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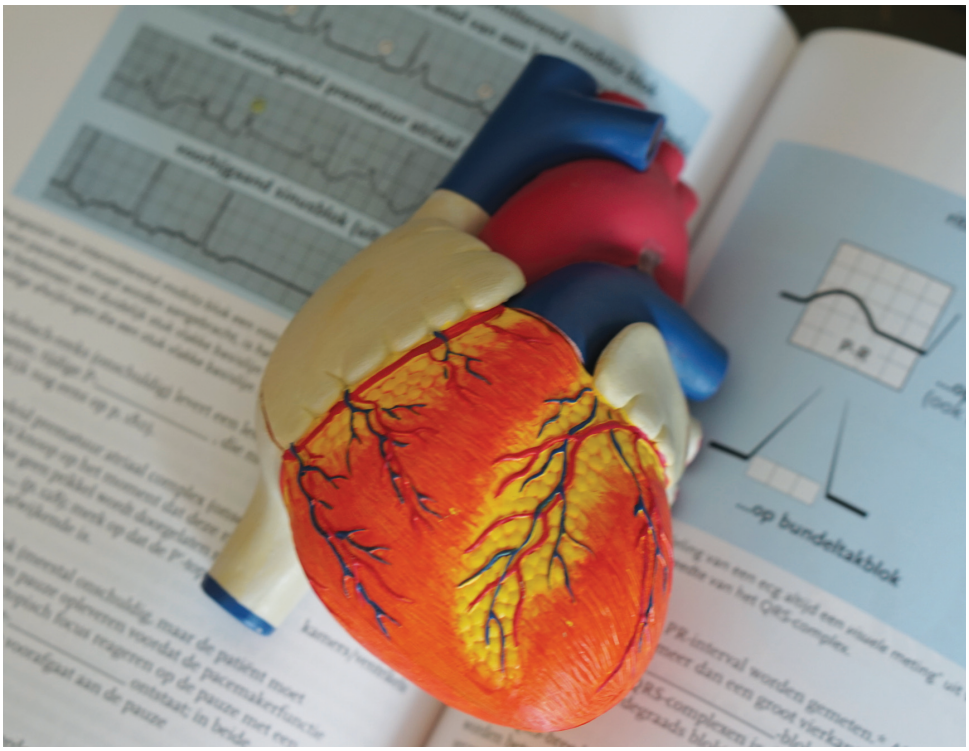


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# From the Editor's Desk

This issue deals with a range of factors related to non-communicable diseases (NCDs), from risk-factor identification and management to special groups that may be affected.

Anthony *et al*. (page 4) examined hypertensive disorders in pregnancy. They outline a clear method of classification, assessment and management. Other countries in Africa also have high levels of hypertensive disease in pregnancy; for example, a study in Botswana showed 22% of pregnancies with hypertensive disease.[1]

Agaba and co-workers (page 11) evaluated NCDs and risk factors among university employees in Nigeria. They used a modified version of the World Health Organisation's STEPwise programme.[2] This is a programme for surveillance and monitoring of NCDs, but interestingly, they included aspects of counselling on lifestyle as part of their study (test and treat). Striking findings were of high levels of inactivity and a poor diet in the group of university employees, indicating that further education and counselling are needed, even in this setting.

Mashiane *et al*., in the Ellisras Longitudinal Study 2017 (page 18), assessed dietary intake and body mass index (BMI). Important findings include the association between BMI and high cholesterol intake, female obesity, high dietary carbohydrate intake and the need for further education and guidance. Female obesity is a key health problem that needs to be addressed, both at the causation and consequences level. Studies examining the impact of obesity on reproductive health and as a risk factor for NCDs have been carried out, but the management is proving to be complicated and difficult.[3] Large epidemiological databases, such as the Ellisras Longitudinal Study 2017, are proving to be valuable in assessing disease burden, risk-factor identification and subsequent healthcare policy.

Ngango and Omole (page 22) demonstrate the high clustering of risk factors for NCDs in the South African primary healthcare setting. Low socio-economic status and risk for NCDs have been shown to be an international problem.[4] Mayosi *et al*. expressed this concern in a concise manner: 'Concerted action is needed to strengthen the district-based primary healthcare system, to integrate the care of chronic diseases and management of risk factors, to develop a national surveillance system, and to apply interventions of proven cost-effectiveness in the primary and secondary prevention of such diseases within populations and health services'.[5]

Sebati and co-workers (page 30) showed that in a group of young people studied in the Ellisras Longitudinal Study 2017, increased waist circumference was associated with hypertension. Obesity levels are of concern. Other studies in South Africa confirm this 'signal' of risk factors for NCDs in the youth,[6] and this is an area that public health officials need to take note of.

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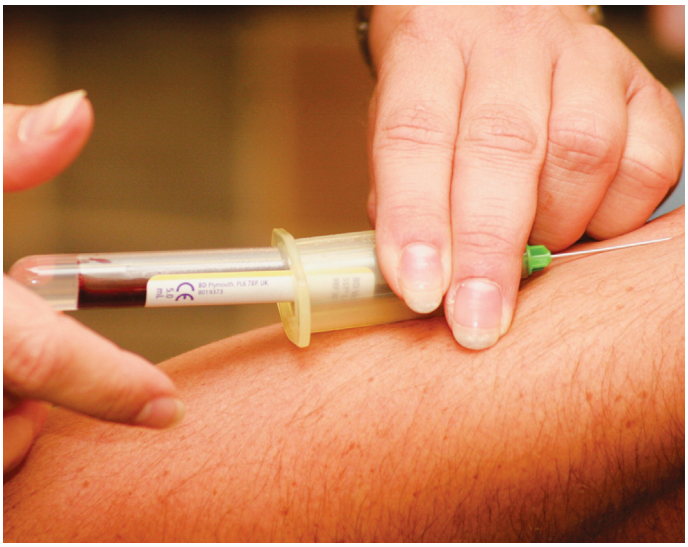

Juma and associates (page 35) assessed cardiovascular risk factors among people living with HIV in Kenya. They found high levels of pre-hypertension and total cholesterol, especially associated with nucleoside reverse-transcriptase inhibitor (NRTI)-based ART regimens. They point out the need to do cardiovascular risk assessments as part of routine management of HIV. Cardiovascular disease in HIV takes many forms.[7] As more patients survive with improved access to and management of HIV treatment, NCDs and risk of cardiovascular disease take over as the major clinical areas.[8]

Ellapen *et al*. (page 40) explain the correct use of exercise in the management of diabetes. Benefits and risks are outlined.

The insert under Drug Trends reveals new ways of protecting insulin from temperature fluctuations, which is a key management aspect to discuss with patients. South African temperatures can range from –5° to +40°C and with intermittent electricity supply and harsh working and sporting environments, this topic becomes a crucial aspect to consider.

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# Hypertensive disorders of pregnancy: what the physician needs to know

JOHN ANTHONY, ALBERTINO DAMASCENO, DIKE OJJII

## Abstract

**Hypertension developing during pregnancy may be caused by a variety of different pathophysiological mechanisms. The occurrence of proteinuric hypertension during the second half of pregnancy identifies a group of women whose hypertensive disorder is most likely to be caused by the pregnancy itself and for whom the risk of complications, including maternal mortality, is highest. Physicians identifying patients with hypertension in pregnancy need to discriminate between pre-eclampsia and other forms of hypertensive disease. Pre-eclamptic disease requires obstetric intervention before it will resolve and it must be managed in a multidisciplinary environment. The principles of diagnosis and management of these different entities are outlined in this review.**

**Keywords:** hypertention disorders, pregnancy

Hypertension during pregnancy is widespread, representing the most common medical complication of pregnancy and affecting 6–8% of gestations in the United States of America. Two hospital-based studies in sub-Saharan Africa have put the prevalence of this disorder at 11.5 and 26.5% of all deliveries, respectively.[1,2] There are four categories of hypertension in pregnancy, chronic hypertension, gestational hypertension, pre-eclampsia, and pre-eclampsia superimposed on chronic hypertension, as defined by the National High Blood Pressure Education Program Working Group in Pregnancy.

Hypertension during pregnancy is not only common but also associated with a risk of morbidity and mortality.[3,4] The risk of adverse outcomes during pregnancy is largely but not exclusively confined to those pregnant women diagnosed to have pre-eclampsia.[4,5] The separation of hypertension during pregnancy into pre-eclampsia or non-pre-eclamptic disease is a foundational consideration when determining the likely course of the disease, the necessary management and the probable outcome.[3]

Pre-eclampsia is uniquely manifest during pregnancy and is associated with a pathophysiological phenotype that encompasses placental disease, growth restriction of the foetus and the development of severe but reversible hypertension during pregnancy.[4,6,7] Chronic hypertension, regardless of the precise diagnosis, is not specifically associated with placental vascular disease or severe intra-uterine growth restriction and will not remit after delivery.8 The necessary level of surveillance, hospitalisation and the need for preterm delivery rests upon the distinction between these hypertensive diagnoses.[9]

In this review we discuss the different types of hypertension during pregnancy, and the physician evaluation, including physical examination and laboratory investigations of the hypertensive pregnant patient.

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## Pre-eclampsia

### *Epidemiology*

Pre-eclampsia affects one in 30 primigravid women and one in 60 women in their second or subsequent pregnancies.[10] Those who have suffered from the condition before are more likely to develop it in subsequent pregnancies (a one-in-seven risk) and women with underlying co-morbidity are also more likely to develop this complication of pregnancy. Specifically, women with chronic hypertension have a 25% risk of developing superimposed pre-eclampsia, and women with collagen vascular disease are also more prone to develop pre-eclampsia.[8,9,11] There is also a hereditary component, and obesity is strongly associated with the risk of developing the condition.[12]

Obstetric risk factors include an increasing risk of developing pre-eclampsia related to multiple and even higher-order multiple pregnancies. A large placenta, such as those seen in women with trophoblastic disease or various kinds of foetal aneuploidy, are also associated with an increased risk of developing pre-eclampsia. Other risk factors that have been identified as leading to an increased probability of pre-eclampsia developing during pregnancy include antiphospholipid antibody syndrome, chronic hypertension, chronic renal disease, a maternal age over 40 years, nulliparity, incidence of pre-eclampsia in a previous pregnancy and pre-gestational diabetes.

The highest incidence of pre-eclampsia is among women having their first baby, whereas the greater prevalence of the disease is in multiparous pregnant women. The disease is described as a condition of primigravidity but it is also, to some extent, associated with primipaternity.[10]

## Clinical phenotype

Pre-eclampsia is a syndrome characterised by the development of hypertension and proteinuria in the latter part of pregnancy, which then remits after delivery.[3] Pre-eclampsia is unlikely to be the cause of hypertension or proteinuria developing before the 20th week of pregnancy.

Hypertension is defined in different ways but the most widely accepted definition is the sustained elevation of diastolic blood

pressure above 90 mmHg over a period of four hours. Proteinuria is similarly defined in different ways but dipstick proteinuria of 1+ or more merits further investigation. The 24-hour urinary excretion of protein greater than 300 mg is regarded as being pathological.

Pre-eclampsia may present in an asymptomatic form. It may also develop acutely or progress to a phase of illness in which multi-organ disease becomes evident.[13] This may include the development of eclampsia, cerebrovascular haemorrhage leading to stroke, renal failure either in consequence of acute kidney injury or associated with a progressive decline in renal function, pulmonary oedema for a variety of reasons, liver injury in the form of the HELLP syndrome (haemolysis, elevated liver enzymes and low platelets) or obstetric haemorrhage caused by abruptio placentae (commonly associated with pre-eclampsia). Many of these complications of pre-eclampsia may be lifethreatening to the foetus and the pregnant woman.[14-16]

Characteristically, the delivery of the baby signals the onset of disease resolution, although the mother may continue to exhibit worsening disease for up to 24 hours after delivery. The hypertension associated with pre-eclampsia may take up to six weeks to resolve completely, even if the risk of fulminant disease abates within 24 hours of parturition.

## Pathology and pathophysiology

Pre-eclampsia is a disease of defective placentation.[6] The vascular adaptation in the vessels supplying blood to the placenta show signs of inadequate dilatation as well as evidence of lumina pathology, similar to atherosclerosis. The placenta itself is usually small and infarcted to a greater extent than is usually seen in normal pregnancy.

The evolution of the clinical phenotype follows these pathophysiological events in the placental bed. The precise mechanisms are not fully elucidated but some combination of systemic immune activation in response to an increasing maternal circulatory burden of trophoblastic tissue released from the ischaemic placenta combines with components of oxidative stress and an imbalance in the production of angiogenic and anti-angiogenic factors to give rise to changes in systemic vascular endothelial function.[17,18]

The volume-overloaded circulation of normal pregnancy is offset by endothelial-dependent vasodilatation to such an extent that normal pregnancy is characterised by falling blood pressure, despite the volume overload.[19] In pre-eclampsia, the endothelial mechanism is disrupted and hypertension based upon vasoconstriction ensues. The pattern of hypertension may evolve through stages where the increased systemic pressure may be partly based upon increased cardiac output, compensatory for the diminished perfusion of the placenta through narrow vessels in the placental bed.[20] The later evolution of the disease is due to defective vasoregulation and vasoconstriction associated with loss of intravascular volume through leaky capillaries and the onset of multi-organ ischaemia.[21-25]

Specific organs show patterns of ischaemic change, and haemorrhage with or without oedema. These include the brain, kidneys, placenta and liver.[26-28] In the brain, the oedema is seen in the watershed areas of perfusion of the occipital lobe and has been designated as 'posterior reversible encephalopathy syndrome'.[29] Large haemorrhages can arise from ruptured vessels, with consequent mass effects, including tonsillar herniation, leading to death. The liver shows periportal ischaemia and haemorrhage in women with the HELLP syndrome, whereas the kidneys show evidence of endotheliosis, associated in some cases with acute tubular and cortical ischaemic damage.[21,28]

The cardiovascular and pulmonary changes seen are those of pulmonary oedema in severe cases, usually without other overt signs of heart failure.[13,30]

## Risk of morbidity and mortality

There are two major causes of death among women with pre-eclampsia, cerebrovascular haemorrhage and pulmonary oedema, and each account for roughly half the number of deaths.[16] Other rarer causes include the rupture of a subcapsular haematoma, which may complicate the HELLP syndrome.

Cerebrovascular haemorrhage is related to severe hypertension.[31] The threshold above which this risk escalates is the mean arterial pressure above which the cerebral autoregulatory function fails. This is commonly considered to be 140 mmHg. It is unusual for women to develop such severe hypertension without associated seizure activity. The development of eclampsia leads to severe hypertension during seizure activity and it is the reason why the case fatality rate for eclampsia is cited as one in 50, whereas the overall case fatality rate of pre-eclampsia is set at one in 1 500.[14,32] The prevention of eclampsia is as important as the treatment of severe hypertension.

Pulmonary oedema may develop for different reasons. The iatrogenic administration of excessive amounts of intravenous fluids may lead to an absolute increase in preload, resulting directly in interstitial pulmonary oedema.[13,22] A very high systemic vascular resistance can also elevate the pulmonary capillary wedge pressure, leading to an increased risk of pulmonary oedema.[33] The left ventricular function may also be abnormal and commonly demonstrates some degree of diastolic dysfunction, although left ventricular systolic dysfunction is unusual.[22,23]

The loss of protein in the urine may lower the colloid osmotic pressure and contribute to development of the generalised oedema so characteristic of pre-eclampsia, with similar effects on the lungs. Changes in capillary permeability and the lymphatic drainage of the lungs all modulate the risk of pulmonary oedema in women with variable changes in vascular resistance and ventricular function. Consequently, the precise mechanism of pulmonary oedema cannot be simply attributed to heart failure in this condition.

## Management principles

Pre-eclampsia is not a condition that can be managed adequately outside a hospital environment.[4] The definitive management of pre-eclampsia is delivery.[4] Once manifest, the condition tends to worsen and it is unusual for delivery to be delayed by more than 10 to 14 days once the patient develops symptoms or signs of the condition. Because the foetus is at risk of impaired growth and likely to deliver prematurely, management needs to take place in an obstetric unit with access to the best available level of paediatric care. Any improvement in neonatal outcome can only be secured by minimising the risks of prematurity. This is accomplished by delaying delivery for as long as the mother's condition can be considered to be satisfactory.[34,35]

The development of symptoms, an uncontrollable spike in blood pressure or the evolution of defined organ dysfunction signal the onset of life-threatening disease, requiring that the focus of treatment shift from the neonatal outcome to protecting the interests of the mother. Delivery at this point is inevitable and the

neonate will need to be cared for in the best available circumstances. The second means of improving perinatal outcome revolve around the use of corticosteroids, given to the mother. These accelerate the maturation of the foetal lungs and lessen the likelihood of neonatal intraventricular haemorrhage in the newborn.[36] In addition to the necessity of effecting delivery by either induction of labour or caesarean section, the obstetrician plays a role in preventing complications.

The prevention of eclampsia is ensured by the use of magnesium sulphate, given as a continuous infusion or as intermittent intramuscular doses.[37,38] This has been shown to be effective in reducing the risk of developing eclamptic seizures (and recurrent seizures) without adversely sedating the foetus.

The mechanism of action is poorly understood and the use of magnesium sulphate needs to be weighed against potential risks. These include the development of toxicity, which is more common in women with renal failure. Toxicity leads to respiratory arrest, which can be reversed with intravenous calcium gluconate.

Women who are fitting should have their seizures aborted with intravenous benzodiazepines. Women who continue to fit despite treatment or those who are unable to protect their airway because of a low Glasgow coma scale need intubation and mechanical ventilation until the pregnancy is over and the mother's condition shows signs of improvement.[13,39]

Proper management of severe hypertension is always a priority. Drugs used to lower the blood pressure are a variety of agents, including direct-acting vasodilators (hydrallazine, dihydrallazine), calcium channel blockers (nifedipine), alphaand beta-blockers (labetalol), and combined arterial and venous vasodilators (nitroglycerine). Potent vasodilators such as sodium nitroprusside or diazoxide should not be used because they are associated with a risk of precipitous decline in blood pressure.

## Specific organ failure is managed according to specific protocols

- Eclampsia requires attention to seizure control as outlined above. Recurrent seizures may only be controllable by continuous infusion of propofol or diazepam; this usually requires intubation and ventilation for up to 24 hours after delivery has been effected. The co-morbidity associated with seizures needs individual management (see below); specific screening and treatment of aspiration pneumonia is important. Any focal neurological signs merit neuro-radiological investigation to exclude haemorrhage and infarction. The differential diagnosis of seizure activity also merits consideration and may extend to other possible diagnoses, including metabolic causes for seizure activity, thrombotic thrombocytopaenic purpura, systemic lupus erythematosis, cerebral venous thrombosis, malaria and amniotic fluid embolus.[40]
- Renal failure may be manifest on the basis of diminished preload together with peripheral, including renal, vasospasm. Acute renal injury may also cause oliguria and azotaemia. This is the consequence of ischaemia (due to pre-eclampsia or pre-eclampsia complicated by hypovolaemia caused by abruptio placentae) and haemoglobinuria. The principles of management are those of cautious intravascular volume expansion (no more than 300 ml of colloidal solution given as a bolus dose) and vasodilatation.[41] Renal failure that fails to respond to these measures should result in a policy of fluid restriction, management of actual or incipient hyperkalaemia and expectant management in anticipation of gradual recovery after delivery.[42] In the acute phase of the illness, dialysis may be necessary.
- Liver injury is associated with the HELLP syndrome. This condition needs to be distinguished from other causes of micro-angiopathic haemolytic anaemia as well as other causes of liver failure. The differential diagnosis therefore includes thrombotic thrombocytopenic purpura, acute fatty liver of pregnancy, auto-immune disease, malaria and sepsis. The hallmark of the HELLP syndrome is that it reverses after delivery, with the nadir of thrombocytopaenia occurring on the third day postpartum.[43] The management is obstetric, meaning delivery. Patients who do not exhibit the characteristic resolution of the thrombocytopaenia merit investigation for other causes of micro-angiopathic haemolytic anaemia. The only lethal complication of the HELLP syndrome is the development of a large subcapsular liver haematoma, which ruptures, causing massive intraperitoneal haemorrhage.[44] The liver injury itself and the elevated liver enzymes seen in HELLP syndrome are not associated with failure of hepatic synthetic function and do not usually lead to coagulopathy or hypoglycaemia. These features, if present, indicate an alternative diagnosis.
- Pulmonary oedema is the most difficult complication of severe pre-eclampsia in which to make a specific diagnosis.[30] The mechanisms of pulmonary oedema are outlined above and the differential diagnosis will include other causes of acute dyspnoea, commonly infection and embolus. Pulmonary oedema itself may be the consequence of pre-eclampsia, or pre-eclampsia complicating underlying illness. These illnesses may include valvular heart disease and ventricular dysfunction due to cardiomyopathy. Regardless of the cause, emergency management is usually the same, involving supportive management of oxygenation and various combinations of diuretic and vasodilator therapy with a view to reducing both afterload and preload. This is commonly accomplished by using direct-acting vasodilators, such as dihydrallazine, together with intravenous furosemide. The development of pulmonary oedema is a signal for investigation by means of radiology, ECG and echocardiography to try to ascertain as closely as possible what the underlying cause may be. In some circumstances, the acute management of critically ill women may be facilitated by the use of pulmonary artery catheters to directly measure haemodynamic variables.[45] Pulmonary oedema complicating pre-eclampsia is also an indication for immediate delivery, to begin reversing the underlying pathophysiology of pre-eclampsia.

## Postpartum management

Delivery of the pre-eclamptic pregnant woman will trigger reversal of the underlying disease. Generalised oedema begins to dissipate as the capillary leak reverses and the pregnancy preload is excreted. Commonly, 48 to 72 hours after delivery, the left ventricular preload may start to increase as the oedema resolves.[46] This is an appropriate time to facilitate a diuresis.

The hypertension itself may persist for up to six weeks after delivery, requiring management for this duration with diuretics and second-line agents. Whereas angiotensin converting enzyme (ACE) inhibitors are commonly used in non-pregnant hypertensives,

often calcium channel blockers are more rapidly effective in these circumstances and are a good choice of treatment for the limited period for which they will be required.

One of the most important aspects of managing the postpartum pre-eclamptic is that of counselling. Pre-eclampsia has been shown to be a marker of long-term risk. Specifically, there is an association between hyperinsulinaemia, dyslipidaemia and the risk of pre-eclampsia. These underlying metabolic disorders are also risk factors for early onset vascular disease (both coronary artery and cerebrovascular disease).[47] Consequently, women with early onset pre-eclampsia are at risk of vascular arterial disease in later life. Attention therefore needs to be paid to primary prevention of these conditions through regular screening, and treatment for metabolic disorders. The second long-term consequence of pre-eclampsia is that of an increased risk of renal failure.[48] This risk correlates with the number of pre-eclamptic pregnancies a woman may have and indicates a need to pay attention to aspects of care in later life that may have a renal protective effect, specifically the early and adequate treatment of hypertension.

## Chronic hypertension in pregnancy

Chronic hypertension during pregnancy may be divided into two groups: uncomplicated chronic hypertension and chronic hypertension with superimposed pre-eclampsia. The latter group requires management according to the principles outlined above, whereas the former requires out-patient care, often with an altered approach to therapeutic intervention. Chronic hypertension is defined as blood pressure of 140/90 mmHg or more on two occasions before 20 weeks of gestation or persisting beyond 12 weeks after delivery.

## Chronic hypertension with superimposed pre-eclampsia

The risk of developing superimposed pre-eclampsia is estimated to be between 10 and 25%.[49] The possibility of decreasing this risk merits consideration. The development of pre-eclampsia cannot be averted by controlling blood pressure and there is no therapy that has any major impact on the risk of developing superimposed pre-eclampsia. However, there is some evidence that the use of low-dose aspirin, given as a daily dose of 57 to 81 mg of aspirin, may reduce the risk of pre-eclampsia developing in about 10% of women who are at risk of the disease.[50] It is not clear why aspirin is effective, and initial theories related to altered prostanoid metabolism have been discounted, with more recent speculation focused on the possible interaction between aspirin and the production of pro-inflammatory cytokines.[51] Aspirin given in this dose is safe and has no effect on the foetus. Despite the modest effect on the incidence of the disease, it remains recommended therapy in women who are at risk.

