the limitations, while providing recommendations for future studies.

1.11 Chapter Summary

The first chapter provided a background on depression and depression prevalence around the world, in South Africa and at University level. It also included the aims and objectives and concluded with the problem statement and relevance of research. The literature review will be presented in chapter 2.

Chapter 2
Literature Review

2.0 Introduction

Chapter two includes the literature review where different aspects of depression and factors of depression will be discussed. These factors include the treatment of depression and the side-effects of these treatments, factors which contribute to depression, depression and comorbidity as well as depression and the factors which are focused on in this study – obesity, dyslidemia, blood pressure, food habits as well as cardiometabolic disease (metabolic syndrome). Furthermore, depression and specific factors affecting university students will also be discussed in this chapter.

2.1 Treatment of depression

There are several antidepressant medications used, which have their own mechanism of action. The benefits of antidepressants stem from how they affect certain brain circuits and the chemicals, called neurotransmitters that pass along signals from one nerve cell to another in the brain. These chemicals include serotonin, dopamine, and norepinephrine. Each antidepressantaffects these neurotransmitters in a unique way (Andrade & Rao, 2010).

Even though there are many treatments for depression, less than 50% of individuals affected around the world (in several countries < 10%) actually receive it (World Health Organization, 2018). Antidepressant medication and psychological counselling (therapy) are the most common treatments of depression. While pharmacological options for treating depression has increased extensively in the last two decades, between one and two thirds of patients will not respond to first-line treatment of antidepressant medications (Simon, 2017). In addition, 15-33% of patients will not be responsive to several interventions. Challenges in public health care as well as by the patients themselves include: a lack of trained professionals (doctors or specialists), a deficiency in resources, difficulty accessing the resources as well asthe social stigmas linked to depression which often deters individuals from attaining help. In addition, there is a lack of sufficient, efficient and accurate assessments (World Health Organization, 2018). Many individuals with depression are often misdiagnosed or those without depression are diagnosedand are prescribed antidepressants. These issues are partially a result of only 14%

of psychologists available to treat almost 85% of the population.

There are several antidepressant medications used, which have their own mechanism of action. The benefits of antidepressants stem from how they affect certain brain circuits and the chemicals, called neurotransmitters that pass along signals from one nerve cell to another in the brain. These chemicals include serotonin, dopamine, and norepinephrine. Each antidepressantaffects these neurotransmitters in a unique way (Andrade & Rao, 2010).

2.2 Older Antidepressants: Tricyclic antidepressants (TCAs) and Monoamine Oxidase Inhibitors (MAOIs)

Tricyclic antidepressants (TCAs) and Monoamine Oxidase Inhibitors (MAOIs) were the first type of antidepressant medication. They are very effective; however, they also have serious side effects. Both TCAs and MAOIs are also known to be very dangerous when a patient has an overdose. These antidepressants were replaced by second generation medication due to their adverse effects. TCAs work by blocking serotonin transporters (SERT) and norepinephrine transporters (NET), causing an enhanced neurotransmission (Gillman, 2007). Consequently, the reabsorption of serotonin and epinephrine back into nerve cells is blocked after these chemicals are released into a synapse (Lodish et al., 2000).

MAOIs are often prescribed only after other medications have been unsuccessful due to their serious side effects. When prescribed MAOIs, it is essential to consume a strict diet due to hazardous and even fatal interactions with particular foods. These foods include certain pickles, cheeses and wines (Flockhart, 2012). Certain medications can be dangerous, and these include pain medications, various herbal supplements as well as decongestants (for nasal congestion). MAOIs also inhibit the body's ability to break down medications which aremetabolized by this enzyme (monoamine oxidase), such as Sudafed (a decongestant). This increases the risk for high blood pressure and increases the concentration of the amino acid tyrosine. Increased levels of tyrosine to a dangerous level may result in damage and even necrosisto the kidneys, liver, the nervous system and other organs (Bouyacoub et al., 2013). These medications also cannot be used in conjunction with other antidepressants such as selective serotonin reuptake inhibitors (SSRIs).

2.2.1 Reuptake Inhibitors:

Selective serotonin reuptake inhibitors (SSRIs), Serotonin and norepinephrine reuptake inhibitors (SNRIs) and Norepinephrine and dopamine reuptake inhibitors (NDRIs)

Various commonly prescribed antidepressant medications are called reuptake inhibitors. Reuptake is the process in which neurotransmitters are naturally reabsorbed by a neuron (nerve cell) after it has sent a message in the brain so that the neurotransmitters can be reused. A reuptake inhibitor prevents this reabsorption process from occurring. Now, instead of being reabsorbed, the neurotransmitter temporarily remains in the gap between the nerves, known as the synapse/neuronal junction (Oler, 2016). The purpose of reuptake inhibitors is to keep the levels of neurotransmitters high which could in turn improve the communication between neurons. This can result in the strengthening of brain circuits that regulate mood. In terms of depression, inhibiting the reuptake of certain chemicals will result in an elevated or improved mood. Each type of reuptake inhibitor targets a different neurotransmitter.

Selective serotonin reuptake inhibitors (SSRIs) and Serotonin and norepinephrine reuptake inhibitors (SNRIs) are the most common prescribed antidepressants today and have mostly replaced the TCAs (Preskorn et al., 2004). SSRIs work by increasing serotonin levels in the brain by blocking the re-uptake of serotonin within the brain, making the serotonin more available.

With increased serotonin, a happier mood and social behaviour can be achieved as well as improved digestion, sleep and sexual function and desire (Jakobsen et al., 2017). SNRIs and SSIRs have the same function, with the addition of blocking the re-uptake of norepinephrine in the brain. By making norepinephrine available, alertness and energy levels of the patient increases (Cashman & Ghirmai, 2009).

Norepinephrine and dopamine reuptake inhibitors (NDRIs) are an additional class of reuptake inhibitors, which target the reuptake of both norepinephrine and dopamine. NDRIs therefore prevent the natural reabsorption process of norepinephrine and dopamine and increase the amount available (Stahl, 2009).

Antidepressants may take several months before positive results are shown. It is estimated that at the beginning of treatment at least 50% of patients suffering from depression willrespond to the treatment, while only one in three people will actually achieve remission (O'Conner, 2009).

Patients on antidepressant medications often complain about side effects of medication as well as access to health care facilities and medication. This often leads to a relapse into depression as patients stop taking their medication. One of the most prevalent forms of

depression is Major Depressive Disorder (MDD), which is categorized by experiencing one or more major depressive episodes (APA, 1994). An episode is defined as having a depressed mood or loss of interest in activities that were once enjoyed in combination with four other symptoms previously mentioned, for at least two weeks (APA, 1994). According to the National Alliance on Mental Illness (NAMI, 2015), approximately 20% of people suffering from MDD will develop psychotic symptoms. Classification of MDD is as follows: mild, moderate or severe. The classification of depression depends on the symptoms of the patient. In other words, how well they are functioning or the level of dysfunction of the patient as well as the degree of suffering that the patient is experiencing (Fava & Kendler, 2000).

2.3 Side effects of antidepressants

The side effects experienced by ingesting TCAs include:

- Constipation
- Dizziness and drowsiness
- Slight blurring of vision
- Dryness of the mouth
- Excessive sweating (especially at night)
- Problems passing urine
- Heart problems such as palpitations and tachycardia(fast heartbeat)
- Weight gain

There are many common unpleasant side effects also caused by the ingestion of SSRIs and SNRIs, these include (APA, 2000):

- nausea
- fatigue and drowsiness
- Ingestion and stomach aches
- insomnia
- dry mouth
- blurred vision
- diarrhoea or constipation
- constipation
- dizziness

- Loss of appetite
- irritable or agitated
- anxiety
- Sexual problems such as low libido and erectile dysfunction
- Weight gain with increased risk of related illness (type 2 diabetes and hypertension)

2.4 Potential health risks

2.4.1 Serotonin Syndrome

Serotonin syndrome occurs when the levels of a chemical called serotonin in your brain become too high. It's usually triggered when you ingest serotonergic medications such as an SSRI or SNRI in combination with another medicine (or substance) that also raises serotonin levels, such as another antidepressant or St John's Wort (Volpi-Abadie et al., 2013). Symptoms may vary from mild to severe and the diagnosis is based on the present symptoms, medical history and the history of medications used. Initial treatment consists of discontinuing the serotonin medication. In more severe cases, a serotonin antagonist is prescribed (Ferri, 2016)

Symptoms include:

- Agitation
- Confusion
- Sweating
- Shivering
- Muscle twitching
- Diarrhoea

Severe symptoms include:

- Arrhythmia
- Seizures(fits)
- Unconsciousness
- Hyponatremia (reduced salt levels)

The symptoms of Serotonin Syndrome are often described as a clinical triad of abnormalities as they present as cognitive, autonomic and somatic effects (Boyer &Shannon, 2005). These

effects are as follows:

- Cognitive effects: agitation, headache, hypomania (mood elevation), hallucinations, mental confusion and coma.
- Autonomic effects: sweating, hyperthermia, shivering, vasoconstriction, nausea, tachycardia, diarrhoea.
- Somatic effects: tremors, hyperreflexia (overactive reflexes), myoclonus (muscle twitching).

2.5 Hyponatraemia

Hyponatraemia particularly affects elderly people who take SSRIs, which may cause a severe reduction in salt levels (hyponatraemia) (Lee et al., 2014). This may lead to fluid build-up inside the cells of the body which can be potentially dangerous. This is because SSRIs block the effects of a hormone that regulates the levels of sodium and fluid in the water. This makes the elderly more vulnerable as fluid and sodium regulation becomes more difficult for the body as people age.

Mild hyponatraemia may cause headaches, muscle pain, confusion and reduced appetite. More severe symptoms of hyponatraemia include listlessness, agitation, psychosis (hallucinations or becoming delusional), disorientation and seizures (fits). The most serious cases of hyponatraemia can cause one to stop breathing or can result in a patient entering a coma (Henry, 2015).

2.6 Contributing factors to depression

MDD is a result of a multifaceted interaction between psychological, social and biological factors. Individuals who have experienced challenging life events (trauma, unemployment or grief) have an increased risk of developing depression. There are several factors which may contribute to or cause depression. These factors include genetics, brain chemistry and hormone imbalances. Other risk factors such as a low self-esteem, physical or sexual abuse, anxiety, non-communicable diseases (NCDs) (diabetes or multiple sclerosis), as well assubstance abuse may also cause depression. With regards to genetics, family history is said to increase the risk of suffering from depression. This is because depression is a complex disease with multiple genes exerting small effects instead of having a single gene that contributes to the disease. Furthermore, researchers have found that people suffering from depression have smaller hippocampus regions, an area in the brain responsible for the storage of memories. This means

that are fewer serotonin receptors. Serotonin is a brain chemical/neurotransmitter that allows communication between regions of the brain involved in processing emotions. Some researchers have found that cortisol (stress hormone), has a shrinking effect on the hippocampus leading to a smaller size, while others theorize that some people are born with a smaller hippocampus, and are thus inclined to suffer from depression (Czéh & Lucassen, 2007). In addition, NCDs may contribute to depression as these illnesses co-exist with other major illnesses, causing anxiety and indolence, often leading to depression (Pan et al., 2019).

2.7 Comorbidities of Depression

When a patient suffers from depression along with another illness, it is described as comorbidity of depression (Ida & Igaku, 1998). It was demonstrated that individuals who suffer fromchronic diseases have an increased chance of experiencing depression, compared to those thatare healthy (Noel et al., 2004 and Harpole et al., 2005). Those who experience depression also have a notable rate of comorbidity with chronic diseases which ranged from 65-71% (Wells, 1991). Furthermore, depression may be the source of additional dysfunctions as well as disabilities compared to other chronic diseases (Hays, 1995). Those suffering from depression usually also suffer from other medical conditions such as diabetes, cardiovascular diseases as well as hypertension (Crockett, 2013). Primary health specialists may find the diagnosis of depression challenging as some patients with other diseases, for example diabetes, demonstrate symptoms highly similar to that of depression (Wells, 1989). Comorbidity of depression may be caused in several ways. For example, the development of type 2 diabetes is associated by an increase in cortisol secretion, which is also seen in individuals suffering from depression (Musselman, 2003). Cortisol, aglucocorticoid, raises blood sugar levels and thus plays a role in the development of type 2 diabetes (Lustman, 2007). Moreover, patients experiencing depression also have a greater risk of developing cardiovascular disease (CVD) (Ariyo et al., 2000). Furthermore, other diseases as well as neurological syndromes have been associated with an increased risk of developing depression. Fava et al (1987), determined that Addison's disease, hyperthyroidism as well as Cushing's syndrome can cause MDD. Another important comorbidity with depression is HIV (Human Immunodeficiency Virus) and AIDS (acquired immunodeficiency syndrome). Depression is associated with a greater severity in those livingwith HIV. In addition, mental health disorders are linked to poorer adherence to antiretroviralmedication. Depression is a risk factor for HIV as it impairs judgement and understandingand is thus linked to high-risk behaviour. A study showed that 19% of individuals receiving normal HIV check-ups in Cape Town have mental disorders –

depression, post-traumatic stress disorder and alcohol dependence.

The University of Michigan carried out a large-scale national survey on the comorbidity of depression. The results showed that > 50% of the patients who were clinically diagnosed as depressed also suffered from anxiety disorders and a 1/3 had comorbidity with either drugs or alcohol. It is clear depression often presents with not only chronic illnesses, but also mental illnesses (Davidson & Ritson, 1993). When depression presents with other mental disorders, they are more severe than when either are present alone (Kang et al., 2015). Furthermore, hospital admissions and suicide rates are higher with poorer prognosis (McCallum et al., 2016). This is predominantly noticeable in young adults with depression. The occurrence of MDD increases significantly across adolescence, with noticeably larger increases among females compared to males (Avenevoli S et al., 2015). The relationship between depression and mental disorders can present in one of three ways: 1) When the other mental disorder occurs before depression it may be the cause of depression; 2) If depression preceded the other disorder, then depression may be the cause of the comorbidity and 3) When depressionand the other disorder occur simultaneously then they may have different causes (Kessler et al., 1996).

The World Health Organisation (WHO) World Health Survey (WHS) performed a study on adults (18+) to investigate the presence of depression with four chronic physical diseases – angina, asthma, arthritis and diabetes (Hyman et al., 2006). There were 245 404 participants from 60 countries. Approximately 9.3%-23.0% with 1 ormore physical diseases also suffered from depression. This was significantly higher than the probability of suffering from depression alone ($p < 0.001$). After correcting for socio-economic factors, the comorbidity of depression showed the largest effect on worsening health scores compared to depression alone, chronic physical diseases alone as well as a combination of any physical disorders (Fortin et al., 2004).

2.8 Depression and obesity

Worldwide, there is a growing incidence of overweight and obesity in both developing and developed countries (Haidar & Cosman, 2011). In the last two decades, developing countries' obesity rate has tripled due to urbanization, greater consumption of fast foods and highcalorie foods as well as adopting a sedentary lifestyle (Popkin et al., 2012).

Depression and obesity are serious illnesses that both create large public health concerns and

repercussions (Mathers & Loncar, 2006). Since depression and obesity both cause a higher riskin the development of cardiovascular disease and because both illnesses have such high prevalence, many studies have observed the association between obesity and depression (Penninx et al., 2001). The real question is whether depression causes obesity or whether obesity is the cause of depression, and whether each is predictive of each other's development or if there is no association between them. Studies have found bidirectional associations between obesity and depression. A systematic review with meta-analysis of longitudinal studies in 2010 (Luppino et al., 2010), indicated that depressed patients had a 58% increased risk of becoming obese, while obese persons had a 55% increased risk of developing depression over time. The association between depression and overweight was weaker than the association found between depression and obesity; thus, the higher the BMI, the higher the risk for depression (Luppino et al., 2010).

There are numerous ways in which obesity can cause depression and vice versa. Obesity is known to be an inflammatory state (Shoelson et al., 2007). Inflammatory states are dividedinto two: acute inflammation and chronic inflammation. Acute inflammation occurs when your body fights infection or when a wound/injury needs healing. When this process occurs in excess, the immune system continually releases white blood cells and chemicals causingthe process to lengthen indefinitely. This is now known as chronic inflammation. This occurs in the state of overweight or obesity (Harvard Health, 2020). The immune system becomes aware of the large number of visceral fat cells surrounding your organs and mistakes them forforeign cells. The white cells then begin to attack these fat cells and the release of whiteblood cells and chemicals continue. As a person remains in an obese state, the longer the body stays in the state of inflammation (Emery et al., 2007). Gaining weight has shown to stimulate inflammatory pathways and studies have shown that inflammation has been associated with depression (Vaccarino V et al., 2007 and de Heer et al., 2014). Inflammation could be seen as a mediator in the association since it contributes to both depression and obesity. Central obesity, also known as abdominal obesity, is a superior predictor of depression than overall adipose mass (Bosomworth, 2019). Rising numbers of research proposes that metabolic irregularities caused by abdominal obesity leading to metabolic disease may be accountable for the increased occurrence of depression found in obese persons. Metabolic disturbances are either directly or indirectly responsible for the control of moods and emotions. In addition, physical pain which isdirectly linked to obesity is also known to cause depression (Beesdo et al., 2009).

Furthermore, the association between obesity and depression could be caused by the dysregulation of the hypothalamic-pituitary-adrenal axis (HPA axis); since dysregulation of the HPA-axis may cause obesity and it is known to be involved in depression and depressive symptoms. Obesity could then cause depression due to the malfunctioning axis (Björntorp., 1996).

In addition, being overweight or obese increases mental discomfort. This even extends to the perception of being overweight, i.e., the person is not necessarily overweight/obese, but feels that they are (Atlantis & Ball, 2008). In many cultures around the world, being "thin" is considered attractive and this is due to the need of social approval and other cultural influences (Derenne & Beresin, 2006). Overweight and obesity could reduce body satisfactionas well as self-esteem. These are both risk factors for depression (Hoek et al., 2005).

2.9 Depression and dyslipidaemia

Dyslipidaemia refers to having incorrect levels of one or more types of lipids (fats) in your blood (Dixon, 2016). The three main types of lipids include low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C) and triglycerides(Rosenson, 2021). LDL-C is known as the "bad" cholesterol as it causes the formation of plaques in artery walls leading to stroke or heart attack (Rosenson, 2020). HDL-C is the "good" cholesterol as it is responsible for the removal of LDL-C from your bloodstream.Low levels of HDL-C may cause a rise in LDL-C as there is not enough HDL-C to remove the LDL-C particles (Dixson, 2016). Low levels of HDL-C are associated with higher cardiovascular disease (CVD) risk. Triglycerides are derived from foods that are consumed (calories) but are not burned right away. They are therefore stored in adipose (fat) cells. Triglycerides are released in the form of energy, however, if you consume more calories than you use/burn, it will result in a rise in triglyceride levels (American Heart Association, 2021). High triglycerides also increase the risk of heart attack and stroke (National Heart, Lung and Blood Institute, 2018). Dyslipidaemia may also include high or low levels oftotal cholesterol. Cholesterol is an organic molecule describes as lipid a fatty, waxy substancefound in all cells of the body (US National Institutes of Health, 2019). Bodily functions require cholesterol to function, such as manufacturing vitamin D and hormones and substances that aid in digesting food (Hanukoglu, 1992). Lipid levels are a biological markers or predictors of depression (Engelberg , 1992). The association between dyslipidaemia and depression stems from the effects of dysregulation of lipids on

serotonergic neuron membranes. Therefore, serotonin transmission and receptor function become impaired (Wu & Comings, 1999). Thus, the incorrect lipid levels result in decreased active serotonin, leading to a depressed mood. Dysregulation of lipids may also result in an altered inflammatory profile which is associated with depression (Myint & Kim, 2003). In addition, the combined effect of dysregulation of lipids, stress and the interaction with the hypothalamic-pituitary-adrenocortical (HPA)-axis may also result in depression (Francis, 1979).

2.10 Depression and blood pressure

Over time, research examining the relationship between blood pressure and depression has been inconsistent. Certain studies indicated that blood pressure increased with depression or even indicated hypertensive stages (Rutledge et al., 2002 and Scherrer et al., 2003). Other research studies observed decreased blood pressure measurements within depressed patients (Hildrum et al., 2007). In addition, some studies found no association between blood pressure and depressive symptoms (Yan et al., 2003 and Shinn et al., 2001).

Psychological factors such as anxiety and depression have been associated with the development of hypertension for many years. The theory that mental strain could be the cause of high blood pressure dates to Moschcowits, in 1919, who described characteristics of a strained personality in persons with hypertension (Moschcowitz, 1919). This theory was supported by numerous studies, indicating that anxiety, depression, and anger lead to hypertension. The theory was then scrutinized over the psychological scales applied (Rutledge, 2002). In recent studies, the results have been contradictory to this theory, as depression has been associated with lower blood pressure (Atlantis & Ball, 2008; Licht et al., 2009 and Lenoir et al., 2008). This was also found when a large population study established that depressive symptoms indicated lower blood pressure 11 years later (Hildrum et al., 2011). Contrary to these findings, a cohort study that used repeated measures of bloodpressure and depressive scores indicated that blood pressure increased with increased depressive episode (Nabi et al., 2011).

In addition, the study conducted by Hildrum and colleges (2009), which indicated lower blood pressure in depressed patients, contradicts the established link betweenpsychopathology, such as depression, and cardiovascular disease (Lenoir et al., 2008). A low cardiac vagal control (CVC) is found in depressed patients, but low blood pressure is usually associated with high

CVC (Licht et al., 2009). This could be explained using antidepressant medication which confounds the relationship between depressionand blood pressure (Hamer et al., 2010). It is found that the relationship between CVC and depression is caused by antidepressants. Patients who used SSRIs, TCAs and noradrenergic and serotonergic (NS) antidepressants showed significant decrease in CVC. TCAs are known to have the greatest effects that cause increase in heart rate which is in response to autonomic effects of the antidepressant (Koschke et al., 2009).

