Insulin resistance and the metabolic syndrome in obese black South African women: a focus on risk factors

by Elmarié Jonker
B.Sc. (Dietetics), M.Sc. (Biochemistry)

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Promotor: Prof. H.H. Vorster
Co-promotor: Dr. A. Kruger

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This study was inspired by a passion for African women

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ABSTRACT

Insulin resistance and the metabolic syndrome in obese black South African women: a focus on risk factors

Introduction

High rates of obesity occur in black South African women, up to double the rate in whites. Concern about the potential health burden of obesity in these women as well as a lack of understanding of the underlying mechanisms of obesity, motivated the POWIRS study (Profiles of Women with the Insulin Resistance Syndrome).

Subjects and methods

The study population consisted of 100 urbanised black women of the North-West Province, South Africa. These women were recruited as apparently healthy, non-pregnant selected volunteers, with ages of 19 to 50 years. Using a cross-sectional comparative study design, the women were divided into a normal-weight, overweight and obese group. Relevant demographic, anthropometric, dietary intake, and serum and plasma variables associated with the metabolic syndrome were compared in these three groups. Descriptive statistics, partial Spearman correlations, odds ratios and effect sizes were calculated. A medium effect size ($d \geq 0.5$) indicated possible practical significance, while a large effect size ($d \geq 0.8$) indicated practical significance.

Results

The acute-phase proteins serum (s)-C-reactive protein, plasma (p)-fibrinogen and possibly p-plasminogen activator inhibitor-1 (PAI-1) had practical significantly higher levels in the obese than the normal-weight women. High-risk levels of s-C-reactive protein (≥ 3 mg/L) occurred in 68% of the obese women compared to 16% of the normal-weight women. Increased p-PAI-1 levels (> 7 U/ml) occurred in 46% of the obese compared to 24% of the normal-weight women. Of the metabolic syndrome components, s-C-reactive protein showed the strongest correlation with body mass index ($r = 0.60$, $p < 0.0001$). Metabolic syndrome

components s-uric acid, s-fasting glucose, s-2h-post-load glucose, s-fasting insulin and the HOMA-insulin-resistance index had practical significantly higher levels in the obese than the normal-weight women, while systolic and diastolic blood pressure, s-gamma-glutamyl-transferase (GGT) and s-triglycerides had higher levels indicative of practical significance ($d \geq 0.5$). The metabolic syndrome was diagnosed in 38% of the obese women, 8% of the overweight women and in none of the normal-weight women. The acute-phase protein s-C-reactive protein, but not p-fibrinogen and p-PAI-1, had higher levels indicative of practical significance in women with compared to those without the metabolic syndrome. Serum-C-reactive protein and p-PAI-1 were positively associated with the HOMA-insulin-resistance index ($r = 0.25$, $p < 0.05$ and $r = 0.22$, $p < 0.05$, respectively).

After exclusion of dietary under-reporters, the obese women compared to the normal-weight women had practical significantly higher daily intakes of the following: total energy (10 591 *versus* 8 419 kJ), total carbohydrates (311 g *versus* 257 g), total protein (97 g *versus* 69 g), animal protein (62 g *versus* 40 g), total fat (86 g *versus* 66 g), saturated fat (28 g *versus* 21 g) and dietary fibre (22 g *versus* 17 g). A high frequency of overeating incidents (38 - 59%) and emotion-induced eating (48%) occurred in women of all body-weight groups, probably representing barriers to successful weight control. Therefore, emotion-induced eating may be a possible risk factor for the development of obesity and a factor in the maintenance thereof in this population group.

Attitudes towards weight control and thinness did not differ practical significantly between the three groups of women. However, with increasing body mass index more women had a positive attitude towards thin people. None of the women had a negative attitude towards thin people and weight control. Obese women had better knowledge ($d \geq 0.5$) of the relationship between obesity, nutrition and health than the normal-weight women.

The majority of the obese women (87%), 81% of the overweight and 12% of the normal-weight women indicated that they should lose weight. Forty-five percent of the obese women, 44% of the overweight women and 16% of the normal-

weight women had been on weight reducing diets before. At the time of the study, 71% of the obese, 60% of the overweight and 11% of the normal-weight women indicated that they were currently trying to lose weight.