The second strategy used to reduce the occurrence of pre-eclampsia is based on the prophylactic administration of large doses of oral calcium. Meta-analysis of the studies conducted to date indicate that calcium administered in doses of up to one gram three times a day may significantly reduce the occurrence of pre-eclampsia and may also reduce the development of severe hypertension.[52] The criticism of this data arises from the observation that the two single largest studies in the meta-analysis failed to reach statistical significance. Despite these reservations, calcium supplementation is widely accepted practice during pregnancy where there is a suspected risk of pre-eclampsia.

Interventions that are not of benefit in preventing pre-eclampsia include bedrest, the use of anti-oxidant vitamins and antihypertensive therapy itself.

Given the imperfect prophylactic measures aimed at preventing pre-eclampsia, care of pregnant women with chronic hypertension requires appropriate precautions to ensure that the development of superimposed disease is detected early in its development because of the attendant risks of foetal and maternal morbidity and mortality. Knowing who will develop superimposed pre-eclampsia before it becomes clinically manifest would be useful information.

The clinical phenotype of pre-eclampsia arises from changes at the level of the foetoplacental unit and any early signs of intra-uterine growth restriction or abnormal uterine artery Doppler velocimetry may precede the onset of the clinical disease.[49] The hallmark of superimposed pre-eclampsia is, however, the development of proteinuria. The difficulty with this is knowing when the proteinuria is a consequence of underlying pre-eclampsia rather than due to renal disease caused by longstanding hypertension or a priori renal disease with secondary hypertension (in many communities HIV-associated nephritis may be a major differential diagnosis).

This distinction may not be easily made on a clinical basis and where a diagnosis of pre-eclampsia enters the differential diagnosis, the patient deserves in-patient care and management for presumptive pre-eclampsia until an alternative diagnosis can be made. The natural history of pre-eclampsia sometimes facilitates the distinction between pre-eclampsia and renal disease as a cause for proteinuria because pre-eclampsia tends to worsen as the pregnancy continues, whereas the chronically hypertensive patient has an indolent condition that changes little with the passage of time.

The recent interest in biomarkers may provide an alternative way of diagnosing which hypertensive conditions have a placental origin. Angiogenic and anti-angiogenic factors [placental growth factor and the soluble receptor for vascular endothelial growth factor (sFlt)] have been shown to be good predictors of placental disease and may provide a ready means of discriminating between various types of hypertensive disease in pregnancy, specifically identifying those most at risk of adverse outcome.[53]

## Uncomplicated chronic hypertension

Uncomplicated chronic hypertension does not usually affect the pregnancy outcome to any significant degree. The drugs used to treat hypertension outside pregnancy may need to be revised and alternatives introduced in order to protect the foetus.

Physiological changes during pregnancy have an impact on chronically hypertensive women as well. Specifically, they will vasodilate during the second trimester, leading to a fall in blood pressure and a reduction in the requirement for treatment at this point in the pregnancy. As the volume expansion during pregnancy continues and peaks at about 32 weeks' gestation, the need for treatment may increase again. The goals of treatment also may need to be revised during pregnancy.

Outside pregnancy, the aim of treatment is prevention of end-organ damage to the heart, vasculature and kidneys. The use of diuretics with ACE inhibitors is common and the goal of therapy is normotensive blood pressure. This strategy does not apply during pregnancy because the drugs may harm the foetus, and placental perfusion (in theory) may be adversely affected by antihypertensive drugs that diminish perfusion pressure.

Diuretics, although used to treat cardiac conditions during

pregnancy, are generally held to be contra-indicated in the management of chronic hypertension during pregnancy because pregnancy relies upon volume expansion to secure an accelerated rate of delivery of oxygenated blood to the peripheral tissues, including the placental bed. ACE inhibitors are also contra-indicated because they may interfere with the physiological regulation of uterine blood flow through local uterine mechanisms. More seriously, they are associated with neonatal renal failure in children of women treated with them during pregnancy. Of the other categories of antihypertensive drugs, beta-blockers are also relatively contra-indicated, being considered to be an independent risk factor for the development of intra-uterine growth restriction.[54]

Antihypertensive therapy during pregnancy in chronically hypertensive women is usually secured through the use of alphamethyldopa or calcium channel blockers. The aim of treatment is to reduce the occurrence of severe hypertension to safer levels of blood pressure. Practically, the threshold for introducing treatment is a sustained increase in blood pressure to above 160/110 mmHg to levels below this without seeking to reduce the pressure to normotensive levels.

The complications of chronic hypertension during pregnancy may extend to various forms of cardiac decompensation, depending on the severity of the condition. Hence, hypertensive cardiomyopathy is rarely seen in relatively young women with chronic hypertension, although it may develop and can give rise to maternal mortality.[55] More commonly, diastolic dysfunction caused by changes in left ventricular morphology may result in the onset of increasing dyspnoea in the third trimester as the volume expansion peaks out. Patients in this category are otherwise well, without any signs of superimposed pre-eclampsia. This is one circumstance where diuretic therapy may result in rapid clinical improvement and resolution of symptoms that will allow the pregnancy to continue to term.

Obstetric intervention is not commonly required in chronically hypertensive women. However, some mild degree of foetal growth restriction may be present and the risk of superimposed pre-eclampsia cannot be excluded with absolute certainty. Consequently, induction of labour is usually recommended for women who do not labour spontaneously before 40 weeks' gestation.

## Latent hypertension

Pregnancy may render overt hypertension that is not yet clinically manifest outside of pregnancy. Women who have a strong familial history of hypertension, whose genetic predisposition will manifest as essential hypertension in later life, may become hypertensive during pregnancy. The mechanism is thought to be related to subnormal pregnancy vasodilatation in vessels, with a hereditary defect in vasoregulation. In this circumstance, the increased intravascular volume of pregnancy cannot be accommodated by adequate vasodilatation, with a rise in blood pressure developing in the late second to third trimester of pregnancy.[56]

This condition should be managed according to the same principles as those outlined for women with chronic hypertension. The outcome of the pregnancy is usually unaffected and the only consideration might be the need for induction of labour in women not yet delivered by 40 weeks' gestation.

## Physiological hypertension

Hypertension does not always indicate disease. Pregnancy is characterised by massive plasma volume expansion, and the cardiovascular adaptation needed to accommodate this increased intravascular volume is that of equally massive peripheral vasodilatation. The net consequence of this is a fall in blood pressure during the second trimester, with increasing levels of blood pressure closer to term. The entire adaptation is mediated by the placenta, and the adequacy of the pregnant physiological change depends on the amount of biochemically active trophoblast in the uterus. Hence women with multiple pregnancies or those who have singleton pregnancies with a large placenta will have a greater degree of volume expansion than those with a smaller placental mass. The consequences of this may be a supraphysiological increase in plasma volume that exceeds the degree of compensatory vasodilatation close to term. These individuals have normal pregnancies in every respect, with normally grown babies and no other signs of pre-eclampsia. This is not a condition requiring treatment or intervention and should be recognised as a variant of normal.[3]

The difficulty of managing these patients lies in being certain that the distinction can be safely made between physiological hypertension and pre-eclampsia. For this reason, many of these women would be allowed to continue to term but induction of labour would be justified at 40 weeks' gestation

## General evaluation of patients with hypertensive disorder of pregnancy

Determining whether high blood pressure identified during pregnancy is due to pre-eclampsia or chronic hypertension is sometimes a challenge to the physician, especially if there are no recorded blood pressures available from the first half of the gestation. Clinical characteristics obtained through a good history, physical examination and some laboratory investigations may be used to help clarify the diagnosis.

### *Relevant history the physician must take*

The time of detection of hypertension is very important. Hypertension occurring before 20 weeks' gestation is almost always due to chronic hypertension, while new-onset hypertension after 20 weeks' gestation should lead to a suspicion of gestational hypertension. Worsening hypertension after 20 weeks of gestation should lead to careful evaluation for the manifestations of pre-eclampsia.

Patients with pre-eclampsia may describe new-onset headache that is frontal, throbbing or similar to migraine headache. They may also have visual disturbances, including scintillations and scotoma, which has been linked to cerebral vasospasm. Gastrointestinal complaints, such as epigastric pain, may be moderate to severe in intensity and due to hepatic swelling and inflammation, with stretch of the liver capsule. Rapidly increasing or non-dependant oedema may be a symptom of developing pre-eclampsia. In addition, rapid weight gain as a result of oedema due to capillary leak, as well as renal sodium and fluid retention could be a pointer to pre-eclampsia. New-onset seizures in pregnancy suggest pre-eclampsia–eclampsia, but primary neurological disorders must always be excluded.

### *Signs the physician must look out for*

Pre-eclampsia is a multi-systemic disease with various physical signs. Oedema can be seen in non-dependent areas such as the face and hands, apart from the dependent areas. Maternal systolic blood pressure above 160 mmHg or diastolic blood pressure above 110 mmHg can occur and denote severe disease.

In measuring the blood pressure, women should be made

to sit quietly for five to 10 minutes before each blood pressure measurement, and blood pressure should be measured in lateral recumbency with the cuff at the level of the heart. Korotokoff sounds I and V should be used to define the systolic and diastolic blood pressure, respectively. In about 5% of pregnant women, an exaggerated gap exists between the fourth and fifth Korotokoff sounds with the fifth sound approaching zero. In this type of case, the fourth sound may more closely approximate the true diastolic blood pressure.

Signs of secondary hypertension such as buffalo hump, wide purple abdominal striae suggesting glucocorticoid excess, systolic bruit heard over the abdomen or in the flanks suggesting renal artery stenosis, and radio-femoral delay or diminished pulses in the lower versus upper extremities suggesting aortic co-arctation should be looked for. The presence of a fourth heart sound on auscultation is not a normal finding in pregnancy and may suggest left ventricular hypertrophy from chronic hypertension. Carotid bruits may also reflect atherosclerotic disease due to longstanding hypertension. In addition, retinal changes of chronic hypertension may be noted. Retinal vasospasm and retinal oedema, which may manifest as severely impaired vision, generally reflects pre-eclampsia.

In pre-eclampsia right upper-quadrant abdominal tenderness stemming from hepatic swelling and capsular stretch may be seen. Although brisk or hyperactive reflexes are common during pregnancy, clonus is a sign of neuromuscular irritability that usually reflects severe pre-eclampsia.

### *Laboratory investigations the physician must order*

Laboratory investigations to evaluate chronic hypertension include testing for target-organ damage, and to exclude secondary causes of hypertension and co-morbid factors. For chronic hypertension in the first trimester, it is very useful to obtain a full blood count, electrolyte, urea and creatinine levels, liver enzyme concentrations and testing for proteinuria. These serve as baseline values to be referred to later in the pregnancy if there is a concern regarding superimposed pre-eclampsia.

Serum lipids usually increase during pregnancy and therefore measurement should be deferred until the postpartum period. Also, the increase in endogenous corticosteroids levels during normal pregnancy makes it difficult to evaluate for secondary hypertension due to adrenal corticosteroid excess.

Useful blood tests when evaluating eclampsia and pre-eclampsia include urinalysis, a full blood count, serum electrolyte levels, urea and creatinine 24-hour urinary protein excretion, and serum uric acid, liver enzyme and bilirubin levels.

## Follow up

The long-term implications of having a pregnancy complicated by pre-eclampsia or hypertension have been highlighted above. It is important that pregnant women with hypertensive disease be given every opportunity to attend appropriate follow-up care in order to prevent long-term premature morbidity and mortality.

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# $HbA_{1c}$ test 'highly unreliable' at diagnosing diabetes

The haemoglobin $A_{1c}$ blood test is 'highly unreliable' at diagnosing diabetes and tends to underestimate the prevalence of the disease, according to a study presented at ENDO 2019, the Endocrine Society's annual meeting in New Orleans. 'Based on our findings, $HbA_{1c}$ should not be solely used to determine the prevalence of diabetes,' said lead researcher Dr Maria Mercedes Chang Villacreses, of City of Hope's Diabetes and Metabolism Research Institute in Duarte, California. 'It should be used in conjunction with the oral glucose test for increased accuracy.'

The $HbA_{1c}$ is a test that shows the average level of blood sugar over the past two to three months. People who have diabetes usually have this test to see whether their blood sugar levels have been staying within a target range. This test is also used to diagnose type 1 and type 2 diabetes. It is often used to diagnose diabetes because no fasting or any preparation is required.

A glucose tolerance test, also known as the oral glucose tolerance test, measures the body's response to sugar (glucose). In this test, a person's blood is taken after an overnight fast, and then again two hours after they drink a sugary drink. The glucose tolerance test can be used to screen for type 2 diabetes.

The study included 9 000 adults without a diabetes diagnosis. The participants got both a $HbA_{1c}$ and an oral glucose tolerance test, and the researchers compared the results. The researchers found the $HbA_{1c}$ test didn't catch 73% of diabetes cases that were detected by the oral glucose test. 'The $HbA_{1c}$ test said these people had normal glucose levels when they didn't,' Chang Villacreses said.

The researchers also found race and ethnicity had a significant impact on the accuracy of $HbA_{1c}$. It was more likely to detect abnormal glucose levels in non-Hispanic whites than in non-Hispanic blacks or Hispanics.

'Our results indicated that the prevalence of diabetes and normal glucose tolerance defined solely by $HbA_{1c}$ is highly unreliable, with a significant tendency for underestimation of the prevalence of diabetes and overestimation of normal glucose tolerance,' Chang Villacreses said.

*Source: Medical Brief 2019*

# A survey of non-communicable diseases and their risk factors among university employees: a single institutional study

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## Abstract

***Background:*** **The incidence of non-communicable diseases (NCDs) is rising globally, with its attendant morbidity and mortality, especially in developing countries. This study evaluated the prevalence of NCDs and their risk factors among members of a university community.**

***Methods:*** **All employees of the university were invited to the University health clinic for screening, using the World Health Organisation's STEPwise approach to NCDs.**

***Results:*** **A total of 883 (521; 59.0% males) employees with a mean age of 44 ± 10 years were studied. The median (IQR) number of NCD risk factors was three (two to three) per participant. The most common NCD risk factors were inadequate intake of fruit and vegetables (94.6%; 95% CI: 92.8–95.9), physical inactivity (77.8%; 95% CI: 74.9–80.5%) and dyslipidaemia (51.8%; 95% CI: 48.4–51.6%). Others included obesity (26.7%; 95% CI: 23.9–29.8%), alcohol use (24.0%; 95% CI: 21.3–27.0%) and cigarette smoking (2.9%; 95% CI: 2.0–4.3). Hypertension was the most common NCD (48.5%; 95% CI: 45.1–51.8%), followed by chronic kidney disease (13.6%; 95% CI: 11.4–16.1) and diabetes mellitus (8.0%; 95% CI: 6.4–10.1). There was no gender-specific difference in the prevalence of NCDs.**

***Conclusion:*** **This study identified that NCDs and their modifiable risk factors are highly prevalent in this community. Workplace policy to support the adoption of healthy living is needed.**

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The incidence of non-communicable diseases (NCDs) is rising globally, with its attendant morbidity and mortality. NCDs (particularly cardiovascular disease, diabetes and cancers) were responsible for 38 million (68%) of the world's 56 million deaths in 2012.[1] Studies have shown that early detection and timely intervention can prevent further morbidity and ultimately prolong life. Additionally, some risk factors for these diseases, when identified, can be modified, thus preventing their onset and progression. Developing countries are currently witnessing an epidemic transition from communicable diseases to non-communicable diseases.[1] Many individuals in these countries are caught in this 'epidemic transition of illnesses' as a result of lifestyle changes.

In Nigeria, the common NCDs include cardiovascular disease, hypertension, diabetes and cancers.[2] Many studies have documented the rising prevalence of NCDs among the general population in Nigeria. Hypertension is said to affect 25 to 48% of the adult population, while nearly 10% are diabetic,[1,6] and the incidence of cancer is on the increase.[3,4]

Recently, attention has focused on special populations, such as healthcare providers, civil servants and bankers, as they are thought to be among the relatively affluent in the community.[5-7] University employees over time have also become affluent (personal communication) and therefore are also likely to be at risk of NCDs due to changes in lifestyle and increasing urbanisation. However, very few studies have addressed NCDs among university employees in Nigeria.[8,9] The magnitude of NCDs and their risk factors in this subset of the population therefore largely remains unknown.

We embarked on this cross-sectional study to describe the prevalence of selected NCDs and their risk factors among the staff members of a university in north-central Nigeria. We also used this project to sensitise the participants on NCDs, as workplace interventions have been found to lead to health promotion.[10]

## Methods

A cross-sectional study of adults, aged 18 years and over employed in the University of Jos, was conducted over a fourmonth period (February to June 2014). The study was resident at the university health centre.

At the end of July 2010, the University workforce comprised a total of 2 603 people (1 793 senior and 810 junior staff). The minimum sample size (380) was calculated from the Kish formula,[11] using the prevalence of hypertension (as this is the NCD with the highest prevalence) and a precision of 5%.

Sensitisation of the university staff members was carried out using invitation letters through the various directorate heads, announcements on the university FM radio station, and banners placed at strategic places such as the entrances and exits of the university and the health clinic two months prior to and during the study period.

All employees of the university who subsequently presented to the university health clinic during the study period were recruited into the study. Pregnant and menstruating women were excluded from the study as anthropometric measurements and urine testing for abnormalities would not be useable.

The Human Research and Ethics Committee of the Jos University Teaching Hospital approved the study. All participants gave written informed consent before participation.

All participants had the opportunity to be counselled on healthy lifestyles, and participants found to have NCDs were referred for appropriate care. All the participants were evaluated using a modified version of the World Health Organisation (WHO) STEPwise approach to non-communicable disease.[12]

STEP 1 entailed history taking, looking particularly for risk factors for NCDs and the lifestyle of the subjects.

STEP 2 involved a physical examination in which the height and weight were measured using an electronic weighing scale, stadiometer and non-stretch tape measure, respectively. The body mass index (BMI) was calculated from the Quetelet index.[13] Blood pressure was measured using the OMRON digital sphygmomanometer.

STEP 3 involved obtaining blood samples for casual plasma glucose, serum creatinine, total cholesterol and high-density lipoprotein cholesterol levels, and urine testing for proteinuria and haematuria. Casual plasma glucose (CPG) level was estimated using the glucose oxidase method. Serum creatinine was assayed using the kinetic enzymatic method, and estimated glomerular filtration rate (eGFR) from the measured serum creatinine level using the CKD-EPI calculator.[14] The laboratory analyses of the tests were carried out at the commercial laboratory of APIN, Jos University Teaching Hospital, Jos.

Generalised obesity, hypertension, diabetes mellitus and dyslipidaemia were defined according to internationally accepted guidelines.[13,15-17] Chronic kidney disease (CKD) was regarded as the presence of proteinuria using urine dipsticks and/or eGFR < 60 ml/min/1.73 m$^2$.[18]

### *Statistical analysis*

Data obtained were analysed using the Epi Info 7 statistical software (CDC, Atlanta, GA). Means ± SD were used to describe normally distributed continuous variables, and proportions for categorical variables. Median with range was used to describe non-normally distributed continuous variables. The Student's *t*-test was used to compare group means and the chi-squared test to compare proportions. The Fisher exact test was used when cells contained less than five observations. The non-parametric Mann–Whitney U-test was used to compare non-normally distributed continuous variables. A *p*-value < 0.05 was considered significant.

## Results

A total of 883 (521; 59.0% males) employees with a slight predominance of junior-cadre workers participated in the study (Table 1). The majority were between 31 and 60 years old with a mean age of 44 ± 10 years. Women were older than the men and half had completed tertiary level education. The majority (80.5%) were married, with a median monthly household income of US$400 equivalent (US$1:00 exchanged for N150:00 as at the time of the study).

The median (IQR) number of NCD risk factors was three (two to three) per participant. The most common NCD risk factors were inadequate intake of fruit and vegetables (94.6%; 95% CI: 92.8–95.9), physical inactivity (77.8%; 95% CI: 74.9– 80.5%) and dyslipidaemia (51.8%; 95% CI: 48.4–51.6%). Details of NCD risk factors by sociodemographic variables are shown in Table 2.

No participant admitted to passive (second-hand) smoking at home or in the work environment and none used smokeless tobacco. As shown in Fig. 1, tobacco use (Fig. 1A), obesity and dyslipidaemia (Fig. 1B) increased with age.

A low intake of fruit and vegetables was common in participants with a formal education (Fig. 1C), as were physical inactivity, obesity and dyslipidaemia (Fig. 1D), compared to those without formal education. Fig. 1F shows that physical inactivity and dyslipidaemia increased with increasing household income.

Hypertension was the most common NCD, being present in nearly half the participants (48.5%; 95% CI: 45.1–51.8%), as

**Table 1.** Characteristics of 883 staff members of the University of Jos evaluated for select non-communicable diseases between February and June 2014

| Variable | Total (*n* = 883) | Males (*n* = 521) | Females (*n* = 362) | *p*-value |
|---|---|---|---|---|
| Mean age, years | 44 ± 10 | 43 ± 10 | 45 ± 9 | 0.002 |
| Age group, years, *n* (%)* | | | | |
| < 20 | 3 (0.3) | 2 (0.4) | 1 (0.3) | < 0.0001 |
| 21–30 | 83 (9.4) | 61 (11.7) | 22 (6.1) | |
| 31–40 | 257 (29.1) | 166 (31.9) | 91 (25.1) | |
| 41–50 | 294 (33.3) | 155 (29.8) | 139 (38.4) | |
| 51–60 | 215 (24.3) | 115 (22.1) | 100 (27.6) | |
| > 60 | 31 (3.5) | 22 (4.2) | 9 (2.5) | |
| Married (*n* = 878); *n* (%) | 707 (80.5) | 437 (84.2) | 270 (75.2) | < 0.0001 |
| Tertiary education completed (*n* = 876); *n* (%) | 440 (50.2) | 243 (46.9) | 197 (55.0) | 0.02 |
| Junior staff (*n* = 843); *n* (%) | 466 (55.3) | 319 (63.0) | 147 (43.6) | < 0.0001 |
| Monthly income, USD, median | 400 | 333.33 | 466.66 | < 0.0001 |
| BMI (kg/m$^2$) | 27.2 ± 5.1 | 25.1 ± 3.5 | 30.2 ± 5.7 | < 0.0001 |
| SBP (mmHg) | 129 ± 19 | 130 ± 19 | 127 ± 20 | 0.06 |
| DBP (mmHg) | 79 ± 12 | 79 ± 12 | 80 ± 11 | 0.4 |
| CPG, median (mg/dl) [mmol/l] | 85.0 [4.72] | 85.0 [4.72] | 86.0 [4.77] | 0.10 |
| Proteinuria (*n* = 883) (%) | 116 (13.2) | 72 (13.8) | 44 (12.2) | 1.15 |
| Serum creatinine (mmol/l) | 74.5 ± 19.3 | 81.8 ± 19.7 | 64.0 ± 13.1 | < 0.0001 |
| eGFR (ml/min/1.73m$^2$) | 114.2 ± 20.5 | 115.1 ± 20.7 | 113.1 ± 20.2 | 0.15 |
| Reduced eGFR | 4 (0.4) | 2 (0.4) | 2 (0.5) | 0.69 |
| TC (mg/dl) [mmol/l] | 193.4 ± 43.9 [5.01 ± 1.14] | 201.4 ± 46.2 [5.22 ± 1.20] | 187.9 ± 41.4 [4.87 ± 1.07] | < 0.0001 |
| HDL-C (mg/dl) [mmol/l] | 56.6 ± 16.4 [1.47 ± 0.42] | 60.7 ± 16.5 [1.57 ± 0.43] | 53.8 ± 15.7 [1.39 ± 0.41] | < 0.0001 |

*Fisher exact test; USD: United States Dollars; BMI: body mass index; SBP: systolic blood pressure; DBP: diastolic blood pressure; CPG: casual plasma glucose; eGFR: estimated glomerular filtration rate; TC: total cholesterol; HDL-C: high-density lipoprotein cholesterol.