2.11 Depression and self-esteem

Self-esteem is having confidence in one's own abilities and worth (Trzesniewski et al., 2003) and is defined as a subjective assessment of self-worth. Studies have indicated that depression and self-esteem are closely related (Coyne et al., 1998) and that having a low self-esteem is a risk factor for depression. Models showing this relationship include the Scar Model, the Vulnerability Model, and the Reciprocal Relation Model. The Scar Model states that low self-esteem is a result of depression which diminishes self-esteem. Depression therefore"scars" the sufferer over time (Shahar & Davidson, 2003). The Vulnerability Model states that the risk of suffering from depression increases with a low self-esteem (Orth & Robins, 2013). Low self-esteem is therefore a risk factor and a possible cause for depression insteadof a result of it. Moreover, the Reciprocal Relation Model combines the first two models - low self-esteem can be the cause or the result of depression. These models have been validated by several longitudinal studies with large sample sizes and intricate statistical analysis (Orth et al., 2008; Shahar G & Henrich, 2010).

2.12 Depression and sugary treats and fast foods

Food has many effects on your mood and emotions. You want food not only when you're hungry but also when upset or angry and when you've eaten you feel delighted. Food consumed has a long-term effect on health, for example, consuming too much sugar increases your risk for mood disorders, such as depression. Sugar is found natural in vegetables, grains, and fruit. However, it is also found in refined food such as bread, pasta, cake, candy, and soda drinks. The consumption of too much simple sugars may increase the risk for mood disorders such as depression as well as other chronic diseases. A study conducted in London by Akbaraly T et al., found that consuming good sugars found in fruits and vegetables decreases the risk for depression, while consuming processed sugars such as fried foods,

dessert, and processed meats increases the risk for developing depression (Akbaraly et al., 2009). Research performed in rats found that sugar was more addictive than cocaine. High levels of sugar stimulate the brain's reward centres and diminish the brain's self-control mechanisms (Lenoir et al., 2007). A diet containing many fruits and vegetables reduces inflammation in the body, whereas, a diet containing a large amount of refined carbohydrates (sugars), promotes inflammation. Chronic inflammation is associated with several health conditions including cardiometabolic disease, cancer as well as depression. Symptoms of inflammation are common with depression, such as increased sensitivities to pain, loss of appetite and changes in sleeping patterns. Thus, depression could also be an indicator of underlying inflammatory conditions. Men are at an increased risk for mental health issues because of sugar compared to women. A study conducted by Knüppel A et al., found that men who consumed 67 grams or more of sugar per day had a 23% increased risk of developing depression, compared to men who consumed 40 grams or less, who had a lower risk of developing depression (Knüppel et al., 2017). Spanish scientists found that consuming high amounts of commercially baked goods increases the risk of developing depression by 38%. The findings also suggest that trans-fats may play a role. Trans fats are known to lead to inflammation and increases the risk for developing cardiovascular diseases (Sánchez-Villegas et al., 2011). In addition, scientists from the University of Granada discovered that a high intake of fast-food results in a 51% increased risk of developing depression compared to a low or no in-take of fast foods (Sánchez-Villegas et al., 2011). The research also revealed that there was a dose relationship between fast-foods and depression, so the more fast-foods consumed, the higher the risk of depression. Fast-food consumption ties in with the same concept as high sugar consumption, where a diet high in inflammatory foods causes systematic inflammation (inflammation of the whole body), which directly increases the risk for developing depression. Systemic inflammation results in the transport of pro- inflammatory molecules to the brain, which also affects neurotransmitters responsible for the regulation of moods (Lassale et al., 2019).

2.13 Depression and cardiometabolic disease/metabolic syndrome

Cardiometabolic disease (CMD) contains a group of risk factors that eventually increases the risk of developing heart disease (cardiovascular disease) as well as diabetes (Fatahi et al., 2020). People who suffer from CMD have a 3-5-fold greater chance of being stricken with cardiovascular disease and death compared to those who do not have CMD. Metabolic

syndrome is diagnosed based on having at least three of the five symptoms of CMD. The five symptoms include visceral obesity, high blood pressure, high triglycerides; high fasting blood sugar levels (glucose) and low HDL-C levels (GhariPour et al., 2006 and Jang, et al 2010). Suffering from more than one symptom is worse than suffering from one alone. A factor that may be associated with CMD is depression (Fatahi et al., 2020). According to research, depression has doubled the risk of developing CMD in the general population (Foley DL et al., 2010). Those suffering from depression are at greater risk for developing CMD as a result of poor health choices (sedentary lifestyle and pooreating habits) (Kinder et al., 2004). These poor lifestyle choices result in weight gain, which result in obesity and lead to type-2 diabetes and cardiovascular disease. Research has shown conflicting results between CMD and depression. Some research has found no association between depression and CMD (Demirci et al., 2011 and Herva et al., 2006), while others have found associations between certain components of CMD and depression (Meitolla J et al., 2008). The worldwide occurrence of depression is rising exponentially and is said to be the second-leading cause of mortality by the year 2030 (WHO, 2020). The association between obesity and diabetes could be linked to neuro-immune pathways (Martins et al., 2019). Dysfunctional adipose tissue (fat-tissue), also known as adipocyte hypertrophy stimulates the production of inflammatory mediators (Gregor & Hotamisligil, 2011). This rise in inflammatory mediators affects the central nervous system, resulting in neuro-inflammation (inflammation of neurons in the brain) in the hippocampus and hypothalamus (Castanon et al., 2015). Research has shown that depression and diabetes aggravate each other. Depression is known to hinder the secretion of islet cells in thepancreas, thus decreasing the regulation of glucose metabolism in those suffering from diabetes (Vancassel et al., 2018). Diabetes also puts the body in a state of chronic inflammation. Depressive patients have elevated levels of pro-inflammatory cytokines. Pro-inflammatory cytokines are signalling molecules that are released by immune cells that promote inflammation. Chronic inflammation causes changes in brain neuro-circuits and neurotransmitters that lead to depressive symptoms. Rising levels of Tumour-Necrosis-Factor-α (TNF-α), in the brain striatum and hippocampus have been linked to depressive and anxiety symptoms (Haji et al., 2012). Further studies should investigate the link between obesity and diabetes with depression and how their connected pathways lead to inflammation and eventually depression.

2.14 Depression and University students

For many students, starting university is an exhilarating experience; however, many students

experience stress and anxiety as they face the challenges of life in their new chapter. Several factors contribute to the risk of developing depression and many students are in fact unprepared for university life.

When attempting to deal with the challenges, many students feel overwhelmed and isolated, which cause some students to suffer from depression. It is not unusual for university students to suffer from depression, as The National Institute of Mental Health reports that 75% of people suffering from anxiety disorders start to show signs and symptoms before 22-years-of-age, which is the age bracket of many university students. Studies have shown that a higher percentage of current students are suffering from depression than those from previous generations (The National Institute of Mental Health, 2012).

2.14.1 Novel surroundings

For a large number of students, starting university means leaving home for the first time and also living away from home for long periods of time. Some students may find this exciting, while others may find it overwhelming. Certain students take the opportunity to explore their identity and grow as an independent individual, while others feel homesick, alone and isolated (McAlpine K, 2020). Living in a new environment comes with changes and challenges, perhaps such as sharing a room with a roommate in university residence or trying to make new friends. Other challenges include managing eating habits as well as sleeping habits as these will also change with the unpredictable schedule in the new environment. All these factors come with just one reason a student may develop depression – leaving home and entering a new environment. When all these factors overwhelm the student, making them sad and anxious and unable to function, they are at high risk of depression (Brennan D, 2021).

2.14.2 Financial strain

Financial burden is something that has been around since the beginning of time; however, students are facing a larger problem than ever before – fewer and poorer job prospects. Today, you can have and own a degree certificate, but it doesn't guarantee a job after graduation. This is a great concern for students, as many will face high amounts of debt as soon as they graduate (Kerr M, 2017). A large number of students come from poor financial backgrounds and their families and themselves cannot afford food or transport, let alone university fees and

everything that comes with it. All students are expected to have access to the internet as well as a computer or laptop to complete university tasks. This is impossible for numerous students who can't even afford to put bread on their table. Ohio State University National Student Financial Wellness Study established that 70% of college students described feeling anxious about their finances. More than 50% worried about paying for fees, 50% worried about their monthly expenses and 32% left their studies due to the debt of money they owed (OSU, 2020). Research from the Hope Centre for College, Community and Justice revealed that food insecurity and housing issues are more prevalent than before. Approximately 13% of students are homeless, while 20-40% of community college students experience food insecurity (The Hope Centre, 2021). The "Hungry for Homeless in College" findings show that first year students who have housing insecurity is linked to a 10% reduction in the chances of obtaining a degree (Goldrick-Rab S, Richardson J and Hernandez A, 2017).

For numerous students, demographic characteristics and housing insecurity is associated with lower academic records and part-time enrolment (compared to full-time). This is usually because low-income students often have part-time jobs. These students have the impossible decision of either choosing to go to class or going to work. Missing work means you don't eat and missing class means you're at an academic disadvantage. Furthermore, low-income students can't afford to take up unpaid internships which may benefit their career in the future (Perman C, 2019).

Worry after worry about financial stress and housing security leads to continual anxiety, stress and often depression. Financial stress prevents students from focusing on long-term life and academic achievements and may also lead to high-risk decision making, which has the potential to be disastrous.

A dire consequence of financial strain includes students dropping out. This is a (very) short-term solution to a long-term problem. This is because without the need to pay for tuition or books, wages last a lot longer. However, the cost of leaving university or college is much higher with much greater consequences (Johnson T, 2011). Leaving university means than perks such as work-study opportunities are lost; scholarships are retracted and subsidized student prices are taken away (transport, internet and accommodation). A greater concern is that student loans are now expected to be paid back, without the higher earning power of a degree (Porter E, 2013).

Generally, financial issues are a major factor for depression, but students are even more affected as they're young, newly independent and have more and more challenges every day.

A few other points about student financial problems, which may come from nativity of being financially independent include Students not tracking their expenditure; students not creating a monthly or weekly budget; students not knowing the difference between what is a need and what is a want and students not creating a plan to pay off debt. These are all learning curves that many students either do not know of or are not prepared for. Putting all these factors together, it is a highly stressful environment which can easily influence depressive symptoms (Noronha P, 2021).

2.14.3 The End of Relationships

University is often seen as a tie for young love, unfortunately, it is not uncommon for breakups to occur. These breakups more often than not bring about depressive emotional states. The risk of developing depression is associated with intrusive thoughts as well as difficulty controlling those negative thoughts. Another problem with breakups is that it causes sleeping difficulties. Approximately 43% of students suffer from insomnia for months after a breakup. Sleep as, mentioned previously is an important factor for depression and lack of sleep is known to cause depression. Students who are adversely affected by breakups often have underlying issues from childhood, such as abuse or neglect. Others feel betrayed or had an insecure attachment to their partner and were unprepared for the breakup. Such strong feelings of betrayal, heartbreak and again, isolation and loneliness put the students at risk for depression (Field T, 2009).

2.14.4 High-risk behaviour

Depression often has serious negative emotional and physical effects on its patients, especially students. Many students are reluctant to seek professional help or don't even know where to find it. Depression not only negatively affects academic achievements but also social interactions. Numerous students are so depressed that they can't function or carry out their daily routine. Depression in university students may result in unhealthy habits as well as risky behaviour (Johnson T, 2008). Certain students may turn to alcohol or substance abuse to manage their feelings. This doesn't necessarily mean that depressed students will consume more than their colleges or friends, but they are more likely to partake in high-risk behaviour such as unsafe sex or turn to heavy drugs to cope with their emotional state (Brennan D, 2021). Depression is also a major risk factor when it comes to suicide. Among people between the ages of 15-24, suicide is the third leading cause of death. Suicide is a global problem and over

the last decade, the rate of suicide has increased exponentially, especially in adolescents and young adults (CDC, 2020).

As a global community, as a university community and a general society, everyone needs to do better in seeing the signs of depression and suicide and also seeking help and guidance to help loved ones, colleges or even strangers get the assistance they need. Too many people are alone, isolated, and unheard. Too many people are afraid of seeking the help that they need.

2.14.5 HIV status

Human Immunodeficiency Virus (HIV) continues to be a worldwide health issue, with increasingly new infections and continued unsafe sexual behaviours by people around the world (UNAIDS, 2019). HIV is a virus which makes people more susceptible to infection and disease by attacking their immune systems, making their bodies too weak to fight infection (Powell MK et al, 2016). HIV is spread via contact with bodily fluids such as blood, semen, vaginal fluid, rectal fluid as well as breast milk from mother to baby (Mabuka J et al, 2012). HIV is most often spread via unprotected sex or sharing syringes for drug abuse. If HIV is left untreated, it will lead to AIDS (Weiss RA, 1993). Someone is considered to have progressed to AIDS when their CD4 cell count (white blood cell count) drops below 200 cells per cubic millimetre of blood (200 cells/mm3). Patients with AIDS are more vulnerable to other infections and are at high risk of death (Kwenti T et al, 2014). Since its discovery in the 1980's, HIV has killed 35 million people globally (UNAIDS, 2018). Most HIV-positive individuals are found in Sub-Saharan Africa, with South Africa being the most affected region (UNAIDS, 2019). Even though HIV infects people of all ages, adolescents and young adults aged 15-24 are accountable for 50% of all new HIV infections. Most young people diagnosed with HIV are women (UNAIDS, 2016).

Universities are seen as high-risk zones that boost the spread of HIV infections as students are known to participate in high-risk behaviour (Smith ML et al, 2014). Students engage in risky sexual behaviours including multiple sexual partners, substance abuse before engaging in sexual acts, having much older sexual partners, sexual intercourse with strangers, incorrect views about condom use as well as partaking in unprotected sex (Agardh A et al, 2011). These acts are high-risk as they make students vulnerable to HIV infection or increase the rate of infection by those who are already HIV-positive. Young students are also more vulnerable to

the risks of contracting HIV as they have misconceptions about HIV and are also easily influenced by their peers to engage in risky sexual behaviour (Andrew PO et al, 2018).

It is therefore of utmost importance that the youth of South Africa are informed about HIV as well as the risks and behaviours that may increase their risk of contracting the virus. The extent of knowledge about HIV has been explored at many universities; however, most of the research was conducted at urban-based universities, while rural-based universities were not included (Reddy P et al, 2011 and Mavhandu-Mudzusi AH, 2014). Generally, university students were knowledgeable about HIV, but no information has been given to or received from rural-based universities.

2.15 Chapter Summary

This chapter provided a literature regarding depression and its co-morbidities and risk factors. It also included the relationship between depression and the variables being looked at in this study (BMI, blood pressure and lipid profiles) according to other literature. It also provided insight to the relationship between depression and self-esteem as well as the link between depression and high sugar levels together with a high in-take of fast or fatty foods. In addition, depression and cardiometabolic disease was also mentioned. Lastly, chapter 2 provided insight to the challenges that university student face and their relationship to depression. Chapter three will present the research methodology and research design.

Chapter 3
Research Methodology

3.0 Introduction

Chapter three describes the research methodologies and protocol followed in this study. This chapter will include the research study design and setting as well as mention the inclusion and exclusion criteria of the study. It will explain the data collection and randomization methods used as well as each protocol used to measure anthropometry, blood pressure, lipid

measurements and lipid profiles. Furthermore, it will state the COVID-19 protocols that were followed, and the statistical analysis methods used. Lastly it will describe the ethical clearance obtained and procedures followed after the participation of the university students.

3.1 Research Design

This study used a cross-sectional study design in which already validated and standardized questionnaires were used to collect qualitative and quantitative data on MDD patients. The dependent variables in this study were the depressive scores from the depression questionnaires.

The initial sample size calculation yielded a result of 65 participants in total. This was based on a difference in mean MADRS scores of 7.7 at 5% significance and a power of 80% (Finzi & Rosenthal, 2014). Students from the University of the Western Cape were included in the study. In total 63 participants were included in the study. No participants were lost during the duration of the study. The 63 students were included out of 128 students who initially completed the surveys, as they were willing to come to campus. Willingness of students to come on to campus duringthe COVID-19 pandemic served as a problem.

This was a comparative study, where the participants were assigned to either a control group or depressed group according to depressive scores. The control group consisted of participants suffering from minimal to mild depression, while the depressed group consisted of participants with moderate to severe depression. According to the Montgomery-Asberg Depression Rating Scale (MADRS) there were 32 participants in the control group and 31 in the depressed group. According to the Beck Depression Inventory scale, there were 30 participants in the control group and 30 participants in the depressed group. This is because

the BDI was a self-report questionnaire sent to participants and three did not complete it. The MADRS questionnaire was a face-face interview and was completed when data collection was observed.

3.2 Research Setting

The study took place at the University of the Western Cape (UWC). All participants included attended the University of the Western Cape, including first years until doctoral level.

3.3 Inclusion criteria

Adults (university students) at the age of 18and above who suffer from symptoms of MDD

were included in the study. Both males and females were included in this study. All students from first year up to PhD students could participate. All participants who completed the questionnaire (MADRS) correctly were included in the study, no matter their depressivescore as the prevalence of depression also needed to be determined.

3.4 Exclusion Criteria

Patients younger than 18-years-old were excluded from the study. Patients were excluded if they showed signs of suicide or if they had a history of substance abuse. If indications of suicide or substance abuse were found, patients were referred to the campus doctor or the psychiatric facility at Tygerberg Hospital. Patients were excluded from the study, if the information provided was incomplete or found to be incorrect/misrepresented.

3.5 Data collection

3.5.1 Recruiting participants and procedure

Students were recruited via advertising platforms. Media usages such as UWC Communication and Instagram was used attract and recruit students. UWC communication sent out a mass email to the entire university advertising the study. Students were initially recruited using the MADRS questionnaire. Potential participants first completed the MADRS questionnaire on their own. These questionnaires were then thoroughly checked, and the scores were tallied. Potential participants who completed the MADRS questionnaire correctly and with no missing information were contacted to participate in the study. Information sheets (Appendix A) were sent to participants before they came for their face- face session and data collection. Extended questions and research survey questions (Appendix F) were also sent to participants to complete before the data collection session. This included the other depression questionnaire, the Beck Depression Inventory (BDI) scale, body image questions, eating habits and sleeping habits. Questions about medication and general health were also included here. A room in UWC Life Science Building was converted into a clinical room where the interviews and data collection took place privately. Participants were scheduled for an hour session, where four participants' data collection was performed at the same time. There were four or five stations per session, so each participant was at one particular station at a time (interview, anthropometry, blood pressure and lipid profiles). COVID-19 protocols were also maintained and always upheld. The MADRS questionnaire was re-done face-face at the interview station to avoid any discrepancies and any other questions or concerns could be aired here. Participants were provided with consent forms (Appendix B) and had to provide written

consent to partake in the study.After completing the interview, the participants went on to the rest of the stations. Low depressive scoring patients (0-19) formed the control group, while moderate to high scoring patients (20 to over 34) formed the depressive group. Compensation in the form of a snack bag was provided for the participants. The study was conducted in an ethical manner, asstated in the ethical considerations.

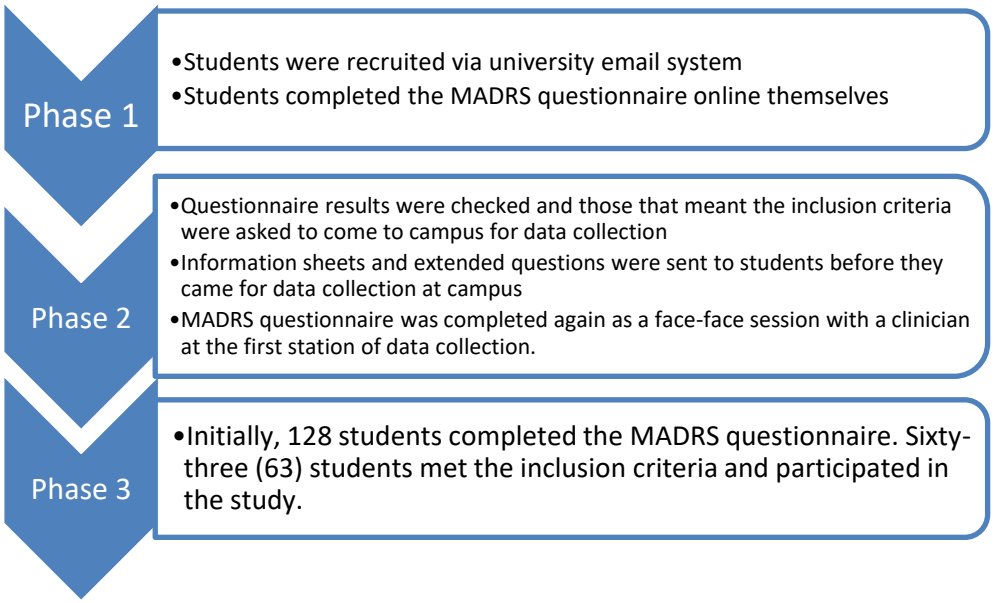

Figure 3.1: Flow-chart of participant recruitment process

3.5.2 Questionnaires

Standardised questionnaires known as Montgomery-Asberg Depression Rating Scale (MADRS) and Beck Depression Inventory (BDI) were used as baseline questionnaires to assess patients' depressive scores as well as their quality of life.

The MADRS questionnaire was a verbal interview performed by an interviewer or trained specialist, while the BDI questionnaire was answered by the participants themselves (Svanborg, & Åsberg, 2001).

3.5.2.1 Beck Depression Inventory (BDI)

The BDI is a multiple-choice questionnaire that consists of 21 questions and that is self-

reported. It is a psychometric assessment that measures risk factors for depression. Each question has 4 possible answers – 0, 1, 2, or 3 (ascending in intensity). The total score ranges from 0 to 63 and it is the sum of all the answers. For example, a score of 0-13 indicates minimal depression while a score of 29-63 indicates severe depression. The questionnaire takes roughly 5 to 10 minutes to complete (Beck AT et al., 1961).

The usual cut-offs are:

- 0–13: minimal depression
- 14–19: mild depression
- 20–28: moderate depression
- 29–63: severe depression.

3.5.2.2 Montgomery-Asberg Depression Rating Scale (MADRS)

This questionnaire consists of 10 items which asses the patients' mood. The items (questions) are scored between 0 and 6. Between the intervals there are also three intermediate levels (1, 3, and 5). The questionnaire includes the following items: 1. Apparent sadness, 2. Reported sadness, 3. Inner tension, 4. Reduced sleep, 5. Reduced appetite, 6. Concentration difficulties, 7. Lassitude, 8. Inability to feel, 9.Pessimistic thoughts and 10. Suicidal thoughts. The total score is calculated by summing the answers of the 10 items, ranging between 0 and 60 (higher scores indicate increased depression or depressive symptoms). It takes approximately 15-20 minutes to complete (Fantino & Moore et al., 2009).