Conclusions

In a group of 100 urbanised black women of the North-West Province, South Africa, levels of the acute-phase proteins C-reactive protein, fibrinogen and possibly PAI-1, were practical significantly higher in obese than normal-weight women. C-reactive protein, but not fibrinogen and PAI-1 may be part of the metabolic syndrome in these women. Since prospective studies have shown that C-reactive protein, PAI-1 and fibrinogen are predictors of myocardial infarction, stroke and non-insulin-dependent diabetes mellitus (NIDDM), this study indicates that obesity may lead to an increased health risk in this population.

The women in this study had a more Westernised attitude towards body size and weight control, which could improve compliance with weight control programmes. However, the barriers to successful weight control in these women should be addressed, for example emotion-induced eating. Since the obese women had better knowledge of the relationship between obesity, nutrition and health than the normal-weight women, lack of knowledge may not represent a barrier to succesful weight control in these women.

Recommendations

It is recommended that the dietary analysis should be repeated in a larger sample of women. These results should motivate urgent development of culturally-sensitive weight control programmes for obese African women. Such programmes could prevent an enormous burden due to obesity and its health consequences on public health resources of this country in the decades to come. Further research of the underlying mechanisms leading to obesity in this population group is strongly recommended, especially the role of emotion-induced eating.

OPSOMMING

Insulienweerstand en die metaboliese sindroom in obese swart Suid-Afrikaanse vroue: 'n fokus op risikofaktore

Inleiding

Obesiteit toon 'n hoë voorkoms in swart Suid-Afrikaanse vroue, tot dubbeld die voorkoms in blanke vroue. Kommer oor die potensiële gesondsheidsgevolge van obesiteit in hierdie vroue, sowel as 'n gebrek aan begrip van die onderliggende meganismes van obesiteit, het die POWIRS-studie (Profiles of Women with the Insulin Resistance Syndrome) gemotiveer.

Proefpersone en metodes

Die studiepopulasie het bestaan uit 100 stedelike swart vroue van die Noordwes-Provinsie, Suid-Afrika. Hierdie vroue is as oënskynlik gesonde, nie-swanger vrywilligers, met ouderdomme vanaf 19 - 50 jaar, gewerf. 'n Dwarssnit vergelykende studie-ontwerp is gebruik en die vroue is in 'n normale-gewig, oorgewig- en obese-groep verdeel. Relevante demografiese, antropometriese, dieet-inname en serum en plasma veranderlikes wat met die metaboliese sindroom geassosieer word, is in die drie groepe vergelyk. Beskrywende statistiek, parsiële Spearman korrelasies, relatiewe kansverhoudings en effekgroottes is bereken. 'n Medium effekgrootte ($d \geq 0.5$) het moontlike praktiese betekenisvolheid aangedui en 'n groot effekgrootte ($d \geq 0.8$) praktiese betekenisvolheid.

Resultate

Die akute-faseproteïene serum (s)-C-reaktiewe proteïen, plasma (p)-fibrinogeen en moontlik p-plasminogeen-aktiveerder-inhibeerder-1 (PAI-1) het prakties betekenisvolle hoër vlakke in die obese in vergelyking met die normale-gewig vroue getoon. Hoë-risiko vlakke van s-C-reaktiewe proteïen (≥ 3 mg/L) het in 68% van die obese vroue teenoor 16% van die normale-gewig vroue voorgekom. Verhoogde p-PAI-1-vlakke (> 7 U/ml) het in 46% van die obese vroue teenoor 24% van die normale-gewig vroue voorgekom. Van die metaboliese

sindroomkomponente het s-C-reaktiewe proteïen die sterkste korrelasie met liggaamsmassa-indeks getoon ($r = 0.60$, $p < 0.0001$). Metaboliese sindroomkomponente s-uriensuur, s-vastende glukose, s-2h-postlading glukose, s-vastende insulien en die HOMA-insulienweerstand-indeks het prakties betekenisvolle hoër vlakke in die obese as die normale-gewig vroue getoon, terwyl sistoliese en diastoliese bloeddruk, s-trigliseriede en s-gamma-glutamiel-transferase (GGT) hoër vlakke aanduidend van prakties betekenisvolheid ($d \geq 0.5$) getoon het. Die metaboliese sindroom is in 38% van die obese vroue, 8% van die oorgewig vroue en geen van die normale-gewig vroue nie, gediagnoseer. Die akute-faseproteïen s-C-reaktiewe proteïen, maar nie p-fibrinogeen en p-PAI-1 nie, het hoër vlakke aanduidend van praktiese betekenisvolheid ($d \geq 0.5$), in vroue met, in vergelyking met dié sonder die metaboliese sindroom, getoon. Serum-C-reaktiewe proteïen en p-PAI-1 was positief geassosieer met die HOMA-insulienweerstand-indeks ($r = 0.25$, $p < 0.05$ en $r = 0.22$, $p < 0.05$, respektiewelik).