**Table 2.** Sociodemographic characteristics and distribution of non-communicable diseases and their risk factors among 883 staff members of the University of Jos, Nigeria

| Variable | Tobacco % (95% CI) | Alcohol % (95% CI) | Diet % (95% CI) | Physical inactivity % (95% CI) | Obesity % (95% CI) | Dyslipidaemia % (95% CI) | HPTN % (95% CI) | DM % (95% CI) | CKD % (95% CI) |
|---|---|---|---|---|---|---|---|---|---|
| All | 2.9 (2.0–4.3) | 24.0 (21.3–27.0) | 94.6 (92.8–95.9) | 77.8 (74.9–80.5) | 26.7 (23.9–29.8) | 51.8 (48.4–51.6) | 48.5 (45.1–51.8) | 8.0 (6.4–10.1) | 13.6 (11.4–16.1) |
| Gender | | | | | | | | | |
| Male | 4.8 (3.2–7.1) | 33.8 (29.8–38.0) | 94.6 (92.2–96.3) | 72.9 (68.9–76.7) | 10.6 (8.1–13.6) | 48.8 (44.4–53.1) | 43.0 (38.7–47.4) | 6.9 (5.0–9.5) | 14.2 (11.4–17.6) |
| Female | 0.3 (0.0–1.8) | 9.9 (7.2–13.6) | 94.5 (91.5–96.5) | 84.8 (80.7–88.3) | 50.0 (44.7–55.3) | 55.0 (39.8–50.3) | 56.4 (51.1–61.5) | 9.7 (6.9–13.3) | 12.7 (9.5–16.7) |
| Marital status | | | | | | | | | |
| Married | 3.0 (1.9-4.6) | 24.3 (21.2-27.7) | 94.6 (92.6-96.1) | 79.5 (76.3-82.4) | 27.0 (23.8-30.5) | 53.2 (49.4-56.9) | 49.4 (45.6-53.1) | 8.8 (6.8-11.2) | 13.0 (10.7-15.8) |
| Unmarried | 2.8 (0.9–6.5) | 22.7 (16.8–29.6) | 94.3 (89.8–97.2) | 71.0 (63.7–77.6) | 25.6 (19.3–32.7) | 46.0 (38.5–53.7) | 44.9 (37.4–52.6) | 5.1 (2.4–9.5) | 15.9 (10.8–22.2) |
| Age group, years | | | | | | | | | |
| < 20 | 0.0 (0.0–70.8) | 0.0 (0.0–70.8) | 100.0 (0.0–29.2) | 100.0 (0.0–29.2) | 0.0 (0.0–70.8) | 33.3 (0.8–90.6) | 33.3 (0.8–90.6) | 0.0 (0.0–70.8) | 0.0 (0.0–70.8) |
| 21–30 | 7.2 (2.7–15.1) | 7.2 (2.7–15.1) | 88.0 (79.0–94.1) | 73.5 (62.7–82.6) | 7.2 (2.7–15.1) | 36.1 (25.9–47.7) | 14.5 (7.7–23.9) | 2.4 (0.3–8.4) | 7.2 (2.7–15.1) |
| 31–40 | 3.1 (1.4–6.0) | 22.2 (17.3–27.8) | 94.9 (91.5–97.3) | 69.3 (63.2–74.8) | 22.2 (17.3–27.8) | 42.4 (36.3–48.7) | 36.2 (30.3–42.4) | 3.9 (1.9–7.0) | 15.6 (11.4–20.6) |
| 41–50 | 2.0 (0.8–4.4) | 29.9 (24.8–35.3) | 96.9 (93.8–98.4) | 80.6 (75.6–85.0) | 29.9 (24.8–35.5) | 53.4 (40.8–52.5) | 49.0 (43.1–54.8) | 5.8 (3.4–9.1) | 12.6 (9.0–16.9) |
| 51–60 | 2.3 (0.8–5.3) | 34.9 (28.5–41.7) | 93.5 (89.3–96.4) | 85.6 (80.2–90.0) | 34.9 (28.9–41.7) | 65.1 (58.3–71.5) | 71.2 (64.6–77.1) | 16.7 (12.0–22.4) | 14.4 (10.0–19.8) |
| 61–70 | 3.2 (0.1–16.7) | 32.3 (16.7–51.4) | 96.8 (83.3–99.9) | 77.4 (58.9–90.4) | 32.3 (16.7–51.4) | 64.5 (45.4–80.8) | 80.6 (62.5–92.5) | 19.4 (7.5–37.5) | 19.4 (7.5–37.5) |
| Education, years | | | | | | | | | |
| None | 0.0 (0.0–60.2) | 0.0 (0.0–60.2) | 0.0 (0.0–60.2) | 0.0 (0.0–60.2) | 0.0 (0.0–60.2) | 25.0 (0.6–80.6) | 75.0 (19.4–99.4) | 0.0 (0.0–60.2) | 0.0 (0.0–60.2) |
| < 7 years | 6.3 (2.1–14.0) | 31.3 (21.3–42.6) | 97.5 (91.3–99.7) | 85.0 (75.3–92.0) | 32.5 (22.4–43.9) | 52.5 (41.0–63.8) | 63.8 (52.2–74.2) | 17.5 (9.9–27.6) | 13.8 (7.1–23.3) |
| 8–11 years | 3.3 (0.7–9.2) | 29.3 (20.3–39.8) | 95.7 (89.2–98.8) | 84.8 (75.8–91.4) | 23.9 (15.6–33.9) | 46.7 (36.3–57.4) | 48.9 (38.3–59.6) | 7.6 (3.1–15.1) | 8.7 (3.8–16.4) |
| > 12 years | 2.7 (1.6–4.3) | 22.0 (18.9–25.5) | 94.0 (91.8–95.7) | 76.3 (72.7–79.5) | 26.4 (23.1–30.1) | 52.7 (48.7–56.6) | 45.8 (41.8–49.7) | 6.6 (4.9–8.9) | 14.3 (11.7–17.3) |
| Staff cadre | | | | | | | | | |
| Junior | 4.4 (2.9–6.8) | 25.9 (22.1–30.0) | 94.5 (92.1–96.3) | 77.2 (73.2–80.7) | 20.8 (17.4–24.7) | 57.7 (52.4–62.4) | 43.2 (38.8–47.7) | 5.7 (3.9–8.2) | 12.9 (10.2–16.3) |
| Senior | 1.0 (0.3–2.8) | 21.6 (17.7–26.2) | 94.6 (91.7–96.5) | 78.6 (74.2–82.6) | 34.3 (29.6–39.3) | 47.3 (42.8–51.8) | 55.2 (50.1–60.2) | 11.1 (8.2–14.7) | 14.4 (11.2–18.4) |
| Income quintile | | | | | | | | | |
| Lowest | 3.4 (1.1-7.9) | 28.4 (21.2-36.5) | 93.0 (87.6-96.6) | 75.0 (67.1-81.8) | 20.1 (13.9-27.6) | 43.1 (34.8-51.6) | 38.1 (30.2-46.6) | 4.9 (2.0-9.8) | 10.4 (5.9-16.6) |
| Second | 6.1 (2.9-10.9) | 28.6 (21.8-36.2) | 93.2 (88.3-96.6) | 81.7 (74.9-87.3) | 20.1 (14.2-27.0) | 48.8 (40.9-56.7) | 40.2 (32.6-48.1) | 4.9 (2.1-9.4) | 15.8 (10.6-22.3) |
| Third | 0.0 (0.0-100.0) | 22.3 (15.8-30.1) | 94.4 (89.2-97.5) | 81.1 (73.7-87.1) | 33.5 (25.8-41.9) | 53.8 (45.3-62.2) | 52.4 (43.9-60.8) | 9.1 (4.9-15.0) | 13.9 (8.7-20.7) |
| Fourth | 3.0 (1.0-6.9) | 18.2 (12.7-25.0) | 95.7 (91.4-33.0) | 82.9 (76.2-88.3) | 25.6 (19.1-33.0) | 57.3 (49.4-65.0) | 56.1 (48.1-63.8) | 11.6 (7.1-17.5) | 11.5 (7.1-17.5) |
| Fifth | 0.6 (0.02-3.5) | 22.7 (16.3-30.1) | 95.4 (90.8-98.1) | 76.6 (90.8-98.1) | 35.0 (27.5-43.1) | 59.7 (51.5-67.6) | 55.1 (46.9-63.2) | 11.0 (6.6-17.1) | 13.6 (8.6-20.0) |

HPTN = hypertension; DM = diabetes mellitus; CKD = chronic kidney disease.

indicated in Table 2. Its prevalence rose with increasing age (Fig. 2A) and household income (Fig. 2B) but decreased with increasing level of education (Fig. 2C). Similar trends were noticed for diabetes mellitus (DM) with regard to age and household income (Fig. 2A, B). CKD also increased with increasing age (Fig. 2A). The prevalence of DM and CKD by sociodemographic characteristics is shown in Table 2.

## Discussion

The main findings of our study were that: (1) the most prevalent NCD risk factors were low intake of fruit and vegetables, physical inactivity and dyslipidaemia, with the majority of participants having multiple factors; (2) nearly half (48.5%) of the participants were hypertensive, and CKD and DM occurred in 13.6 and 8.0%, respectively; and (3) the sociodemographic characteristics of age, income and education impacted on the prevalence of the common NCDs and their risk factors. Inadequate consumption of fruit and vegetables was the most prevalent risk factor in this study, followed by physical inactivity and obesity. This is in accord with the findings of many researchers. Sufficient consumption of fruit and vegetables was lacking in 96.6% of respondents in a Nepalese community.[19] Zaman and co-workers[20] reported that 92% of participants in a nationally representative sample in Bangladesh reported inadequate intake of fruit and vegetables (median serving of 1.6 portions/day).

In a study of university employees in western Nigeria, 96% of participants had inadequate consumption of fruit and vegetables.[8] Similarly, a high prevalence (90%) has been reported from northern Nigeria.[6] However, in a community survey in eastern Nigeria, a slightly lower proportion (70.4%) of respondents had inadequate intake of fruit and vegetables.[21] Nonetheless, a high prevalence of inadequate intake of fruit and vegetables exists in the general population and that needs to be addressed.

Physical inactivity has been found to contribute significantly to NCD-related mortality.[22] Three-quarters of the participants in our study were physically inactive. This is in accord with the findings of Oladimeji and co-workers,[6] who reported that 91% of workers in the public sector were physically inactive. Likewise, nearly 80%

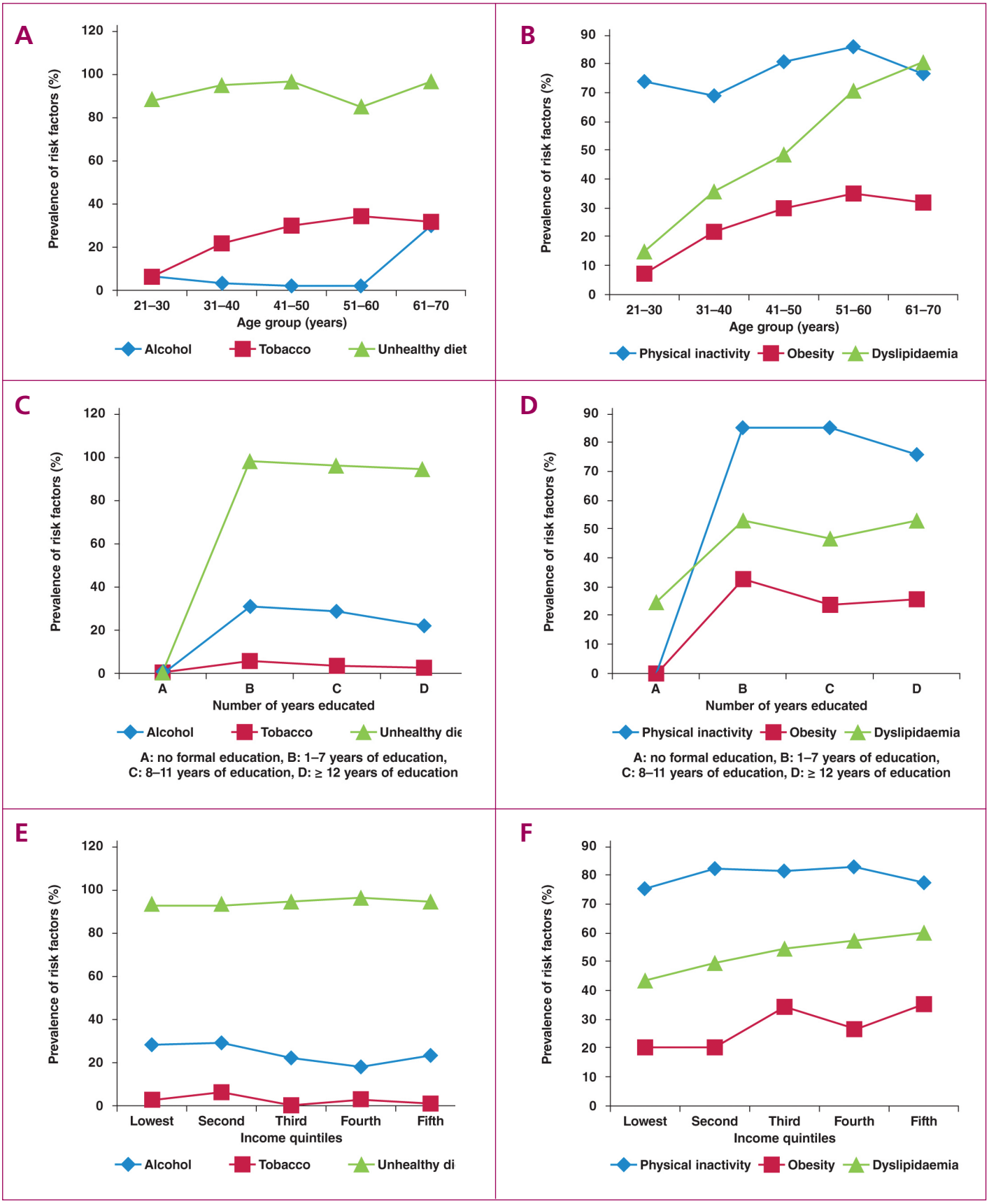


**Fig. 1.** Prevalence of non-communicable disease risk factors in relation to some sociodemographic characteristics in 883 staff members of the University of Jos.

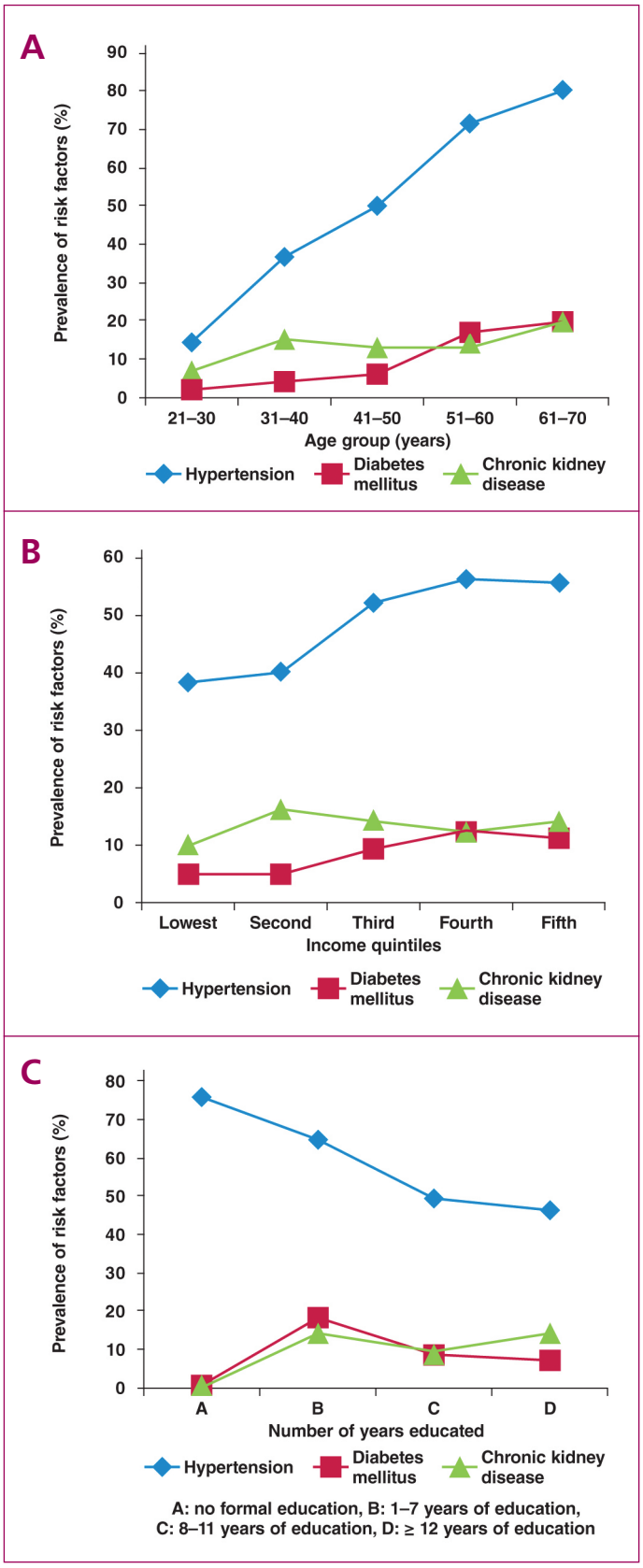


**Fig. 2.** Prevalence of non-communicable diseases in relation to some sociodemographic characteristics among 883 staff members of the University of Jos.

of hospital workers in Nigeria have been reported to be physically inactive.[23] However, a lower prevalence of physical inactivity has been reported in earlier studies.

Of the 2 000 persons studied in Togo, 41% were sedentary, while 35% was reported from Bangladesh.[24,25] In a study among workers at a medical college in Ghana, only 25% were physically inactive.[26] The reason for the disparity between our findings and those of prior studies reporting low prevalence of physical inactivity may be related to the highly selective nature of our study participants.

We noted the rarity of both active and passive cigarette smoking in our participants. This is in keeping with previous reports that document a paucity of smoking among Nigerians.[8,6,27] Generally, this finding is in contrast to the findings in southern Africa, Asia[20,28] and the Western world,[29-31] where smoking constitutes a major public health hazard.

Clustering of risk factors was prevalent in this study, with the median number of risk factors being three (IQR 2–3) per participant. This finding corroborates the findings of previous studies. In a study of over 3 800 South African adults aged 50 years and above, Phaswana-Mafuya and associates[32] reported a mean incidence of risk factors of three. In a recent German survey, 45.1% of participants had multiple risk factors.[33] Similar clustering has been reported by the SAGE wave 1 study that evaluated older adults across six countries.[34]

A study among Senegalese private sector workers revealed that more than half of the participants had two or more cardiovascular risk factors.[35] Villegas and co-workers[36] reported that 67.6% of men and women sampled across 17 general practice settings in Ireland had more than one cardiovascular risk factor. This scenario is the typical clustering in patients and deserves attention to reverse or limit their contribution to NCD and its related mortality.

The prevalence of the selected NCDs parallels that obtained in the literature from the Western world and the African region. Hypertension was present in nearly half of the participants; CKD was present in a little over a 10th of the population, and DM in nearly a 10th. In the SAGE wave 1 study, the prevalence of hypertension ranged from as low as 17.9% in Bangladesh to as high as 78% in South Africa among older persons.[34] A prevalence of 47.2% was reported among Irish hospital attendees in a study that evaluated over 1 000 patients recruited from several general practices.

Oluyombo and colleagues,[37] working in south-west Nigeria, reported a prevalence of 47.2% among residents of a semi-urban community. A slightly lower prevalence of 31.4% was recently reported from south-east Nigeria.[21] In a large community survey that evaluated 5 206 adults in Malawi, Msyamboza and associates[38] reported a prevalence of 33% among persons aged 25 to 64 years. A recent review by Bosu[7] demonstrated that the prevalence of hypertension among workers in the West African sub-region has steadily increased from 12.9% in the 1980s to 37.5% in 2014, while figures up to 51.6% (95% CI: 49.8–53.4) and 43% (95% CI: 42.1–43.9) have been recently reported in Nigeria among urban and rural populations, respectively.[4]

CKD, an emerging NCD, has gained attention in recent times as it is both an end-point of communicable and non-communicable diseases and a strong cardiovascular risk factor. It has become a pandemic, affecting both developed and developing countries. CKD was present in a significant proportion of the participants in our study. Similar reports exist regarding the prevalence of CKD from the Western world and Asia.[39-41]

However, varying reports from the African region exist. In a recent community survey from Senegal that studied 1 037 adults,

CKD was present in 4.9% of the participants.[42] In a similar study from Cameroun, the prevalence of CKD ranged from 11.0 to 14.2%, depending on the prediction equation used.[43] In a study that evaluated 402 private sector IT workers in Dakar, Senegal in late 2010, 22.4% had CKD.[35] The prevalence of CKD in Nigeria in various subsets of the population has been reported to range from 7.8% among public sector employees,[44] to 11.4% in the community[45] and 43.5% among retirees,[46] depending on the criteria used.

The prevalence of DM in this study parallels the estimated global prevalence of 9%, the WHO estimated prevalence of 7.9% in Nigeria in 2014,[1] and the 9.7% recently reported from Senegal.[35] It is however slightly lower than the 11% obtained among university employees in south-western Nigeria.[8] However, our study differed from theirs as they relied on selfreported diagnosis, which is subject to recall bias. Oluyombo and associates[37] recently reported that 6.8% of 750 respondents had DM. Our finding together with the foregoing support the assertion that the prevalence of DM is on the increase in Nigeria. However, the prevalence of DM in our study was higher than the 2.5% reported by Oladapo and co-workers[47] in south-west Nigeria, and the 3.6% by Okpechi and colleagues[21] in southeastern Nigeria.

That sociodemographic characteristics impact on NCDs and their risk factors was confirmed by the findings of our study. The prevalence of hypertension, CKD and DM rose with increasing age, as expected. Their prevalence also increased with increasing income, as a result of the concomitant rise in the prevalence of some of the risk factors with increasing income. It is noteworthy that hypertension decreased with increasing educational level. This confirms the results of prior studies that reported an inverse relationship between educational level and hypertension.[19,48] This provides an opportunity for intervention in order to halt the rising trends in NCD.

Together with the existing literature, our study has implications for the subset of employees at this university and the general population at large, as large numbers of these individuals are at an elevated risk of NCD-related events. In a recent review of national policies addressing NCDs in low- and middle-income countries, Lachat and colleagues[22] demonstrated the disconnect that exists between the burden of NCDs and the response of the respective governments, including Nigeria. Concerted efforts are needed to stem the high prevalence of NCDs and their risk factors in our environment, so as to achieve the 2025 voluntary global targets of the Global NCD Action Plan.[1]

### *Limitations*

The findings of this study must be interpreted within the limitations inherent in the study design. We studied only employees of the university hence the generalisability of the findings is limited. The purposive sampling process used may also have introduced selection bias in the study. A stratified systematic sampling would have yielded a more representative sample. However we invited all the staff members of the university to participate in the study.

We were unable to measure triglyceride levels so we used non-fasting blood samples for the determination of lipid levels. At first glance, one may assume that assessing lipid abnormalities using casual plasma samples (and not in the fasted state) as we did in this study would constitute a limitation. However, the lack of effect of fasting on levels of serum total cholesterol and reduced high-density lipoprotein cholesterol has been documented and therefore casual plasma sampling is used in field studies.[49,50]

We were also unable to repeat proteinuria assessments or eGFR after three months and therefore the prevalence of CKD may have been spuriously high. Finally, we could not establish causality as our study was cross-sectional in design. Despite these limitations, we have studied the largest sample of university employees in Nigeria to date. Our study therefore provides the fulcrum for further studies of this nature to elucidate the burden of NCDs in this category of workers.

## Conclusion

This study identified that the most prevalent NCD risk factors among employees of a university are behavioural and therefore modifiable. We also demonstrated that the NCDs and their risk factors are impacted upon by sociodemographic characteristics. Given the burden of NCDs and their risk factors among this subset of the general population, there is a need for workplace policies aimed at health promotion to be put in place in order to stem the rising trend of NCDs. Multicentre studies addressing the burden of NCDs among university employees are imperative.