The usual cut-off points are:

- 0 to 6 – normal /symptom absent
- 7 to 19 – mild depression
- 20 to 34 – moderate depression
- >34 – severe depression

3.6 Randomization

The participants were not randomized. All participants who completed the initial MADRS were selected to participate. This was directly correlated to the response rate to the advertised study. Therefore, whoever responded to the study was allowed to participate, providing they were

within the inclusion criteria and that the information provided was correct and complete.

3.7 Anthropometric measurements

After the face-face consultation with the patients, the anthropometric measurements of weight, height, BMI (body mass index) and waist circumference were performed according tothe Centre for Disease Control (CDC) protocol (CDC, 2015).

3.7.1 Weight

A digital scale was be used to measure the subject's weight. A spring-loaded scale should not be used. The scale was placed on a firm surface such as a wooden floor or on tiles instead of carpet flooring. The subject removed his/her shoes and removed all heavy clothing and other accessories (tie or scarf). The participant stood on the scale with both feet in the centre of the scale, while looking directly ahead. The weight of the subject was then recorded to the nearest kilogram (kg). The weight was recorded three times and an average score wascalculated for accuracy (Centre for Disease Control and Prevention, 2021).

A study conducted by Kumar SN and colleges, provided evidence for the reliability and validity of digital weight scales (DWS) (Kumar SN et al, 2014). The study compared MatScan measurements – an objective evaluation of asymmetrical weight bearing of lower limbs – with digital weight scale measurements. The DWS measurements were valid and accurate when compared the MatScan measurements. Thus, DWS could be used objectively by clinicians to provide an accurate measurement.

3.7.2 Height

All heavy clothing and shoes were removed before measuring the height. Hair was unbraided or untied if it interfered with the height measurement. The measurement of the subject's height was taken on flat surface as well as against the wall. The subject stood with his/herfeet together, against the wall, with their legs straight and with their arms at their side. The subject looked straight ahead, and the position of the subject's head was adjusted if necessary. The measurement was taken when the subjects head, shoulders, buttocks, and heels were touching the wall. All these points may not always touch the wall, due to different body shapes. A flat head piece or ruler was used to form a right angle with the wall. The rulerwas lowered until it

reached the crown of the head. The eyes of the person taking the measurements were in line with the head piece. The height point was marked lightly on the wall at the height where the ruler met the crown of the head. The subject then stepped away from the wall and the height was measured using a tape measure from the floor to the mark on the wall. The height was recorded to the nearest centimetre (cm). Height was also measured three times, with the average calculated to maintain accuracy (Centre for Disease Control and Prevention, 2021).

Research conducted by Baharudin A and colleges investigated the accuracy and validity of using a portable stadiometer (height ruler) compared to a mechanical scale. The study showed that using a stadiometer was reliable and that it produced accurate measurements when compared to the mechanical scale measurements. The coefficient of reliability (R), coefficient of variation (CV) and technical error of measurement (TEM) were within the acceptable limits (Baharudin A et al, 2017).

3.7.3 BMI

The Body Mass Index was calculated using the following formula: kg/m^2. BMI categories (underweight, normal weight, overweight, obesity class 1, obesity class 11 and obesity class 111) were according to World Health Organization standards.

3.7.4 Waist circumference

The measurement was taken when the subject exhales to measure the true waist circumference. The measuring tape was placed around the body in line with the umbilicus (from the umbilicus back to the umbilicus). The measurement was taken to the nearest centimetre (cm). Waist circumference was measured three times to avoid discrepancies.
Waist circumference "risk levels" (normal, low risk, high risk and very high risk) for cardiometabolic complications were according to World Health Organization (WHO) standards (WHO, 2020).

A study investigating the validity of self-measured waist, hip and neck circumference compared to technician measurements (using a measuring tape), found that self-measurements were as accurate as technician measurements. The technical error measurements of self vs technician measurements had very high reliability, while the re-test reliability of self and technician measurements was also high (Barrios P et al, 2016).

3.7.5 Hip circumference

Hip circumference (the distance around the largest part of the hips) was measured using a tape measure. The measuring tape was placed around the body at widest part of the buttocks and the measurement was recorded to the nearest cm. Hip circumference was measured three times to maintain accuracy (WHO, 2020).

3.7.6 Waist to hip ratio (WHR)

Waist to hip ratio was calculated using the formula: waist circumference (cm)/hip circumference (cm)

Waist to hip ratio risk levels (normal, moderate risk and very high risk) for cardiometabolic complications were according to WHO standards (WHO Waist Circumference and Waist-Hip Ratio Report).

3.7.7 Waist to height ratio (WtHR)

Waist to height ratio was calculated using the formula: waist circumference (cm)/height (cm)

Waist to height ratio risk levels (no increased risk, increased risk, and very high risk) were according to WHO standards (WHO, 2020).

3.8 Blood pressure and heart rate

The blood pressure and heart rate protocol were performed according to the Mayo Clinic procedure with an AfriMedics blood pressure monitor machine (American Heart Association, 2011).

A study conducted in England, assessing the accuracy of home blood pressure monitors compared to blood pressure taken in a professional setting, found that home blood pressure monitors, such as the AfriMedics blood pressure monitor, are accurate, provided that the machines are ≤ 4-years-old. Furthermore, cuff failure in home blood pressure machines were more frequent, thus should be checked frequently and the correct cuff size must be used (Hodgkinson JA et al, 2020).

The subject was seated at a table with their less-dominant arm placed on the desk. His/her arm was relaxed and faced upwards and in line with their heart. If the subject was anxious or nervous, 5 minutes were given for the subject to relax. Before measuring the blood pressure, the blood pressure cuff was checked to the correct size. The cuff's bladder length should be at least 80% of the circumference of the subject's upper arm. The process began by wrapping the cuff around the upper arm of the subject. The bottom edge of the cuff was 1 inch or two finger spaces above the antecubital fossa (middle of the arm/inner side of the elbow). A moment passed before proceeding with pressing the "Start" button on the blood pressure machine. While the machine measures the blood pressure, the subject and the person taking the blood pressure remained quiet and refrained from moving. The cuff then inflated and thenslowly deflated as the measurements were taken. Once the measurement was complete, the machine displayed the subject's blood pressure and heart rate. These steps were repeatedthree times and the average reading was used in the analysis. Blood pressure measurements recorded from the machine included systolic blood pressure, diastolic blood pressure, heart rate and mean arterial pressure.

Blood pressure categories (low, normal and high) were according to World Health Organization (WHO) standards (WHO, 2020).

3.9 Lipid Profiles

The lipid profile and glucose levels tests were performed according to the protocol stated on the instructions which accompany the CardioChek plus analyser machine. Fasting blood samples were used in this study.

The lipid strips and finger prick method, specifically using the CardioChek machine has been validated by the UK NHS Purchasing and Supply Agency, who compared finger prick blood samples (lipid strips) with a standard venous blood sample that was tested in a lab. The correlation coefficient between the CardioChek method and lab sample was >0.84, which is deemed accurate (Centre for Evidence Based Purchasing, 2010).

Firstly, gloves were worn as a protective measure as we were working with blood (infectious diseases). A station was set-up which contained cotton swabs, alcohol swabs, capillary tubes and a sterile finger prick (lancet). The CardioChek plus machine was set-up and steps were followed according to the machine instructions. Prior to the pricking process, the hand that the blood was drawn from was warmed. The 3rd finger was cleaned and wiped using the alcohol swab. Next, the 3rd finger was wiped with a dry cotton swab. The finger was then pricked with the lancet and the first drop of blood (the size of a matchstick) was removed with the cotton swab. The finger was pressed, and the second drop of blood was used. The HealthCheck System capillary tube was used to the blood up until the line which could be seen on the capillary tube. The top of the capillary tube was pressed to release the blood onto the ImportItAll lipid panel test strip, which was already positioned in the CardioChek machine. After 90-120 seconds, the results were displayed on the machine which was recorded. The lipogram strips were disposed aseptically. The restart button was pressed to thetake the next measurement. Measurements recorded from the machine include total cholesterol, LDL-C, HDL-C, triglycerides, and total cholesterol/HDL-C ratio.

Lipid cut-off points for low, normal, and high values for these lipids were according to World Health Organization standards (WHO, 2020).

3.10 COVID-19 Protocols

COVID-19 protocols were upheld and maintained throughout the study. When participants arrived, they were provided with sanitizer or could wash their hands at a washing station. Sanitizer was available at each station. All researchers and supervisors wore PPE (gloves and masks) during the interaction with participants as well as throughout the data collection. Social distancing was maintained with 1.5m spacing. Workstations were sanitized and wiped down after each participant. Only 4 participants were allowed in the data collection room at a time, as each participant was at one station at a particular time. If the participants of the next session arrived early, they waited in the waiting room next door, with social distancing (a chair space or more per participant). Gloves were changed regularly, and hands were washed constantly. Protocols were upheld according to World Health Organization guidelines (WHO,2020).

3.11 Statistical analysis

The measurements acquired through the questionnaire and anthropometric measurements were

compiled and put through IBM SPSS™. Descriptive statistics were calculated and used to organise and summarise the data. Calculations were performed to determine the means and correlation coefficient measures of participants' characteristics. Spearman's rhocorrelation coefficients were used to determine the associations between the non-parametric continuous variables, while point-biserial correlations were used to determine correlations between categorical and continuous variables. Chi-square analysis was used to determine associations between categorical variables only. Furthermore, linear regression was used to determine the strength of the relationships between the variables. Results with a p value <0.05 were considered statistically significant.

The dependent variable(s) in this study were the depressive scores (MADRS and BDI). The independent variables were the BMI categories, lipid profiles and blood pressure measurements.

3.12 Ethical clearance and considerations

Ethical clearance was obtained from the University of the Western Cape (Ref. Code: BM21/2/14). At screening, participants were briefed and made aware of what the study entails. Participants were informed about each procedure and each station was explained. In addition, participants were informed about the duration as well as the benefits of volunteering for the study. Students were made aware of the confidentiality agreement – no names and surnames would be used (a number code would replace the names) and no data would be shared with external parties. Furthermore, all files and documents collected during the study was kept in a secure and locked room to maintain anonymity and confidentiality. Consent forms, questionnaires and information sheets were completed before commencing data collection of each participant. Moreover, patients were informed that the study is voluntary; thus, discontinuation of the study could be done freely and at any time. Participants were not advised to discontinue any medication for the purpose of this study, although participants were asked not to eat two hours before their data collection session. When an anomaly was found within the results of the students, either with weight (high/low BMI), blood pressure, lipids or depressive scores, the students were referred to the campus medical doctor or the Tygerberg medical centre. The ethical clearance certificate can be found under Appendix G.

3.13 Chapter Summary

Chapter three provided insight to the methodologies used to complete the research. The research design and procedure were explained as well as how participants were recruited. It

also included the inclusion and exclusion criterions. In addition, data collection methods and tools were described, and ethical clearance was stated. Furthermore, statistical analysis methodologies were also mentioned. Chapter four will display the results of the study.

Chapter Four
Results

4.0 Introduction

The results of the study are included in this chapter. It begins with the frequencies and descriptive statistics of the study population. It includes demographic variables, depressive scores, BMI categories, lipid profile categories and blood pressure categories. It also includes the objectives of the study, such as the associations between BMI, blood pressure and lipids with depressive scores and the secondary objectives, where the variables are associated with each other (excluding depressive scores). Independent T-tests, ANOVA and regression analysis are also included. Lastly, chi-square tests show the associations between categorical variables.

4.1 Descriptive Statistics

Table 4.1 below indicates the descriptive statistics of all participants (overall), the control group (BDI and MADRS scores) as well as the depressed group (BDI and MADRS scores). Table 4.1 shows that there were no significant differences across control and depressive groups for any variable in the study. All variables were compared to both BDI depressive scores as well as MADRS depressive scores.

Table 4.1 indicates that the mean overall age of the participants was 22.71 ± 6.67. The average overall BDI score was 20.37 ± 11.41 and average overall MADRS score was 19.22 ± 9.60. There was a significant difference in the MADRS_control group vs MADRS_ group, where $p < 0.001$. The average overall BMI was 25.20 ± 5.98. The BDI_control average BMI was 24.67 ± 6.06 compared to BDI_depressed average BMI, which was 25.77 ± 6.00. The MADRS_control group average WC was higher (79.40 cm ± 18.67) than the MADRS_depressed group WC (77.99 cm ± 15.52). The average WHR overall was 0.78 ± 0.08 and average overall WtHR was 0.48 ± 0.098, while the average MADRS_control WtHR was 0.48 ± 0.11 and the MADRS_depressed average WtHR was 0.49 ± 0.09.

Table 4.1 indicates that there were no statistically significant differences between MADRS (control and depressed) or BDI (control and depressed) lipid profiles ($p > 0.05$). The average overall total cholesterol level was 4.01 mmol/ ± 1.06. The average overall total cholesterol for MADRS_control group was 4.21 mmol/L ± 1.13 and MADRS_depressed group was 3.82

mmol/L ± 0.99. The mean LDL-C level overall was 1.83 mmol/L ± 1.02 compared to the average LDL-C level of the MADRS_control group which was 2.08 mmol/L ± 1.10 and 1.57 mmol/L ± 0.89 for MADRS_depressed group.

Table 4.1 shows that the average HDL-C level for the BDI_control group was 1.50 mmol/L ± 0.38, compared to the BDI_depressed group with an average HDL- C of 1.57 mmol/L ± 0.43. The average overall triglyceride level was 1.23 mmol/L ± 0.86. The average triglyceride level for the MADRS_depressed group was higher at 1.45 mmol/L ± 1.07, compared to the BDI_depressed group (1.40 mmol/L ± 1.06). The difference was not statistically significant ($p > 0.05$).

According to Table 4.1, the MADRS_control average total cholesterol/HDL-C ratio was 2.79 mmol/L ± 0.80 and the MADRS_depressed average was 2.57 mmol/L ± 0.64. Table 4.2 shows that there were also no significant differences between MADRS and BDI control and depressive groups in the blood pressure measurements. The average MADRS_control SP was 115.93 mmHg ± 12.22 and average MADRS_depressed SP was 116.51 mmHg ± 16.37. The average BDI_control DP was 73.63 mmHg ± 6.68, while the average BDI_depressed DP was 74.92 mmHg ± 9.00. The average overall MAP was 95.59 mmHg ± 13.31 and the mean overall HR was 80.42 bpm ± 12.45. The average BDI_control HR was 78.10 bpm ± 10.93 and average BDI_depressed HR was 83.47 bpm ± 13.79. A non-parametric Mann-Whitney U Independent T-test was performed to determine significant differences between control and depressive scores for both MADRS and BDI scores.

Table 4.1: Descriptive table across MADRS groups and BDI groups

	Overall	Control (MADRS)	Depressed (MADRS)	P-value	Control (BDI)	Depressed (BDI)	P-value
	N= 63	N= 32	N= 31		N= 30	N= 30	
	mean (SD)	mean (SD)	mean (SD)		mean (SD)	mean (SD)	
Actual Age	22.71 ± 6.67	23.63 ± 7.61	21.77 ± 5.50	0.610	23.00 ± 5.78	21.53 ± 5.48	0.207
Depressive scores							
BDI score	20.37 ± 11.14	12.41 ± 5.59	27.81 ± 9.63	0.123	11.60 ± 4.99	29.13 ± 8.30	0.680
MADRS	19.22 ± 9.60	11.78 ± 5.48	26.90 ± 6.29	0.001**	13.17 ± 6.70	26.23 ± 7.18	0.149
Anthropometry							
BMI (Kg/m^2)	25.20 ± 5.98	25.27 ± 6.18	25.14 ± 5.88	0.807	24.67 ± 6.06	25.77 ± 6.00	0.549
Waist circumference (WC) (cm)	78.80 ± 17.06	79.40 ± 18.67	77.99 ± 15.52	0.511	78.35 ± 17.84	78.81 ± 15.7	0.521
Waist to hip ratio (WHR)	0.78 ± 0.08	0.79 ± 0.09	0.77 ± 0.08	0.757	0.78 ± 0.08	0.78 ± 0.09	0.800
Waist to hight ratio (WtHR)	0.48 ± 0.098	0.48 ± 0.11	0.49 ± 0.09	0.923	0.48 ± 0.10	0.49 ± 0.09	0.606
Lipid Profiles							
Total cholesterol (mmol/L)	4.01 ± 1.06	4.21 ± 1.13	3.82 ± 0.99	0.990	3.93 ± 0.98	3.95 ± 1.02	0.611

LDL-C (mmol/L)	1.83 ± 1.02	2.08 ± 1.10	1.57 ± 0.89	0.848	1.85 ± 1.03	1.69 ± 0.96	0.192
HDL-C (mmol/L)	1.55 ± 0.43	1.56 ± 0.44	1.55 ± 0.43	0.823	1.50 ± 0.38	1.57 ± 0.43	0.669
Triglycerides (mmol/L)	1.23 ± 0.86	1.03 ± 0.55	1.45 ± 1.07	0.678	1.06 ±0.58	1.40 ± 1.06	0.191
Tot. cholesterol/HDL-C (mmol/L)	2.68 ± 0.73	2.79 ± 0.80	2.57 ± 0.64	0.906	2.72 ± 0.75	2.59 ± 0.60	0.837
Blood pressure							
Systolic Pressure (SP) (mm/Hg)	116.21 ± 14.29	115.93 ± 12.22	116.51 ± 16.37	0.488	115.61 ± 11.50	116.17 ± 16.35	0.351
Diastolic pressure (DP) (mm/Hg)	74.49 ± 8.04	74.19 ± 7.19	74.81 ± 8.96	0.301	73.63 ± 6.68	74.92 ± 9.00	0.061
Mean Arterial Pressure (MAP) (mm/Hg)	95.59 ± 13.31	87.99 ± 8.29	88.74 ± 10.96	0.346	87.50 ± 7.57	88.71 ± 11.01	0.132
Heart Rate (bpm)	80.42 ± 12.45	78.77 ± 12.63	82.13 ± 12.25	0.787	78.10 ± 10.93	83.47 ± 13.79	0.527

Note: * indicates statistically significant differences < 0.05; ** indicates statistically significant differences < 0.01. N: number of participants; P vale: indicates significance and SD indicates standard deviation. BDI: Beck Depression Inventory (depressive score); MADRS: Montgomery Asberg Depression Rating Scale; BMI: body mass index and bpm indicated beats per minute.

Table 4.2 below indicates the descriptive statistics overall, in males as well as in females. The average overall age of the participants was 22.71 ± 6.67, and males and females did not have a significant difference in age ($p > 0.05$). Table 4.2 shows that there's a significant difference between male and female BDI score ($p < 0.026$). Females had a higher mean BDI score (21.67 ± 11.36) compared to males (14.55 ± 8.25).

The average MADRS score overall was 19.22 ± 9.60. Females also had a significantly higher MADRS score compared to males ($p < 0.008$) at 95% CI level (-12.818 - -2.143). The average BMI overall was 25.20 ± 5.98. Males and females did not have significantly different BMI scores. Females had a mean BMI of 25.56 ± 6.064 and males had a mean BMI of 23.69 ± 5.73. Table 4.2 shows that males had a mean WC of 81.15cm ± 19.79 and females had a mean WC of 78.13cm ± 16.53. In addition, the average WHR overall was 0.78 ± 0.08. Females had a higher average WtHR of 0.487 ± 0.099, while males had an average WtHR of 0.475 ± 0.119. Table 4.2 shows that there were no significant differences in total cholesterol levels between males and females ($p > 0.05$). The overall mean total cholesterol levels among the participants was 4.01 mmol/L ± 1.06. The overall mean LDL-C level was 1.83 mmol/L ± 0.80 and overall mean HDL-C was 1.55 mmol/L ± 0.43. There were no significant differences between males and females in LDL-C or HDL-C levels ($p < 0.05$). The mean triglyceride level in males was 1.41 mmol/L ± 0.66 and mean female triglyceride level was 1.19 mmol/L ± 0.905.There were no significant differences between male and female total cholesterol/HDL-C ratios ($p > 0.05$).

Table 4.2 indicates that males had a significantly higher average SP compared to females ($p < 0.019$) at 95 % CI level (2.843 – 26.43). Males had a mean SP of 128.05 mmHg ± 18.06, while females had an average SP of 113.42 ± 11.83. The overall mean DP was 74.49 ± 8.04. Males also had significantly higher mean MAPs ($p < 0.044$), where the average male MAP was 95.59mmHg ± 13.31 and the average females MAP was 86.55mmHg ± 7.77. There was no significant difference between average HR of males and females ($p > 0.05$). The overall HR was 80.42 bpm ± 12.45. A non-parametric Mann-Whitney U Independent T-test was performed to determine significant differences between males and females.