Na uitsluiting van dieet-onderrapporteerders, het die obese vroue in vergelyking met die normale-gewig vroue, prakties betekenisvolle hoër daaglikse innames van die volgende getoon: totale energie (10 591 *versus* 8 419 kJ), totale koolhidrate (311 g *versus* 257 g), totale proteïene (97 g *versus* 69 g), dierlike proteïene (62 g *versus* 40 g), totale vet (86 g *versus* 66 g), versadigde vet (28 g *versus* 21 g) en dieetvesel (22 g *versus* 17 g). 'n Hoë frekwensie van ooreet-insidente (38 – 59%) en emosie-geïnduseerde eetgedrag (48%) het in vroue van alle gewigsgroepe voorgekom en verteenwoordig waarskynlik hindernisse vir suksesvolle gewigsbeheer. Gevolglik mag emosie-geïnduseerde eetgedrag 'n potensiële risikofaktor vir die ontwikkeling obesiteit en 'n faktor in die instandhouding daarvan in hierdie populasiegroep wees.

Houding teenoor gewigsbeheer en slankheid het nie prakties betenisvol tussen die drie groepe vroue verskil nie. Met toenemende liggaamsmassa-indeks het meer vroue 'n egter positiewe houding teenoor skraal persone getoon. Geen vroue het 'n negatiewe houding teenoor skraal persone en gewigsbeheer getoon

nie. Obese vroue het beter kennis as die normale-gewig vroue ($d \geq 0.5$) van die verwantskap tussen obesiteit, voeding en gesondheid getoon.

Die meerderheid van die obese vroue (87%), 81% van die oorgewig vroue en 12% van die normale-gewig vroue het aangedui dat hulle gewig behoort te verloor. Vyf-en-veertig persent van die obese, 44% van die oorgewig en 16% van die normale-gewig vroue het voorheen gewigsverminderings-diëte gevolg. Tydens die studie het 71% van die obese vroue, 60% van die oorgewig-vroue en 11% van die normale-gewig vroue aangedui dat hulle tans gewig probeer verloor.

Gevolgtrekkings

In 'n groep van 100 stedelike swart vroue van die Noordwes-Provinsie, Suid-Afrika, was vlakke van die akute-faseproteïene C-reaktiewe proteïen, fibrinogeen en moontlik PAI-1 prakties betekenisvol hoër in obese as normale-gewig vroue. C-reaktiewe proteïen, maar nie fibrinogeen en PAI-1 nie, is moontlik deel van die metaboliese sindroom in hierdie vroue. Aangesien prospektiewe studies getoon het dat C-reaktiewe proteïen, PAI-1 en fibrinogeen voorspellers van miokardiale infarksie, beroerte en nie-insulien-afhanklike diabetes mellitus (NIDDM) is, dui hierdie studie aan dat obesiteit tot 'n verhoogde gesondheidsrisiko in hierdie populasiegroep mag lei.

Die vroue in hierdie studie het 'n meer Westerse houding teenoor liggaamsgrootte en gewigsbeheer getoon, wat meewerkendheid in gewigsbeheerprogramme mag verbeter. Hindernisse vir suksesvolle gewigsbeheer in hierdie vroue, byvoorbeeld emosie-geïnduseerde eetgedrag, moet egter aangespreek word. Aangesien die obese vroue beter kennis van die verwantskap tussen obesiteit, voeding en gesondheid as die normale-gewig vroue getoon het, blyk dit dat gebrek aan kennis nie 'n hindernis vir suksesvolle gewigsbeheer verteenwoordig nie.