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# Ellisras Longitudinal Study 2017: The relationship between dietary intake and body mass index among young rural adults in South Africa aged 18 to 30 years (ELS 18)

JULIA TM MASHIANE, KOTSEDI D MONYEKI, ANDRE P KENGNE, NKWANA M ROSINA, MAFOLOA S MONYEKI

## Abstract

***Aim:*** **To assess the relationship between dietary intake and adiposity in young rural South African adults.**

***Methods:*** **A total of 728 young adults participated and dietary intake was assessed using the 24-hour recall method. Linear regression models were used to determine the association between dietary intake and body mass index (BMI) before and after adjustment for age and gender.**

***Results:*** **Females showed higher mean BMI values than males in all age groups. An age group of 27- to 30-year-old females had a mean value of 28.1 kg/m² while males had a mean value of 21.9 kg/m². The distribution of BMI categories (underweight, normal weight, overweight, obese) was 20.5, 61.7, 9.3 and 3.1% in males, and 8.6, 42.5, 23.1 and 25.8% in females ($p \leq 0.05$). Cholesterol intake was significantly ($p \leq 0.05$) associated with BMI (beta = 0.002, 95% CI: 0.00–0.004) as well as overweight and obesity (odds ratio = 1.734; 95% CI: –1.09–2.75) after adjustment for age and gender.**

***Conclusion:*** **There was a high prevalence of overweight and obesity among rural Ellisras females. Moreover, increasing cholesterol intake was associated with overweight and obesity in the overall sample.**

**Keywords:** dietary intake, body mass index, adults, overweight and obesity

The prevalence of obesity continues to increase at an alarming rate worldwide, with approximately two billion people being overweight and one-third of them obese.[1] Over-consumption of macronutrients contributes to overweight and obesity among the adult population.[2] A diet characterised by a decrease in dietary fibre and an increase in saturated fats, accompanied by a lack of physical activity, results in weight gain.[3,4] This is the result of a positive energy balance, where energy intake is higher than energy expenditure.[2]

Traditional eating habits of South Africans residing in rural areas consist mostly of a prudent diet, which is associated with a low prevalence of overweight and obesity.[5-8] However, the shift towards a Western diet has become apparent among rural Africans, increasing their likelihood of having modifiable risk factors for chronic diseases of lifestyles, which include physical inactivity, increased alcohol consumption, stress and smoking.[5]

Preliminary results from the Ellisras cohort study showed a significant association between intake of mono-unsaturated fats and body mass index (BMI) among rural Ellisras children.[9] Furthermore, Sekgala *et al*.[10] reported a potential link between dietary fibre intake and fasting blood glucose and high-density lipoprotein cholesterol levels with both systolic and diastolic blood pressure among young rural Ellisras adults. With the Ellisras sample reaching the young adult stage, the relationship between BMI and dietary intake has received little attention. This cross-sectional study aimed to investigate the relationship between dietary intake and BMI among young rural Ellisras adults aged 18 to 30 years.

## Methods

This study is part of the ongoing Ellisras longitudinal study (ELS), of which the details of the sampling procedure and geographical area were reported elsewhere.[11] The subjects participating in this cross-sectional study included 728 young adults (356 males and 372 females), aged 18 to 30 years, who are part of the Ellisras longitudinal study (ELS).

The ethics committee of the University of Limpopo granted ethical approval prior to the survey. The participants were provided with informed consent forms and signed the form after receiving verbal assent from the project leader.

All participants underwent a series of anthropometric measurements according to the standard procedures recommended by the International Society for the Advancement of Kinanthropometry (ISAK).[12] Weight was measured on an electronic scale to the nearest 0.1 kg, with light clothing and without shoes. Martin anthropometric was used to measure height, to the nearest 0.1 cm, with no shoes. BMI was defined as weight (kg)/height (m²). All participants were classified as underweight, normal, overweight and obese, according to World Health Organisation cut-off points for adults.[13]

Diet was measured using the 24-hour recall method, which is a valid method to determine group dietary intake.[14] In December

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2015, senior Northern Sotho-speaking dietetics students of the University of Limpopo, specifically trained in using the 24-hour recall method, interviewed the parent/caregiver at home regarding the dietary intake of the young adults over the previous 24 hours. For each participant, an interview took place on one weekday and one weekend day. An average of two days of 24-hour dietary intake was then taken for each participant.

Estimated portion sizes of foods consumed were recorded in as much detail as possible, using a pre-tested questionnaire and food models simulating average portions of local foods.[15,16] Dietary data were analysed using local food tables and Food Finder dietary software, and compared with recommended intakes.[15-18]

### *Statistical analysis*

Variables were summarised as descriptive statistics. Linear regression models were used to assess the continuous association between dietary intake and BMI, while logistic regression models were used to assess the association between low/high dietary intake and prevalent overweight and obesity, both in invariable analyses and after adjusting for age and gender. All data were analysed using the statistical package for social sciences (SPSS) version 23 and a *p*-value < 0.05 was used to characterise statistically significant results.

## Results

The mean BMI was 20.3–21.9 kg/m² in males and 23.2–28.1 kg/m² in females ($p \leq 0.05$). Mean BMI increased from 20.3 kg/m² in the age group 18–20 years to 21.9 kg/m² in the age group 27–30 years in males, and from 23.2 to 28.1 kg/m² in females (Fig. 1).

The distribution of BMI categories in the overall sample was 8.6–20.5% for underweight, 9.3–23.1% for overweight and 3.1–25.8% for obesity. Equivalent figures were 20.5, 61.7, 9.3 and 3.1% in males, against 8.6, 43.5, 23.1 and 25.8% in females ($p \leq 0.05$ for the difference in the distribution of BMI categories in males and females) (Fig. 2). Males had a higher incidence of underweight (20.5%) than females. However, females (23.1 and 25.8%) showed a higher incidence than males (9.3 and 3.1%) of overweight and obesity, respectively.

Fried chicken (23.8%), pap (22.6%), cold drink (16.9%) and white sugar (14%) were the foods most frequently consumed by the young Ellisras adults, while samp (2.6%), yogurt (2.4%) and spinach (2.0%) were the least frequently consumed foods (Table 1). Carbohydrates ranged between 78.2 and 84.5% while total fats and saturated fats ranged between 31.6 and 42%, and 4.1 and 6.0%, respectively, for all BMI categories for the overall population (Fig. 3).

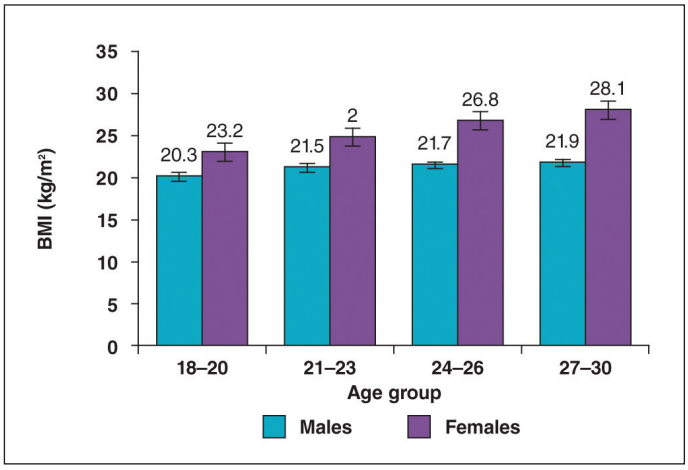


**Fig. 1.** Descriptive statistics of mean body mass index by age group and gender among young rural Ellisras adults aged 18–30 years.

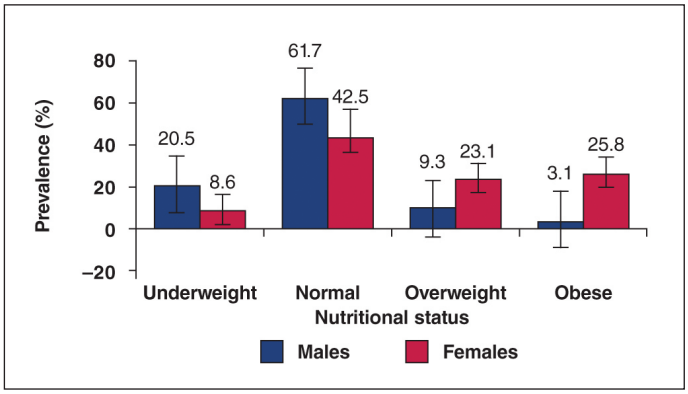


**Fig. 2.** The prevalence of malnutrition by gender among young rural Ellisras adults aged 18–30 years.

**Table 1.** The most frequent food items in the diets for the overall sample, from the most common food liked to the least liked

| Variables | Percentage |
|---|---|
| Fried chicken with skin | 23.8 |
| Pap | 22.6 |
| Cold drink | 16.9 |
| White sugar | 14 |
| Vetkoek | 5.8 |
| Fried beef | 4.7 |
| Peanut butter | 4.4 |
| Samp | 2.6 |
| Yoghurt | 2.4 |
| Spinach | 2.0 |
| Pilchards | 0.5 |

In linear regression analyses, there was a borderline positive association between cholesterol intake and BMI ($p = 0.058$), with further enhancement after adjustment for age and gender (beta = 0.002, $p = 0.035$) (Table 2). Table 3 presents logistic regression for the association between overweight/obesity and low dietary intake. In logistic regression analyses, there was a positive association between cholesterol intake and overweight and obesity ($p = 0.084$), and after adjustment for age and gender, the association of cholesterol intake with overweight and obesity was significant ($p = 0.020$) (Table 3).

## Discussion

This study aimed to investigate the relationship between dietary intake and BMI among young rural Ellisras adults aged 18 to 30 years. There was a significant association between cholesterol intake and BMI. Furthermore, a high prevalence was reported of overweight and obesity among females compared to males in the Ellisras population. These findings were in line with previous studies conducted in rural black communities in the Limpopo province.[3] This may be due to culture-related attitudes, physical inactivity, poor nutritional value of food, and high intake of calorie-dense food in rural populations.[3]

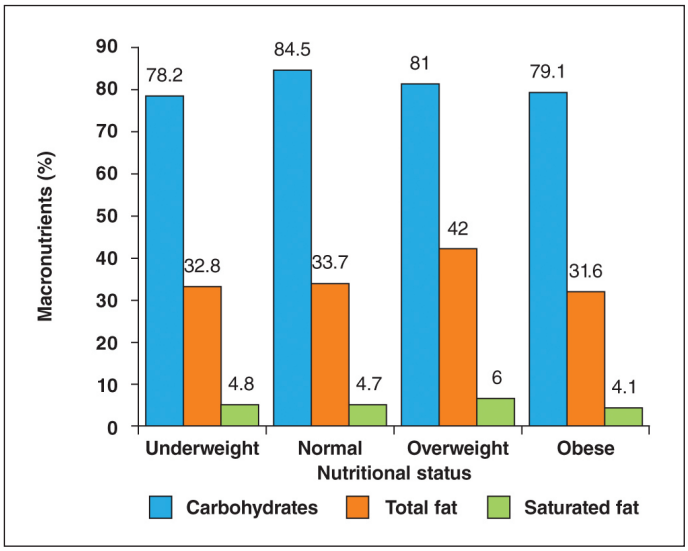


**Fig. 3.** Descriptive statistics for 24-hour recall of dietary intake by nutritional status of young rural Ellisras adults aged 18–30 years.

Obesity is a risk factor for cardiovascular diseases such as hypertension and type 2 diabetes, and it is a global public health concern.[13] Van Den Ende *et al*.[9] reported a low prevalence of overweight and obesity among the same sample at a younger age (7–15 years). The present study revealed a high prevalence of obesity (3–26%) and overweight (9–23%) as the ELS sample grew older. This is a serious concern.

The findings are in line with other studies in Africa and the prevalence of overweight and obesity continues to increase, with from 25 to 60% of urban females being overweight.[3,4] The influence of a Western diet together with low levels of physical activity, particularly among women, as reported by Sekgala *et al*.,[10] Mchiza *et al*.[6] and Jaffer *et al*.[7] among the South African population, could be contributing to this escalating high prevalence of obesity and overweight.

Furthermore, several studies have reported the overconsumption of macronutrients to be one of the leading causes of the high prevalence of overweight and obesity among the adult Saudi population.[19,20] An increase in urbanisation, in terms of social, political and economic factors, explains the dietary transition in South Africa among females.[21] It is projected that the population of overweight and obesity worldwide will increase to 2.3 billion for overweight and 700 million for obesity.[3] According to the Global Burden of Metabolic Risk Factors of Chronic Diseases Collaboration Group, 9.1 million adults are affected with overweight and obesity.[22] This has caused the tendency of overweight and obesity to double worldwide.

**Table 2.** Linear regression coefficient, 95% CI and p-value in the association with body mass index and dietary intake

| | Unadjusted | | | | Adjusted (age and gender) | | | |
|---|---|---|---|---|---|---|---|---|
| **BMI variables** | **β** | **95% CI** | | ***p*-value** | **β** | **95% CI** | | ***p*-value** |
| Total fat | –0.002 | –0.011 | 0.007 | 0.665 | –0.001 | –0.010 | 0.007 | 0.738 |
| Animal protein | 0.000 | –0.016 | 0.015 | 0.988 | 0.004 | –0.010 | 0.018 | 0.538 |
| Plant protein | –0.001 | –0.041 | 0.038 | 0.951 | 0.008 | –0.028 | 0.044 | 0.667 |
| Total sugar | 0.009 | –0.010 | 0.028 | 0.366 | –0.002 | –0.019 | 0.015 | 0.827 |
| Carbohydrates | 0.001 | –0.002 | 0.004 | 0.545 | 0.001 | –0.002 | 0.004 | 0.459 |
| Total dietary fibre | 0.016 | –0.040 | 0.073 | 0.570 | 0.019 | –0.032 | 0.071 | 0.460 |
| Total protein | 0.000 | –0.013 | 0.014 | 0.972 | 0.005 | –0.007 | 0.017 | 0.451 |
| Cholesterol intake | 0.002 | 0.000 | 0.004 | 0.058 | 0.002 | 0.000 | 0.004 | 0.035* |
| Mono-unsaturated fatty acids | –0.008 | –0.032 | 0.016 | 0.527 | –0.005 | –0.027 | 0.016 | 0.634 |
| Polyunsaturated fatty acids | –0.002 | –0.033 | 0.028 | 0.876 | –0.002 | –0.029 | 0.026 | 0.899 |
| Saturated fatty acids | –0.007 | –0.033 | 0.019 | 0.600 | –0.007 | –0.030 | 0.017 | 0.583 |

CI: confidence interval, β: beta-coefficient. *Significant at $p < 0.05$.

**Table 3.** Logistic regression for the association between overweight/obesity and low dietary intake

| | Unadjusted | | | | Adjusted for age and gender | | | |
|---|---|---|---|---|---|---|---|---|
| **Variable** | **OR** | **95% CI** | | ***p*-value** | **OR** | **95% CI** | | ***p*-value** |
| Overweight/obesity | | | | | | | | |
| Total fat | 0.78 | 0.56 | 1.10 | 0.154 | 0.86 | 0.59 | 1.22 | 0.430 |
| Total sugar | 1.18 | 0.67 | 2.08 | 0.561 | 0.96 | 0.52 | 1.78 | 0.900 |
| Saturated fat | 1.23 | 0.89 | 1.69 | 0.215 | 1.32 | 0.924 | 1.894 | 0.127 |
| Mono-unsaturated fat | 0.61 | 0.20 | 1.88 | 0.388 | 0.48 | 0.14 | 1.694 | 0.255 |
| Polyunsaturated fat | 1.48 | 0.25 | 8.93 | 0.668 | 1.46 | 0.20 | 10.81 | 0.708 |
| Cholesterol intake | 1.43 | 0.95 | 2.16 | 0.084 | 1.73 | 1.09 | 2.75 | 0.020* |

OR: odds ratio; CI: confidence interval. *Significant at $p < 0.05$.

The intake of carbohydrates and fats in the present study was higher than that reported by Van Den Ende *et al*.9 in the same sample at a younger age. Singh *et al*.[2] recommended 60% carbohydrate, 30% total fats and 10% protein as the total daily kilocalories for an individual. The high consumption of fats in our study therefore reveals that there is a peak in the nutritional transition, and weight status has therefore changed among Ellisras females. The high intake of saturated fat reported in this study is in agreement with that in healthy young adults in Saudi Arabia.[23]

The significant association between dietary intake and BMI predicts that the higher the percentage of kilojoules, the higher the risk of overweight and obesity. This finding is consistent with Van Den Ende *et al*.[9] Sengwayo *et al*.[3] found a significant association of dyslipidaemia with high BMI among females in Limpopo. This is associated with a shift in the nutritional pattern, which predisposes to the development of atherosclerosis due to a high cholesterol intake.

A limitation of this study is the cross-sectional design, which does not allow an analysis of cause and effect regarding the association between BMI and dietary intake. Also we did not consider blood sample analysis to support the findings of dietary intake. However, Steyn *et al*.[21] confirm that dietary intake can be reliably evaluated by assessing the macronutrient intake. All anthropometric data were measured, not self-reported by the participants, which allows the comparison of our study with other studies in South Africa to be accurate.[4,21] Furthermore, we used intervieweradministered questionnaires, which are more effective than a self-administered questionnaire.[5]

## Conclusions

There was a high prevalence of overweight and obesity among rural Ellisras females. Cholesterol intake was associated with a raised BMI in the overall sample. Therefore, dietary knowledge and access to resources are important to improve health and nutrition in a sustainable way. The need to assess the changes that occur over

time in serum levels of a variety of biochemical and haematological parameters related to cardiovascular diseases and/or diabetes in rural African settings is vital.

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# Prevalence and sociodemographic correlates of cardiovascular risk factors among patients with hypertension in South African primary care

JM NGANGO, OB OMOLE


## Abstract

***Objective:*** **To determine the prevalence and sociodemographic correlates of cardiovascular risk factors among patients with hypertension at Johan Heyns Community Health Centre, Sedibeng district, South Africa.**

***Methods:*** **A total of 328 participants were systematically sampled. A researcher-administered questionnaire collected information on: socio-demography, presence of diabetes, family history of hypercholesterolaemia, family history of fatal cardiovascular (CV) events, and engagement in physical activities. Other measurements included: blood pressure (BP), weight, height, abdominal circumference and electrocardiography (ECG). Data analysis included descriptive statistics, chi-squared test and regression analysis. Main outcome measures included the proportions of participants with each CV risk and their significant sociodemographic determinants.**

***Results:*** **Participants' mean age was 57.7 years. Most participants were black (86.0%), female (79%) and pensioners (43.6%). The mean BP was 139/84 mmHg, and 60.7% had their BP controlled to targets. There was an average of 3.7 CV risk factors per participant and the prevalence of CV risk factors was: abdominal obesity (80.8%), physical inactivity (73.2%), diabetes (30.2%), alcohol use (28.0%), hypercholesterolaemia (26.5%), smoking (11.9%), past family history of fatal CV event (14.9%), and left ventricular hypertrophy (5.2%). Sociodemographic factors significantly associated with each CV risk factor were: obesity and being female ($p$ = 0.00); alcohol use and young age ($p$ = 0.00); smoking, being male and race other than black ($p$ = 0.00 and $p$ = 0.00, respectively); physical inactivity, being a pensioner and male ($p$ = 0.02 and $p$ = 0.02, respectively); diabetes and being male ($p$ = 0.03); hypercholesterolaemia and race other than black ($p$ = 0.03); family history of hypercholesterolaemia and race other than black ($p$ = 0.00); and family history of fatal CV event and race other than black ($p$ = 0.00).**

***Conclusion:*** **There is a high burden of CV risk factors among patients with hypertension in South African primary care, signifying a substantial risk of cardiovascular disease (CVD) in this setting. Interventions aimed at CVD risk reduction need to take cognisance of the sociodemographic correlates of CV risk factors.**


**Keywords:** prevalence, cardiovascular risk factors, hypertension, primary care

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Hypertension is a major risk factor for cardiovascular disease (CVD).[1] It affects a quarter of the world's adult population and accounts for 80% of deaths and 87% of disability in developing countries.[2,3] Its burden is greater in low- and middle-income than in high-income countries.[4]

In the past four decades, African countries have experienced an increased prevalence of CVD due to increasing urbanisation and lifestyle changes.[5] CVD is the second leading cause of mortality after HIV/AIDS in South Africa, accounting for up to 40% of deaths among adults.[6]

In South Africa, of all CVD risk factors, hypertension is the commonest, with a prevalence that is highest among the poor in urban settings.[7,8] Hypertension often co-exists with other CV risk factors, resulting in a significantly increased risk for CVD.[9] Of serious concern is that the prevalence of these other CV risk factors, such as diabetes, tobacco use, high cholesterol levels, obesity, physical inactivity and unhealthy diets are also on the increase, both in urban and rural settings in Africa.[10]

Most CV risk factors are modifiable, providing opportunities for reduction in CVD-attributable morbidity and mortality rates.[11] Simultaneously addressing CV risk factors substantially reduces the risk of adverse CV events more than controlling for any single risk factor alone.[12] In this vein, it is well established that lowering only blood pressure (BP) does not adequately minimise the risk of CV morbidity and mortality. Despite this knowledge, co-existing CV risk factors remain inadequately managed among patients with hypertension.[9]

In South Africa, clinicians at the primary healthcare (PHC) level manage a substantial proportion of patients with hypertension. This level of care is expected to provide primary and secondary prevention interventions through patient education on healthy lifestyle, screening, and prompt management of CV risk factors. To carry out these tasks effectively for patients with hypertension, it is important to know which of the CV risk factors are prevalent in a PHC setting, especially since the burden of CV risks may differ across populations.[13] Patients with hypertension in health facilities have a higher baseline CV risk burden than reported in community-based studies.

Although the South African literature has reported urban–rural disparity and higher-than-expected CV risk prevalence in the general population,[14] few studies have focused on patients with

hypertension in PHC facilities. The aim of this study was to determine the prevalence and determinants of CV risk factors among patients with hypertension in a large, peri-urban PHC facility in Gauteng province, South Africa.

## Methods

This cross-sectional study was conducted in a large community health centre (CHC) south of Johannesburg. At the time of the study, from March to May 2012, the CHC provided ambulatory curative, preventative and rehabilitation services, and served as a referral centre for five smaller clinics. In the out-patient department (OPD), there were three doctors, three PHC nurse clinicians and five staff nurses.

In this facility, patients with hypertension are booked by appointment for their monthly follow-up visits. A day before the appointment, an administrative clerk retrieves patients' medical records from the archives for all patients booked. On the day of the clinic visit, patients collect their files and proceed to sit in the waiting hall, from where a nurse invites a group of 10 patients for measurement of vital signs at the nurses' station. Thereafter, the patients are directed to a doctor or PHC nurse clinician, depending on the complexity of the presenting problem.

In 2011, an estimated 1 974 patients with hypertension attended this CHC. Assuming an annual increase of 10%, the estimated patients with hypertension for the year 2012 was rounded off to be 2 100. To determine the sample size, the researchers used the Raosoft sample size calculator.[15] The required sample size was calculated to be 328, based on a 5% margin of error, 95% confidence level and a response distribution of 50%.

Patients were recruited into the study by the first author and two staff nurses who were trained as research assistants. During the sampling, a research assistant approached patients as they presented for measurement of vital signs at the nurses' station. Every third patient with hypertension was sampled for recruitment until the sample size was attained. If the third patient refused, the next willing patient (fourth or fifth) who met the inclusion criteria was approached and recruited.

To be selected, a patient had to consent to participate in the study, be 18 years or older and have attended the CHC for at least two months of hypertension treatment. Patients who participated in the pilot study, emergencies, those with mental impairment and those who presented after hours were excluded. Patients willing to participate in the study were taken to an adjacent room where the researcher and the second research assistant obtained informed written consent.

The following CV risk factors were considered in this study, informed by relevance to PHC and financial costs: advancing age, gender, obesity, left ventricular hypertrophy (LVH) on electrocardiograph (ECG), tobacco and alcohol use, diabetes, physical inactivity, hypercholesterolaemia, family history of hypercholesterolaemia and past family history of fatal CV events.

After obtaining informed consent, the first author and second research assistant (when interpretation was necessary) administered the questionnaire, performed anthropometric and clinical measurements, and performed ECGs in an adjacent room.