Table 4.2: General characteristics overall, in males and in females

Variables	Gender	N	Mean (SD)	95% CI (lower ; upper)	P value
	Overall	63	22.71 ± 6.67		
Actual Age	Males	12	28.08 ± 12.38	(-1.276 ; 14.541)	0.092
	Females	51	21.45 ± 3.591		
Depressive scores	Overall	60	20.37 ± 11.14		
BDI score	Males	11	14.55 ± 8.25	(-13.332 ; -0.092)	0.026*
	Females	49	21.67 ± 11.36		
	Overall	63	19.22 ± 9.60		
MADRS	Males	12	13.17 ± 7.56	(-12.818 ; -2.143)	0.008**
	Females	51	20.65 ± 9.535		
Anthropometry	Overall	63	25.20 ± 5.98		
BMI	Males	12	23.69 ± 5.73	(-5.781 ; 2.054)	0.330
	Females	51	25.56 ± 6.046		
	Overall	63	78.70 ± 17.06		
WC (cm)	Males	12	81.15 ± 19.79	(-10.121 ; 16.182)	0.630
	Females	51	78.13 ± 16.53		
	Overall	63	0.78 ± 0.08		
WHR	Males	12	0.83 ± 0.11	(-0.016 ; 0.137)	0.111
	Females	51	0.77 ± 0.077		
	Overall	63	0.48 ± 0.098		
WtHR	Males	12	0.475 ± 0.119	(-0.093 ; 0.06512)	0.715
	Females	51	0.487 ± 0.099		
Lipid profiles	Overall	63	4.01 ± 1.06		
Total cholesterol (mmol/L)	Males	12	3.88 ± 1.08	(-0.897 ; 0.570)	0.643
	Females	51	4.05 ± 1.074		
	Overall	63	1.83 ± 1.02		
LDL-C cholesterol (mmol/L)	Males	12	1.84 ± 0.80	(-0.564 ; 0.588)	0.966
	Females	51	1.83 ± 1.08		
	Overall	63	1.55 ± 0.43		
HDL-C cholesterol (mmol/L)	Males	12	1.35 ± 0.44	(-0.549 ; 0.053)	0.099
	Females	51	1.599 ± 0.420		
Triglycerides (mmol/L)	Overall	62	1.23 ± 0.86		

	Males	12	1.41 ± 0.66	(-0.248 ; 0.707)	0.329
	Females	50	1.19 ± 0.905		
Total cholesterol/HDL-C ratio (mmol/L)	Overall	63	2.68 ± 0.73		
	Males	12	3.02 ± 0.97	(-0.209 ; 1.068)	0.171
	Females	51	2.60 ± 0.65		
Blood pressure					
Systolic Bloodpressure (SP) (mm/Hg)	Overall	63	116.21 ± 14.29		
	Males	12	128.05 ± 18.06	(2.834 ; 26.427)	0.019*
	Females	51	113.42 ± 11.83		
Diastolic Blood pressure (DP) (mm/Hg)	Overall	63	74.49 ± 8.04		
	Males	12	79.33 ± 11.32	(-1.378 ; 13.338)	0.103
	Females	51	73.35 ± 6.72		
Mean Arterial Pressure (MAP) (mm/Hg)	Overall	63	88.35 ± 9.62		
	Males	12	95.59 ± 13.31	(0.2909 ; 17.581)	0.044*
	Females	51	86.55 ± 7.77		
Heart Rate (HR) (bpm)	Overall	63	80.42 ± 12.45		
	Males	12	79.36 ± 17.33	(-12.621 ; 9.997)	0.804
	Females	51	80.67 ± 11.23		

Note: * indicates statistically significant differences < 0.05; ** indicates statistically significant differences < 0.01. N = number of participants; Mean (SD): standard deviation; 95% CI: 95% confidence interval and P value indicates the significance. BDI: Beck Depression Inventory; MADRS: Montgomery Asberg Depression Rating Scale; BMI: Body mass index; WC: waist circumference; WHR: waist to hip ratio; WtHR: waist to height ratio; LDL-C: low-density lipoprotein cholesterol; HDL-C: high-density lipoprotein cholesterol; bpm: beats per minute.

4.2 Frequencies

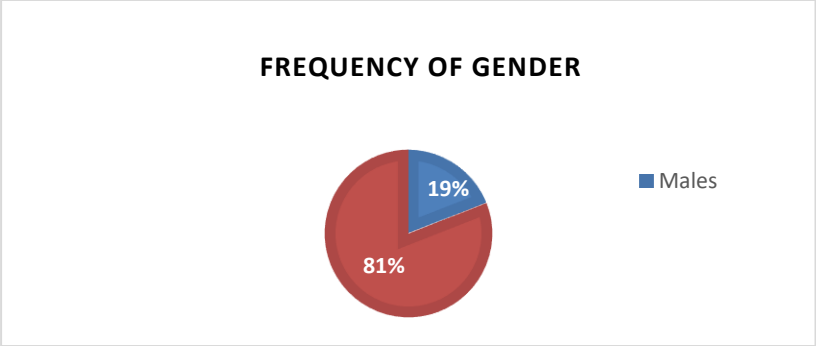

Figure 4.1: The frequency of male and female participants in the study.

Figure 4.1 indicates that 81% of females participated in the study, while the remaining 19% were males.

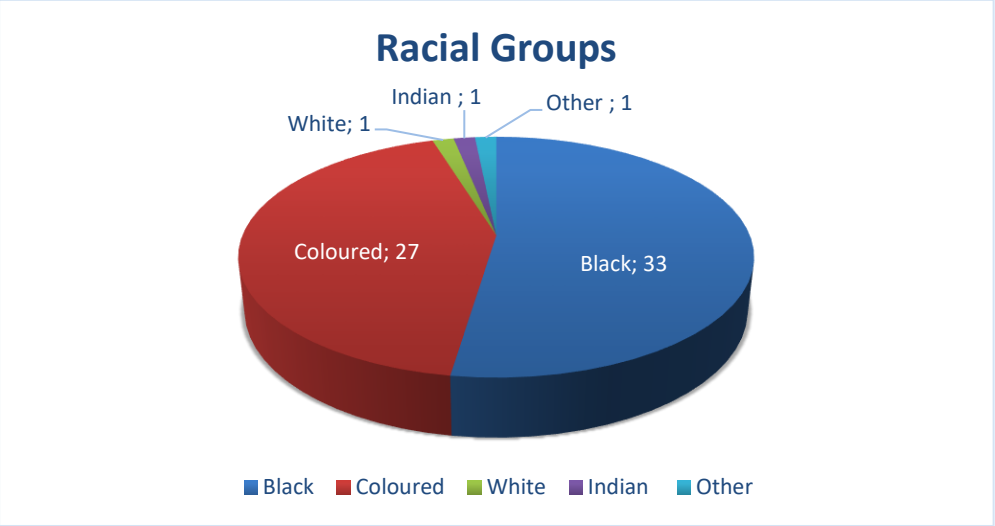

Figure 4.2: The number of participants per race category

Figure 4.2 indicates the number of participants in each racial group. Majority of participants were Black (33) and Coloured (27), while the remaining racial groups – White, Indian, and Other, each had 1 participant respectively.

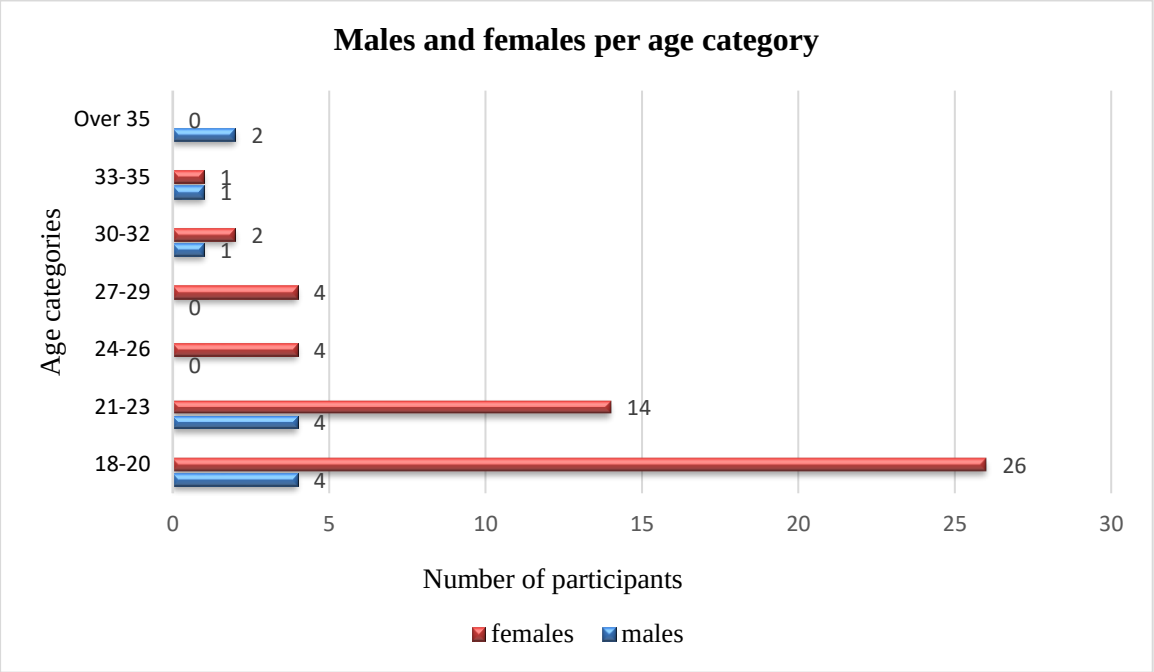

Figure 4.3: The prevalence of males and females per age category.

Figure 4.3 indicates that most participants were females aged 18-20. Fourteen females and four males were aged 21-23, while eight participants were between the ages 24-29. Only seven participants were aged 30 and above.

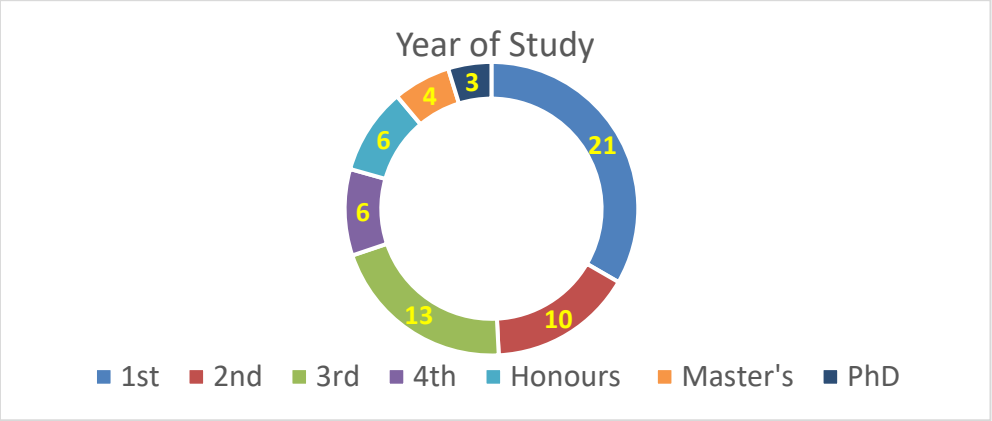

Figure 4.4: The frequency of participants per year of study.

Figure 4.4 indicates that most participants were registered undergraduate students (1st-3rd year). The remaining participants were honours', master's, and PhD students, with the least number of students registered for their PhD (3 participants).

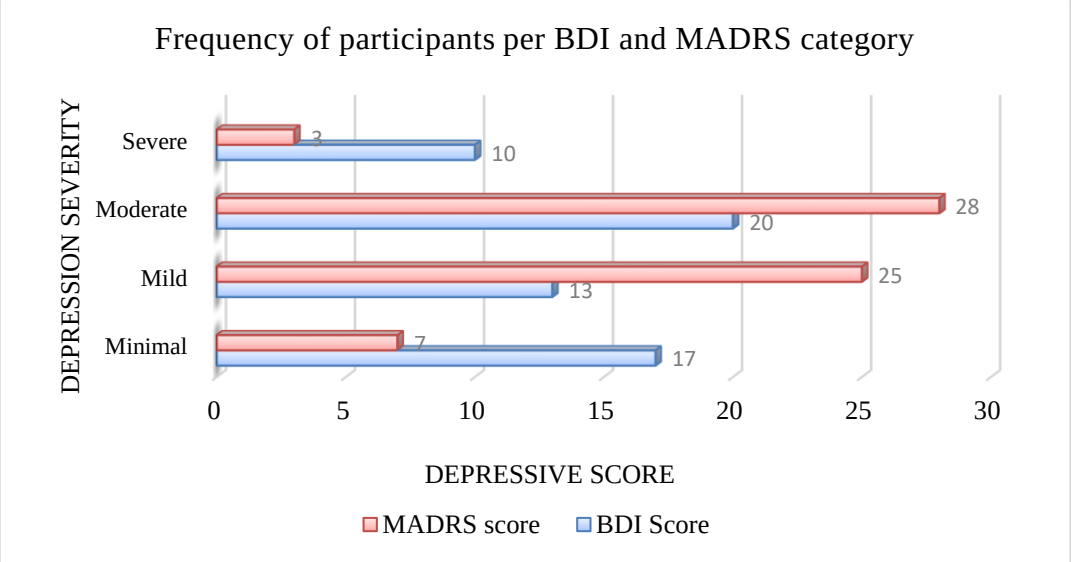

Figure 4.5: Depression severity among the participants according to BDI and MADRS scores.

Figure 4.5 shows that most participants were moderately depressed, according to both BDI and MADRS scores. Many participants were also mildly depressed (25 MADRS and 13 BDI scores). The lowest number of participants had severe depression, where 10 had a severe BDI score and only 3 participants had a severe MADRS score.

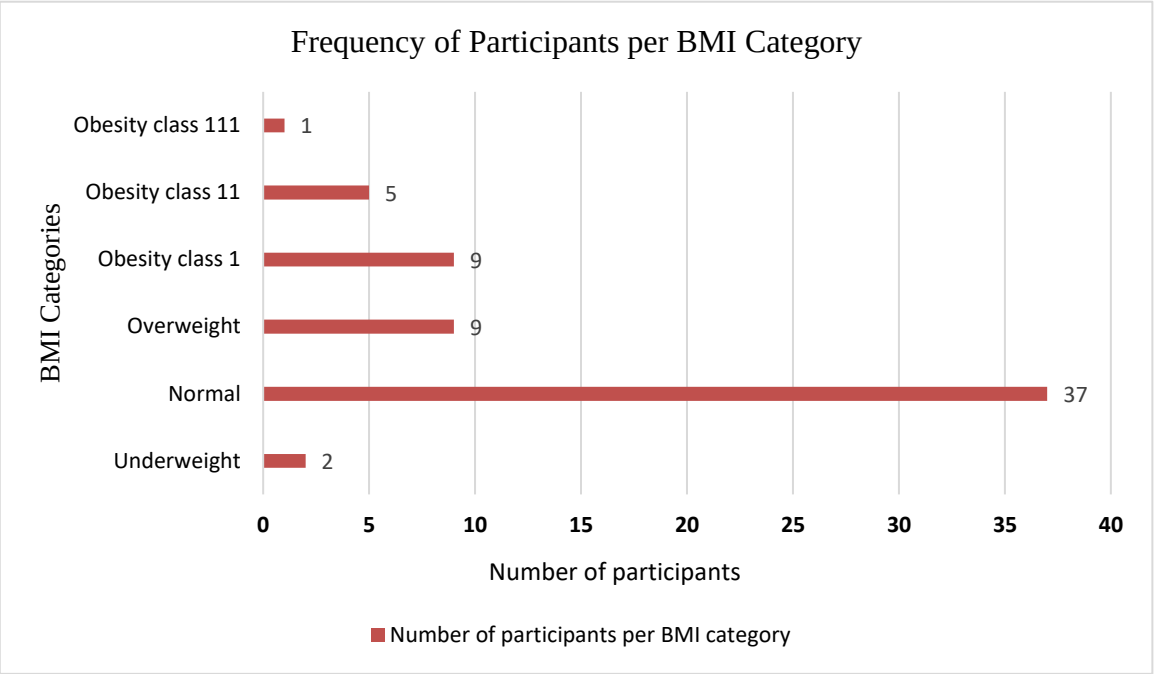

Figure 4.6: The frequency of participants per BMI category.

Figure 4.6 indicates that most participants were within in the normal weight range according to BMI categories (37 participants). A total of nine participants were overweight and 14 were classed between obesity class 1 and obesity class 11. On either extreme, one participant was found to be overweight, while two were found to be underweight.

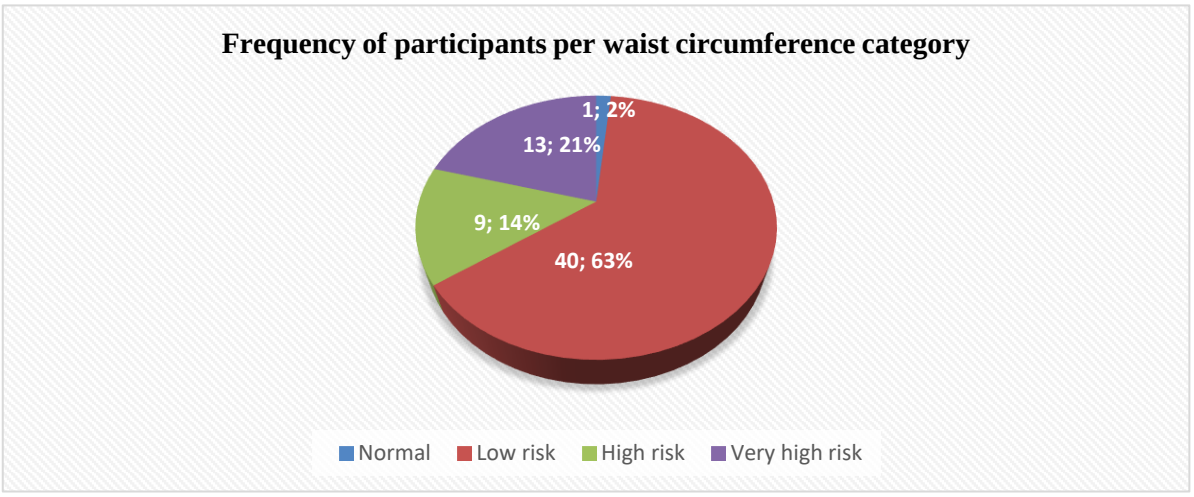

Figure 4.7 The frequency of participants in each category of waist circumference.

Figure 4.7 indicates that 63% of participants were classified as “low risk” according to waist circumference categories. The remaining 35% of participants were classified as “high risk” and “very high risk” according to waist circumference. Only one participant was classified as having a normal waist circumference. Risk according to waist circumference means to be at risk of developing cardiometabolic risk factors.

Most participants also had normal waist to hip ratios (WHR) (77.8%), while 15.9% were

classified as having a "very high risk" according to WHR categories. Risk according to WHR means to be at risk of developing cardiometabolic (CMB) risk factors. Furthermore, 63.5% of participants were at "no increased risk" of developing CMB risk factors according to waist to hip ratio (WtRH), while 19% and 17.5% were at "increased risk" and "very high risk" respectively.

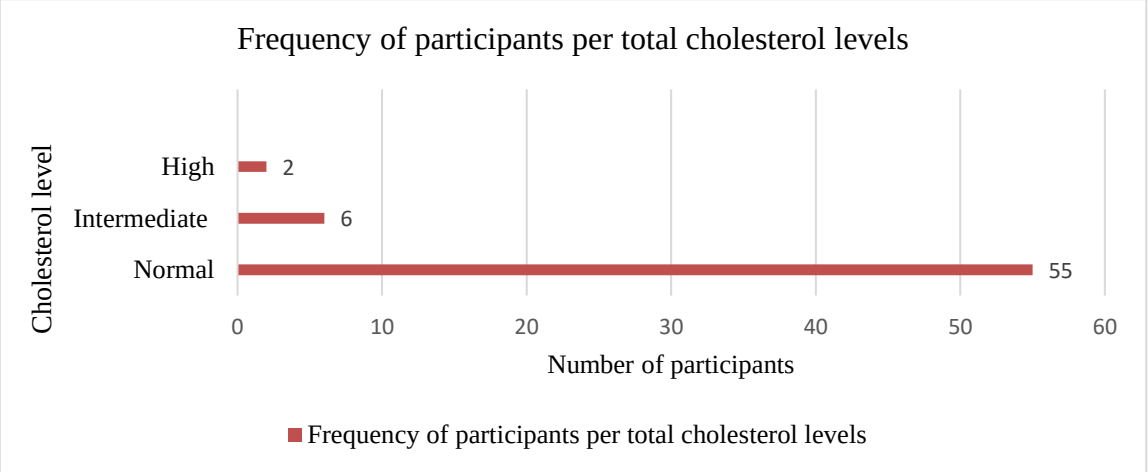

Figure 4.8: The frequency of participants in each category of total cholesterol categories.

In Figure 4.8, it is seen that most participants had normal total cholesterol levels, while six participants had intermediate levels and only two had high total cholesterol levels. Regarding the low-density lipoprotein cholesterol (LDL-C) levels, over half of the participants also had normal levels (55.6%), while 38.1% was classified as having low LDL-C. Only 6.4% of participants were classified as having intermediate to high LDL-C levels.

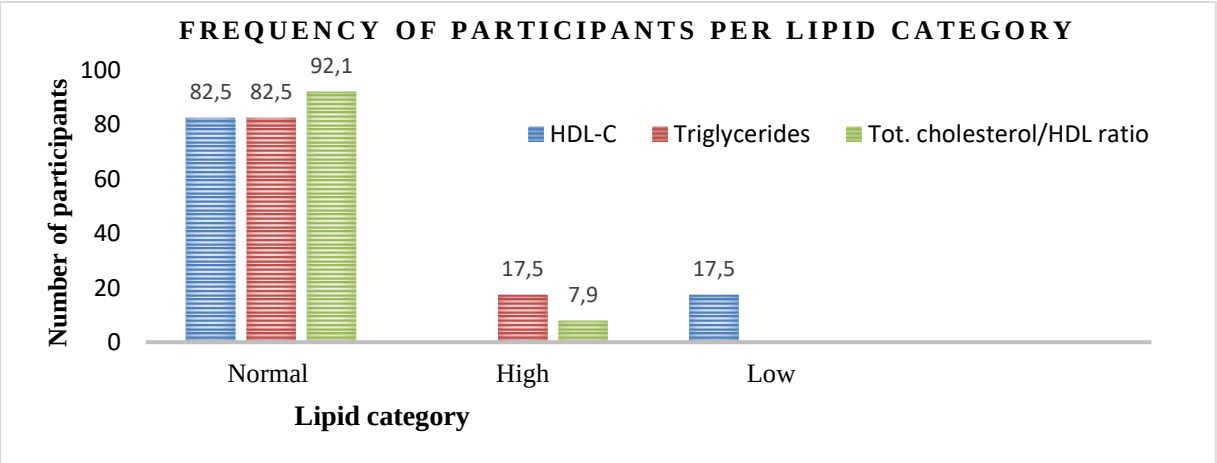

Figure 4.9: The frequency of participants per lipid category according to high-density lipoprotein cholesterol (HDL-C) levels, triglyceride levels and total cholesterol/HDL-C ratio levels.

Regarding HDL-C levels, 82.5% of participants had normal HDL-levels, while 17.5% was

classified as having low HDL-C levels. Figure 4.9 indicates that 82.5% of the participants had normal triglyceride levels, while 17.5% had high triglyceride levels respectively. Majority of participants had normal total cholesterol/HDL-C ratios (92.1%), while the remaining 7.9% was classified as having high total cholesterol/HDL-C ratios.