Aanbevelings

Dit word aanbeveel dat die studie herhaal word in 'n groter groep vroue van 'n ewekansige steekproef. Hierdie resultate behoort dringende ontwikkeling van kultuur-sensitiewe gewigsbeheerprogramme vir obese Afrika-vroue te motiveer.

Sodanige programme mag 'n enorme las as gevolg van obesiteit en die gesondheisgevolge daarvan, op publieke gesondheidshulpbronne van Suid-Afrika in komende dekades voorkom. Verdere navorsing oor die onderliggende meganismes wat in hierdie populasiegroep tot obesiteit lei, word sterk aanbeveel, veral die rol van emosie-geïnduseerde eetgedrag.

CHAPTER 1

Introduction

1.1 Introduction

Obesity has been classified by the Word Health Organization as a disease and as one of the major health problems facing mankind in this century (WHO, 2004). Obesity is a problem among adults and children (Vega, 2001), not only in the developed world (Europe, North America, Japan, Australia and the United Kingdom), but also in the developing world (South America, Asia and Africa) (Popkin & Doak, 1998; Wang *et al*, 2002).

The literature indicates that obesity is a complex multifactorial disease (Prentice, 2001) and uncertainty about the etiology impairs appropriate action to combat this problem (Prentice & Jebb, 1995). It is also an expensive disease, with 51% of the medical costs of obesity in the USA being due to obesity-related diseases (Vega, 2001). In developing countries, overnutrition-related diseases such as obesity, diabetes and cardiovascular disease are emerging, while undernutrition and poverty-related diseases are still prevalent, creating a double burden of disease. This places an enormous burden on the fragile health budgets of developing countries. In South Africa, the situation may be even worse, since a quadruple burden of disease is experienced: poverty-related infectious disease, violence-related trauma, HIV/AIDS, and non-communicable diseases (NCD) due to overnutrition (Vorster *et al*, 1999; Bourne *et al*, 2002).

Obesity in South Africa has certain unique features. These include the high prevalence among women compared to men (Bourne *et al*, 2002), the cultural tolerance of obesity (Walker *et al*, 2001), the contribution of food insecurity to the development of obesity (Drenowski & Specter, 2004; Townsend *et al*, 2001), the coexistence of obesity with undernutrition (Vorster *et al*, 1997) and the often observed absence of the obesity-related comorbidities in African women, which has led to the concept of 'healthy obesity' (Walker *et al*, 1989; Walker *et al*,

1990). New research and opportunities regarding obesity in African women have highlighted some of these features and questioned some of the conclusions of earlier research (Ndlovo *et al,* 1999; Mvo *et al,* 1999; Vorster *et al,* 2000; Kruger, 2000; Kruger *et al,* 2001; Puoane *et al,* 2004).

The purpose of this thesis is to focus on these new developments and to examine obesity amongst African women in more depth, looking not only at the known causes and consequences, but also to explore often neglected causes such as the role of culture, behavioural and other aspects. Special attention is given to the biochemical risk factors and markers related to NCD in this population, since the biological profiles of obese women could be compared to those of overweight and normal-weight women.

1.2 Structure of the thesis

This thesis consists of a number of separate manuscripts (Chapter 2, 3, 4 and 5) and a brief combined discussion and conclusion with appropriate recommendations (Chapter 6).

The first manuscript 'Obesity in black South African women – a review' (Chapter 2) consists of a narrative review of the literature on what is known about obesity in black South African women. This manuscript has been submitted to the *'South African Journal of Clinical Nutrition'.*

The second manuscript 'Obesity is associated with an acute phase response in black South African women' (Chapter 3), describes the association of acute-phase proteins C-reactive protein, fibrinogen and plasminogen-activator inhibitor-1 with obesity, and compares these and other health-risk factors, such as dietary intakes and metabolic syndrome components between normal-weight, overweight and obese women. This manuscript has been prepared for submission to *'Public Health Nutrition'.*

The third manuscript 'Insulin resistance and the metabolic syndrome in black South African women' (Chapter 4), describes the metabolic syndrome in these

women, with special reference to the acute-phase proteins and insulin resistance. This manuscript has been prepared for submission to the *'South African Journal of Diabetes and Vascular disease'.*

The fourth manuscript 'Weight-related attitude, knowledge and behaviour in black South African women' (Chapter 5), compares weight-related attitudes, knowledge and behaviour between normal-weight, overweight and obese women, and identifies barriers to successful weight control. The manuscript has been submitted to *'Health SA'.*

Chapter 6 gives a brief summary of the results of the study, conclusions and recommendations. This is followed by an addendum, consisting of all the questionnaires used in the study.