A structured researcher-administered questionnaire was developed *de novo* in English, based on the literature. Each questionnaire had a serial number identical to the corresponding participant's file number, in case of need for retrieval. Personal identifying information was not collected. The questionnaire collected information on:

- Sociodemography: age, gender, marital status, educational attainment, employment status and ethnic group, tobacco use (cigarette smoking, exposure to second-hand smoke and snuff use) and alcohol consumption. Current smoking was defined as self-reported active smoking within the last year before examination.[16] Current alcohol use was defined as a patient who regularly consumed alcohol (regardless of the type) in the past 12 months.[17]
- Clinical co-morbidity: self-reports of diabetes mellitus, family history of hypercholesterolaemia and family history of fatal CV events (defined as death of a close family member due to heart attack or stroke before the age of 55 years in men or 65 years in women).[18]
- Engagement in physical activity: level of involvement and duration of time spent on physical activity, whether at the workplace, during leisure time or as household chores. Engagement in physical activity was defined as reporting moderate-intensity activities in a usual week for ≥ 30 minutes per day, ≥ five days per week; or vigorous-intensity activities in a usual week, ≥ 20 minutes per day, ≥ three days per week or both.[9] The following examples of physical activities were used to categorise participants. Moderate-intensity physical activity: brisk walking, dancing, gardening, housework, domestic chores, walking domestic animals, active involvement in games and sport with children. Vigorous-intensity physical activity: running, walking up hill, fast cycling, aerobics, competitive sport and games such as soccer, hockey basketball, fast swimming and heavy shovelling or digging.

Anthropometric measurements included abdominal circumference, weight, height and body mass index (BMI). These were done using equipment and procedures as recommended in the South African hypertension guideline of 2011.[2] Each instrument was calibrated before use and research assistants were trained to minimise inter-rater differences. Measurements were done using:

- Weight and height: Seca® scale, manufactured by Seca®CE in Germany (model: 767-1321004, serial number: 1767002071575). Heights and weights were measured in metres and kilograms to two decimal points. The research assistant used the technique recommended by de Onis *et al*.[19] for weight and height measurements. Thereafter, the researcher computed the BMI in kg/m$^2$. Obesity was considered if BMI was > 30 kg/m$^2$.
- Waist circumference: flexible, non-elastic metric tape. The research assistant used a flexible non-elastic tape measure 154 cm long and 15 mm wide, with 1-cm graduation intervals for the waist circumference measurement. The measurement was recorded at the end of a normal expiration at the midpoint between the lower margin of the last palpable rib and the top of the iliac crest on the mid-axillary line, with the tape parallel to the floor at the level at which the measurement was done.[20] Each measurement was repeated twice and if the measurements came within 1 cm of one another, the average was calculated. If there was > 1 cm difference, the two measurements were repeated. Central obesity was present if waist circumference was ≥ 102 cm in men and ≥ 88 cm in women.[18] In the Asian population, central obesity was considered in men if the abdominal circumference was ≥ 90 cm and in women if the waist circumference was ≥ 80 cm.[21]

Clinical measurements were done including:

- BP: automated Dinamap®, General Electric Medical Systems (model: DPC321N-EN, item number: 2019194-001). After resting for five minutes, the BP was measured according to the method described in the JNC VII and South African Hypertension Society guidelines.[2] The readings were recorded in the patients' files.
- ECG: 12-lead digital electrocardiogram, Shenzhen Biocare Electronics Ltd (model E.C.G-1200). A resting 12-lead ECG was done using the technique recommended by Noble and colleagues.[22] The ECG was interpreted by the researcher with LVH assessed using the Romhilt–Estes five-point score. This has been reported to yield a specificity of 99%.[23]

Participants with problematic alcohol use or smoking were counselled and referred for assistance. To compensate for time lost due to participating in the study, all participants were attended to by a dedicated doctor and arrangements were made with the pharmacy to immediately dispense medications ahead of the queue. Data were captured on Microsoft Excel spreadsheets daily and cross-checked with the second author.

A pilot study was conducted using 30 patients at a nearby CHC in the same sub-district to assess the feasibility of the study. The results of the pilot study are not included in the main study but informed minor adjustments to some questions for ease of participants' understanding, for example, that a drink of alcohol should be expressed in ml and not in oz, and that three possible responses should be allowed for the question on assessment of hypercholesterolaemia.

Ethics clearance was obtained from the Human Research and Ethics Committee of the University of the Witwatersrand (number M10929). Permission was obtained from the Sedibeng District Health Services management. To ensure anonymity, the questionnaires were coded using the corresponding file number and we did not collect personal identifiable data. Patients who were found to have a problem with alcohol use or smoking and with worrying ECG findings were referred for further assistance.

### *Statistical analysis*

Captured data were imported into STATA statistical analysis software, version 10. A statistician assisted with analysis. Descriptive statistics were performed to describe participants' sociodemographic and clinical characteristics. Chi-squared and *t*-tests were used to compare groups, and variables that showed significant associations on bivariate analysis were inputted into multivariate analysis. A $p$-value < 0.05 was considered statistically significant. Main outcome measures included: proportions of participants with each CV risk factor (tobacco use, alcohol use, physical inactivity, diabetes, hypercholesterolaemia, family history of hypercholesterolaemia and fatal CV event) and the socio-demographic correlates of each CV risk.

## Results

There were 328 participants and their characteristics are shown in Table 1. The mean age of participants was 57.7 years and most participants were black (86.0%), female (79%) and pensioners (43.6%). The mean systolic BP was 139/84 mmHg, with 60.7% (199) having their BP controlled to targets.

In addition to hypertension, the 328 participants reported a total of 1 232 cumulative CV risk factors; an average of 3.7 CV risk factors per participant. Table 2 shows that the prevalence of CV risk factors was as follows: abdominal obesity (80.8%), physical inactivity (73.2%), diabetes (30.2%), alcohol use (28.0%) and smoking (11.9%).

**Table 1.** Participants' characteristics

| Variable | % (*n*) |
|---|---|
| Age, years | |
| Gender | |
| Female | 79 (260) |
| Male | 21 (68) |
| Marital status | |
| Divorced | 6.4* (21) |
| Living together | 3* (10) |
| Married | 51.8* (170) |
| Not married | 12.8* (42) |
| Widowed | 25.9* (85) |
| Ethnic group | |
| Asian | 0.3 (1) |
| Black | 86.0 (282) |
| Coloured | 0.9 (3) |
| White | 12.8 (42) |
| Employment status | |
| Employed | 30.8 (101) |
| Pensioner | 43.6 (143) |
| Unemployed | 25.6 (84) |
| Educational level | |
| None | 10.7 (35) |
| Primary | 33.5 (110) |
| Secondary | 53.7 (176) |
| Tertiary | 2.1 (7) |
| Mean age, years (SD) | 57.7 (10.8) |
| Mean weight: study population | 85.4 |

*The total percentage with decimals was slightly less than 100% (98.9%), but rounded to the nearest integer, it became 100%.

**Table 2.** Prevalence of cardiovascular risk factors

| Variable (*n* = 328) | % (*n*) |
|---|---|
| Mean BP, mmHg | |
| Systolic (SD) | 139.0 (20.9) |
| Diastolic (SD) | 84.3 (12.57) |
| BP controlled to target | 60.7 (199) |
| Tobacco use | |
| Current smoker | 11.9 (39) |
| Second-hand smoker | 16.0 (47) |
| Current snuffer | 19.5 (64) |
| Alcohol use | |
| Current alcohol use | 28.0 (92) |
| Physical activity | |
| Active | 26.8 (88) |
| Inactive | 73.2 (240) |
| Clinical risk factors | |
| Diabetes mellitus | 30.2 (99) |
| Elevated cholesterol | 26.5 (87) |
| Family history of hypercholesterolaemia | 5.2 (17) |
| Family history of fatal CV event (among females < 65 years and males < 55 years) | 14.9 (49) |
| Left ventricular hypertrophy, % | 5.2 (17) |
| Anthropometric measures | |
| Mean weight (kg) 85.4 | |
| Mean BMI (kg/m$^2$) 33.7 | |
| Increased waist circumference (> 88 cm for women, > 102 cm for men) | 80.8 (265) |

**Table 3.** Cardiovascular risk factors by age group

| | Age group, years | | | | | |
|---|---|---|---|---|---|---|
| Risk factor | 20–39 (*n* = 15) | 40–59 (*n* = 168) | 60–79 (*n* = 140) | ≥ 80 (*n* = 5) | Total (*n* = 328) | *p*-value |
| Alcohol use, *n* (%) | 9 (60) | 44 (26.2) | 38 (27.10) | 1 (20) | 92 (28.04) | 0.037* |
| Cigarette smoking, *n* (%) | 2 (13.33) | 22 (13.1) | 15 (10.71) | 0 (0) | 39 (11.89) | 0.7173 |
| Snuff use, *n* (%) | 5 (33.33) | 36 (21.4) | 22 (15.7) | 1 (20) | 64 (19.5) | 0.1962 |
| Exposure to smoking, *n* (%) | 0 (0.0) | 31 (18.5) | 13 (9.3) | 3 (60) | 47 (14.3) | 0.0015* |
| Physical inactivity, *n* (%) | 11 (73.3) | 105 (62.5) | 119 (85) | 5 (100) | 240 (73.2) | 0.0001 |
| Type 2 diabetes, *n* (%) | 3 (20.0) | 50 (29.8) | 44 (31.4) | 2 (40) | 99 (30.2) | 0.7809 |
| Hypercholesterol-aemia, *n* (%) | 7 (46.6) | 37 (22) | 41 (29.3) | 2 (40) | 87 (26.5) | 0.4379 |
| Family history of hypercholesterol-aemia, *n* (%) | 2 (13.3) | 11 (6.5) | 4 (2.8) | 0 (0.0) | 17 (5.2) | 0.2184 |
| Fatal CV event, *n* (%) | | | | | | |
| Female | 2 (13.3) | 18 (10.7) | 13 (9.3) | 1 (20) | 34 (10.4) | 0.8400 |
| Male | 1 (6.6) | 4 (2.4) | 10 (7.1) | 0 (0.0) | 15 (4.6) | 0.2252 |
| BMI ≥ 30 kg/m², *n* (%) | 9 (60) | 118 (70.2) | 87 (62.1) | 2 (40) | 216 (65.8) | 0.4945 |
| Waist circumference, *n* (%) | | | | | | |
| Female | 10 (83.3) | 130 (91) | 91 (91) | 5 (100) | 236 (90.7) | 0.7765 |
| Male | 1 (6.6) | 8 (4.8) | 20 (14.3) | 0 (0.0) | 29 (42.6) | 0.4484 |
| Left ventricular hypertrophy, *n* (%) | 0 (0.0) | 9 (5.3) | 7 (5) | 1 (20) | 17 (5.2) | 0.3137 |

**p*-values include very small numbers in the extreme age groups to be statistically reliable.

Most participants (60.4%, *n* = 198) had normal tracings on ECG with only 5.2% (*n* = 17) showing LVH. Abnormalities other than LVH were found in 34.4% (*n* = 113) of participants and included: sinus bradycardia (52.2%), left-axis deviation (14.2%), premature ventricular contractions (7.1%), right bundle branch block (4.4%), T-wave changes (4.4%) and left bundle branch block (2.6%).

On tests of associations between participants' characteristics and CV risk factors (Tables 3–5), age was significantly associated with current alcohol use ($p = 0.04$), exposure to second-hand smoke ($p = 0.00$) and physical inactivity ($p = 0.00$). Gender was significantly associated with being diabetic ($p = 0.03$), physically inactive ($p = 0.02$), current alcohol use ($p = 0.00$), obesity ($p = 0.00$), snuff use ($p = 0.00$) and cigarette smoking ($p = 0.00$). Race was significantly associated with cigarette smoking ($p = 0.00$), snuff use ($p = 0.01$), hypercholesterolemia ($p = 0.01$) and family history of fatal CV event ($p = 0.02$ for females and $p = 0.00$ for males). Marital status was associated with cigarette smoking ($p = 0.03$) and family history of fatal CV event ($p = 0.02$). Educational level was significantly associated with snuff use ($p = 0.03$) and family history of hypercholesterolaemia ($p = 0.00$). Lastly, employment status was significantly associated with physical inactivity ($p = 0.00$).

**Table 4.** Cardiovascular risk factors and gender

| Risk factors | Female (*n* = 260) | Male (*n* = 68) | Total (*n* = 328) | *p*-value |
|---|---|---|---|---|
| Alcohol use, *n* (%) | 56 (21.5) | 36 (53.4) | 92 (28) | 0.0000 |
| Cigarette smoking, *n* (%) | 19 (7.3) | 20 (29.4) | 39 (11.9) | 0.0000 |
| Snuff use, *n* (%) | 63 (19.2) | 1 (1.5) | 64 (19.5) | 0.0000 |
| Exposure to smoking, *n* (%) | 43 (16.5) | 4 (5.9) | 47 (14.3) | 0.08832 |
| Physical inactivity, *n* (%) | 182 (70) | 58 (85.3) | 240 (73.2) | 0.0221 |
| Diabetes mellitus, *n* (%) | 71 (27.3) | 28 (41.2) | 99 (30.2) | 0.0322 |
| High cholesterol, *n* (%) | 70 (26.9) | 17 (25) | 87 (26.5) | 0.3784 |
| Family history of cholesterol, *n* (%) | 14 (5.4) | 3 (4.4) | 17 (5.2) | 0.1626 |
| Fatal CV event, *n* (%) | 34 (13) | 15 (22) | 49 (15) | 0.4332 |
| BMI ≥ 30 kg/m², *n* (%) | 190 (73) | 26 (38.2) | 216 (65.8) | 0.0000 |
| Waist circumference, *n* (%) | 236 (90.8) | 29 (42.6) | 265 (80.8) | 0.0529 |
| Left ventricular hypertrophy, *n* (%) | 10 (3.8) | 7 (10.3) | 17/328 (5.2) | 0.07153 |

**Table 5.** Cardiovascular risk factors and race

| Risk factors | Asian (*n* = 1) | Black (*n* = 282) | Coloured (*n* = 3) | White (*n* = 42) | Total (*n* = 328) | *p*-value |
|---|---|---|---|---|---|---|
| Alcohol use, *n* (%) | 0 (0.0) | 73 (26) | 2 (66.6) | 17 (40.5) | 92 (24.2) | 0.0717 |
| Cigarette smoking, *n* (%) | 0 (0.0) | 24 (8.5) | 0 (0.0) | 15 (35.7) | 39 (11.9) | 0.0000* |
| Snuff use, *n* (%) | 0 (0.0) | 63 (22.3) | 1 (33) | 0 (0.0) | 64 (19.5) | 0.0081* |
| Exposure to smoking, *n* (%) | 0 (0.0) | 38 (13.5) | 0 (0.0) | 9 (21.4) | 47 (12.6) | 0.0866 |
| Physical inactivity, *n* (%) | 1 (100) | 203 (72) | 3 (100) | 33 (78.6) | 240 (73.2) | 0.5292 |
| Diabetes mellitus, *n* (%) | 1 (100) | 87 (30.1) | 2 (66.6) | 9 (21.4) | 99 (30.2) | 0.122 |
| High cholesterol, *n* (%) | 1 (100) | 63 (22.3) | 1 (33) | 22 (52.4) | 87 (26.5) | 0.0079* |
| Family history of cholesterol, *n* (%) | 1 (100) | 5 (1.8) | 1 (33) | 10 (24) | 17 (5.2) | 0.0000* |
| Fatal CV event, *n* (%) | | | | | | 49 (15) |
| Female | 0 (0.0) | 24 (8.5) | 0 (0.0) | 10 (24) | 34 (69.4) | 0.0215 |
| Male | 0 (0.0) | 7 (2.5) | 0 (0.0) | 8 (19) | 15 (30.6) | 0.0000 |
| BMI, *n* (%) | 1 (100) | 192 (68.1) | 2 (66.7) | 21 (50) | 216 (65.8) | 0.8794 |
| Waist circumference, *n* (%) | | | | | 265 (80.8) | |
| Female | 1 (100) | 203 (72) | 1 (33) | 31 (73.8) | 236 (90.8) | 0.3502 |
| Male | 0 (0.0) | 23 (8.1) | 1 (33) | 5 (12) | 29 (8.8) | 0.2968 |
| Left ventricular hypertrophy, *n* (%) | 0 (0.0) | 16 (5.7) | 0 (0.0) | 1 (2.4) | 17 (5.2) | 0.7213 |

**p*-value includes very small numbers of Asian and coloured participants to be statistically reliable. These two races with the white race were allocated as other race in the logistic regression (Table 6).

Table 6 shows the sociodemographic correlates of each CV risk factor in multivariate regression analysis. Compared to those aged 20–39 years, older patients were significantly more likely to report being physically inactive but less likely to report alcohol use.

Compared to women, men were more likely to report alcohol use, cigarette smoking, being physically inactive and having diabetes. Women on the other hand, were more likely to report using snuff and being obese. Black participants were significantly more likely to report snuff use compared to other racial groups, but less likely to report cigarette smoking, family history of hypercholesterolaemia, family history of fatal CV event and having hypercholesterolaemia. Compared to those in employment, pensioners were significantly more likely to report being physically inactive. Educational level and marital status did not correlate with any CV risk factor.

## Discussion

This study found that the prevalence of other CV risk factors among patients with hypertension was high. In addition, there were

**Table 6.** Sociodemographic determinants of CV risk factors

| Risk factor | Odds ratio | 95% | CI *p*-value |
|---|---|---|---|
| **Alcohol use** | | | |
| Age group, years | | | |
| 20–39 | 1.00 | | |
| 40–59 | 0.2227 | 0.0723–0.6853 | 0.0088 |
| 60–79 | 0.1830 | 0.0581–0.5764 | 0.0037 |
| 80+ | 0.2119 | 0.0185–2.4242 | 0.2121 |
| Gender | | | |
| Female | 1.00 | | |
| Male | 4.2939 | 2.3918–7.7088 | 0.0000 |
| **Cigarette smoking** | | | |
| Race | | | |
| Other | 1.00 | | |
| Black | 0.1543 | 0.0668–0.3567 | 0.0000 |
| Gender | | | |
| Female | 1.00 | | |
| Male | 6.2782 | 2.7958–14.0980 | 0.0000 |
| **Current snuff use** | | | |
| Education level | | | |
| Below secondary | 1.00 | | |
| Secondary or higher | 0.6100 | 0.3376–1.1021 | 0.1015 |
| Race | | | |
| Other | 1.00 | | |
| Black | 10.9513 | 1.4475–82.8551 | 0.0204 |
| Gender | | | |
| Female | 1.00 | | |
| Male | 0.0477 | 0.0065–0.3520 | 0.0028 |
| **Physical inactivity** | | | |
| Age group, years | | | |
| 20–39 | 1.00 | | |
| 40–59 | 0.6033 | 0.1806–2.0147 | 0.4114 |
| 60–79 | 0.8299 | 0.1753–3.9292 | 0.8141 |
| 80+ | 118865.6277 | 0.0000– > 1.0312 | 0.9641 |
| Employment | | | |
| Employed | 1.00 | | |
| Pensioner | 3.4727 | 1.1946–10.0953 | 0.02 |
| Unemployed | 1.7198 | 0.9188–3.2192 | 0.10 |
| Gender | | | |
| Male | 1.00 | | |
| Female | 0.4342 | 0.2162–0.8719 | 0.02 |
| **Diabetes mellitus** | | | |
| Gender | | | |
| Female | 1.00 | | |
| Male | 1.8634 | 1.0701–3.2448 | 0.0279 |
| **Hypercholesterolaemia** | | | |
| Race | | | |
| Other | 1.00 | | |
| Black | 0.3201 | 0.1131–0.9063 | 0.0319 |
| **Family history of hypercholesterolaemia** | | | |
| Race | | | |
| Other | 1.00 | | |
| Black | 0.1210 | 0.0296–0.4941 | 0.0033 |
| Education | | | |
| Below secondary | 1.00 | | |
| Secondary or higher | 0.7258 | 0.1094–4.8153 | 0.7399 |
| **Family history of fatal CV event** | | | |
| Race | | | |
| Other | 1.00 | | |
| Black | 0.1210 | 0.0296–0.4941 | 0.0033 |
| **BMI > 30 kg/m²** | | | |
| Gender | | | |
| Female | 1.00 | | |
| Male | 0.1859 | 0.1053–0.3283 | 0.0000 |

significant sociodemographic differences in the prevalence of each CV risk factor, and for each CV risk factor, the prevalence found in this study was higher than previously published in population-based studies in South Africa.[24-27] These findings have clinical and policy implications in that they suggest the presence of a high inherent risk of CVD among patients with hypertension in South Africa and call for interventions to address gaps in CV-risk screening and management, especially that each additional CV risk exponentially increases the risk of CVD in a patient with hypertension.[28]

Since most CV risk factors are modifiable, lowering BP alone without intervening in co-existing CV risk factors can therefore not be deemed optimal care. Regrettably, only four to 7% of patients with multiple CV risk factors receive appropriate risk-management interventions during clinical encounters,[12] signifying enormous missed clinical opportunities and the need for strategies to close this gap. Such strategies should include academic detailing of CV risk factors in the management of hypertension to improve healthcare providers' screening behaviours and prompt them to initiate management for these risk factors. Even when the burden of CV risk factors is assessed to be low, primary prevention should still be done, since the prevalence of CV risks tends to increase with age and if not attended, a relatively low burden of CV risks in the present may translate into higher lifetime risks of CVD in a patient with hypertension.[29]

In line with other African studies,[24,30] this study found that obesity is prevalent (65.8%) among patients with hypertension; higher than reported in two recent nationally representative population surveys in South Africa: SADHS 2016 (29.01%)[26] and SANHNES-1 (29.07%).[27] This is possibly due to clustering of CV risk factors in patients with hypertension.

The concurrent high prevalence of increased abdominal circumference (80.8%) in this study also reiterates the substantially higher risk of CVD in this population compared to the general population, especially since abdominal circumference is a strong predictor of adverse CV outcomes. Measurement of abdominal circumference should therefore form part of the vital signs in patients with hypertension during clinic visits in PHC. This is to ensure that healthcare providers respond to abnormal values by counselling on the need for weight loss, healthy diets and increased physical activity.[31-33] In addition, health education needs to be offered to dispel cultural myths that purport obesity as a symbol of wealth and wellbeing.[34]

Physical inactivity is a leading cause of mortality and there is a graded inverse relationship between physical activity and risk of CVD.[33] In this study, most participants (73.2%) reported being physically inactive (Table 3), far greater than the prevalence reported in previous South African studies.[35,36] A Libyan study has found a similar prevalence (74.5% among men and 75.5% in women).[37]

The implication of this finding is the enormous clinical and financial burden it places on the ever-stretched healthcare system in South Africa. This is dire, considering the relative risk for developing hypertension in sedentary men and women with normal BP at rest is 35 to 70% higher than in their physically active peers.[38] It is therefore important that clinic visits in primary care be used as opportunities to promote a physically active lifestyle, especially among patients with one or more CV risks. This is imperative in the light of the emerging epidemic of non-communicable diseases in South Africa.

Pensioners, men, blacks and participants of lower socioeconomic status were significantly more likely to report being physically inactive (Table 6). While previous studies in South Africa have reported poor engagement of old people in regular exercise,[39] the significantly higher odds of physical inactivity among men

(compared to women) is contrary to the literature[26,27] and may point to a potential measurement bias in that household chores were classified as moderate-intensity physical activity. It is well established that women engage in household chores more than men.[27]

Participants of lower socio-economic status were more likely to be physically inactive because of poor knowledge of the health benefits of physical exercise and/or the unavailability of social environment and amenities for engaging in physical exercise.[39] Population-based interventions need to address these gaps through health education, campaigns and provision of public facilities for exercise.

Cigarette smoking increases the risk of hypertension two-fold,[40] and environmental exposure to cigarette smoke increases the risk of adverse effects by at least 10%.[41] Although the smoking prevalence of 11.9% reported in this study is lower than the South African national figure (16.2%), it closely aligns with the racial, gender and age trends described in previous national surveys.[26,27] This result reflects the gains of the tobacco-control programme in South Africa. However, the 16.3% of participants who were non-smokers but exposed to environmental tobacco smoke raises serious cause for concern and indicates that screening for tobacco use should include enquiry about exposure to second-hand smoke, and if present, prompt discussions on how the patient can be protected, including exploring the enforcement of anti-smoking legislation.