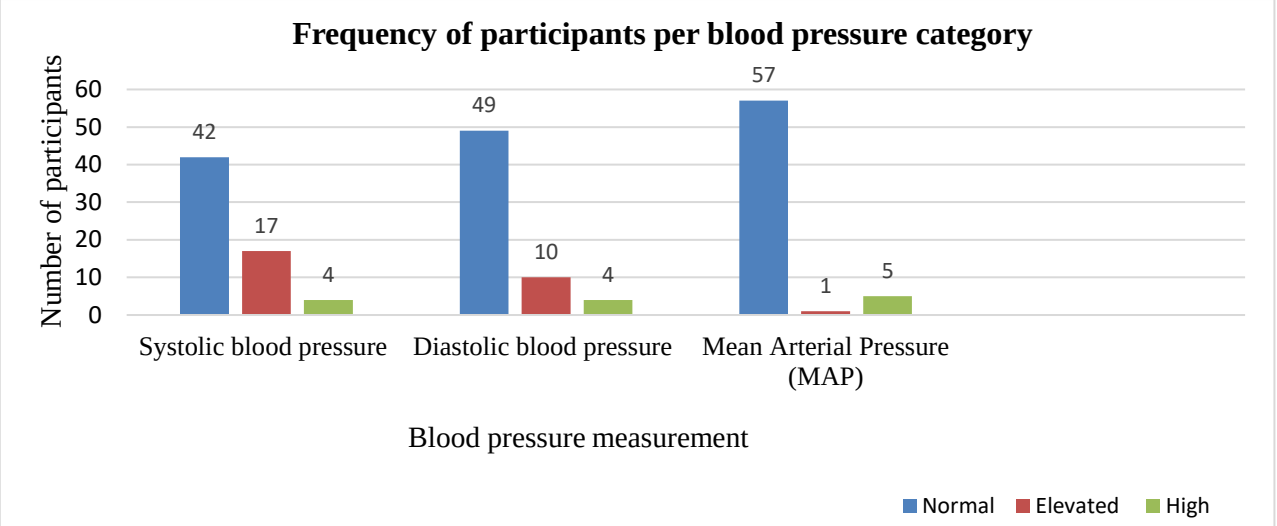

Figure 4.10: The number of participants in each category according to blood pressure measurements.

Figure 4.10 indicates that most participants had normal systolic, diastolic, and mean arterial blood pressure measurements. Furthermore, 17 participants had elevated systolic blood pressure, while 10 had elevated diastolic blood pressure and 1 had an elevated mean arterial pressure (MAP) measurement respectively. Four participants had both high systolic and diastolic blood pressures, while five participants had high MAP measurements. With regards to heart rate, most students had normal heart rates, while 4.8% was classified as having low heart rates and 6.3% was classified as having high heart rates respectively.

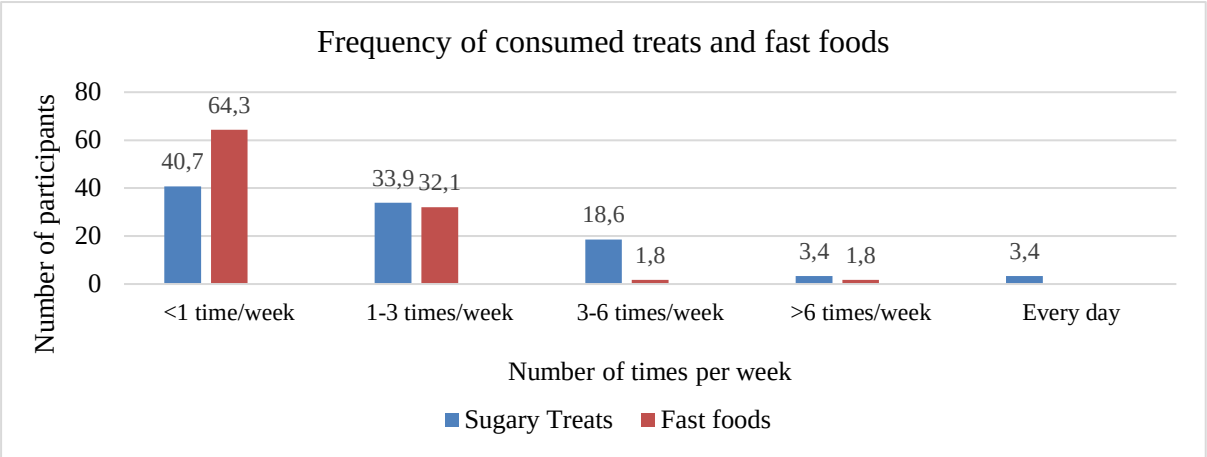

Figure 4.11: The number of times per week sugary treats and fast foods are consumed by the participants.

According to Figure 4.11, most participants consume both sugary treats and fast foods less than once a week. Just over 1/3 of participants consume treats and fast foods 1-3 times a week, while 18.6% of participants consume sugary treats 3-6 times a week respectively. Fast food is consumed 3-6 times a week and more than six times a week by 1.8% of participants (1 participant).

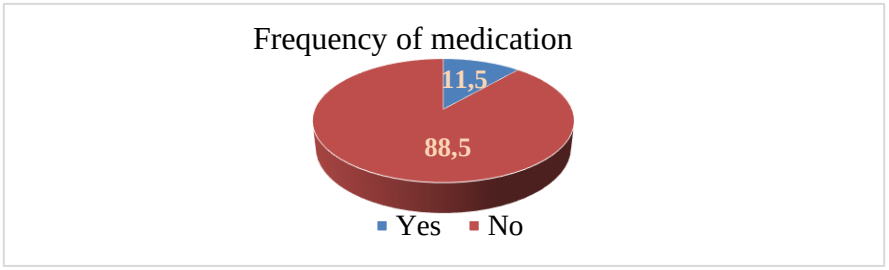

Figure 4.12 Frequency of participants on medication.

Figure 4.12 indicates that 88.5% of participants were not medicated, while 11.5% were medicated.

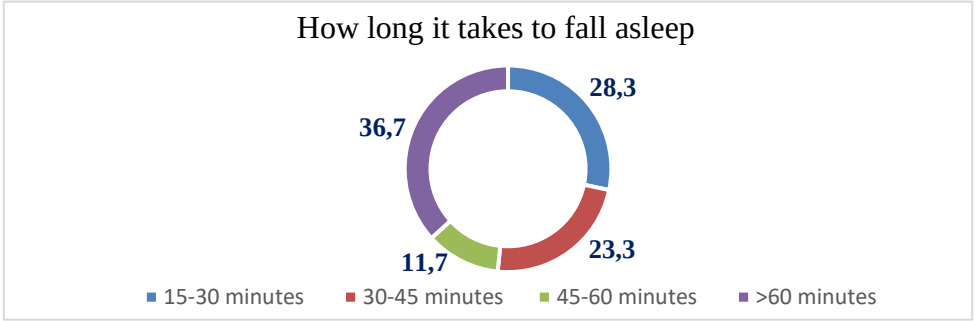

Figure 4.13 Frequency of participants per time category of falling asleep.

According to Figure 4.13, more than 1/3 of participants take more than 60 minutes to fall asleep. A total of 51.6% of participants take between 15-45 minutes to sleep, while 11.7% fall asleep within 45-60 minutes.

4.3 Analysis of Variance (ANOVA)

Table 4.3 Results of One-way ANOVA according to age categories.

Variables	N	F	df	P-value	Mean Square
BMI	60	2.79	5	0.026*	85.957
WC	60	5.579	5	0.001**	1063.075
WHR	60	4.784	5	0.001**	0.021
WtHR	60	4.418	5	0.002**	0.031

Note: * indicates statistically significant differences < 0.05; ** indicates statistically significant differences < 0.01. N: Number of participants, F: F-statistic, df: degrees of freedom and P-value: significance. BMI: body mass index, WC: waist circumference, WHR: Waist to hip ratio, WtHR: Waist to height ratio

A One-way ANOVA test was completed including all variables – depressive scores (BDI and MADRS), anthropometry (BMI, WC, WHR and WtHR), lipid profiles (total cholesterol, LDL-C, HDL-C, triglycerides, and total cholesterol/HDL-C ratio) and blood pressure measurements (SP, DP, MAP and HR) according to age.

Table 4.3 shows the only significant results after running the Oneway ANOVA test. Table 4.3 shows that there was a significant difference between the age groups) for their average BMI score, where $F(5,55) = 2.790$ and $p < 0.026$. When performing a Post Hoc Tukey comparison, it showed that BMI differed between age groups 1 (18-20) and 6 (33-35) and age groups 2 (21-23) and 6 (33-35). A significant difference is seen between the age groups for their average waist circumference (WC), where $F(5,55) = 5.759$ and $p < 0.001$. When performing a Post Hoc Tukey comparison, it showed that WC differed between age groups 1-5 (18-20;21-23;24-26;27-29 and 30-32) and group 6 (33-35). There was also a significant difference found for the average waist to hip ratio (WHR) between the 6 groups, where $F(5,55) = 4.484$ and $p < 0.001$. When performing a Post Hoc Tukey comparison, it showed that WHR differed between age groups 1-5 (18-20;21-23;24-26;27-29 and 30-32) and group 6 (33-35). Thus, there was a significant difference in WHR found between all the age groups with group 6 (33-35). A significant difference between the 6 age groups can also be seen with the average wait to height ratio (WtHR) measurements, where $F(5,55) = 4.418$ and $p < 0.002$. When performing a Post hoc Tukey comparison, it showed that WtHR differed between age groups 1-4 (18-20;21-23;24-26;27-29) and group 6. Thus, there was a significant difference of WtHR between group

1 and 6, between group 2 and 6, between group 3 and 6 and group 4 and 6. All outliers of age were removed from the ANOVA test (age >35, thus 3 missing values).

4.4 Correlations

Table 4.4 Correlation between depressive scores and anthropometry, lipid profiles and blood pressure.

Variables	Gender	BDI score N=60	MADRS N=63
Demographics			
Race	Overall	0.057	0.107
	Males	-0.064	0.122
	Females	0.052	0.0116
Actual Age	Overall	0.217	-0.161
	Males	-0.237	-0.366
	Females	-0.157	-0.046
Anthropometry			
BMI	Overall	0.064	0.090
	Males	0.036	-0.109
	Females	-0.007	0.041
WC	Overall	-0.011	0.004
	Males	-0.082	-0.014
	Females	-0.028	-0.037
WHR	Overall	0.064	0.090
	Males	-0.118	0.081
	Females	0.008	0.020
WtHR	Overall	0.030	0.064
	Males	-0.137	-0.030
	Females	-0.020	0.030
Lipid profiles			
Total cholesterol	Overall	0.009	-0.153
	Males	-0.101	-0.431
	Females	0.001	-0.025
LDL-C	Overall	-0.070	-0.228
	Males	0.247	-0.137
	Females	-0.096	-0.209
HDL-C	Overall	0.136	-0.010
	Males	-0.237	-0.344
	Females	0.150	-0.059
Triglycerides	Overall	-0.040	0.037
	Males	-0.205	-0.235
	Females	0.029	0.156
Tot.chol./HDL-C	Overall	-0.125	-0.090
	Males	0.265	0.093
	Females	-0.151	-0.033
Blood pressure			
SP	Overall	-0.054	-0.050
	Males	0.456	0.635**

	Females	-0.022	0.033
	Overall	0.006	-0.053
DP	Males	0.354	0.272
	Females	-0.004	-0.026
	Overall	-0.035	-0.067
MAP	Males	0.528	0.481
	Females	-0.036	-0.019
	Overall	0.048	0.0702
HR	Males	0.565	0.425
	Females	-0.69	-0.023

Note: * indicates statistically significant differences < 0.05; ** indicates statistically significant differences < 0.01. N: Number of participants, BMI: body mass index, WC: waist circumference, WHR: Waist to hip ratio, WtHR: Waist to height ratio, LDL-C: low-density lipoprotein cholesterol, HDL- C: high-density lipoprotein cholesterol, Tot.Chol./HDL: Total cholesterol/HDL ratio, SP: systolic blood pressure, DP: diastolic blood pressure, MAP: mean arterial pressure and HR: heart rate.

A non-parametric Spearman rho correlation was run to assess the relationship between depressive scores (BDI and MADRS) with anthropometric measurements (BMI, WC, WtHR, WHR), lipid profiles (LDL-C, HDL-C, Triglycerides, Total cholesterol, Total cholesterol/HDL-C) and blood pressure measurements (SP, DP, MAP and HR) in males and females.

Table 4.4 indicates that there was only a significant correlation between SP and MADRS scores in males, where $r = 0.635$ and $p < 0.01$. Since there were no significant relationships between depressive scores with anthropometry and lipids, further analysis was performed.

After running a Point-biserial correlation between MADRS and BDI scores with all the categorical variables (anthropometry, lipids, and blood pressure), MADRS scores were negatively and significantly correlated to total cholesterol levels with $r = -0.214$ and $p < 0.041$. MADRS scores also negatively and positively correlated with LDL-C categories, where $r = -0.212$ and $p < 0.041$. There was also a significant positive correlation between MADRS scores and triglyceride levels, where $r = -0.214$ and $p < 0.044$. There were no significant correlations with BDI scores. MADRS and BDI scores were continuous variables, whereas total cholesterol, triglycerides and LDL-C were all categorical variables.

Table 4.5: Correlation between demographics, blood pressure, lipid profiles and anthropometry.

	DEMOGRAPHICS	BLOOD PRESSURE				LIPID PROFILES					ANTHROPOMETRY			
VARIABLES	**GENDER**	**Race**	**HR**	**MAP**	**DP**	**SP**	**TOT/HDL**	**TRIGS**	**HDL**	**LDL**	**TOT.C**	**WtHR**	**WHR**	**WC**	**BMI**
	Overall	0.413**	-0.029	0.013	0.037	0.025	-0.042	0.254*	0.104	0.048	0.099	0.248*	0.251*	0.216	0.197
AGE	Males	0.621*	-0.049	0.392	0.599*	0.166	0.276	0.388	0.106	0.611*	0.664*	0.721**	0.653**	0.716**	0.681*
	Females	0.381**	0.000	-0.123	-0.101	-0.051	-0.173	0.184	0.149	-0.069	-0.053	0.139	0.120	0.081	0.113
	Overall	0.209	0.078	0.174	0.200	0.174	0.251*	0.102	-0.131	0.163	0.210	0.926**	0.622**	0.906**	
BMI	Males	0.706*	0.140	0.427	0.697*	0.273	-0.021	0.049	0.245	0.476	0.408	0.757**	0.587*	0.860**	
	Females	0.063	0.127	0.164	0.158	0.228	0.334*	0.131	-0.240	0.124	0.165	0.942**	0.701**	0.935**	
	Overall	0.299*	0.064	0.244	0.281*	0.221	0.315*	0.124	-0.217	0.175	0.185	0.953**	0.843**		
WC	Males	0.877**	0.056	0.497	0.690*	0.427	0.060	0.084	0.119	0.455	0.373	0.960**	0.846**		
	Females	0.159	0.113	0.195	0.231	0.274	0.170	0.294*	0.032	-0.228	0.121	0.137	0.863**		
	Overall	0.367**	-0.059	0.309*	0.331*	0.282*	0.260*	0.094	-0.239	0.127	0.117	0.810**			
WHR	Males	0.833*	-0.035	0.524	0.623*	0.462	-0.074	-0.035	0.028	0.230	0.190	0.928**			
	Females	0.293*	-0.014	0.213	0.274	0.170	0.294*	0.032	-0.228	0.121	0.137	0.863**			
	Overall	0.367**	-0.062	0.203	0.241	0.181	0.289*	0.086	-0.176	0.176	0.289*				
WtHR	Males	0.879**	-0.025	0.445	0.614*	0.354	-0.553	0.035	0.119	0.333	0.317				
	Females	0.156	0.063	0.194	0.213	0.219	0.389**	-0.100	0.260	0.161	0.195				
	Overall	0.159	0.126	0.132	0.198	-0.001	0.368**	0.023	0.471*	0.866*					
TOT.CHOL	Males	0.317	0.014	0.063	0.314	-0.190	0.222	0.275	0.479	0.843**					
	Females	0.123	0.161	0.179	0.218	0.057	0.413**	0.016	0.475**	0.872**					
	Overall	0.198	0.133	0.268*	0.341**	0.118	0.537**	-0.148	0.224						
LDL	Males	0.333	0.375	0.466	0.600*	0.165	0.437	0.197	0.210						
	Females	0.215	0.092	0.255	0.316*	0.112	0.565**	-0.151	0.232						
	Overall	0.051	0.123	-0.155	-0.055	-0.260*	-0.598**	-0.109							
HDL	Males	0.167	0.126	-0.224	0.126	-0.483	0.694*	-0.444							
	Females	0.017	0.101	-0.066	-0.030	-0.138	-0.562**	-0.033							
	Overall	-0.038	0.034	-0.016	-0.021	-0.009	0.150								
TRIGS	Males	0.254*	-0.629*	-0.133	-0.244	0.168	0.746**								
	Females	-0.072	0.140	-0.090	-0.025	-0.123	0.048								
	Overall	0.083	0.013	0.256*	0.207	0.259*									
TOT/HDL-C	Males	-0.028	-0.084	0.242	0.056	0.396									
	Females	0.113	0.049	0.230	0.228	0.190									
	Overall	0.126	-0.130	0.897**	0.727**										
SP	Males	0.570	0.119	0.818**	0.581*										
	Females	0.102	-0.202	0.902**	0.735**										
	Overall	0.314	0.133	0.946**											
DP	Males	0.615*	0.592*	0.886**											
	Females	0.304*	0.001	0.942**											
	Overall	0.230	0.047												
MAP	Males	0.013	0.542												
	Females	0.215	0.992												
	Overall	-0.138													
HR	Males	-0.148													
	Females	-0.029													

Note: * indicates statistically significant differences < 0.05; ** indicates statistically significant differences < 0.01. BMI: body mass index, WC: waist circumference, WHR: Waist to hip ratio, WtHR: Waist to height ratio, TOT. CHOL: total cholesterol, LDL-C: low-density lipoprotein cholesterol, HDL- C: high-density lipoprotein cholesterol, TRIGS: triglycerides, Tot.Chol./HDL: Total cholesterol/HDL ratio, SP: systolic blood pressure, DP: diastolic blood pressure, MAP: mean arterial pressure and HR: heart rate.

A non-parametric Spearman rho's correlation was run to assess the relationship between age, anthropometric measurements (BMI, WC, WHR and WtHR), lipid profiles (Total cholesterol, LDL-C, HDL-C, Triglycerides, Total cholesterol/HDL-C ratio) and blood pressure measurements (SP, DP, MAP and HR).

Table 4.5 shows that race had a significant positive correlation with actual age overall, in males and in females, where r = 0.413, r = 0, 381 and r = 0.621 and where $p < 0.001$ and $p < 0.05$. Race also had a significant positive relationship with BMI in males where r = 0.706 and $p < 0.05$. Overall, race was positively and significantly associated with WC, where r = 0.299 and $p < 0.05$. WC was also significantly and positively associated with race in males, where r = 0.877 and $p < 0.001$. Race was significantly and positively associated with WHR overall, where r = 0.367 and $p < 0.001$. WHR was also significantly and positively associated with race in both males and females where r = 0.833 and r = 0.293 and $p < 0.05$ respectively. Overall as well as in males, WtHR had a significant and positive relationship with race, where r = 0.367 and r = 0.879 and $p < 0.001$ respectively. Race also had a significant and positive relationship with triglycerides in males, where r = 0.254 and $p < 0.05$. Lastly, race was also significantly and positively associated with DP in both males and females, where r = 0.615 and r = 0.304 and $p < 0.05$ respectively.

Table 4.5 indicates that HR had a significant negative relationship with triglycerides in males, where r = -0.629 and $p < 0.05$. HR also had a significant positive association with DP in males, where r = 0.592 and $p < 0.05$. MAP had a significantly positive association with WHR overall, where r = 0.309 and $P < 0.05$. MAP also had a significant and positive association overall with LDL-C and Total cholesterol/HDL-C ratio, where r = 0.268 and r = 0.256 and $p < 0.05$ respectively. MAP had significantly positive associations with SP overall, in males and in females, where r = 0.897, r = 0.818, r = 0.902 and $p < 0.001$ respectively. Diastolic blood pressure was also significantly and positively associated with MAP overall, in males and in females, where r = 0.946, r = 0.886, r = 0.942 and $p < 0.001$.

Table 4.5 shows that DP had a significant positive relationship with age, BMI, WC and WtHR in males, where r = 0.599, r = 0.697, r = 0.690, r = 0.614 and $p < 0.05$ respectively. Overall, LDL-C was significantly and positively associated with DP, where r = 0.341 and $p < 0.001$. LDL-C was also significantly and positively associated with DP in males and females, where

r = 0.600 and r = 0.316 and $p < 0.05$ respectively. Diastolic blood pressure was also significantly associated with SP overall and in females, where r = 0.727 and r = 0.735 and $p < 0.001$. Systolic blood pressure was also significantly and positively associated with DP in males, where r = 0.581 and $p < 0.05$. Furthermore, Table 4.5 indicates that SP had a significant positive relationship with WHR overall, where r = 0.282 and $p < 0.05$. There was also a significant negative relationship between SP and HDL-C overall where r = -0.260 and $p < 0.05$. Overall, SP was significantly and positively associated with total cholesterol/HDL-C ratio, where r = 0.259 and $p < 0.05$.

Table 4.5 shows that Total cholesterol/HDL-C ratio had a significant positive relationship with BMI overall and in females, where r = 0.251 and r = 0.334 and $p < 0.05$. Total cholesterol/HDL-C ratio also had a significant positive relationship overall with WC, WHR and WtHR, with r = 0.315, r = 0.260 and r = 0.289 and $p < 0.05$ respectively. Total cholesterol/HDL-C was also significantly and positively associated with WtHR in females, where r = 0.389 and $p < 0.001$. In addition, total cholesterol/HDL-C had a significant positive relationship with total cholesterol and LDL-C overall and in females, where r (overall) = 0.368 and r (overall) = 0.537 and r (females) = 0.413 and r = 0.565 and $p < 0.001$. Overall and in females, Total cholesterol/HDL-C had a significantly negative association with HDL-C, where r = -0.598 and r = -0.562 and $p < 0.001$. There was also a significant association with males, where r = 0.694 and $p < 0.05$. Lastly, triglycerides had a significant positive relationship with HDL-C in males, where r = 0.746 and $p < 0.001$.