1.3 Acknowledgements

The experimental data presented in this thesis were generated in the POWIRS study (Profiles of Women with the Insulin Resistance Syndrome). This multi- and transdisciplinary study took place from 1 March 2003 – 31 April 2003. The author was an active researcher in this study and her role is evident from Table 1.1 (see Addendum 1, p. 119), which also shows the contributions and signatures of all the other co-workers, giving permission for the results to be used in this thesis.

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CHAPTER 2

OBESITY IN BLACK SOUTH AFRICAN WOMEN – A REVIEW

Jonker E (M.Sc), Vorster HH (D.Sc), Kruger A (Ph.D)
School for Physiology, Nutrition, and Consumer Sciences, North-West University, Potchefstroom Campus, South Africa

ABSTRACT

The objective of this paper is to review the causes and consequences of obesity in black South African women. One-third to half of these women are obese, almost double the prevalence in white women. It is particularly problematic in this population, because large numbers of people are still undernourished and with the additional HIV/AIDS epidemic, the health budget of South Africa will have to cope with a double burden of disease. Lack of understanding of obesity in African women impairs appropriate action to prevent and treat the problem. Suggested contributing factors are urbanisation, low physical activity, low metabolic rate, excessive energy intake, genetic susceptibility, food insecurity, emotion-induced eating, cultural values, and low birth weight. It seems that South African black women are conscious about weight control and are beginning to experience obesity negatively, in contrast with previous suggestions. The known health consequences of obesity have recently become more evident in these women and include a state of chronic low-grade inflammation, insulin resistance and the metabolic syndrome, all associated with diabetes mellitus and cardiovascular diseases. This points to an increased health risk in these women. It is concluded that intervention programmes to address obesity could be motivated by the associated comorbidities in black South African women.

Key words: *obesity, black women, South Africa*

INTRODUCTION

Since the last quarter of the 20th century, obesity rates have been escalating worldwide (Popkin & Doak, 1998; Flegal *et al*, 2002). Between 1991 and 1998, obesity has increased by almost 50% in all race groups in the USA (Weinsier *et al*, 2002). In 1999, 61% of USA adults were overweight or obese (McCrory *et al,* 2002). These trends are also observed in children (Caprio, 2002; Wang *et al,* 2002). The national obesity rate in the USA according to the 1999 – 2000 NHANES survey compared to those of 1988-1994 showed that the highest increase in the obesity rate of 11.5% occurred in the black female population (Flegal *et al,* 2002). In South Africa, from 1969 to 1989, obesity has increased from 9.3% to 19.9% in the black population (Walker *et al,* 2001).

Obesity was previously thought to be a disease of the wealthy (Popkin & Doak, 1999; Prentice, 2001), but higher levels of obesity are found in minority populations of many countries. Examples are African-Americans, Mexican-Americans, Pima-Indians, Hispanics and Maori's (Cannan & Walker, 1996; Foster *et al,* 1999; Baskin *et al*, 2001; Flegal *et al,* 2002; Barakat *et al*, 2002). Especially in developing countries like South Africa, high obesity rates are problematic, since a large proportion of the population is still undernourished and also affected by HIV/AIDS. Therefore, a double burden of disease results which put an enormous burden on health resources of this country (Vorster *et al*, 1999).

The increase in obesity could be ascribed to be mostly a consequence of a dramatic decrease in physical activity, both occupational and in leisure-time (Prentice & Jebb, 1995; Popkin, 1998) and an increasing availability of highly palatable high-fat foods that encourage overeating and are easily accessible to most people (Golay & Bobbioni, 1997; Grundy, 1998). Furthermore, commercial portion sizes are increasing. In the USA, portion sizes of fast foods are now two to five times larger than when first introduced (Abraham, 2004). Restaurants and fast-food stores offer super-sized 'value meals' in order to increase and maintain their market share (Malthiessen *et al,* 2003). Therefore, it is not

surprising that obesity rates are increasing to pandemic proportions in the USA (Manson & Bassuk, 2003).