The prevalence of snuff use found in this study was significantly higher than the South African national average (19.5 vs 6.7%),[36] and has implications in that a previous study among South African women reported higher but statistically insignificantly increased BPs among snuff users compared to non-users.[42] Such BP increases in a setting of high snuff use and multiple co-existing CV risks (as in this study), may translate into substantial risk of CVD at the population level. It is therefore imperative to promote cessation of snuff use among patients with hypertension, until results of well-designed longitudinal studies clarify the nature of this relationship.

Previous studies have shown that sociodemographic variables such as education, religious beliefs and socio-economic status influence smoking behaviours.[26,43,44] High smoking prevalence among the whites in this study can, firstly, be explained by income differentials, in that whites are less responsive to price and tax hikes implemented in the South African tobacco-control programme and continue to smoke at high rates. Secondly, the coloured (mixed ancestry) population, who are known to smoke more than other racial groups at a national level, were underrepresented in the population groups in the current study setting.

Studies have shown varying relationships between alcohol use and the odds of being hypertensive. While a higher mean number of standard drinks consumed[45] increases the odds, a reduction in alcohol consumption is associated with a reduction in blood pressure in a dose-dependent manner in both healthy and hypertensive participants, with an apparent threshold effect at two drinks per day.[45] The findings on alcohol use in this study (Tables 2, 6) are consistent with prevalence and sociodemographic trends described in recent nationally representative studies in South Africa; the highest prevalence occurring among whites (male or female) living in urban areas, who have more than secondary education and the highest wealth quintile.[26,27,46]

The findings that participants aged 20 to 39 years had a higher prevalence of alcohol use and were more likely to be physically inactive have been reported in a previous South African article.[46] Considering that these are young people, the cumulative effects of unattended co-existing CV risks over many years may place this cohort at substantially elevated risk of premature CVD-related morbidity and mortality later in life. This is more so since a dose–response relationship (strongest among black men) has been reported between alcohol use and coronary calcification.[47] Young patients with hypertension who have risky alcohol consumption behaviours should therefore be prioritised for intensified CV risk assessment and management.

The prevalence of type 2 diabetes found in this study (30.2%) was high and mirrors findings from other studies among patients with co-existing CV risks: physical inactivity (78.8%), obesity (66.7%), dyslipidaemia (41.4%), alcohol use (21.2%) and smoking (11.1%).[25,48-51] This clustering of CV risks in patients with diabetes underscores the necessity for more intensified screening and management of CV risks in this group.

Although previous studies have suggested increased risk of diabetes among women,[26] this study finds to the contrary. Being male was the only correlate of diabetes. This may reflect variations in the prevalence of CV risk across different populations. However, these findings may have clinical implications, especially that men in this study were also more likely to have other CV risks (Table 4).

Hypercholesterolaemia is a major risk factor for CVD[29] and was found in 26.5% of study participants. However, the true prevalence of hypercholesterolaemia could have been higher since 58.5% of participants either did not know their lipid profile or had never been tested. This highlights a significant gap in clinical practice in South African PHC and calls for strategies to increase healthcare providers' adherence to national guidelines on hypertension.

Most CVDs have hereditary and environmental risk components,[52] and a 14.9% prevalence of positive family history of premature fatal CVD suggests a high burden of familial predisposition to CVD in this population. Clinicians should therefore routinely screen for family history of CVD, noting that the odds of reporting a positive family history of fatal CVD is four times higher among races other than black people.[52] Since the pathological processes conferring increased risk of CVD in those with a positive family history of CVD (particularly macrovascular complications) start long before they become clinically evident, primordial and primary prevention at PHC level are crucial to deter or delay the onset of CVD.

In this study, most participants (60.7%) had their BP controlled to target, more than in a previous study in the same setting.[53] However, in the context of multiple risk factors, a systolic BP below 140 mmHg may still confer significant risk of CVD, since CV risk factors have differential effects on various CVD outcomes, and a patient with moderate levels of multiple risk factors could have a greater overall risk of CVD than a patient with a high risk in only one factor.[11]

While BP needs to be controlled to targets, CVD risk assessment needs to be personalised, and individualised interventions instituted during clinic visits. Poor BP control is generally commoner among black patients with hypertension, as is LVH.[53-55] It is therefore not surprising that most of the 5.2% of participants who had LVH in this study were black. Given that LVH is associated with a two- to four-fold increase in the risk of premature CV morbidity and mortality,[55] black patients need to be targeted for intensive BP control, LVH screening and management interventions that promote left ventricular remodelling.

### *Limitations and strengths*

Several limitations must be borne in mind in this study. Firstly, this was a cross-sectional study and the associations found are not causal in nature. Secondly, there was the potential for social desirability

bias, recall bias and reliance on self-reports, all of which could have resulted in information bias and possible misclassification. Thirdly, a substantial proportion of patients in the research setting were referred from other clinics for uncontrolled hypertension, which could have resulted in selection bias. Fourthly, only CV risk factors relevant to PHC were investigated and exclusion of some investigations, such as echocardiogram, could have underestimated CV risks such as LVH. Lastly, the study setting had an under-representation of coloured and Asian ethnic groups and the study findings may therefore not be representative of the overall South African population.

Most studies on CV risk factors in South Africa have been community or hospital based. One of the strengths of this study includes that it is one of the few studies that focused on CV risks among patients with hypertension in PHC. It also uncovered a high prevalence of co-existing CV risks among patients with hypertension in a peri-urban setting and highlights the substantial risk of CVD in South African PHC. Based on its findings, the study indicates that the PHC level of care must play a significant role in curbing the epidemic of CVD in South Africa.

## Conclusion

This study shows that the prevalence of CV risk factors among patients with hypertension in South African PHC is high, reflecting the clustering of CV risk factors and a high CVD risk in this population. While urgent preventative interventions are needed to address this enormous risk, such interventions must take cognisance of the sociodemographic disparities in prevalence of CV risk factors in South Africa.

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# Ellisras Longitudinal Study 2017: The relationship between waist circumference, waist-to-hip ratio, skinfolds and blood pressure among young adults in Ellisras, South Africa (ELS 14)

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## Abstract

***Background:*** **Obesity and hypertension are major risk factors for non-communicable diseases in the world today. The relationship between indicators of obesity and blood pressure needs attention in the rural South African population.**

***Aim:*** **This study examined the relationship between anthropometric parameters and blood pressure (BP) among young adults in the Ellisras rural area of South Africa.**

***Methods:*** **A total of 742 (365 females and 377 males) young adults aged 22 to 30 years, who were part of the Ellisras Longitudinal Study (ELS), participated in the research. Anthropometric and BP measurements were taken using the protocol of the International Society for the Advancement of Kinanthropometry (ISAK). Linear regression was used to determine the relationship between anthropometric parameters and BP. The risk of developing hypertension among young Elisras adults was evaluated using logistic regression.**

***Results:*** **The results indicted a higher but non-significant prevalence of hypertension in men (2.7%) than women (2.4%). Linear regression showed a significant positive ($p < 0.05$) association between waist circumference and systolic BP (beta = 0.273, 95% CI: 0.160–0.386), even after being adjusted for age and gender (beta = 253, 95% CI: 0.127–0.343). The risk for developing hypertension was significant ($p < 0.05$) for waist circumference (OR = 2.091, 95% CI: 1.129–3.871) after adjustment for age and gender.**

***Conclusion:*** **Of all anthropometric parameters, waist circumference was most significantly associated with BP ($p < 0.05$). Anthropometric indicators of obesity were strong predictors of hypertension among young adults in the Ellisras rural area.**

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**Keywords:** hypertension, skinfolds, waist circumference, waist-to-hip ratio, central obesity, adults

Hypertension is a significant global challenge that contributes to high risk of cardiovascular and kidney disease.[1] The prevalence of hypertension continues to increase in low- and middle-income countries,[2] and is mainly due to population growth, aging and behavioural risk factors such as unhealthy diet, which lead to obesity.[3]

Obesity usually refers to excessive storage of energy in the form of adipose tissue,[4] and remains a strong predictor for the risk of developing hypertension.[5] Moreover, a high prevalence of hypertension is found in individuals who are more obese or overweight than normal individuals, thus further increasing the risk for cardiovascular disease.[6] Studies have also shown that obesity increases fatty tissue, which heightens vascular resistance and overworks the heart to pump blood throughout the body, thereby elevating blood pressure.[7]

Studies have established that anthropometry is an easy and reliable method to use for predicting cardiovascular diseases (CVDs), including hypertension.[8,9] However, such studies were mostly conducted in children and fewer in adults, especially in rural areas.[10,11] In the same rural population, Monyeki *et al*.[12] reported skinfold thickness, waist circumference (WC) and body mass index (BMI) to have a significant positive association with blood pressure at a younger age (seven to 13 years). However, little is known about the association between WC, waist-to-hip ratio (WHR) and skinfold thickness with BP in young adults. Therefore, the aim of this study was to assess the relationship between anthropometric (WC, skinfold thickness, WHR) parameters and BP among young Ellisras adults aged between 22 and 30 years.

## Methods

Ellisras, now known as Lephalale, is a rural area based in Limpopo province, South Africa. Ellisras has approximately 42 settlements, with a population of about 5 000 people.[13] Ellisras village (23° 40'S, 27° 44'W) is about 70 km from the nearest settlement on the Botswana border.

In Ellisras, the Iscor coal mine and Matimba electricity station are the main sources of employment for the people, while other sources of livelihood include crop farming and cattle rearing, as few individuals are educated. Unemployment and poverty appear to be a major concern in South African rural areas, including Ellisras.[14]

Details of the Ellisras Longitudinal Study (ELS) research design and sampling procedure have been reported elsewhere.[15,16] For the purpose of this analysis, a total of 742 young adults aged 22 to

30 years (365 females and 377 males), who were part of the ELS, participated in this survey.

The ethics committee of the University of the North, now known as the University of Limpopo, granted approval prior to the survey. Participants read and signed informed consent forms. All subjects went through a series of anthropometric measurements based on the standard procedures recommended by the International Society for the Advancement of Kinanthropometry (ISAK).[17] Skinfold (triceps, biceps, subscapular and supraspinale) and height were measured using both the Martin anthropometer and Slim Guide skinfold calliper. Height was rounded off to the nearest 0.1 cm and skinfolds were measured three times, where the values were rounded off to the nearest 0.1 mm. A flexible steel tape was used to measure WC in centimetres as participants assumed a standing position. WC was measured sideways from midway between the lowest portion of the rib cage and iliac crest and anteriorly, midway between the xiphoid process of the sternum and the umbilicus.

Measurements for both systolic (SBP) and diastolic blood pressure (DBP) were taken at least three times with the electronic Micronta monitoring kit at an interval of five minutes, after the participants had been sitting for at least 15 minutes in a well-ventilated room.[18,19] The device contained an infrasonic transducer that keeps records of BP and pulse rate on the display screen. The device has been used for research and clinical purposes.[20] Readings taken with a conventional mercury sphygmomanometer and an automated device showed a high correlation ($r = 0.93$), based on a pilot study conducted before the actual survey.

Readings for intra- and inter-tester technical errors of measurement (% TEM) for height, skinfolds and WC ranged from 0.04 to 4.16 cm (0.2–5.01%), 0.2 to 6 mm (0.4–6.8%) and 0 to 3.4 cm (0–4%), respectively.[12]

### *Statistical analysis*

Descriptive statistics for WC, WHR and skinfold thickness were computed. Independent *t*-tests were calculated to examine whether there were any significant gender differences in the participants' anthropometric and haemodynamic measurements. Hypertension was defined as SBP ≥ 140 mmHg or DBP ≥ 90 mmHg, according to WHO.[21] Central obesity was assessed based on WC and WHR as follows: WC in men ≥ 102 cm and in women ≥ 88 cm, and WHR ≥ 0.5 cm in both men and women.[22] The sum of four skinfold measurements (triceps, biceps, subscapular and supraspinale), categorised as above the 85th percentile, was used to determine general obesity, where young adults were characterised as normal or excessively fat.[23]

**Table 1.** Descriptive statistics for anthropometric parameters, skinfold thickness and blood pressure among young Ellisras adults

| Variable | Men ($n$ = 364) | Women ($n$ = 375) | $p$-value |
|---|---|---|---|
| Age, years | 25.44 ± 2.60 | 25.52 ± 2.53 | 0.636 |
| SBP, mmHg | 125.33 ± 12.95 | 114.32 ± 10.23* | 0.000 |
| DBP, mmHg | 71.67 ± 10.11 | 69.43 ± 9.12* | 0.002 |
| Biceps skinfold, cm | 3.62 ± 2.10 | 10.70 ± 6.44* | 0.000 |
| Triceps skinfold, cm | 6.46 ± 4.30 | 12.41 ± 7.20* | 0.000 |
| Subscapular skinfold, cm | 8.71 ± 3.28 | 12.59 ± 6.80* | 0.000 |
| Supraspinale skinfold, cm | 5.11 ± 3.57 | 12.34 ± 7.19* | 0.000 |
| Waist-to-height ratio | 0.434 ± 0.082 | 0.509 ± 0.122* | 0.000 |
| Waist circumference, cm | 74.74 ± 9.56* | 82.49 ± 14.73* | 0.000 |

Mean ± SD; *$p < 0.05$.
SBP, systolic blood pressure; DBP, diastolic blood pressure.

**Table 2.** Prevalence of central obesity, overweight and hypertension among young rural Ellisras adults

| Variable | Men, $n$ (%) | Women, $n$ (%) | $p$-value* |
|---|---|---|---|
| High WC | 5 (1.4) | 134 (35.4) | 0.000 |
| High WHR | 110 (30.2) | 263 (69.6) | 0.000 |
| Overweight | 56 (15.4) | 57 (15.1) | 0.142 |
| High SBP | 51 (14.0) | 7 (1.9) | 0.013 |
| High DBP | 11 (3.0) | 9 (2.4) | 0.312 |

Hypertension 8 (2.2) 5 (1.3) 0.003WC, waist circumference; WHR, waist-to-hip ratio; SBP, systolic blood pressure; DBP, diastolic blood pressure.

The association between BP, WHR, WC and skinfolds, adjusted for age and gender, was assessed using a linear regression model. The risk of developing hypertension among young Ellisras adults who were obese or overweight was assessed with multinomial logistic regression analysis. Data were analysed using the Statistical Package for the Social Sciences (SPSS) (version 23), with the level of significance set at $p < 0.05$.

## Results

Table 1 presents descriptive statistics for anthropometric parameters, skinfolds and BP among young Ellisras adults aged 22 to 30 years. Women showed significantly ($p < 0.05$) higher mean average WC (82.49 ± 14.73 cm) than men (74.74 ± 9.56 cm) ($p < 0.000$). Men showed a significantly higher mean SBP (125.33 ± 2.60 mmHg) than women (114.32 ± 10.23 mmHg).

Presented in Table 2 are the summary data on the prevalence of hypertension, overweight and central obesity among the participants as evaluated using anthropometric parameters (WC, WHR and skinfold thickness). There was a higher significant ($p < 0.000$) prevalence of central obesity in women (69.6%) than in men (1.4%), while the prevalence of overweight was higher in men (15.4%) than women (15.1%), although not significant. High SBP was significantly ($p < 0.05$) more prevalent in men (14.0%) than in women (1.9%).

Table 3 indicates the Pearson correlation coefficients between anthropometric parameters, skinfold thickness and BP measurements. There were significant ($p < 0.05$) correlations between triceps ($r = 0.022$), biceps ($r = 0.021$) and subscapular ($r = 0.053$) skinfolds and SBP in women, while DBP in women was significantly ($p < 0.05$) associated with triceps ($r = 0.046$), biceps ($r = 0.007$) and subscapular ($r = 0.013$) skinfolds. In men, SBP significantly ($p < 0.05$) correlated with triceps ($r = 0.012$) and biceps ($r = 0.015$) skinfolds, while DBP was substantially ($p < 0.05$) correlated with biceps ($r = 0.017$) and subscapular ($r = 0.054$) skinfolds.

**Table 3.** Pearson's correlation coefficient between blood pressure and anthropometric parameters (skinfold thickness, WC and WHR)

| Variable | Triceps | Biceps | Subscapular | Supraspinale | WHR | Waist |
|---|---|---|---|---|---|---|
| Women | | | | | | |
| SBP | 0.022** | 0.021** | 0.053** | 0.140 | 0.175 | 0.237 |
| DBP | 0.046** | 0.007** | 0.013** | 0.093 | 0.136 | 0.684 |
| Men | | | | | | |
| SBP | 0.012** | 0.015** | 0.120 | 0.016 | 0.231 | 0.303 |
| DBP | 0.059 | 0.017** | 0.054** | 0.068 | 0.171 | 0.211 |

**Correlation is significant at the 0.05 level (two-tailed).
SBP, systolic blood pressure; DBP, diastolic blood pressure.

**Table 4.** Liner regression analysis for the association of WC, WHR and skinfold thickness with blood pressure

| | Unadjusted (for age and gender) | | | Adjusted (for age and gender) | | |
|---|---|---|---|---|---|---|
| **Variable** | **β** | ***p*-value** | **95% CI** | **β** | ***p*-value** | **95% CI** |
| Systolic blood pressure | | | | | | |
| Triceps | 0.229 | 0.171 | 0.556–0.099 | 0.397 | 0.013 | 0.709–0.085 |
| | | | 0.527–1.182 | | | 0.010–0.647 |
| | | | 1.420–0.697 | | | 0.603–0.160 |
| | | | 0.465–0.088 | | | 0.163–0.378 |
| | | | 0.143–0.064 | | | 0.025–0.059 |
| | | | 16.654–8.327 | | | 9.419–14.301 |
| | | | 0.160–0.386 | | | 0.127–0.343 |
| Subscapular | 0.854 | 0.000 | 0.318 | 0.057 | | |
| Biceps | 1.058 | 0.000 | 0.222 | 0.254 | | |
| Supraspinale | 0.188 | 0.181 | 0.108 | 0.435 | | |
| Sum of 4 skinfolds | 0.103 | 0.000 | 0.017 | 0.433 | | |
| WHR | 4.163 | 0.513 | 2.441 | 0.686 | | |
| WC | 0.273 | 0.000 | 0.253 | 0.000 | | |
| Diastolic blood pressure | | | | | | |
| Triceps | 0.377 | 0.004 | 0.633–0.122 | 0.412 | 0.002 | 0.669–0.155 |
| | | | 0.013–0.525 | | | 0.137–0.404 |
| | | | 0.392–0.172 | | | 0.216–0.412 |
| | | | 0.225–0.207 | | | 0.165–0.218 |
| | | | 0.053–0.006 | | | 0.037–0.031 |
| | | | 7.766–11.726 | | | 6.243–13.286 |
| | | | 0.053–0.230 | | | 0.036–0.213 |
| Subscapular | 0.269 | 0.039 | 0.133 | 0.333 | | |
| Biceps | 110 | 0.443 | 0.098 | 0.541 | | |
| Supraspinale | 0.009 | 0.935 | 0.058 | 0.608 | | |
| Sum of 4 skinfolds | 0.024 | 0.118 | 0.003 | 0.856 | | |
| WHR | 1.980 | 0.690 | 3.522 | 0.479 | | |
| WC | 0.141 | 0.002 | 0.124 | 0.124 | | |

Dependent variables: DBP and SBP.
WC, waist circumference; WHR, waist-to-hip ratio.

**Table 5.** Logistic regression analysis of association of anthropometric variables with hypertension among young Ellisras adults

| | Unadjusted (for age and gender) | | | Adjusted (for age and gender) | | |
|---|---|---|---|---|---|---|
| **Variable** | ***p*-value** | **OR** | **95% CI** | ***p*-value** | **OR** | **95% CI** |
| High systolic blood pressure | | | | | | |
| Abdominal obesity (WC) | 0.952 | 0.983 | 0.566–1.707 | 0.019 | 2.091 | 1.129–3.871 |
| | | | 1.012–2.801 | | | |
| | | | 0.415–1.051 | | | |
| Overweight | 0.045 | 1.634 | 0.460 | 1.229 | | 0.712–2.122 |
| Abdominal (WHR) | 0.080 | 0.660 | 0.830 | 1.061 | | 0.621–1.812 |
| High diastolic blood pressure | | | | | | |
| Abdominal obesity (WC) | 0.989 | 1.005 | 0.491–2.059 | 0.273 | 1.543 | 0.711–3.343 |
| | | | 0.741–2.590 | | | |
| | | | 0.592–2.009 | | | |
| Overweight | 0.308 | 1.385 | 0.676 | 1.147 | | 0.604–2.177 |
| Abdominal obesity (WHR) | 0.782 | 1.090 | 0.308 | 1.396 | | 0.735–2.653 |
| Hypertension | | | | | | |
| Abdominal obesity (WC) | 0.041 | 2.775 | 0.891–8.585 | 6.186 | 0.049 | 1.0073–7.993 |
| | | | 0.221–4.614 | | | |
| | | | 0.514–4.896 | | | |
| Overweight | 0.987 | 1.012 | 0.532 | 0.548 | | 0.068–4.175 |
| Abdominal obesity (WHR) | 0.416 | 1.596 | 1.906 | 0.314 | | 0.543–6.699 |

Dependent variables: DBP, SBP, hypertension.
WC, waist circumference; WHR, waist-to-hip ratio.

Table 4 presents linear regression coefficients for the association between anthropometric parameters and BP. The results exhibited a significant positive ($p < 0.000$) relationship between WC and SBP (beta = 0.273; 95% CI: 0.053–0.230), even after being adjusted for age and gender (beta = 2.091; 95% CI: 1.129–3.871). There was a significant positive ($p < 0.002$) association between WC and DBP (beta = 0.141; 95% CI: 0.053–0.230) when the data were unadjusted for age and gender. Triceps skinfold ($p < 0.004$) was significantly associated with DBP (beta = 0.377; 95% CI: 0.633–0.122), even after the data were adjusted for age and gender ($p < 0.002$) (beta = 0.412; 95% CI: 0.669–0.155).

Table 5 presents logistic regression analyses to determine the risk of developing hypertension among young Ellisras adults. High SBP was associated with abdominal obesity (WC) after adjusting for age and gender (OR = 2.091, 95% CI: 1.129–3.871). There was a significant association between high SBP and overweight (OR = 1.634, 95% CI: 1.012–2.801).

## Discussion

The purpose of the study was to determine the association between anthropometric parameters and BP among young Ellisras adults aged 22 to 30 years. In this study, WC was significantly associated with both SBP and DBP. This confirms the results of previous studies in which a significant positive association between WC and both SBP and DBP was reported among adults aged 23 to 40 years.[24,25]

Although studies have been conducted in different parts of the world, subjects of similar ages were targeted, therefore resulting in similar findings. Furthermore, a study carried out in adolescents aged 13 to 19 years found similar results.[9] However, Ashwell *et al*.[22] found that WHR was positively associated with SBP among adults. The study focused on individuals aged 60 years and older, therefore making the age difference a plausible explanation for the disparity in published research findings.

Our study also found that there was no significant association between both SBP and DBP and WHR. Contrary to this, Barbosa *et al*.[26] found WHR to be significantly associated with both SBP and DBP. Regarding skinfold thickness, the present study found that both SBP and DBP were significantly correlated with triceps, biceps and subscapular skinfolds among young Ellisras adults. Similarly, Birmingham *et al*.[27] reported a significant positive correlation between subscapular, triceps and biceps skinfolds and both SBP and DBP in individuals aged 18 to 40 years. Furthermore, the results agree with those of Dua *et al*.[28] and Timpson *et al*.,[29] which indicated a significant positive association between triceps, biceps and subscapular skinfolds and BP in adults.

In our study, men (1.9%) had a higher prevalence of hypertension compared to women (1.3%). Tesfaye *et al*.[30] also found the prevalence of hypertension to be higher in men (21.0%) than in women (16.4%). It has been reported that gender differences in the association between anthropometric variables and blood

pressure could be influenced by both biological and behavioural risks.[31] Biological factors include chromosome differences and sex hormones, which serve as a mechanism of protection against hypertension in most young women until they reach menopause.[32]

In contrast to our study, Luz *et al*.[33] reported the prevalence of hypertension to be 54.6 and 71.3% in men and women, respectively. The study focused more on older adults rather than young adults. As women grow older, their oestrogen levels decrease while the pituitary hormones increase, thus putting older women at a greater risk of developing hypertension than men.[34]

In our study, the prevalence of central obesity was found to be higher in women (35.4–69.6%) than in men (1.4–30.2%). Barbosa *et al*.[26] also found a higher prevalence of central obesity in women (65.1%) than men (40.1%), as did Munaretti *et al*.[35] (women 63.2%; men 18.7%). Women have a larger amount of body fat compared to men.[36] In addition, lifestyle risks such as excessive consumption of diets rich in refined fats, oil and carbohydrates contribute to the elevation of central obesity.[37] Most studies are in agreement with our study as they have also reported the incidence of general obesity to be higher in women than in men.[38,39] Al-Hazzaa *et al*.[40] reported that general obesity can be high in either men or women, taking into account their behavioural risk factors such as smoking and alcohol consumption.