In Table 4.5, triglycerides are shown to be significantly and positively associated with age overall, where r = 0.254 and $p < 0.05$. Waist circumference is also significantly and positively associated with triglycerides in females, where r = 0.294 and $p < 0.05$. High-density lipoprotein cholesterol had a significant and positive relation overall and in females, where r = 0.471 and r = 0.475 and $p < 0.05$ and $p < 0.001$ respectively. Low-density lipoprotein cholesterol had a significant positive relationship with age in males, where r = 0.611 and $p < 0.05$. There was also a significant positive association between LDL-C and total cholesterol, overall, in males and in females, where r = 0.866, r = 0.843 and r = 0.872 and $p < 0.05$ and $p < 0.001$ respectively. Total cholesterol had a significant positive relationship with age in males and overall, with WtHR, where r = 0.664 and r = 0.289 and $p < 0.05$.

Table 4.5 shows that waist to height ratio had a significant positive association with age overall and in males where $r = 0.248$ and $r = 0.721$ and $p < 0.05$ and $p < 0.001$ respectively. Waist to height ratio was also significantly and positively associated with BMI overall, in males and in females, where $r = 0.926$, $r = 0.757$, $r = 0.942$ and $p < 0.001$. Waist to height ratio also had a significant positive relationship with WHR overall, in males and in females, where $r = 0.843$, $r = 0.864$ and $r = 0.863$ and $p < 0.001$. In addition, waist to hip ratio had a significantly positive association with age overall and in males, where $r = 0.251$ and $r = 0.653$ and $p < 0.05$ and $p < 0.001$. Waist to hip ratio had a positively significant association with BMI overall in females and in males, where $r = 0.622$, $r = 0.701$, $r = 0.587$ and $p < 0.001$ and $p < 0.05$ respectively. In addition, WHR also had a significantly positive relationship with WC overall, in males and in females, where $r = 0.843$, $r = 0.846$, $r = 0.863$ and $p < 0.001$. Waist circumference had a positive and significant association with age in males, where $r = 0.716$ and $p < 0.001$. There was also a significantly positive relationship between WC and BMI overall, with males and with females, where $r = 0.906$, $r = 0.860$, $r = 0.953$ and $p < 0.001$ respectively. Lastly, BMI also had a significant positive correlation with age in males, where $r = 0.681$ and $p < 0.05$.

Table 4.6: Relationship between depressive scores and extended variables.

Variables	Sugary Treats	Fast Foods	Falling Asleep
BDI control and depressed	0.068	0.116	0.340**
MADRS control and depressed	0.124	0.089	0.381**

Note: * indicates significance at $p < 0.05$ level and ** indicates significance at $p < 0.01$ level.

Table 4.6 indicates that BDI scores had a significantly positive relationship with how long it takes to fall asleep, where $r = 0.340$ and $p < 0.001$. MADRS scores was also positively and significantly associated with how long it takes to fall asleep, where $r = 0.381$ and $p < 0.001$.

4.5 Regression Analysis

Table 4.7 Logistic regression models illustrating the greatest independent predictors of variables after adjusting for confounders.

Variables	R^2	*B*	df	P value	F
Anthropometry					
Predictor: BMI					
Dependent variables:					
WC	0.862	0.928	1	0.001**	381.220
WHR	0.505	0.771	1	0.001**	62.187
WtHR	0.891	0.944	1	0.001**	500.493
Lipid profiles					
Predictor: Total Cholesterol					
Dependent variables:					
LDL-C	0.720	0.849	1	0.001**	157.172
Predictors:					
LDL-C	0.283	0.523	1	0.001**	24.132
HDL-C	0.283	0.532	1	0.001**	24.091
Dependent variables:					
Total cholesterol/HDL-C					
Blood pressure					
Predictor: WHR					
Dependent variables:					
SP	0.293	0.542	1	0.001**	25.332
DP overall	0.225	0.474	1	0.001**	17.674

Note: * indicates statistically significant differences < 0.05; ** indicates statistically significant differences < 0.01. R^2: the percentage of variance in the dependent variable that can be explained by the independent variable (variance), *B*: Beta-coefficient, df: degrees of freedom, P value: indicates significance, F: F-statistic. BMI: body mass index, WC: waist circumference, WHR: Waist to hip ratio, WtHR: Waist to height ratio, LDL-C: low-density lipoprotein cholesterol, HDL- C: high-density lipoprotein cholesterol, SP: systolic blood pressure, DP: diastolic blood pressure.

Table 4.7 indicates that $R^2 = 0.862$ in the regression model of BMI (predictor) and WC (dependent variable). Thus, 86.2% of the variability of waist circumference can be explained by BMI. The regression model indicates statistical significance with F (1, 61) = 381.220 and $p < 0.001$. The β-coefficient ($\beta = 0.928$), with the significance of $p < 0.001$, indicates that BMI is a significant indicator of waist circumference. Thus, BMI is an independent predictor of waist circumference.

Table 4.7 also indicates that R2 = 0.505 in the regression model of BMI (predictor) and WHR (dependent variable). Thus, 50.5% of the variability of waist to hip ratio (WHR) can be explained by BMI. The regression model indicates statistical significance with F (1, 61) =

62.187 and $p < 0.001$. The β-coefficient ($\beta = 0.771$), with the significance of $p < 0.001$, indicates that BMI is a significant indicator of waist to hip ratio. Thus, BMI is an independent predictor of waist to hip ratio.

In addition, Table 4.7 shows that $R^2 = 0.891$, in the regression model for BMI (predictor) and WtHR (dependent variable). Thus, 89.1% of the variability of waist to height ratio can be explained by BMI. The regression model indicates statistical significance with $F(1, 61) = 500.493$ and $p < 0.001$. The β-coefficient ($\beta = 0.944$), with the significance of $p < 0.001$, indicates that BMI is a significant indicator of waist to height ratio.Thus, BMI is an independent predictor of waist to height ratio.

Furthermore, Table 4.7 shows that $R^2 = 0.720$ for the regression model between Total cholesterol (predictor) and LDL-C (independent variable). Thus, 72.0% of the variability of LDL-C can be explained by total cholesterol. The regression model indicates statistical significance with $F(1, 61) = 157.172$ and $p < 0.001$. The β-coefficient ($\beta = 0.849$), with the significance of $p < 0.001$, indicates that total cholesterol is a significant indicator of LDL-C. Thus, total cholesterol is an independent predictor of LDL-C.

Table 4.7 also indicates that both LDL-C and HDL-C are predictors for total cholesterol/HDL-C ratio, where R2 = 0.283, thus, 28.30% of the variability of total cholesterol/HDL-C ratio can be explained by LDL-C and 28.30% can also be explained by HDL-C. The regression model indicates statistical significance with $F(1, 61) = 24.123$ and $F(1, 61) = 24.091$, with $p < 0.001$. The β-coefficient ($\beta = 0.532$), with the significance of $p < 0.001$, indicates that LDL-C and HDL-C is a significant indicator of total cholesterol/HDL-C ratio. Thus, LDL-C and HDL-C are independent predictors of total cholesterol/HDL-C ratio.

Table 4.7 shows that $R^2 = 0.293$ in the regression model between WHR (predictor) and SP (dependent variable). Thus, 29.30% of the variability of systolic blood pressure can be explained by waist hip ratio (WHR). The regression model indicates statistical significance with $F(1, 61) = 13.6681$ and $p < 0.001$. The β-coefficient ($\beta = 0.542$), with the significance of $p < 0.001$, indicates that WHR is a significant indicator of systolic blood pressure. Thus, WHR is an independent predictor of systolic blood pressure.

Lastly, Table 4.7 shows that R2 = 0.225 in the regression model between WHR (predictor) and DP (dependent variable). Thus, 22.5% of the variability of diastolic blood pressure (DP) can be explained by waist to hip ratio (WHR). The regression model indicates statistical significance with F (1, 61) = 17.674 and $p < 0.001$. The β-coefficient ($\beta = 0.474$), with the significance of $p < 0.004$, indicates that WHR is a significant indicator diastolic blood pressure. Thus, WHR is an independent predictor of diastolic blood pressure.

4.6 Chi-square Analysis

Table 4.8: The association between MADRS control and depressed categories with lipid profile categories and lipid profiles with blood pressure measurements.

Variables		X^2	df	P value	OR (95% CI)	Count	Fisher's Exact Test
MADRS control and depressed	TRIGS	5.671	1	0.017*	0.215 (0.050 – 0.918)	9	0.022
	TOT.CHOL	4.940	1	0.026*	0.807 (0.665 – 0.980)	7	0.053
	LDL-C	4.138	1	0.042*	0.875 (0.768 – 0.997)	31	0.113
LDL-C	MAP	8.121	1	0.004**	0.136 (0.035 – 0.529)	6	0.042

Note: * indicates statistically significant differences < 0.05; ** indicates statistically significant differences < 0.01. X^2: Pearson Chi-square value, df: degrees of freedom, P value: indicates significance, OR(95% CI): Odds ratio at 95% confidence interval. MADRS: Montgomery Asberg Depression Rating Scale, TRIGS: triglycerides, TOT. CHOL: total cholesterol and LDL-C: low-density lipoprotein cholesterol and MAP: mean arterial pressure.

After performing chi-square analysis between both BDI and MADRS control and depressive scores with all categorical variables (anthropometry, lipid profiles and blood pressure measurements), Table 4.8 above indicates the only significant results obtained.

Table 4.8 shows that the Pearson chi-square test between MADRS control and depressive score with triglycerides resulted in a Pearson Chi-square score of 5.671. The result was significant where $p < 0.017$, thus MADRS scores and triglycerides levels had significant association. The OR of having high depressive scores with high triglyceride levels was 0.125 at 95% confidence interval (0.050 - 0.918). Total Cholesterol was also significantly associated with MADRS scores, where $p < 0.026$, with a Pearson chi-square statistic of 4.940. The odds ratio for MADRS scores with total cholesterol was 0.807, with a 95% confidence interval of 0.665-0.980. The participants are still at risk of having high cholesterol even if they have normal depressive scores.

The Pearson chi-square test between MADRS scores and LDL- C levels resulted in a Pearson Chi-square score of 4.138. The result was significant with $p < 0.042$. Thus, MADRS scores had a significant association with LDL-C. The odds ratio between MADRS scores with LDL-C levels was 0.875 with 95% confidence interval of 0.786-0.997. The odds of having low LDL-C with high depressive scores was 0.875.

The Pearson chi-square test between LDL-C and MAP resulted in a Pearson Chi-square score of 8.121.The result was significant with $p < 0.004$. Thus, LDL-C had a significant association with MAP. The odds ratio for LDL-C and MAP is 0.136 with a confidence interval of 0.035-0.529. Thus, the odd of having high MAP with high LDL-C is 0.136.

4.7 Chapter summary

Chapter four included the descriptive statistics and frequencies of all the variables in the study. It provided the severity of depression according to both MADRS and BDI scores. Chapter 4 provided the associations between depression and BMI, blood pressure and lipid profiles and further associations between the variables themselves. This chapter also provides further statistical analysis, indicating regression analysis and Chi-square tests. Chapter five provides the discussion for the results obtained.

Chapter five
Discussion

5.1 Introduction

The aim of the study was to determine the association of body mass index (BMI), blood pressure and lipid profiles with Depressive Scores in students at the University of the Western Cape (UWC). Associations between the variables (BMI, blood pressure and lipid profiles) were also investigated as these are modifiable risk factors which contribute to cardiometabolic syndrome. Depression and cardiometabolic syndrome are known to be significantly associated (Whooley et al., 2008 and Hadidi et al., 2009). The results of this study will be discussed and dissected in this chapter.

The prevalence of depression among this study population, according to the Beck Depression Inventory (BDI) scale was 47.61%, as 30 participants were classified as having moderate to severe depression. This depression prevalence is high when comparing the prevalence across different studies of a similar study population. Studies carried out by Berhanu et al, Melese et al, Birhanu et al, and Dessie Y et al, had a prevalence ranging from 21.6% to 32.2% of depression prevalence (Berhanu et al., 2015; Melese et al., 2015; Birhanu et al., 2016 and Dessie et al., 2013). Studies with higher prevalence include a study performed in Jimma and Addis Ababa, where the depression prevalence were 58.4% and 51.3% respectively (Gebreegziabher et al., 2019 and Kebede et al., 2019). The difference in prevalence in this could be the result of asmaller sample size, different socio-economic factors, such as age, level of education andfinancial status. Another study performed in India reported that 37.7% of their studypopulation suffered from moderate depression (Deb et al., 2016). This study coincidessimilarly, as 33.3% of participants suffered from moderate depression, according to the BDI. This study includes mainly black and coloured students. UWC is well-known for its richcultural background. UWC has always been on the forefront, fighting for rights againstoppression and discrimination and always including those from a disadvantaged background.UWC prides itself with equality, access, and quality high education. One of UWC's mainobjectives is to help the previously marginalized population to be free and participate freelyin South Africa's society (University of the Western Cape, 2021).

This study includes a far higher female participant to male participant ratio. It is known that young females are twice as likely to be depressed compared to males, although this ratio alters and decreases with an increase in age (Albert, 2015). The increased risk of depression starts at puberty for females, due to fluctuating hormones and other social or psychological factors such as gender-discrimination, poor economic circumstance, sexual abuse, sexuality, and identity issues. Females typically reach puberty before males, and thus have a higher risk of developing depression and developing depression at an earlier stage (Lewis et al., 2018 and Kuehner, 2017). The higher female to male ratio could be explained by few males responding to the study or because males are more reluctant to seek help, due to "old-fashion" masculinity ideals, such as being "unmanly" or "weak" (Staiger et al., 2020).

When completing a non-parametric Mann Whitney-U Independent T-test according to gender, a significant difference was found in Montgomery-Asberg Depression Rating Scale (MADRS) scores between males and females, where females had a higher average MADRS scores than males. Further significant differences were found in systolic blood pressure (SP) and mean arterial pressure (MAP), where males had higher SP and MAPs than females. When performing a one-way analysis of variance (ANOVA) according to gender, there were also significant differences between males and females across MADRS scores, SP and MAP. There was also a significant difference across gender for diastolic blood pressure (DP). Thesefindings are expected and coincide with studies previously performed. As mentioned previously, females are twice as likely to be depressed compared to males due to the commencement of puberty at an early age, causing a change of hormones, gender inequality and other socio-economic factors (Reiss, 2013). The higher blood pressures (SP, DP, and MAP) found in males compared to females is also expected, as males generally have a higher blood pressure compared to females (Reckelhoff , 2001). Men have a higher blood pressureto females in similar ages, only after menopause do women's blood pressure increase to levels higher than males (Maranon & Reckelhoff, 2013). It was also found that males have higher waist to hip (WHR) ratios than females. This result coincides with Barnaby, 2016, where males have a higher waist to hip ratio to females by 0.10-0.15 (Dixson, 2016).

Majority of participants were in the 18-20 or 21-23 age categories, and the frequencies decreased with an increase of age. These students were also found to be in their undergraduate degree (first, second or third year). There are many factors which may

contribute to depression at this level of study and at this age. For many students, University would come as a new environment, which may be exciting to some, but is daunting and overwhelming to others. Undergraduate students have the battle of fitting in, finding their footing and trying to perform academically. Some psychological factors that contribute to depression include, low self-esteem, underlying mental health issues before starting university and loneliness (Mofatteh , 2020). Apart from these struggles, they may have socio-economic challenges such as victimization, fearing for safety on campus and studying in a non-native language. Students may also have a fear of failure and beoverwhelmed with the pressure of workload. Being a student is highly demanding, while trying to juggle these concerns and trying to create their own identity (Ratanasiripong et al., 2018). There are also financial factors that may increase the risk of depression or depressive symptoms, these include, poverty in childhood, lack of family income and no adequate financial aid (Sznitman et al., 2011). Students need to deal with these stresses before their brains (the pre-frontal cortex) are fully developed. The prefrontal cortex aids in reasoning and controlling impulses. Undergraduates therefore rely on the amygdala,so emotions are used to analyse information (Pletnikoff, 2021). The amygdala of the brain is responsible for processing emotions and fear memories (Salzman, 2021). Thus, students become overwhelmed with stress due to several contributing factors, which may result in depression. Students who participated in this study may also have had other issues such as being overwhelmed to start University on their own, at home, and online due to the Coronavirus disease 2019 (COVID-19) pandemic. While some are comfortable learning on their own, many feel overwhelmed by having to process everything by themselves.

When completing a One-way Analysis of variance (ANOVA) according to age, there were only significant differences across the anthropometric measurements. According to the one- way ANOVA, there was a significant difference between the groups according to age across body mass index (BMI), waist circumference (WC), waist to hip ratio (WHR) and waist to height ratio (WtHR). All these anthropometric measurements had a difference between group 1 (age 18-20) and group 6 (age 33-35). This is to be expected as anthropometric measures areexpected to increase with an increase in age (Kobani, 1994; Stevens et al., 2010 and Taking et al., 2016).

5.2 Anthropometric measurements and depression

According to Spearman's non-parametric correlation, there were no associations between the anthropometric measurements (BMI, WC, WHR, WtHR) with depressive scores (MADRS or BDI scores). Thus, BMI was not significantly associated with depression in this study.Results concerning the association between BMI and depression are inconsistent. Somestudies have found a positive association, others have found a negative association, and some have found no significant association. Data obtained from the Third National Health and Nutrition Examination Survey showed that BMI is associated with depression and that the higher the BMI, the higher the level of depression (Onyike, 2003). A systematic review and meta-analysis completed by Luppino FS and colleges in 2010, found a reciprocal link between obesity and depression. Obesity was found to increase depressive scores, while depression predicted development of obesity. Researchers attending the University of Exeter (United Kingdom), together with scientists from the University of South Australia, used genetic data to explore the relationship between depression and BMI. The scientists wanted todetermine whether higher BMI was associated with increased risk of depression. There was a positive association between BMI and depression, where a higher BMI associated with higherdepressive scores (Tyrell et al., 2019). A study completed in the Netherlands wanted to investigate if there was a U-shaped association between BMI and depression, meaning that both underweight and overweight categories could be associated with depression (de Wit et al., 2009). A very significant U-shape association was found between BMI categories and depression, while no linear association was found. Thus, a U-shaped association should be investigated within this study to determine a significant association. This is also because the data had varied results, so participants with high BMIs didn't necessarily have a high depressive score, but some with low BMI's had high depressive scores. Thus, a linearassociation was not obtained. No association between BMI and depressive scores could also be a result of a smaller sample size in this study.

5.3 Lipid profiles and depression

After performing a non-parametric Spearman's correlation, there were no significant correlations between lipid profiles (total cholesterol, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), triglycerides, total cholesterol/HDL-C ratio) and depression (MADRS and BDI scores). However, after running a point-biserial correlation, Total cholesterol and LDL-C were negatively associated with

MADRS scores, but not BDI scores. Triglycerides were positively associated with MADRS scores. Thus, lower total cholesterol and lower LDL-C measurements will result in higher depressive scores and higher triglyceride levels mean higher depressive scores (an inverse relationship). A study conducted by Rabe-Jablonska, J and colleges indicated that low cholesterol levels was associated with the severity of depression (Rabe-Jablonska & Poprawska, 2000). A meta-analysis including associations between total cholesterol, LDL-C and HDL-C and depression, found that a higher level of total cholesterol resulted in a lower depression rate (inverse relationship) (Shin et al., 2008).

Research has suggested that cholesterol is an important organic molecule associated with behavioural changes as well has mood disorders (Vevera et al., 2005). This is because the brain is a cholesterol-rich organ, accounting for approximately 25% of the body's total cholesterol (Singh et al., 2007). Cholesterol is also an important component in normal brain cell functioning such as cell membrane integrity as well as myelin production (Sooksawate et al., 2001). Cholesterol is also partly responsible for managing neurotransmitters and the formation of synapses. An important neurotransmitter involved in mood is serotonin, which stabilizes mood and feelings of happiness and well-being. Low cholesterol levels may influence serotonergic functioning by lowering the number of activated serotonin receptors and thus decrease overall serotonergic activity (Singh et al., 2007). Low cholesterol levels may also reduce the function of serotonin transporter activity. Decreased levels of serotonin have been linked to depression and overall low mood (Chung et al., 2007). Medications such as serotonin re-uptake inhibitors (SSRIs) may improve mood by increasing the level of serotonin in the brain (Harmer et al., 2017). Even though 87.3% of the participants in this study had normal total cholesterol levels, they are still at risk of developing depression. With regards to risk estimation and odds ratio, total cholesterol is independent of MADRS scores and participants are still at risk of developing depression evenif they have normal cholesterol levels in this sample of 63 participants, as the Pearson Chi- square value is 4.940 with $p < 0.026$ and an odds ratio of 0.807 with confidence interval (CI) (95%) 0.665-0.980. A cross-sectional study including 19 527 participants, found that the oddsratio for low levels of cholesterol associating with depression was 0.64 with CI (0.42-0.98) (Cepeda et al., 2020).

Research regarding the relationship between LDL-C and depression is inconsistent. Some studies have found no association between LDL-C (Sagud et al., 2009) and depression, while

others have found a positive association (Oh & Kim, 2017). Other findings include a U-shaped relationship between LDL-C and depression (Tedders et al., 2011). This study found a significant negative association with depression. A study investigating the "causal relationship between blood lipids and depression phenotypes" foundthat lower LDL-C was related to more severe depressive symptoms (So et al., 2021). Another study conducted by Sadeghi M and colleges, found that depression severity was linked to reduced apolipoprotein A (LDL-C) (Sadeghi et al., 2011). A study investigating the role of lipoproteins in cognitive performance and depression found that there was a significant negative correlation between LDL-C and depression (Jia et al., 2020).