There is concern about the high obesity rates due to the known health consequences therof (Pi-Sunyer, 1993; Grundy, 1998). However, there is a lack of knowledge about the underlying mechanisms acting in obesity, especially concerning the causes. This impairs development of effective culturally-sensitive weight control programmes. This article gives an overview of up to date reports on obesity in black South African women and incorporates some results of the recent POWIRS study (Profiles of Women with the Insulin Resistance Syndrome) (Schutte *et al,* 2004).

Prevalence of obesity in black South African women

Table 1 compares the prevalences of obesity in some populations of South Africa, Africa and the USA. Obesity is particularly common in black women, much more so than in men and other population groups (Health Systems Trust, 2004) (Table 1). Black women in South Africa have a prevalence of 31.2% (Health Systems Trust, 2004), while in some areas more than 50% are obese (Mollentze *et al,* 1995). White women have a prevalence of 25.5% (Health Systems Trust, 2004). In the USA, the same trend is observed, with black women being more obese than whites (Flegal *et al*, 2002). Black men have a much lower prevalence of obesity than black women, namely 7.8 % (Health Systems Trust, 2004).

Urbanisation and the nutrition transition have been associated with the observed increases in obesity in South Africa and other developing countries (Popkin, 1999; Martorell *et al,* 2001; Vorster, 2002). Obesity rates in poorer African countries are still very low, for example in Tanzania, where only 2.6% of black women are obese (Table 1).

Table 1 ***Obesity rates in some populations***

Population	***Women %***	***Men %***	***Reference***
South Africa			
Whites	25.5	20.1	http://www.hst.org.za/healthstats/46/data/int
Blacks	31.2	7.8	http://www.hst.org.za/healthstats/46/data/int
Coloureds	28.5	9.2	http://www.hst.org.za/healthstats/46/data/int
Indians	21.3	9.0	http://www.hst.org.za/healthstats/46/data/int
Rural blacks (Free State)	53.1	23.0	Mollentze *et al*, 1995
Urban blacks (Free State)	53.4	32.9	Mollentze *et al*, 1995
Rural Zulu	31.6		Walker *et al*, 2001
Rural Venda	19.9	2.9	Walker *et al*, 2001
Urban blacks (North West)	29.3		Kruger *et al*, 2002
Tanzania	2.6		Martorell *et al*, 2001
Uganda	1.2		Martorell *et al*, 2001
Zimbabwe	2.5		Martorell *et al*, 2001
Kenya	2.4		Martorell *et al*, 2001
Zambia[5]	2.3		Martorell *et al*, 2001
USA			
Whites	30.1	27.3	Flegal *et al*, 2002
Blacks	49.7	28.1	Flegal *et al*, 2002

Obesity classified as BMI ≥ 30 kg/m^2

FACTORS CONTRIBUTING TO OBESITY

Obesity is a multifactorial disease, where social, behavioural, cultural, physiological, metabolic and genetic factors interact in an individual to determine the development of obesity (Prentice, 2001; Expert Panel on overweight and obesity, 1998). Suggested contributing factors in black women are age (Puoane *et al*, 2002), urbanisation and the nutrition transition, low physical activity, low resting metabolic rate, diet, cultural norms and beauty standards, socio-economic status, education and higher pregnancy rates. Another possible contributor in developing countries, which are in a process of urbanisation, is a history of early nutritional deprivation, as reflected in low birth weight (Vorster *et al*, 1999). There is still a large gap in knowledge about the causes of obesity in black women. The above-mentioned contributing factors are discussed below.