We found that WC and overweight were significantly associated with hypertension and can best be used to predict the risk of hypertension in individuals who are obese. Sakurai *et al*.[25] also reported a strong association between WC and hypertension among Asians aged 19 to 33 years. Furthermore, Zhu *et al*.[24] found that WC, overweight and hypertension were significantly correlated in white Americans living in an urban setting.

However, our findings contradict those reported by Munaretti *et al*.[35] in which WHR was shown to be a significant predictor of hypertension. The contradiction between the two studies is probably because WHR is considered to have greater accuracy because of the nature of the measurements required, compared to WC and participants' age categories. The study setting was also different.[22] Individuals with high WC in our study were at a greater risk of developing hypertension.

Although the current study found WC and overweight to be the best predictors for hypertension, Hou *et al*.[41] reported that the prediction of individuals who are at a greater risk of developing CVD, specifically hypertension, can be improved by combining WC, WHR and BMI. These findings are consistent with those published previously.[24,42] However, Hans *et al*.[43] reported that the WC parameter has several advantages compared to other parameters because of its ease of measurement and interpretation in most clinical settings.

The present study did not include predisposing factors such as diet, lifestyle and level of physical activity for central obesity. Other important factors associated with hypertension, such as medical history, family history, alcohol intake and smoking, were not assessed. The study had a low sample size therefore its findings cannot be representative of all young adults in South Africa.

## Conclusion

The prevalence of hypertension was high in men compared to women. Hypertension was significantly related to WC and overweight (the sum of four skinfolds). It is vital to investigate the relationship between lifestyle and biological risk factors for cardiovascular disease over time.

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# Cardiovascular risk factors among people living with HIV in rural Kenya: a clinic-based study

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## Abstract

***Objectives:*** **To determine the prevalence of cardiovascular risk factors and their association with antiretroviral therapy (ART) among HIV-infected adults in a rural sub-county hospital in Kenya.**

***Methods:*** **This was a descriptive survey of patient charts characterising cardiovascular risk among adult patients (> 18 years) at Ukwala sub-county hospital between June 2013 and January 2015. Post-stratification survey weights were applied to obtain prevalence levels. Adjusted odds ratios (AOR) for each variable related to cardiovascular risk factors were calculated using logistic regression models.**

***Results:*** **Overall, the prevalence of diabetes mellitus was 0.4%, 0.3% of patients had had a previous cardiovascular event (heart attack or stroke), 40.4% had pre-hypertension, while 10.4% had stage 1 and 2.9% stage 2 hypertension. Up to 14% of patients had elevated non-fasting total cholesterol levels. Factors associated with hypertension were male gender (AOR 1.59, $p$ = 0.0001), being over 40 years of age (AOR 1.78, $p$ = 0.0001) and having an increased waist circumference (OR 2.56, $p$ = 0.0014). Raised total cholesterol was more likely in those on tenofovir disoproxil fumarate (TDF) (AOR 2.2, $p$ = 0.0042), azidothymidine (AZT) (AOR 2.5, $p$ = 0.0004) and stavudine (D4T)-containing regimens (AOR 3.13, $p$ = 0.0002).**

***Conclusions:*** **An elevated prevalence of undiagnosed cardiovascular risk factors such as hypertension and raised total cholesterol levels was found among people living with HIV. There was an association between raised total cholesterol and nucleoside reverse-transcriptase inhibitor (NRTI)-based ART regimens. Our findings provide further rationale for integrating routine cardiovascular risk-factor screening into HIV-care services.**

**Keywords:** people living with HIV, cardiovascular risk factors, antiretroviral therapy, hypertension, diabetes, hypercholesterolaemia, sub-Saharan Africa

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With the use and effectiveness of antiretroviral therapy (ART), people with HIV are living longer.[1] Non-AIDS events, of which cardiovascular disease (CVD) mediated by inflammation and atherosclerosis predominate, are becoming more prevalent.[2,3] A meta-analysis found that people living with HIV have a significantly higher risk for CVD when compared to HIV-negative persons.[4] This may be due to traditional cardiovascular risk factors such as smoking and hypertension, which have been found to be increased in some HIV-positive cohorts,[2,5] as well as ART,[6] exposure to HIV itself or immune activation and a pro-inflammatory state induced by HIV,[7] or a combination of these factors.

Although there are accumulating data on cardiovascular risk factors in people living with HIV in developed countries,[3] there are limited data from Africa. We report on the prevalence of risk factors for CVD among HIV-infected adults enrolled in HIV care and treatment at a sub-county hospital in Kenya, and describe the association with ART.

## Methods

This was a cross-sectional survey of patient charts characterising cardiovascular risk among adult patients (> 18 years) at Ukwala sub-county hospital between June 2013 and January 2015. Within this period, individuals with HIV attending Ukwala sub-county hospital for HIV care were screened for cardiovascular risk factors as part of a pilot project for integration of non-communicable disease care into HIV programmes supported by Grand Challenge Canada (GCC).

Ethical approval for this study was obtained from the Maseno University ethics review committee. Data used in this study were obtained from patient charts routinely collected at the clinic, and a written informed consent was provided before screening by each participant while attending the HIV clinic. Confidentiality, anonymity and privacy of all participants were guaranteed at all levels of this study by excluding all unique identifiers for the participants.

Baseline assessment included demographic variables, risk factors for CVD and measurement of body mass index (BMI), blood pressure, non-fasting total cholesterol and random blood glucose levels. World Health Organisation (WHO) cardiovascular risk score was calculated for patients aged above 40 years[8] and the information included in the patients' medical record files. All people with HIV

attending the Ukwala HIV clinic were included. Those who declined consent for the cardiovascular risk-factor screening and pregnant women were excluded.

Patients fulfilling national eligibility criteria (CD4 count > 350 cells/mm$^3$ at time of the study) were treated with standard ART according to national guidelines.[9] Standard regimens at that time included tenofovir, lamivudine and efavirenz (TNF/3TC/EFV) or zidovudine, lamivudine and efavirenz (AZT/3TC/EFV). Some were still receiving stavudine, lamivudine and efavirenz (D4T/3TC/EFV), which was being phased out at the time. A minority received a lopinavir/ritonavir (LPV/r)-containing regimen.

Prior to commencing CVD screening within the HIV clinics at Ukwala sub-county hospital, healthcare providers (including nurses, laboratory technologists, clinicians and data clerks) in the health facility received a two-day training, followed by regular intensive theoretical and practical skills training and mentoring in measuring and interpreting cardiovascular risk factors. The facility was also provided with regularly calibrated point-of-care diagnostic equipment for cardiovascular risk assessment.

Blood pressure (BP) was measured using a hospital-grade Omron M3® (Omron, Netherlands) digital automatic blood pressure machine. Hypertension diagnosis was based on standard guidelines, and included blood pressure measurements, medical history, physical examination, assessment of absolute cardiovascular risks (where deemed necessary by the examining physician) and laboratory investigations. A comprehensive assessment of BP involved multiple measurements taken on separate occasions, at least twice or three times, one or more weeks apart or sooner if the hypertension was severe.

Hypertension was defined as per the seventh report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure (JNC 7)10 as follows: pre-hypertension: systolic 120–139 mmHg, diastolic 80–89 mmHg; stage 1 hypertension: systolic 140–159 mmHg, diastolic 90–99 mmHg; stage 2 hypertension: systolic ≥ 160 mmHg, diastolic ≥ 100 mmHg, and those currently on antihypertensive drugs.

Total cholesterol and blood glucose levels were measured in the clinic using finger-prick blood by a Humansence® (Human, Wiesbaden, Germany) meter calibrated with a control strip on the first and after every 10th specimen. Raised total cholesterol level was defined according to US National Cholesterol Education Program ATP III guidelines.[11]

Data collection involved the extraction of data from the patients' charts using a standardised data tool by trained data clerks. Charts for patients who attended the clinic from June 2013 to January 2015 were targeted. Those with missing details on key variables such as cardiovascular risk-factor screening results and ART regimen were excluded from the data.

Detailed abstraction was then conducted on the remaining patients' charts using a data tool that was made up of four sections, including: (1) anthropometric measures (age, body mass index, waist circumference and blood pressure), (2) behavioural and biomedical cardiovascular risk factors (including smoking status, excessive use of alcohol and non-fasting total cholesterol level), (3) clinical information (such as on HIV infection and HIV treatment, ART regimen and duration), and (4) medical history. Data extracted were entered in a paper data tool then later transferred into an EpiData software version 3.1 for cleanup in readiness for analysis using SPSS software.

### *Statistical analysis*

Statistical analysis was performed using SPSS software version 22 (IBM SPSS Statistics, Armonk, NY: IBM Corp). Descriptive statistics involved calculating the median and interquartile range (IQR) for continuous data and proportions for categorical variables. Comparisons of median duration between groups were done using the Mann–Whitney test with a 5% level of significance. Associations were assessed using a logistic regression model, and crude and adjusted odds ratios are reported with their corresponding confidence intervals.

## Results

A total of 1 510 subjects was screened, of whom eight were excluded from analysis because of incomplete data (Fig. 1). Data collected included demographic variables, risk factors for CVD and determination of BMI, measurement of blood pressure, and non-fasting total cholesterol and random blood glucose levels. Cardiovascular risk score was calculated for those above 40 years using the WHO (Afri-E) risk-screening chart.[8]

Of the subjects screened, 69% (1 036) were women. The median age was 30 (IQR 31–48) years and median CD4 count was 430 (IQR 308–574) cells/mm$^3$; 79% of subjects were on ART with a documented regimen. Current smokers were 1.9% (29), whereas 0.4% (seven) had known diabetes and 0.3% (four) had had a previous cardiovascular event (heart attack or stroke).

The median BMI was 21 (IQR 20–23) kg/m$^2$ with 11% of subjects underweight, 12% overweight and 2% obese. Waist circumference was > 100 cm (102 cm) in 1% of men and > 90 cm (88 cm) in 7.5% of women (Table 1). The median duration on ART was 32.5 (17.4–50.6) months. Cardiovascular risk-factor distribution stratified by ART status is shown in Table 2.

Of the 1 502 individuals screened, 40.4% (609/1502) had pre-hypertension, 10.4% (157/1502) were stage 1 and 2.9% (43/1502) were stage 2 hypertension. In multivariate analysis, hypertension was associated with being male [adjusted OR 1.59 (1.26–2.01), $p$ = 0.0001], being 40 years or older [adjusted OR 1.78 (1.44–2.21), $p$ = 0.0001], and having an increased waist circumference [adjusted OR 2.56 (1.44–4.55), $p$ = 0.0014].

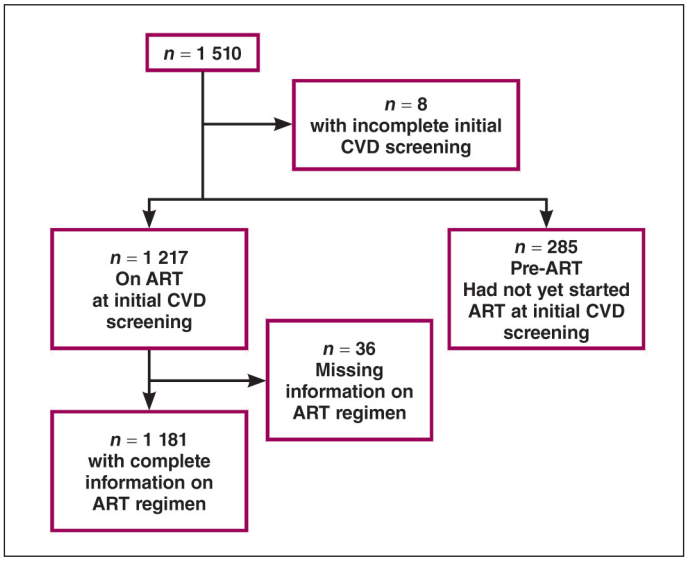


**Fig. 1.** Data flow chart for cardiovascular risk screening.

**Table 1.** Prevalence of cardiovascular risk factors among people living with HIV

| Variables | Frequency (*n*) | Overall % |
|---|---|---|
| Age ≥ 40 years | 716 | 47.4 |
| Male gender | 471 | 31.2 |
| Current smokers | 29 | 1.9 |
| Increased waist circumference* | 89 | 5.9 |
| Total cholesterol ≥ 5.2 mmol/l | 207 | 13.7 |
| Body mass index ≥ 25 kg/m² | 182 | 12.1 |
| Random blood glucose ≥ 7.8 mmol/l | 31 | 2.1 |
| Known diabetes at screening | 7 | 0.5 |
| Cardiovascular risk score ≥ 10% | 8 | 0.5 |
| Pre-hypertension | 609 | 40.4 |
| Hypertension stage 1 | 157 | 10.4 |
| Hypertension stage 2 | 43 | 2.9 |

*Females ≥ 90 cm, males ≥ 100 cm.

While the association between lower CD4 count and prevalence of hypertension was not certain, lower CD4 count was indicative of a lower prevalence of hypertension among those with low CD4 counts. There was no association between hypertension and current ART regimen (Table 3). The median duration on ART was not significantly different for those with or without hypertension (Mann–Whitney test, $p = 0.6794$).

A total of 207 (14%) patients had an elevated non-fasting total cholesterol level (> 5.2 mmol/l). On multivariate analysis, being above 40 years of age [adjusted OR 1.95 (1.42–2.69), $p = 0.001$] and having an increased waist circumference [adjusted OR 2.06 (1.14–3.71), $p = 0.0164$] were associated with having a raised total cholesterol level. In addition, raised total cholesterol was more likely in those on TDF [adjusted OR 2.20 (1.28–3.78), $p = 0.0042$], AZT [adjusted OR 2.50 (1.50–4.18), $p = 0.004$] and D4T-containing regimens [adjusted OR 3.13 (1.72–5.71), $p = 0.002$]. However, the median duration on ART was not significantly different for those with or without a raised total cholesterol level (Mann–Whitney test, $p = 0.1261$). There was no

**Table 2.** Cardiovascular risk factors stratified by ART status

| CVD risk factors | ART, *n* (%) | Pre-ART, *n* (%) | *p*-value | OR (95% CI) |
|---|---|---|---|---|
| Male gender | 396 (32.5) | 74 (26.0) | 0.0312 | 1.38 (1.03–1.84) |
| Age ≥ 40 years | 629 (51.7) | 87 (30.5) | 0.0001 | 2.43 (1.85–3.21) |
| Current smokers | 24 (2.0) | 5 (1.8) | 0.8100 | 1.13 (0.43–2.98) |
| Total cholesterol ≥ 5.2 mmol/l | 186 (15.3) | 19 (6.7) | 0.0001 | 2.53 (1.55–4.13) |
| Body mass index ≥ 25 kg/m² | 151 (12.4) | 31 (10.9) | 0.4761 | 1.16 (0.77–1.75) |
| Elevated waist circumference* | 76 (6.2) | 13 (4.6) | 0.2786 | 1.39 (0.76–2.55) |
| Random blood glucose ≥ 7.8 mmol/l | 25 (2.1) | 5 (1.8) | 0.7447 | 1.17 (0.45–3.10) |
| Cardiovascular risk score ≥ 10% | 6 (0.5) | 2 (0.7) | 0.6630 | 0.70 (0.14–3.49) |
| Known diabetes | 6 (0.5) | 1 (0.4) | 1.000 | 1.41 (0.17–11.70) |
| Hypertension | 666 (54.7) | 140 (49.3) | 0.0985 | 1.24 (0.96–1.61) |
| Pre-hypertension | 494 (47.3) | 113 (44.0) | 0.3415 | 1.14 (0.96–1.61) |
| Hypertension stage 1 | 132 (19.3) | 24 (14.3) | 0.1303 | 1.44 (0.90–2.30) |
| Hypertension stage 2 | 40 (6.6) | 3 (2.0) | 0.0291* | 3.48 (1.06–11.42) |

*Females ≥ 90 cm, males ≥ 100 cm; Fischer's exact two-sided test.

**Table 3.** Unadjusted and adjusted odds ratios for hypertension

| | Unadjusted | | OR Adjusted OR | |
|---|---|---|---|---|
| Characteristic | OR (95% CI) | *p*-value | OR (95% CI) | *p*-value |
| Male gender | 1.65 (1.32–2.06) | 0.0001 | 1.68 (1.32–2.14) | 0.0001 |
| Age ≥ 40 years | 2.06 (1.67–2.53) | 0.0001 | 1.78 (1.43–2.22) | 0.0001 |
| Current smokers | 1.42 (0.67–3.02 | 0.3653 | 1.02 (0.47–2.24) | 0.9574 |
| Body mass index ≥ 30 kg/m² | 3.14 (1.35–7.31) | 0.0079 | 1.47 (0.55–3.94) | 0.4421 |
| Random blood glucose ≥ 7.8 mmol/l | 1.13 (0.54–2.34) | 0.7459 | 1.09 (0.51–2.33) | 0.8225 |
| Increased waist circumference* | 2.95 (1.79–4.87) | 0.0001 | 2.49 (1.39–4.43) | 0.0020 |
| ART regimen | | | | |
| No ART | Ref | Ref | Ref | Ref |
| TDF-based | 1.18 (0.88–1.58) | 0.2794 | 1.10 (0.81–1.49) | 0.5506 |
| AZT-based | 1.40 (1.07–1.84) | 0.0152 | 1.26 (0.95–1.68) | 0.1090 |
| D4T-based | 1.42 (0.97–2.06) | 0.068 | 1.22 (0.82–1.81) | 0.3226 |
| LPV-based | 0.91 (0.41–2.03) | 0.8212 | 0.97 (0.42–2.25) | 0.9385 |
| CD4 count (cells/mm³) | | | | |
| Missing | 0.71 (0.53–0.94) | 0.0165 | 0.72 (0.54–0.97) | 0.0307 |
| 0–100 | 0.47 (0.23–0.92) | 0.0287 | 0.49 (0.24–0.99) | 0.0472 |
| 101–200 | 0.78 (0.47–1.29) | 0.3369 | 0.67 (0.40–1.14) | 0.1406 |
| 201–350 | 0.67 (0.50–0.91) | 0.0112 | 0.59 (0.43–0.82) | 0.0015 |
| 351–500 | 0.84 (0.62–1.12) | 0.2315 | 0.77 (0.56–1.04) | 0.0882 |
| > 500 | Ref | Ref | Ref | Ref |

*Females ≥ 90 cm, males ≥ 100 cm.

**Table 4.** Unadjusted and adjusted odds ratios for elevated total cholesterol

| | Unadjusted | | OR Adjusted OR | |
|---|---|---|---|---|
| **Characteristic** | **OR (95% CI)** | ***p*-value** | **OR (95% CI)** | ***p*-value** |
| Male gender | 0.85 (0.61–1.17) | 0.3194 | 0.83 (0.59–1.17) | 0.2806 |
| Age ≥ 40 years | 2.21 (1.63–3.00) | 0.0001 | 1.95 (1.42–2.69) | 0.0001 |
| Smoker | 0.22 (0.03–1.64) | 0.1404 | 0.22 (0.03–1.67) | 0.1434 |
| BMI ≥ 30 kg/m² | 2.15 (0.95–4.86) | 0.0647 | 1.03 (0.39–2.74) | 0.946 |
| Random blood glucose ≥ 7.8 mmol/l | 1.96 (0.83–4.62) | 0.1252 | 1.99 (0.82–4.81) | 0.1278 |
| Increased waist circumference* | 2.68 (1.64–4.36) | 0.0001 | 2.06 (1.14–3.71) | 0.0164 |
| ART regimen | | | | |
| No ART | Ref | Ref | Ref | Ref |
| TDF-based | 2.47 (1.45–4.22) | 0.0009 | 2.20 (1.28–3.78) | 0.0042 |
| AZT-based | 2.84 (1.72–4.71) | 0.0001 | 2.50 (1.50–4.18) | 0.0004 |
| D4T-based | 3.86 (2.14–6.95) | 0.0001 | 3.13 (1.72–5.71) | 0.0002 |
| LPV-based | 1.98 (0.55–7.17) | 0.2968 | 1.85 (0.50–6.80) | 0.3536 |
| CD4 count (cells/mm³) | | | | |
| Missing | 0.74 (0.49–1.11) | 0.147 | 0.87 (0.57–1.33) | 0.5217 |
| 0–100 | 1.04 (0.42–2.60) | 0.9306 | 1.13 (0.44–2.92) | 0.7964 |
| 101–200 | 0.48 (0.20–1.16) | 0.1029 | 0.46 (0.19–1.13) | 0.0885 |
| 201–350 | 0.79 (0.51–1.22) | 0.2884 | 0.79 (0.50–1.25) | 0.3174 |
| 351–500 | 0.93 (0.62–1.39) | 0.7106 | 0.92 (0.60–1.41) | 0.6951 |
| > 500 | Ref | Ref | Ref | Ref |

*Females ≥ 90 cm, males ≥ 100 cm.

significant association between CD4 count and total cholesterol level (Table 4).

Thirty-one (2.1%) subjects had a random blood glucose level of > 7.8 mmol/l. These patients were referred to the physician for fasting glucose determination and/or oral glucose tolerance tests.

Eight (0.55%) of those above 40 years of age had more than 10% risk of developing a major adverse cardiovascular event in 10 years, according the WHO (Afri-E) risk score performed on these clients.

## Discussion

In this study, cardiovascular screening of people living with HIV revealed a significant prevalence of undiagnosed hypertension (13.3%) and raised total cholesterol levels (14%), two of the major cardiovascular risk factors. Possible aetiological factors for hypertension include traditional risk factors (such as age, gender, smoking and obesity), ART, or possibly HIV infection itself. Our analysis of risk factors indicated significant associations between the occurrence of hypertension and male gender, older age (> 40 years) and increased waist circumference. There was however no association between ART regimen and hypertension, suggesting that other factors may have been contributory.

In a population survey targeting a peri-urban community in Nairobi, prevalence of hypertension was 22%,[12] which is higher than seen in this study. One of the possible reasons for this disparity is that despite living with HIV, the age of this cohort was relatively young and with fewer smokers compared to those reported in the general population (2015 Kenya STEPS survey). Also, the prevalence of other known risk factors for hypertension such as overweight and obesity was at 14%, well lower than reported in the national STEPS survey (27%).

In another retrospective review of data from an HIV-positive population in western Kenya, the prevalence of hypertension was 11.2% in men and 7.4% in women.[13] The figures observed in this review compare well with those found in our study.

Possible aetiological factors for high cholesterol levels include genetic factors, diet, ART or HIV infection itself. After adjusting for confounders, elevated cholesterol level was associated with three ART regimens (TDF, AZT and D4T) suggesting a potential causal relationship. However, since a full lipid profile was not performed, it remains unclear if this was due to a raised low-density lipoprotein cholesterol level.

A study in Tanzania showed a high prevalence of dyslipidaemia (low high-density lipoprotein cholesterol and elevated triglyceride levels) in an ART-naïve cohort of HIV patients.[5] There is therefore a need for further research to illustrate the role of ART therapy on the patterns of dyslipidaemia.

The prevalence of smoking, obesity, glucose intolerance and diabetes were low in this population at 1.9, 12.1 and 2.6%, respectively, and only 0.6% had a WHO cardiovascular risk score > 10%. This is much lower compared to the peri-urban population study of Nairobi where 10% were smokers, 5% had diabetes, and more than 40% had central obesity.[12] Our rural hospital setting may present a different HIV population where disease and lifestyle advice provided to the patients may have altered risk factors, particularly smoking incidence.