Low levels of high-density lipoprotein cholesterol (HDL-C), the "good" cholesterol, puts one at an increased risk of cardiovascular disease and hypertension, while having high levels of LDL-C may cause a stroke or heart attack (Boytsov et al., 2017). Generally, having high levels of HDL-C and low levels of LDL-C is ideal in terms of cardiovascular disease. However, research has shown that there is a relationship between (very) low levels of LDL-C and increased risk of developing mood disorders such as depression (Bandyopadhyay et al., 2018). Scientists are still unsure as to why at a particular threshold, low LDL-C becomes an issue (Weissglas-volkov & Pajukanta, 2010).

LDL-C is a lipoprotein, so its function involves transporting lipids to cells in the body (CDC, 2017). Persistently low LDL-C impairs brain function as well as hormone production and activity and increases the chance of developing a condition known as hypobetalipoproteinemia. This condition hinders the body's ability to transport and absorb fats. This condition has been linked to several disorders such as haemorrhagic stroke, cirrhosis, cancers as well as depression (Michos, 2017). Hypobetalipoproteinemia is also associated with mutations of the ANGPTL3 gene (promotes the uptake of free triglycerides), which causes abnormal decreases in both LDL-C and HDL-C, leading to depression (Welty, 2014). Low levels of LDL-C are similarly associated with the unbalanced regulation of tumor necrotizing factor alpha (TNF-a), which may be correlated with depression, cancer, and Alzheimer's disease (Ruan et al., 2006). The risk of having low LDL-C with high depressive symptoms (high MADRS scores) in this study was 0.875 with CI (95%) 0.768 – 0.997, where the Pearson Chi-square value was 4.138 with a significance of $p < 0.042$ in this sample of 63 participants. A study completed by Liu J et al., involving 26 819 Canadian adults

found a similar odds ratio to this study, with an OR = 1.05 and CI (95%) 0.98-1.12 (Liu et al., 2020).

There has been varied data regarding whether triglycerides are associated with depression. A study conducted by Sheikh N and colleges investigated whether triglycerides and cholesterol have a relationship with depression and found no significant association between the lipids and depression (Sheikh et al., 2004). Other studies also investigating the levels of lipids (total cholesterol, HDL-C, LDL-C, and triglycerides) with depression also found no significant associations between triglycerides and depression (Martinac et al., 2007 and Sheikh et al., 2004). In contrast, research conducted in Sweden resulted in low levels of triglycerides associating with depression in women only (Lindberg et al., 1994). Research resulting in the opposite conclusion, includes a study conducted by The Korea National Health and Nutrition Examination Survey (KHANES), found that high levels of triglycerides was associated with depression (Oh & Kim, 2017). This study found a positive association between triglycerides and depression. Another study performed by the Netherlands Study of Depression and Anxiety (NESDA), found that triglyceride levels were higher in patients with major depressive disorder (van Reedt Dortland et al., 2010). A study conducted by Toker S and colleges found that there was a positiveassociation between raised triglyceride levels and depression (Toker et al., 2008). A cross- sectional study conducted by Enoch RH, found that triglyceride levels were elevated in patientswith depression compared to control patients (Enoch, 2020).

Research conducted investigating the relationship between metabolic syndrome (in this case, triglycerides) found that elevated triglycerides were associated with higher depressive symptoms in children above the age of 12.68 (Michael, 2017). Furthermore, a study investing psychological conditions with metabolic factors in elderly also found that there was a significant positive association between depression and triglycerides (Bove et al., 2010). Increased levels of triglycerides are strongly associated with a rise in psychological disorders, including schizophrenia, bipolar disorder, and depression. It is also associated with increased levels of aggression and hostility (Fowkes et al., 1992). In a study conducted by Dr Glueck et al., elevated triglycerides as well as high total cholesterol and low HDL-C were the only causes of mild to severe depression in patients with hypertriglyceridemia (Glueck et al., 1993). The patients in this study underwent a lipid-lowering diet to investigate whether lowering lipids would improve depressive symptoms. Triglyceride levels decreased by 47%, resulting in a significant decrease in depressive scores. The greater the decrease in

triglyceride levels, the higher the percentage reduction in depressive scores. There could therefore be a reversible causal relationship between triglycerides and depression, where a reduction in triglycerides leads to a reduction in depressive symptoms (Glueck et al., 1993). In this study, the odds of having high triglycerides with high depression is 0.215 with CI (95%) 0.050-0.918, where the Pearson Chi-square value is 5.671 with a significance of $p < 0.017$. A Dutch cohort study, including 2981 participants found the odds of having high triglycerides with high depression to be 1.89 with CI (95%) 1.15-3.11. The difference between this study's odds ratio and the cohort study could be explained by a larger sample size in the cohort study (Watson et al., 2021).

The link between high triglycerides and depression also involves insulin resistance. This is due to the ingestion of carbohydrates (sugars, starch, and processed grains), which release insulin. Insulin is a hormone produced by the pancreas that regulates the metabolism of carbohydrates by absorbing glucose and converting it to glycogen through glycogenesis(Stryer, 1995). Insulin resistance occurs when the cells in the body's fat, muscles and liver no longer respond to insulin, resulting in a rise in blood sugar levels. This may lead to type-2 diabetes mellitus (National Institute of Diabetes and Kidney Disease, 2018). Scientists from Stanford University of Medicine found thatinsulin resistance is linked to an increased risk of developing depression and that if you are insulin resistant; you are twice as likely to be depressed compared to someone that is not insulin-resistant (Goldman, 2021). Brain insulin resistance occurs when the brain itself no longer responds to insulin (Arnold et al., 2018). The central nervous system (CNS) regulates insulin within the brain, which is found mostly in the hypothalamus, hippocampus, and cortex areas (Sripetchwandee et al., 2018).

There are three possible pathophysiologic mechanisms in which depression is associated with insulin resistance. This includes the hypothalamic-pituitary-adrenal (HPA) stress axis, the gray matter capacity in particular regions of the brain as well as the brain's reward system (Lyra e Silva et al., 2019). Research proposes that insulin resistance could impair the function of the HPA-stress axis which controls the brain's response to stress. A dysregulated HPA axis affects the regulation of glucocorticoid (steroid hormones which regulate the use ofsugar and control inflammatory responses) release which leads to abnormal stress responses. These abnormal stress responses lead to depressive symptoms (Kullmann et al., 2016). Relationships between insulin resistance and abnormal structure and function of the brain has linked to depression, specifically in the Anterior Cingulate Cortex (ACC) and the

hippocampus. High insulin resistance has resulted in reduced volumes in the ACC as well as the hippocampal gray matter and have resulted in higher depressive symptoms compared to low insulin resistance (Singh et al., 2019). The hippocampus is associated with emotional memory recalling and learning (Schumacher et al., 2018), while the ACC is involved in processing emotions and making decisions. Decreased capacities in these areas of the brain may explain the impairments observed in depressed patients (Hamer et al., 2019).

With regards to insulin resistance and the brain's reward system, brain insulin regulates dopamine brain pathways (Lyra e Silva et al., 2019), therefore, disruptions in insulin signalling results in impairment of dopamine signalling which affects the brains' motivation and reward mechanisms. These disruptions may lead to depressive symptoms. Patients with insulin resistance and depression have exhibited disturbances in the ability to signal satiation, which results in an increase level of anhedonia (the inability to feel pleasure), resulting in a low mood (Hamer et al., 2019).

5.4 Blood pressure measurements and depression

There were no significant associations between any blood pressure measurements (systolic blood pressure, diastolic blood pressure, mean arterial pressure and heart rate) with depressive scores (MADRS or BDI) in this study after completing a Spearman's rho correlation. However, when filtering for gender, there was a significant moderate positive association between MADRS scores and systolic blood pressure (SP) in males using Spearman rho's association. Neither MADRS nor BDI scores correlated with blood pressure measurements in females in this study. The significance of depression with systolic blood pressure in males could stem from males generally having a higher blood pressure compared to females, which starts at puberty and continues through adulthood, until women begin menopause (Himmelmann et al., 1994 and Harshfield et al., 1994). This study found no significant differences across blood pressure measurements between depressed and control participants. Research conducted by Wiehe M et al., also found no differences in blood pressure between patients with or without depression (Wiehe et al., 2006). A study conducted by Licht CM et al investigated whether decreased blood pressure was associated with depression and found that depressed participants had lower systolic blood pressure than controls. Low systolic blood pressure rather than hypertension associated with depression (Licht et al., 2009). After running a Pearson Chi-square test there was no significant

association between high or low systolic blood pressure with depressive scores, so high or low systolic blood pressure with depression could not be determined in this study. Research conducted by Stroup-Benham CA found a significant positive association between low systolic blood pressure and depression ($p < 0.01$) (Stroup-Benham et al., 2000). Scientists in Dublin, at Trinity College, propose that depression may be caused by decreased blood flowto the brain in the case of low blood pressure (hypotension) (Sexton et al., 2017). Irish researchers conducted a study including 4500 men and women over the age of 50. Participants with constantly low systolic blood pressure had a 30% increased risk of having mental illness (Briggs et al., 2017). A large study carried out by the Norwegian Institute of Science and Technology measured the blood pressures and depressive scores of 60 000 men and women aged 20-89. The study found that participants with low systolic blood pressure had a 22% increased risk of developing depression compared to those with normal blood pressure (Hildrum et al., 2007). A retired professor at the University of Glasgow, showed that participants consuming beta blockers, which lower blood pressure, had two-fold the risk of developing depression. He did note that even though there was an association, it did not mean that there is a causal relationship between depression and low systolic blood pressure,so having low blood pressure doesn't necessarily cause depression (Boal et al., 2016).

In contrast to low blood pressure, independently, depression and hypertension (high blood pressure) have a high prevalence worldwide, there have been few studies investing how these diseases associate with each other. A meta-analysis conducted by Scalo AZ et al., found an increased prevalence of hypertension in patients with depression (Scalo et al., 2005). Research has suggested that there is a deficiency of dopamine sites in the brain which will initiate depression (Mycek et al., 2000). In addition, the blood deficiency in the brain as a result of hypertension may also put individuals with hypertension at risk for depression (Thomas et al., 2003). This is because both hypertensive and depressive patients have an increased sympathetic activity as well as increased secretion of cortisol (stress hormone) and adrenocorticotropic hormone (regulates production and release of cortisol from the adrenal gland) (Meng et al., 202).

Many epidemiological studies have also researched the association between blood pressure and depression. These studies have indicated that depression has nonlinear association with systolic and diastolic blood pressure, thus high depressive scores will be found with both highand low systolic and diastolic pressures (Kim et al., 2010; Meng et al., 2012 and

Tikhonoff et al., 2014). Research studies are still finding mixed reviews with regards to the relationship between blood pressure and depression. Further research needs to be done as to whether there's a causal relationship or a linear/non-linear relationship between the two diseases. This study may have had no blood pressure associations with depression as most participants were classified as normal in all blood pressure measurements (systolic blood pressure, diastolic pressure, mean arterial pressure and heart rate). Finding an association between systolic blood pressure and depression in men and not in women could be due to men generally having higher blood pressures than females. A larger sample size with a higher number of participants in high or low blood pressure categories may have resulted in significant associations between the blood pressure measures and depressive scores. Age could also have impacted the blood pressure measures as most participants were in the 18– 20-year-old category, also with normal blood pressure, resulting in no associations between blood pressure measurements and depression.

5.5 Depression and Extended Questions

Both MADRS and BDI depressive scores positively and strongly correlated with how long it takes to fall asleep. Most participants reported that they take more than 60 minutes to fall sleep. Most people who have suffered through depression have experienced sleeping problems. Patients suffering from depression often have trouble falling asleep and staying asleep throughout the night. According to Johns Hopkins Medicine, 75% of patients sufferingfrom depression have trouble with their sleep. Sleeping problems often aggravate depression symptoms, leading to a harmful cycle between sleep and depression. A lack of good sleep results in difficulties regulating emotions as well as shortens the restorative slow-wave sleep that is needed every night. Short-wave sleep is considered to be the deepest sleep, the phase in which a person dreams or even sleepwalks. Short-wave sleep is important for the ability to transform short-term memory into long term, long lasting memories (memory consolidation). In addition, people who have interrupted sleep had a 31% decrease in positive mood the following day, according to Finan P (Finan et al., 2015). This is because lack of sleep leads to a reduction in emotional resilience, which is having the ability to be positive to get through the challenges and stresses of everyday life (Johns Hopkins Medicine, n.d).

5.6 Variables in relation to cardiometabolic disease

Cardiometabolic disease (CMD), also known as cardiometabolic syndrome (CMS) is classified as non-communicable diseases which are preventable but are very common worldwide. CMD is a combination of metabolic dysfunctions which includes impaired glucose tolerance, dyslipidaemia (high cholesterol, low HDL-C, and high LDL-C), high blood pressure, insulin resistance and well as increased visceral adiposity ("belly fat") (Hossain et al., 2007). The prevalence of CMD is increasing at an exponential rate and epidemiological studies have shown that disease incidence is increasing in Sub-Saharan Africa (Njelekela et al., 2009; Oladapo et al., 2010; Tran et al., 2011 and Wai et al., 2012). CMD is driven by a variety of factors, such as sedentary levels, dietary habits, stress levels as well as economic development (Reddy & Yusuf, 1998). Research is now including depression as a risk factor for co-morbidity with CMD (Tomlinson et al., 2009 and Katon et al., 2010). Research has also found associations between CMD and depression (Celano & Huffman, 2011 and Glassman et al., 2011). Physiological mechanisms (mechanism of action) and causal relationships between CMD and depression are still being investigated, but the knowledge of the characteristics of each of these diseases may help in controlling disease prevalence (Katon et al., 2010).

In this study, three out of the five risk factors (glucose, dyslipidaemia, blood pressure, insulin resistance and visceral fat) of CMD were investigated to determine their association with depression. The three risk factors included in this study were body mass index (BMI), dyslipidaemia and blood pressure. Many variables investigated in this study were not significantly associated with depression; however, the variables were associated with each other. These variables increase the risk of CMD. The findings associated with these CMD risk factors will be discussed here as well as the number of participants in each risk factor category (whether they have no risk factors, one, two or three risk factors). The higher the number of risk factors, the higher the risk of CMD. Most participants (49%) had no risk factors for cardiometabolic disease. However, almost 27% of participants had one metabolic risk factor and 20% had at least two metabolic risk factors. Only two participants had all three metabolic risk factors. These numbers are high and concerning considering the participants were aged 18-35 years old and are already at risk for cardiometabolic diseaselater in life.

The significant findings regarding each risk factor in relation to cardiometabolic disease will be discussed.

5.6.1 BMI and anthropometric associations and their relationship to cardiometabolic disease

Even though BMI was not associated with depressive scores, it was associated with the other anthropometric measures which are risk factors for cardiometabolic disease. BMI was significantly, strongly, and positively associated with waist circumference, waist to hip ratio and waist to height ratio. Most participants had a normal BMI (58.7%), while 14.3% were overweight and 14.3% were classed as obesity class 1. Almost 8% of the participants were classified as obesity class 111. Waist circumference has been found to be a superior way of determining fat distribution compared to BMI. Waist circumference measures abdominal obesity or visceral adiposity which is characterized by an increase adipose tissue surrounding the intra-abdominal organs (Ritchie & Connell, 2007). A study performed by Chinedu and colleges also determined that waist circumference can be a positive indicator of overweight or obese participants (Chinedu et al., 2013). Obesity as defined by BMI and increased waist circumference is a risk factor for insulin resistance (Schulze et al., 2006) and hence type 2 diabetes mellitus, as well as increases the risk for cardiovascular disease (Yusuf et al., 2004). Excess visceral adiposity is related to dysregulation of fatty deposits in artery walls as well as dysregulation of hormones regulating blood-glucose levels (Tchernof & Després, 2013). An increase in fatty deposits in artery walls leads to atherosclerosis, which may also result in cardiovascular disease.

A study conducted by Reza S and colleges investigated whether BMI correlated with waist to hip ratio and found that waist to hip ratio correlated to BMI significantly in females only. In this study, BMI was significantly associated with waist to hip ratio overall, in males and in females (Reza et al., 2020). In addition, a study conducted in Santo Andre in 2019 found a significant correlation between BMI and waist to height ratio. They found and increase in the association with an increase in age (Faria et al., 2021). Most participants had a normal weight to height ratio (63.5%), while 19% had an increased risk and 17.5% had a very high risk of obesity-related cardiovascular risk. Research has found that WtHR is superior to both BMI and waist circumference in assessing cardiometabolic risk in adults. BMI cannot differentiate between adiposity or muscle mass and it cannot reflect the body's fat

distribution and WC does not take height into account (Yajnik et al., 2004 and Zimmet et al., 2005). In addition, children with increased WtHR have a higher chance of having more cardiometabolic risk factors (Spolidoro et al., 2013 and Khoury et al., 2013). The higher the value of waist to height ratio, the higher the risk of developing obesity-related CVD. WtHR is an indicator for the risk of developing heart disease, stroke, hypertension, obesity, and diabetes (Browning et al., 2010). Increased waist means increased abdominal adiposity. Increased visceral fat affects organs such as the liver, heart, and kidneys with regards to CMD (World Health Organization, 2008). This is because visceral fat releases molecules and hormones, including adiponectin, tumor necrosis factor, interleukin-6, leptin and resistin. Adiponectin is an important hormone with regards to CMD as increased release of adiponectin causes a reduction in the growth of new blood vessels (Pinthus et al., 2008). This causes and increase in blood pressure which may result in hypertension, leading to CMD. Rush et al determined that black South African women have a lower BMI at a given percentage of body fat, compared to that of white European women and have less abdominal visceral fatty deposition. This was determined with dual x-ray absorptiometry. This coincideswith this study as most participants were black female participants with normal waist to height ratios (Rush et al., 2007). Further smaller studies show that African women have less abdominal fat than white women (Lewis et al., and van der Merwe et al.,2000). BMI was the greatest predictor of waist circumference, waist to hip ratio and waist to height ratio, while adjusting for all other variables in this study.

5.6.2 Lipid profile associations

Even though not all lipid measurements associated with depression, some lipid measurements did associate with each other and other variables. Triglycerides had a significant positive association with age. This coincides with Sweeney MET, who also found that triglycerides increase with an increase of age (Sweeney, 2021). LDL-C significantly and positively associated with total cholesterol and total cholesterol/HDL-C ratio. These are expected associations as LDL-C is used to calculate total cholesterol (American Heart Association, 2021). LDL-C significantly, strongly, and positively associated with total cholesterol in females. Most participants had normal LDL-C (55.6%), while 38, 1% had low LDL-C, which was associated with depression. Only 6.4% of participants were between intermediate and high LDL-C groups. LDL-C is in control of transporting cholesterol to arteries. When LDL-C

levels are increased, it causes the cholesterol levels within artery walls to build-up. This contributes to the formation of plaque within artery walls, leading to a condition known as atherosclerosis (Brown & Goldstein, 1977). The plaque narrows blood vessel walls and blocks blood flow to the heart and brain (Davies et al., 1993). Plaques can also rupture and cause a blood clot. The blockages and clots may both result in a heart attack or stroke (Lee & Libby, 1997). In South Africa, black women have a more favourable lipid profile compared to white women, with lower total cholesterol levels, lower LDL-C and lower triglyceride levels compared to white women. In addition, black women are seen to have a "protective" lipid profile as they have significantly different genes and alleles compared to white women, which result in these lower lipid profiles (Ellman et al., 2015). Total cholesterol was seen tobe the greatest independent predictor of LDL-C when controlling for all other variables in this study.

HDL-C overall significantly and negatively associated with total cholesterol/HDL-C ratio in this study. This is expected because HDL-C is used in calculating total cholesterol (AHA, 2021). HDL-C had a significant moderate negative association with total cholesterol/HDL-C ratio in males but not in females. A reduction in HDL-C levels is associated with an increased risk for CMD and could also be an independent risk factor for CMD (Ascaso et al.,2007). Most participants had normal HDL-C (82.5%) levels, while 17.5% had low HDL-C, putting them at risk for CMD. 92% of participants had a normal total cholesterol/HDL-Cratio, while 7.9% had a high total cholesterol/HDL-C ratio. A higher ratio indicates a higher risk for CVD. Participants with a higher total cholesterol/HDL-C ratio have a greater CMD risk because of the disproportion in the cholesterol components carried by the protecting lipoproteins. This may be a result of a rise in the atherogenic factors (promoting the formation of fatty deposits in artery walls) in the numerator and a reduction in the anti- atherosclerotic (agents counteracting atherosclerosis) trait of the denominator, or both, with regards to the total cholesterol/HDL-C ratio (Criqui & Golomb, 1998). HDL-C also had a positive correlation with total cholesterol in females in this study. In females, total cholesterol/HDL-C ratio had positive associations with BMI, waist circumference (WC) and waist to hip ratio. In a study conducted by Brenner DR et al., it was found that WC was correlated positively to total cholesterol/HDL-C ratio in men and women, but BMI hadno significant correlation (Brenner et al., 2010). A study conducted by Komiya S et al., investigating the relationship of serum lipids to waist to hip ratio found that obese women with high WHR had a high total cholesterol/HDL-C ratio, while those with normal WHR had

lower ratios (Komiya & Masuda, 1989). A high intra-abdominal proportion results in a high total cholesterol/HDL-C ratio. As mentioned previously, a higher ratio indicates an increased risk for CMD. In males, total cholesterol/HDL-C also had a strong positive correlation with triglycerides in this study. A study conducted by Lemieux I et al., found that men with higher triglycerides had higher total cholesterol/HDL-C ratios (Lemieux et al., 2001). The greatest predictor(s) of total cholesterol/HDL-C ratio in this study was LDL-C and HDL-C (with the same R = 0.532), while adjusting for all other variables.

5.6.3 Lipids associating with anthropometry and blood pressure

LDL-C correlated significantly and positively with diastolic pressure (DP) and mean arterial pressure (MAP). A study conducted by Lamarche F et al., also concluded that LDL-C had a significant positive association with diastolic pressure (Lamarche et al., 2018).Again, elevated LDL-C can cause and increase in fatty deposits in artery walls leading to narrowing and stiffening of the arteries, causing increased blood pressure and increased riskof CMD (AHA, 2021). LDL-C correlated moderately and positively with diastolic blood pressure. Most participants had normal diastolic pressure (77.8%), while 22.2% had moderate-high diastolic pressure. Increased diastolic pressure as a result of high LDL- C may lead to diastolic heart failure. This is described as a stiff left ventricle with reduced levels of compliance and weakened relaxation leading to a higher end diastolic pressure (Gutierrez & Blanchard, 2004).