With increasing longevity of people living with HIV, the prevalence of hypertension, hyperlipidaemia and glucose intolerance is likely to increase. Therefore routine and systematic screening for cardiovascular risk factors among this population is crucial. The majority of cardiovascular risk factors, also seen in people with HIV, such as smoking, hypertension and obesity, are modifiable, therefore early identification and treatment of these conditions provides an opportunity to improve the quality of care and possibly survival rate in this population. Existing studies conducted in sub-Saharan Africa suggest there is little knowledge of the risk posed by CVD in this population.[14] There is therefore a need to establish CVD care in HIV programmes to potentially mitigate adverse cardiovascular events in these patients.[15]

This study has several limitations, including collecting data from patient charts at one time point. Further studies are needed to establish how screening, referral and evidence-based interventions could reduce cardiovascular risk of people living with HIV in rural Kenya and beyond. Cardiovascular risk was determined after a median duration of 32 months of ART. A longer period of observation may be required to detect transition in cardiovascular risk. However the high prevalence of hypertension indicates that there was a considerable amount of undiagnosed incipient and actual hypertension in this population. Lastly, fasting lipid profiles were not performed where elevated non-fasting values were found, and inferences from an elevated total cholesterol level may not accurately reflect the prevalence of hypercholesterolaemia. However, recent guidelines advocate the use of non-fasting cholesterol tests.[16] Our data are from 2013 to 2016, and the situation in terms of ART regimens and cardiovascular risk may have changed since then.

## Conclusion

CVD screening in a primary HIV-care clinic revealed a high prevalence of undiagnosed hypertension and raised total cholesterol levels,

and suggests an association between raised total cholesterol level and nucleoside reverse-transcriptase inhibitor (NRTI)-based ART regimens in an HIV-infected African population. Our findings provide further rationale for integrating routine cardiovascular risk-factor screening into HIV-care services in resource-limited settings. Larger studies with more detailed investigations and longer follow up are recommended.

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# Weight gain in early childhood increases heart and metabolic risk in adolescence

Earlier onset of weight gain, under two years of age, is associated with higher cholesterol, higher blood pressure, and more central fat in adolescence, compared with onset of weight gain in children aged three to five, finds a 14-year University of Sydney study.

The study tracked the body mass index (BMI) of children from birth to 14 years and found that earlier onset of high BMI (in children under two years) resulted in higher cholesterol levels, higher blood pressure, and more central (unhealthy) fat in adolescence, compared with onset of high BMI in children aged three to five years.

Teenage obesity is a major health problem in Australia, but the pathways to and the consequences of obesity in teenagers has not been well studied. This is the first study to look at the consequences of weight gain at two different stages of early childhood and its impact on developing cardiovascular disease as an adult.

'Our study found that there are two main pathways to obesity as a teenager – rapid weight gain in the first two years of life (early weight gain) or rapid weight gain between ages two and five years of age (later weight gain),' said senior author University of Sydney's Professor David Celermajer, Scandrett professor of cardiology at Sydney Medical School and the Heart Research Institute.

'The data show that there are consequences of the timing of the onset of excess BMI in early childhood. Earlier onset of a rising BMI that persisted through childhood results in greater central fat and higher cholesterol in teenagers, independent of their BMI at 14 years.'

A group of 410 Australian children were assessed from birth throughout childhood to age 14 years, recording their weight, height, and waist circumference. Of the 410 children, 190 had detailed measurements of cholesterol, blood pressure and central weight recorded at age 14 years. Three groups were identified in the study: normal BMI, 'early rising' excess BMI from two years, and 'late rising' excess BMI from five years.

Lead author Dr Jennifer Barraclough, cardiologist and PhD student at University of Sydney and the Heart Research Institute said: 'The early weight-gain group have more centrally placed or unhealthy fat than the later weight gain group. Fat around the middle is a key risk factor for cardiovascular disease in adulthood. The early weight-gain group also had significantly higher cholesterol levels compared to a group of healthy weight teenagers.

'Our study shows that the earlier the onset of excess fat before five years of age, the more likely the individual is to have fat around the middle by adolescence. The study also found that both early and late-weight gain groups were more likely to have mothers with overweight or obesity and a high BMI, than healthy weight teenagers.'

Co-author Professor Louise Baur, head of child adolescent health at the University's Sydney Medical School and The Children's Hospital Westmead said: 'This study has shown that it is important for families and the community to understand the risks of excess weight gain in early life and to ensure healthy eating and activity are supported from a very young age.

'These findings may provide an opportunity to identify "high risk" young children and trial interventions at an early age, prior to the development of high cholesterol and centrally placed fat, which becomes evident in adolescence and increases the risk of heart disease as an adult.'

Baur highlighted the importance of healthy infant feeding. 'Breastfeeding should be supported where possible until at least 12 months, with solids introduced from around six months. Healthy eating and physical activity for all family members is also an important factor promoting healthy weight gain in the young child. Family doctors and early childhood nurses can also help to monitor weight gain in this critical period of life,' she said.

*Source: Medical Brief 2019*

# The effect of exercise on diabetes management

TJ ELLAPEN, A BROODRYK, Y PAUL, P BUYS

## Abstract

**Regular exercise is a fundamental component of any strategic diabetes-management plan. Habitual exercise provides numerous physiological benefits to diabetic patients. It is however naïve to believe that exercise does not induce risk factors that may adversely impact on the health of a diabetic patient. The objective of this clinical commentary is to increase awareness of both the benefits and risks induced by exercise among diabetic patients.**

**Keywords:** diabetes, exercise, benefits, risks

Diabetes mellitus is classified as a metabolic disease, which entails either an absolute (type 1 diabetes mellitus) and/or a relative (type 2 diabetes mellitus) insulin deficiency.[1] Insulin-dependent diabetes (type 1 diabetes mellitus) is an autoimmune condition, caused by the assault on the person's pancreas by the body's own antibodies. This has an impact on the beta-cells within the pancreas, resulting in altered insulin production. Treatment for insulin-dependent diabetes involves the injection of exogenous insulin.

Non-insulin-dependent diabetics (type 2 diabetes mellitus) produce insulin, which may be insufficient or their cells are resistant to it. Type 2 diabetes can produce significant health complications, such as diabetic retinopathy, diabetic neuropathy, diabetic kidney disease, and increase the risk of coronary artery diseases (CAD) and strokes. Non-insulin-dependent diabetes can be managed through medication, a proper nutritional plan, regular exercise and reduction in body fat.

In 2017, it was estimated that 451 million people were diagnosed with diabetes globally; a number which, as predicted by the International Diabetes Federation, may escalate to 693 million by the year 2045.[2]

Hunter-Adams *et al*. reported that the incidence of diabetes in Africa will increase by 110% in the period from 2013 to the 2035. Furthermore, the prevalence of diabetes in South Africa will increase concomitantly with the overarching African continental epidemic.[3,4] A concerning statistic is that only 43 to 50% of all diabetic patients in Africa are presently diagnosed.[3]

Durstine *et al*. reported that 80% of type 2 diabetes patients are obese, adversely contributing to their pathology by increasing insulin resistance.[1] Obesity is characterised by excessive body fat, with an inverse decrease in lean muscle mass, decreasing an individual's blood glucose uptake and thereby predisposing the person to diabetes mellitus.[1] The 2015 Global Burden of Disease report identified hyperglycaemia and obesity as predisposing risk factors contributing to premature death and disability among many South Africans.[5]

The World Health Organisation (WHO) has identified physical inactivity (a sedentary lifestyle), commonly observed among diabetic patients, as a leading cause of premature death.[6] A person is considered physically active if the individual performs moderate accumulative aerobic activity for 150 minutes/week, with an exercise intensity ranging between three and six METs (metabolic equivalent) or vigorous physical activity for 75 minutes/week at an intensity greater than six METs.[6] One who does not comply with these criteria may be considered physically inactive. Mickelsfield *et al*. reported that 43 to 49% of all South Africans are physically inactive.[7] In 2012, the South African Health Review reported that of approximately 1 032 981 people identified as diabetic, only 57.4% were pharmacologically treated.[4] In 2016, the Global Health Estimate reported that 972 0000 South Africans had died from diabetes.[8]

Presently diabetes-management strategies are centred on identification, chronic medication, and dietary and exercise counselling.[9] An electronic Google Scholar search (limited to articles published in 2018) revealed 40 800 publications indicating the benefits of exercise for diabetic patients. Despite the impressive number of publications regarding the benefits of exercise for diabetic patients, many South Africans still do not incorporate regular exercise into their diabetes-management strategy.

Anecdotal reports suggest that numerous diabetic patients interested in making regular exercise a part of their management strategy simply join a health and wellness or fitness club and commence exercising without proper nutritional and exercise supervision. This commentary aims to increase awareness regarding the beneficial impact of exercise among diabetic patients, taking the potential risks induced by exercise into consideration and offering recommendations in order to enable patients to better avoid these risks.

## The benefits of exercise and physical activity

Regular structured exercise is beneficial to diabetic patients and offers the following advantages.

- Regular structured exercise lowers blood glucose concentration during and after an exercise session.[10] Regular prolonged aerobic exercise at moderate intensity level increases glucose absorption, which reduces the hyperglycaemic state of diabetic patients. This, coupled with increased insulin sensitivity, further increases glucose absorption, thereby maintaining a lower glucose state among diabetic patients. It is therefore important that the diabetic patient carry snacks with him/her during exercise in the event of exercise-induced hypoglycaemia. Moreover, post-exercise meals are essential for the maintenance of a

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normal energy metabolism. Regular exercise decreases diabetic patients' hyperglycaemic state, which warrants adjustment in insulin medication.[11]

- Chronic resistance training increases skeletal muscle mass, which, in turn, is responsible for 80% of insulin-mediated glucose uptake.[12] The increased insulin-mediated glucose uptake is derived from the breakdown of triglycerides into fatty acids and glycerol (lipolysis) for the provision of energy. This physiological benefit decreases blood glucose and adipose tissue, which is beneficial to diabetic patients with the co-morbidity of obesity.[12]
- Enhanced insulin sensitivity warrants exogenous insulin adjustment.[13] Insulin is a hormone that converts glucose to glycogen, thereby reducing blood glucose levels. Increased insulin sensitivity allows lower dosages of insulin to more readily facilitate this function, thereby preventing the pancreas from secreting copious amounts of insulin, and thus helping to prevent pancreatic hyperactivity. Insulin binds to the insulin receptor, which then results in subcellular signalling and GLUT-4 translocation. Diabetic patients experience decreased insulin sensitivity due to a myriad possible errors ranging from problems with the binding of insulin to the insulin receptor, to erros in the subcellular signalling or GLUT-4 translocation. When a diabetic patient exercises, there is a concomitant decrease in the amount of insulin secreted, which upregulates the sensitivity of the insulin receptors, enabling them to better identify the presence of blood glucose, thus increasing glucose absorption into the muscle groups that are being exercised.[12] Habitual muscle strength training increases the resting metabolic rate of the patient, increasing blood glucose uptake without augmenting insulin secretion.[12]
- Regular structured exercise results in reduced plasma triglyceride and cholesterol levels. During prolonged aerobic exercise, triglycerides are broken down into fatty acids and glycerol in order to be converted into glucose.[12] This is an additional benefit for diabetic patients with the co-morbidities of obesity and hypertension. Regular exercise helps to reduce obesity and hypertension.[1] Obesity has been associated with insulin resistance, which inhibits cells from readily identifying insulin (decreasing insulin sensitivity).[1] Exercise-induced hyperglycaemia may be affected by the exercise intensity. Higher exercise intensities are more likely to promote enhanced hepatic glycogenolysis (the decomposition of glycogen to glucose, occurring in the liver in response to hormonal and neural signals), resulting in hyperglycaemia.

## The risks that diabetic patients should be aware of when exercising

During exercise, diabetic patients undergo various cardiovascular and hormonal changes aimed at ensuring an adequate supply of glucose in order to meet the energy demand required by the physical activity in which they are engaged. As a result, the patient may experience exercise-induced hypoglycaemia, exercise-induced hyperglycaemia, exercise-induced ketosis and post-exercise hypoglycaemia.

- Exercise increases post-exercise insulin sensitivity, which increases glucose re-absorption in both exercised muscles and the liver in an attempt to replenish glycogen stores. This physiological phenomenon is called exercise-induced hypoglycaemia, which can have severe, harmful effects on diabetic patients. It is therefore imperative to amend the injected exogenous insulin dosage after exercising in order to maintain a healthy energy balance.[14,15]
- Exercise increases insulin absorption, which alters glucose metabolism. Exercise-induced insulin absorption is further increased when a patient injects insulin shortly before an exercise bout or uses fast-acting insulin. It is therefore recommended that patients exercise 60 to 90 minutes after insulin injections only. The high level of insulin during exercise increases the conversion of glucose to glycogen, while supressing glycogenolysis (breakdown of glycogen to glucose) and gluconeogenesis (glucose formation from non-carbohydrates), which may lead to exercise-induced hypoglycaemia.[15]
- Many diabetic patients may furthermore run the risk of developing exercise-induced ketosis. Prolonged exercise increases peripheral glucose absorption of the exercising muscles, thereby stimulating lipolysis (breaking down of triglycerides into glycerol and fatty acids for the use of energy) and hepatic glucose production (the formation of glucose from lactate and amino acids within the liver, primarily regulated by insulin and glucagon) and ketogenesis (the breaking of fatty acids for energy, producing ketones).[16] It is recommended that diabetic patients check their blood glucose and urine ketone levels before commencing an exercise session. If their blood glucose concentration is higher than 250 mg/dl (13.88 mmol/l) and/or ketones or blood are visible in their urine, exercise should be postponed and the exogenous insulin dosage adjusted.[15]
- After exercise the individual experiences a state of exercise-induced hyperglycaemia for a period of five to 15 minutes. This is due to the need for increased glucose re-absorption into the exercised muscle in order to replenish the glycogen stores. In diabetic patients, the state of exercise-induced hyperglycaemia may however occur for a period far longer than the normal five to 15 minutes, and this may lead to adverse conditions.[14] Careful monitoring of blood glucose levels during and after exercise is therefore essential in order to prevent this scenario. Many diabetic patients experience enhanced insulin sensitivity, resulting in increased re-absorption of glucose, producing a state of post-exercise hypoglycaemia. Frontera *et al*. and Trefts *et al*. postulated that this occurs due to increased insulin sensitivity, which allows more glucose to be absorbed into the previously exercised muscles, only to be converted and stored as glycogen.[16,17] Various recommendations have been proposed concerning overcoming post-exercise hypoglycaemia, including decreasing the pre-exercise dosage of insulin, avoiding fast-acting exogenous insulin immediately before exercising, and the consumption of a balanced and appropriate post-exercise meal.[15,17]

## Exercise therapists in South Africa

Physiotherapists and biokineticists are Health Professions Council of South Africa-affiliated paramedical exercise therapists. Exercise rehabilitation and therapy falls within their scope of profession.[18] Type 1 diabetic adolescents and adults who employ exercise regimes in order to enhance their sports performance at elite competitive levels are strongly encouraged to consult both a biokineticist and an exercise scientist due to their complicated intrinsic energy metabolism and exercise programme prescription requirements. Non-insulin-dependent diabetics with CAD and obesity co-morbidities should consult a biokineticist before starting to exercise, to avoid the inherent exercise-induced risks. Diabetic

**Table 1.** Exercise rehabilitation prescription for diabetic patients[1,19]

| Exercise components | Types of exercise | Objectives | Intensity/frequency/duration |
|---|---|---|---|
| Aerobic:<br>Large muscle group activities | Walking, jogging, running, swimming, simulated stair climbing, cycling | Increase aerobic capacity (cardiorespiratory fitness)<br>Reduce co-morbidity cardiometabolic risk factors (obesity, cardiac artery diseases) | Intensity:<br>50–90% heart rate maximum<br>50–85% $VO_{2max}$<br>Monitor Borg RPE scale<br>Frequency: 4–7 days/week<br>Duration: 20–60 min/session |
| Strength:<br>All major muscle groups (shoulders, biceps, triceps, forearms, chest, back, lumbopelvic hip complex, quadriceps, hamstrings, adductors, abductors, calves | Free weight exercises<br>Calisthenics<br>Resistance gym machines<br>Isokinetic machines | Increase muscle strength<br>Increase muscle endurance<br>Enhance sports performance among elite diabetic athletes | Rehabilitation:<br>Low resistance/weight, high repetition for patients<br>Sport performance:<br>Athlete-designed strengthening programme |
| Anaerobic:<br>Only for diabetic athletes with good energy metabolism control | High-intensity interval | Increase anaerobic capacity | High-intensity interval training (e.g. track running interval training or pyramid swimming interval training) |
| Flexibility | Static, proprioception neuromuscular facilitation, dynamic stretching | Increase muscle extensibility<br>Increase joint range of motion (passive and active) | Minimum of 2 sessions per week |
| Neuromuscular/ Proprioception | Biodex balance system<br>Romberg stance | Improve balance<br>Improve proprioception<br>Improve neuromuscular co-ordination | Minimum of 2 sessions per week |
| Functional activities | Daily living activities<br>Sport-specific activities for athletes | Dependent on patient lifestyle and activities | Minimum of 2 sessions per week |

How to calculate heart rate maximum using Karvonen formula
Target heart rate = (maximum heart rate – resting heart rate) × % intensity) + resting heart rate
Maximum heart rate = 220 – age of patient
Resting heart rate is resting pulse rate.
$VO_{2max}$ is the maximal oxygen consumption to be determined by the sub-maximal cardiorespiratory treadmill or cycle test
50 to 90% heart rate maximum corresponds to 50 to 85% $VO_{2max}$.
Borg rate of perceived exertion (RPE) scale
This is a self-evaluation of the patient's exercise intensity effort. The scale ranges from 6–20, with a rating of 10 being equal to a heart rate of 100 bpm.



patients should have regular contact with a biokineticist and/or a physiotherapist as part of their multi-disciplinary diabetes-management team. All diabetic patients should obtain medical clearance from their endocrinologist before starting an exercise rehabilitation programme. The diabetes multi-disciplinary medical team should include an endocrinologist, medical nurse, dietician, pharmacist, biokineticist and an exercise scientist for athletes.

## Clinical exercise testing

All diabetic patients should undergo a clinical test protocol as recommended by the American College of Sports Medicine (ACSM).[1,19] The testing protocol entails a sub-maximal aerobic cycle or treadmill test with an electrocardiogram (ECG). The sub-maximal aerobic test is used to evaluate the patient's cardiorespiratory endurance ($VO_{2max}$), while the ECG is used to identify cardiac dysrhythmias, especially among patients with cardiac pathologies. The strength of the patient can be tested isokinetically and/or isotonically. The joint range of motion (flexibility) should be measured through the use of a goniometer and proprioception can be evaluated either through the use of an electronic balance system or through a Romberg stance test.[1,19]

Peripheral neuropathy is a neurological condition, which results from injury to the nerves (deafferentation) that relay messages to and from the brain to the spinal cord and to other parts of the body. Peripheral neuropathy can be caused by diabetes mellitus,

adversely influencing the patient's proprioception. Proprioception is a person's awareness of their body position. Should a patient be diagnosed with this condition, proprioceptive exercises will help to rehabilitate the deafferentated nerves over a long period of time.[20] Proprioceptive exercise enhances the ability of patients to perform daily activities and reduces their risk of falling.[20,21] Many protocols also include biomechanical gait analyses aimed at identifying deviant walking patterns.[1,19]

## Exercise rehabilitation prescription

All diabetic exercise rehabilitation and/or sports performance programmes should include the following components: warm-up, stretching, aerobic, strengthening, proprioception, and functional exercises and activities, as well as a gentle cool-down.[1,19] While the ACSM prescribes a generic rehabilitation programme for diabetic patients, with specific goals (Table 1),[1,19,21] the types of exercise prescribed to a patient will vary depending on the patient's cardiorespiratory, muscular and flexibility conditioning and their desired outcomes (improved quality of life, enhanced health and fitness or competitive sports performance). The primary objectives of non-insulin-dependent diabetics would be to decrease body fat percentage, prevent obesity and lower hypertension. Insulin-dependent diabetics should strive to incorporate exercise to reduce their hyperglycaemia, which will allow them to lower the exogenous insulin intake. Table 1 is an overview of the ACSM's diabetes rehabilitative programme.[1]

Despite the fact that the prescription of stretching and muscle-strengthening exercises depends on the patient's capability, these exercises should nevertheless pertain to all major muscle groups. It is furthermore important that when diabetic patients start an exercise rehabilitation programme, they exercise at conversational heart rate zone: they should be able to exercise but simultaneously be comfortable talking to their biokineticist and/or training partner.[12]

## Conclusion

Regular exercise is an essential component of a diabetic patient's lifestyle-management strategy. However there are several exercise-induced metabolic complications that warrant recognition and it would therefore be prudent for all diabetic patients to consult a biokineticist or physiotherapist prior to commencing an exercise rehabilitation programme. It is furthermore clear that post-exercise exogenous insulin supplementation and meals must be adjusted to appropriately maintain sound energy metabolic homeostasis.

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# Drug Trends in Diabetes

## New technologies launched to protect insulin from temperature fluctuations

Insulin exposed to temperatures outside the recommended range starts losing its effectiveness and is at further risk when exposed to heat by diabetic subjects on the move. This is according to research conducted in Europe that found that insulin stored in domestic fridges is at risk.

Dr Brian Kramer, an endocrinologist from the Cardiovascular Diabetes Education, concurs with the research findings, saying: 'if insulin is accidentally frozen or exposed to high temperatures, it becomes ineffective.'

To counteract this risk, local company, MoveIT Solutions, has launched two innovative products in South Africa, MedAngel ONE and FRIO® cooling cases, which work hand-in-hand to monitor insulin temperature and store insulin at the correct temperature at home or on the go.

Developed in Europe, the products are ideally suited to local conditions that include temperature extremes and unreliable electricity supply. Around 6% of our population, or 3.5 million people, live with diabetes and a further five million are estimated to have pre-diabetes.

Founder and CEO of MoveIT Solutions, Gary Broomberg, says: 'As a diabetic myself, I became increasingly concerned about the effect temperature plays in the deterioration of insulin stored outside the recommended temperature range, which for most brands is 2–8°C. I'm an active outdoorsman and my insulin was often exposed to high temperatures for extended periods.'

This led Broomberg to investigate possible solutions, and ultimately fly to Europe to engage with MedAngel and FRIO®, securing licences to represent the products locally.

MedAngel ONE is an app that links a sensor via Bluetooth to mobile phones, providing a reliable record of insulin temperature. The app alerts users when insulin is above or below the recommended temperature. The waterproof sensor allows for monitoring insulin stored in a fridge and insulin on the move.

Independently tested, FRIO® cooling cases consist of a range of pouches to store insulin pens, vials and insulin pumps and work through a process of evaporation to keep the contents safe and cool. An inner pocket is filled with specially developed crystals that activate when immersed in water, expanding into a gel and remaining cool for 45 hours. It will keep insulin between 18 and 26°C, even in a constant environmental temperature of 37.8°C.

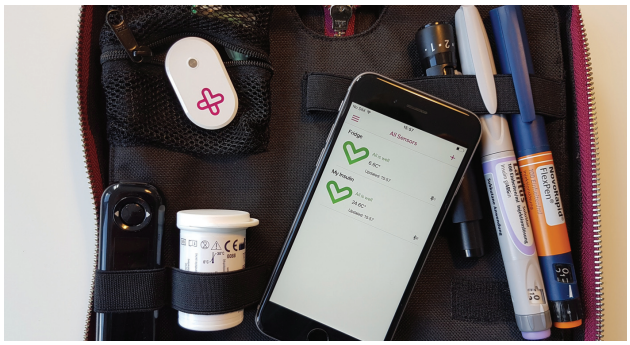
FRIO® cooling cases come in a range of sizes, styles and colours and are ideal for storing insulin pens, insulin pumps and vials. Paired with the MedAngel ONE sensor, diabetics have complete peace of mind that their insulin is safe.

'Maintaining the correct temperature for insulin is critical for diabetics, not only when stored in a domestic fridge, which is subject to temperature fluctuations, but also for travel or during sporting activities. Children, many of whom use insulin pumps, are especially at risk as they tend to spend long periods outdoors at school and at home,' says Broomberg.

The MedAngel ONE can be ordered online at za.medangel.co and the FRIO® cooling cases at friosouthafrica.com. The products are delivered by an express courier service.

**FRIO® cooling** case on belt.


**MedAngel ONE** sensor in the fridge.



**MedAngel ONE** sensor in a supplies bag.

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