On the other hand, HDL-C had a significant negative correlation with systolic pressure. A study performed by Nasri H et al also concluded that HDL-C had an inverse association with systolic blood pressure (Nasri & Yazdani, 2006). Low levels of HDL-C have also been associated with increased risk for CMD (Di Angelantonio et al., 2009). This is because reduced HDL-C cholesterol levels fast-tracks the progress of atherosclerosis as cholesterol transport is impaired (the removal of LDL-C or “bad” cholesterol becomes insufficient) also resulting in thickened arteries, leading to CVD and CMD. HDL-C is also responsible for preventing LDL-C from oxidizing, which plays an important role in the development of atherosclerosis (Singh, 2021).

In males, triglycerides had a moderate negative association with heart rate. A study conducted by Balikai FA et al., also found a negative association between triglycerides and heart rate (Balikai et al., 2020). No other research has been conducted to find the association between triglycerides and heart rate variability. Much more research needs to be conducted to determine the relationship between triglycerides and heart rate, especially in men.

5.6.4 Blood pressure and anthropometry

Overall, systolic blood pressure (SP) had a significant correlation with waist to hip ratioin this study. A study conducted by Chaudhary S et al., found that systolic blood pressure was positively correlated with WHR and that diastolic blood pressure was correlated significantly and positively with waist circumference (WC) (Chaudhary et al., 2019). This coincides with this study as diastolic blood pressure (DP) was also correlated positively with WC. DP also positively correlated with waist to hip ratio (WHR) in males in this study. A study conducted by Gupta R et al., found that in males, with an increase in DP, there was an increase in WHR (Gupta & Mehrishi, 1997). Diastolic blood pressure also correlated moderately and positively with BMI in males. Waist to hip ratio was the greatest independent predictor of both systolic and diastolic pressure when controlling for all other variables in this study. This could be explained by 34.9% of the participants in this study falling into the high risk to very high-risk WC category, while 22.2% fell into the moderate risk to very high risk for cardiometabolic complications according to WHR. As mentioned earlier, increased WC and WHR means an increased risk for cardiometabolic disease. Excess abdominal adiposity is associated with a variety of cardiometabolic complications as well as cardiovascular disease. Higher visceral (abdominal) adiposity leads to the build-up of fatty deposits in the arteries, leading to heart attack and stroke. Furthermore, a larger mid-section makes it more difficult for the body to produce insulin, which controls blood-sugar levels (glucose). This may lead to type-2 diabetes and hence cardiometabolic disease (British Heart Foundation).

5.6.5 Blood pressure measurements associated with each other

Systolic blood pressure (SP) was significantly and positively correlated with diastolic blood pressure (DP) and mean arterial pressure (MAP) overall, in males and in females. Gavish B et

al also found a significant positive correlation between SP and diastolic pressure (Gavish et al., 2008).

5.6.6 Associations of race with anthropometry and blood pressure

Overall, race had a positive association with waist circumference and waist to hip ratio. Race also had a positive correlation with waist to height ratio. In addition, race had a positive correlation with diastolic pressure. Both males and females had these same correlations, with the addition of race having positive correlation with BMI in males. There are debates concerning the correct anthropometric measurements relative to race and sex. Research has shown that WC and WHR are important tools in determining cardiometabolic diseases and cut-off values are different between race and gender (Gharipour et al., 2013, Bener et al., 2013, Lee et al., 2016 and Tran et al., 2011). Thus, using European standards for African-decent individuals is not ideal. Each country needs to validate their own cut-off points with regards to ethnicity to screen for visceral obesity and hence CMD. This is a particular concern in African countries and in South Africa, as black Africans are more prone to hypertension (high blood pressure), insulin resistance and low levels of HDL-C compared to white South Africans (Gaillard et al., 2010). These are all risk factors for CMD and are therefore of utmostimportance to address.

5.7 Limitations of the study

- The BDI scores were self-reported, which could have resulted in bias. Three participants had missing values for BDI scores as a result of it being self-report.
- More demographic data needed to be collected as factors such as financial status and other environmental factors influence depression.
- Habits such as smoking, and exercise should also have been recorded, even if it wasn't the research question, as these are modifiable risk factors of depression.
- The extended questions such as eating habits and sleeping habits were self-reported, so could also result it bias.
- A relatively small sample size may have resulted in no significance in some variables.
- There was a far higher female to male ratio.

5.8 Anticipated impact

Depression is a serious debilitating disease that is still not been taken seriously. Globally and nationally, depression is the cause for job loss, decreased social networking and increased suicide. This study aims to expose the prevalence of depression in students at the University of the Western Cape, as many students cope with depression and other mental illness daily. If statistics and numbers are shown, they can create an impact and awareness of the number of people suffering from this disease. Students, teachers, lecturers, and everyone part of the student community can speak about their sufferings and help each other, instead of not knowing where to turn to for help. This may spark a change in the student community toreach out to others and offer help. Furthermore, potential reasons may be discovered as to why participants are depressed (BMI, blood pressure or lipids). If any of these measurements are out of place or worrying, participants will be contacted and informed.

5.9 Dissemination of Results

- The data will be shared among peers and colleges as well as with lecturers and Professors in the form of a master's presentation at the end of the year.
- The information and data collected will be disseminated through the written scientific report and possibly a journal publication.
- Presentation of project at a national and international conference
- Participants will receive their results after the scientific write-up
- Participants may schedule a date and time with our Medical Dr and supervisor, who will explain all the results and make recommendations to campus psychologist or Tygerberg medical centre for severe depressive participants.

5.10 Recommendations

- An even ratio or relatively even ratio of males to females could result in more accurate results.
- A larger sample size may also yield in more significant results.
- Bias could be kept to a minimum by only having face-face interviews and not self-report surveys.
- The inclusion of more demographic data could enhance the literature and discussion.

Conclusion

In conclusion, it was seen that overall, no anthropometric measurements associated with depression. Furthermore, total cholesterol and LDL-C significantly associated negatively with depressive scores. Triglycerides associated significantly and positively with depressive scores. Overall, there were no significant associations between anyblood pressure measurements with depression, but systolic blood pressure did significantly and positively associate with MADRS depressive scores in males. There were also significant associations with regards to the secondary objectives, in which associations were madebetween the variables themselves (and not with depressive scores), as these variables are important factors in cardiometabolic disease.

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Appendices

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Appendix A: Information sheet

Project Title: The association of BMI and blood pressure measurements with depressive and quality of life scores in University students.

What is this study about? This is a research project being conducted by Dr Julèy De Smidt and Professor Oelofse from the University of the Western Cape in South Africa. We are inviting you to partake in this research project because you meet the set criterion for the population of interest and your participation will benefit yourself and others. The aim of this research project is to investigate whether Body Mass Index, blood pressure and lipid profiles are associated with depression in students aged 18 and above. Your participation will therefore be of great importance and much appreciated.

What will I be asked to do if I agree to participate? You will be asked to sign a consent form agreeing to participate in the study. You will be asked to complete a depressionquestionnaire (Montgomery-Asberg Depression Scale - MADRS) to determine if you qualify to participate. Should you be selected to participate (we will inform you) you will be asked toparticipate in a face-face consultation where we will re-do the depression questionnaire in an interview manner to avoid any discrepancies. Thereafter we will send you a Google form linkto complete the other depression questionnaire (Beck Depression Inventory (BDI) and lifestyle questions which will be self-reported. The study will be conducted at the University of the Western Cape, in the Life Science building. You will then be asked to give consent to proceed with the data collection. Anthropometric measurements (height, weight, waist circumference) as well as blood pressure and lipid measurements will be taken after the face- face consultation. The lipid measurement includes a finger-prick method, which we have gained ethical clearance for (ethics reference number: BM21/2/14).

Would my participation in this study be kept confidential? Your personal information will be kept confidential. To help protect your confidentiality, your real names will not be included in the data collection sheets (but represented in numbers/student number) and all information collected will be locked in cabinets and password protected computers. Your names will be represented as codes and only the researcher will have access to the information which links the code to your collected data. Furthermore, your identity will be kept confidential during data collection. Data collection sheets and audio tapes will be kept safely in a lockable filling cabinet and accessed only by the researcher. All raw data includingwritten documents will be destroyed after three months of the final dissertation being marked and graded. If we write a report or article about this research project, your identity will be protected.

What are the risks of this research? Risks from participating in this research study mainly include discomfort around providing private or sensitive information. There are no other known risks associated with participating in this research project. If any of the questions asked during the interview make you feel uncomfortable, you can refrain from answering it.

What are the benefits of this research? You may become aware of depressive symptoms you were unaware of before or confused about before. We can help you understand our symptoms and can also refer you to the campus doctor or the psychiatric facility at Tygerbergso may receive beneficial help. By participating in this research study, you will help identify the prevalence of depression in students. This will help open platforms for those students whoare struggling and those who are in need. This study will help raise awareness about how many students are being affected by this illness and how serious it is.

Do I have to be in this research, and may I stop participating at any time? Your involvement in this investigation is completely voluntary. You may choose to participate or to not partake at all. If you decide to participate in this research, you may stop participating at any time. If you decide not to participate in this study or if you stop participating at any time, you will not be penalized or lose any benefits to which you otherwise qualify.

Is any assistance available if I am negatively affected by participating in this study? If at any time of the study, you feel uncomfortable and need assistance, the researcher will refer you for counselling through a social welfare office in your area or to the counsellor on campus grounds. As stated above, the campus doctor will be the referral on-hand and we will also further refer the patients to Tygerberg Psychiatric Facility.

What if I have questions? If you have any questions about the research study itself, please contact Ammaarah Gamieldien on 0738771148 or 3568307@myuwc.ac.za, Dr Juley DeSmidt on 082 835 1243, jjdesmidt@gmail.com or Prof Andre Oelofse on 0835313545 or aoelofse@uwc.ac.za. Should you have any questions regarding this study and your rights as a research participant or if you wish to report any problems you have experienced related to the study, please contact any of the above mentioned.

This research has been approved by the University of the Western Cape's Senate Research Committee (Ethics Reference Number: BM21/2/14).

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Appendix B: Consent Form

Title of Research Project: The association of BMI and blood pressure measurements with depressive and quality of life scores in University Students

The study has been described to me in language that I understand, and I freely and willingly agree to participate. My questions about the study have been answered. I recognize that my identity will be always kept confidential and that I may withdraw from the study without giving a reason. I understand that I may do so at any time, and this will not negatively affect me in any way.

Participant's name ……**Participant's signature…….**

Date……

Should you have any questions concerning this study or wish to report any problems you have experienced related to the study, please contact the study coordinator:

Study Coordinator's Name: Dr Juley De Smidt

Tel: 021 9592182

Email: jdesmidt@uwc.ac.za

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Appendix C: Montgomery-Asberg Depression Rating Scale

Montgomery-Åsberg Depression Rating Scale (MADRS)

Instructions: The ratings should be based on a clinical interview moving from broadly phrased questions about symptoms to more detailed ones which allow a precise rating of severity. The rater must decide whether the rating lies on the defined scale steps (0, 2, 4, 6) or between them (1, 3, 5). It is important to remember that it is only rare occasions that a depressed patient is encountered who cannot be rated on the items in the scale. If definite answers cannot be elicited from the patients, all relevant clues as well as information from other sources should be used as a basis for the rating in line with customary clinical practice. This scale may be used for any time interval between ratings, be it weekly or otherwise, but this must be recorded.

1. Apparent Sadness: Representing despondency, gloom and despair, (more than just ordinary transient low spirits) reflected in speech, facial expression, and posture.

Rate on depth and inability to brighten up.

0 No sadness
1
2 Looks dispirited but does brighten up without difficulty
3
4 Appears sad and unhappy most of the time
5
6 Looks miserable all the time - Extremely despondent

2. Reported Sadness: Representing reports of depressed mood, regardless of whether it is reflected in appearance or not. Includes low spirits, despondency or feeling of being beyond help without hope.

Rate according to intensity, duration and the extent to which the mood is reported to be influenced by events.

0 Occasional sadness in keeping with the circumstances
1
2 Sad or low but brightens up without difficulty
3
4 Pervasive feelings of sadness or gloominess - The mood is still influenced by external circumstances
5
6 Continuous or unvarying sadness, misery or despondency

3. Inner Tension: Representing feelings of ill-defined discomfort, edginess, inner turmoil

mounting to either panic, dread or anguish.

Rate according to intensity, frequency, duration and the extent of reassurance calledfor.

0 Placid. Only reflecting inner tension
1
2 Occasional feelings of edginess and ill-defined discomfort
3
4 Continuous feelings of inner tension or intermittent panic which the patient can only master with some difficulty
5
6 Unrelenting dread or anguish. Overwhelming panic

4. Reduced Sleep: Representing the experience of reduced duration or depth of sleep compared to the subject's own normal pattern when well.

0 Sleeps as usual
1
2 Slight difficulty dropping off to sleep or slightly reduced light or fitful sleep
3
4 Sleep reduced or broken by at least two hours
5
6 Less than two or three hours sleep

5. Reduced Appetite: Representing the feeling of loss of appetite compared with when well.

Rate by loss of desire for food or the need to force oneself to eat.

0 Normal or increased appetite
1
2 Slightly reduced appetite
3
4 No appetite. Food is tasteless
5
6 Needs persuasion to eat

6. Concentration Difficulties: Representing difficulties in collecting one's thoughts mounting to incapacitating lack of concentration.

Rate according to intensity, frequency, and degree of incapacity produced.

0 No difficulties in concentrating.
1
2 Occasional difficulties in collecting one's thoughts.
3
4 Difficulties in concentrating and sustaining thought which reduces ability to read or hold a conversation.

7. Lassitude: Representing a difficulty getting started or slowness initiating and performing everyday activities.

0 Hardly no difficulty in getting started. No sluggishness.
1
2 Difficulties in starting activities.
3
4 Difficulties in starting simple routine activities which are carried out with effort.
5
6 Complete lassitude. Unable to do anything without help

8. Inability to Feel: Representing the subjective experience of reduced interest in the surroundings, or activities that normally give pleasure. The ability to react with adequate emotion to circumstances or people is reduced.

0 Normal interest in the surroundings and in other people.
1
2 Reduced ability to enjoy usual interest.
3
4 Loss of interest in surroundings. Loss of feelings for friends and acquaintances.
5
6 The experience of being emotionally paralyzed, inability to feel anger, grief or pleasure and a complete or even painful failure to feel for close relatives and friends

9. Pessimistic Thoughts: Representing thoughts of guilt. Inferiority, self-reproach, sinfulness, remorse and ruin.

0 No pessimistic thoughts.
1
2 Fluctuating ideas of failure, self-reproach or self-depreciation.
3
4 Persistent self-accusations, or definite but still rational ideas of guilt or sin. Increasingly pessimistic about the future.
5
6 Delusions of ruin, remorse or unredeemable sin. Self- accusations which are absurd and unshakable

10. Suicidal Thoughts: Representing the feeling that life is not worth living, that a natural death would be welcome, suicidal thoughts, and the preparations for suicide. Suicidal attempts should not in themselves influence the rating.

0 Enjoys life or takes it as it comes.

1
2 Weary of life. Only fleeting suicidal thoughts.
3
4 Probably better off dead. Suicidal thoughts are common, and suicide is considered as a possible solution, but without specific plans or intention.
5
6 Explicit plans for suicide when there is an opportunity. Active preparations for suicide

TOTAL:

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Appendix D: Beck's Depression Inventory

Beck's Depression Inventory This depression inventory can be self-scored. The scoring scale is at the end of the questionnaire.

Beck's Depression Inventory

1.

0 I do not feel sad.

1 I feel sad

2 I am sad all the time and I can't snap out of it.

3 I am so sad and unhappy that I can't stand it.

2.

0 I am not particularly discouraged about the future.

1 I feel discouraged about the future.

2 I feel I have nothing to look forward to.

3 I feel the future is hopeless and that things cannot improve.

3.

0 I do not feel like a failure.

1 I feel I have failed more than the average person.

2 As I look back on my life, all I can see is a lot of failures.

3 I feel I am a complete failure as a person.

4.

0 I get as much satisfaction out of things as I used to.

1 I don't enjoy things the way I used to.

2 I don't get real satisfaction out of anything anymore.

3 I am dissatisfied or bored with everything.

5.

0 I don't feel particularly guilty

1 I feel guilty a good part of the time.

2 I feel quite guilty most of the time.

3 I feel guilty all of the time.

6.

0 I don't feel I am being punished.

1 I feel I may be punished.

2 I expect to be punished.

3 I feel I am being punished.

7.

0 I don't feel disappointed in myself.

1 I am disappointed in myself.

2 I am disgusted with myself.

3 I hate myself.

8.

0 I don't feel I am any worse than anybody else.

1 I am critical of myself for my weaknesses or mistakes.

2 I blame myself all the time for my faults.

3 I blame myself for everything bad that happens.

9.

0 I don't have any thoughts of killing myself.

1 I have thoughts of killing myself, but I would not carry them out.

2 I would like to kill myself.

3 I would kill myself if I had the chance.

10.

0 I don't cry any more than usual.

1 I cry more now than I used to.

2 I cry all the time now.

3 I used to be able to cry, but now I can't cry even though I want to.

11.

0 I am no more irritated by things than I ever was.

1 I am slightly more irritated now than usual.

2 I am quite annoyed or irritated a good deal of the time.

3 I feel irritated all the time.

12.

0 I have not lost interest in other people.

1 I am less interested in other people than I used to be.

2 I have lost most of my interest in other people.

3 I have lost all of my interest in other people.

13.

0 I make decisions about as well as I ever could.

1 I put off making decisions more than I used to.

2 I have greater difficulty in making decisions more than I used to.

3 I can't make decisions at all anymore.

14.

0 I don't feel that I look any worse than I used to.

1 I am worried that I am looking old or unattractive.

2 I feel there are permanent changes in my appearance that make me look unattractive

3 I believe that I look ugly.

15.

0 I can work about as well as before.

1 It takes an extra effort to get started at doing something.

2 I have to push myself very hard to do anything.

3 I can't do any work at all.

16.

0 I can sleep as well as usual.

1 I don't sleep as well as I used to.

2 I wake up 1-2 hours earlier than usual and find it hard to get back to sleep.

3 I wake up several hours earlier than I used to and cannot get back to sleep.

17.

0 I don't get more tired than usual.

1 I get tired more easily than I used to.

2 I get tired from doing almost anything.

3 I am too tired to do anything.

18.

0 My appetite is no worse than usual.

1 My appetite is not as good as it used to be.

2 My appetite is much worse now.

3 I have no appetite at all anymore.

19.

0 I haven't lost much weight, if any, lately.

1 I have lost more than five pounds.

2 I have lost more than ten pounds.

3 I have lost more than fifteen pounds.

20.

0 I am no more worried about my health than usual.

1 I am worried about physical problems like aches, pains, upset stomach, or constipation.

2 I am very worried about physical problems and it's hard to think of much else.

3 I am so worried about my physical problems that I cannot think of anything else.

21.

0 I have not noticed any recent change in my interest in sex.

1 I am less interested in sex than I used to be.

2 I have almost no interest in sex.

3 I have lost interest in sex completely.

INTERPRETING THE BECK DEPRESSION INVENTORY

Now that you have completed the questionnaire, add up the score for each of the twenty-one questions by counting the number to the right of each question you marked. The highest possible total for the whole test would be sixty-three. This would mean you circled number three onall twenty-one questions. Since the lowest possible score for each question is zero, the lowest possible score for the test would be zero. This would mean you circles zero on each question.You can evaluate your depression according to the Table below.

Total Score	Levels of Depression
1-10	These ups and downs are considered normal11-16
	Mild mood disturbance
17-20	Borderline clinical depression
21-30	Moderate depression

31-40_____Severe depression over

40_____Extreme depression

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Appendix E: Data Collection sheet

Participant ID:
Age:
Gender:
Date

1) Anthropometric data

Anthropometric measurement	Measurements			
	Attempt 1	Attempt 2	Attempt 3	Average
Weight (kg)				
Height (cm)				
Waist circumference (cm)				
Hip circumference (cm)				

Index/Ratio	How it is calculated	Result
Body mass index (BMI)	Weight (kg)/height (cm)	
Waist-to-hip ratio (WHR)	Waist circumference (cm)/hip circumference (cm)	
Waist-to-height ratio (WtHR)	Waist circumference (cm)/height (cm)	

2) Clinical data

a. Blood pressure (BP)

Clinical measurement	Measurements			
	Attempt 1	Attempt 2	Attempt 3	Average
Systolic BP				
Diastolic BP				
Mean arterial pressure (MAP)				
Heart rate (HR)				

b. Lipid profile

Lipid profile	Result
Total cholesterol	
LDL	
HDL	
Triglycerides	
Total chol/HDL	

3) Depressive Scoring

Standardized questionnaire	Score	Severity
Montgomery-Asberg Depression Rating Scale (MADRS)		
Beck Depression Inventory (BDI)		

4) Inflammatory conditions

Inflammatory condition	Yes/No	Comment
Seasonal/chronic allergies		
Chronic urinary tract disease (UTI)		

Chronic cough		
Asthma		
Gout		
Type 1 diabetes		
Gastro-duodenal ulcer		
Rheumatoid arthritis		
Other		

Appendix F: Link to the questionnaire survey

Link to the questionnaire: https://forms.gle/LpGCbJvjBH8btz2X9

Appendix G: Ethical Clearance Certificate

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60 YEARS of hope, action & knowledge

6 September 2021

Ms A Gamaldien
Medical Biosciences
Faculty of Natural Sciences

Ethics Reference Number: BM21/2/14

Project Title: The association of BMI and blood pressure measurements with Depressive and Quality of Life Scores in University students

Approval Period: 6 September 2021 – 6 September 2024

I hereby certify that the Biomedical Science Research Ethics Committee of the University of the Western Cape approved the scientific methodology and ethics of the above mentioned research project.

Any amendments, extension or other modifications to the protocol must be submitted to the Ethics Committee for approval.

Please remember to submit a progress report annually by 30 November for the duration of the project.

Permission to conduct the study must be submitted to BMREC for record-keeping.

The Committee must be informed of any serious adverse event and/or termination of the study.

Ms Patricia Josias
Research Ethics Committee Officer
University of the Western Cape

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FROM HOPE TO ACTION THROUGH KNOWLEDGE.