Urbanisation and the nutrition transition

Urbanisation and the nutrition transition have undoubtedly contributed to the increase in obesity rates in black women (Popkin, 1999; Puoane *et al*, 2002; Vorster, 2002). During the last three to four decades, urbanisation of black South Africans has increased rapidly, mostly due to socio-economic and political changes (Olatunbosun *et al*, 2000). From 1993 to 1996, the percentage of

blacks that became urbanised increased from 36 % to 43%, while in other population groups the rates remained relatively constant (Vorster, 2002). Urbanisation is associated with an epidemiologic transition (shift in disease and mortality patterns) and nutrition transition (shifts in dietary patterns). This process occurs globally, mostly due to decreased infant mortality and mortality from infectious diseases and famine, and increased longevity associated with an increase in non-communicable (NCD) (degenerative) diseases in later life (Popkin, 1994). The nutrition transition is central in the epidemiologic transition, where the diet becomes more atherogenic, containing more fat, animal products, refined carbohydrates and added sugar, in comparison with the indigenous diets, high in unrefined carbohydrate, low in fat and animal protein. More foods are eaten away from home and more processed foods are consumed (Drenowski & Popkin, 1997; Puska *et al,* 2002). Accompanying this process is a large decrease in physical activity, both occupational and in leisure-time (Popkin, 1998). The nutrition transition is a process well described by Popkin (1999), occurring in five stages: 1) collecting food, 2) famine 3) receding famine, 4) degenerative diseases and 5) behavioural change. Many first world countries, and also some lower income countries, such as China and Mauritus, have already entered the behavioural change stage, where healthier lifestyles are promoted in order to decrease the development of degenerative diseases (Popkin, 1994; Popkin, 1999).

In black South Africans, and also in other developing populations, the nutrition transition has occurred at an accelerated pace (Popkin, 1994; Bourne, *et al,* 2002). From 1940 to 1990, fat intake has increased with 60%, while carbohydrate intake has decreased with 11%. Protein intake as a percentage of total energy has remained fairly constant (Bourne & Steyn, 2000). This change is higher than observed in other population groups over longer periods of time (Bourne *et al,* 2002).

The traditional African diet was very low in fat (17% of total energy) (Bourne *et al,* 1993) and animal protein, and high in coarse grains (Walker, 1998). With

urbanisation, increased socio-economic status and the nutrition transiton, this diet became more varied and therefore more nutritionally adequate (Vorster *et al,* 2000; MacIntyre *et al,* 2002). Increased dietary variety as well as the accompanying increase in palatability have been described to result in increased energy intake and are associated with increased risk to develop obesity (McCrory *et al,* 2002).

Apart from the lifestyle changes associated with urbanisation, it seems that populations in transition are more vulnerable to NCD. It has been observed that migrants have higher NCD rates than host populations. For example, in Japannese men who resided in the USA, the incidence of non-insulin-dependent diabetes mellitus (NIDDM) was higher than in those living in Japan (Fujimoto *et al,* 2002). Other studies found the same phenomenon, where obesity increased from a more to a less traditional environment (Hodge & Zimmet, 1994). Also in the Zulu, it has been observed that social stressors increase the likelihood to develop hypertension (Seedat, 1983). Those most likely to become hypertensive were the ones that struggled the most to adapt to their new environment (Walker, 1964). Therefore, it seems that the stress to adapt to urban life increase the vulnerability to develop NCD. This may explain to some extent the higher rates of female obesity, non-insulin-dependent diabetes mellitus (NIDDM) and hypertension in black compared to white women in South Africa (Punyadeera *et al,* 2001).

Energy expenditure

Low physical activity has been widely suggested to be a strong determinant of obesity (King *et al,* 2001; Trost *et al,* 2001; Schmitz *et al,* 2000). Badenhorst and Walker (1994) suggested that a decrease in energy expenditure of only 200 kcal per day could theoretically lead to an increase of 9 kg body weight per year.

Data on the prevalence of physical activity in black South Africans are limited, but two cross-sectional studies in the Western Cape in urban blacks, have indicated that between 30% and 40% of men and women do not engage in any physical

activity, neither occupational nor leisure-time. A further 40 - 60% of them took part in minimal to moderate activity (Lambert *et al,* 2001).

In other countries, black women were found to be less physically active than white women (Lovejoy *et al,* 2001, Weinsier *et al,* 2002). In South African black women, low physical activity was also found to be a determinant of obesity (Kruger *et al,* 2002). Traditionally, rural black women had physically demanding lifestyles. For example, Zulu women were responsible for all the farming work, while men took care of the cattle (Walker, 1995). Currently, physical activity is low, even in rural blacks. It is suggested that since they no longer need to hunt, gather food from the field or fetch water, there is no need for some activities, such as walking (Kruger *et al,* 2003).

A consistent finding in American black women was that they have lower resting metabolic rates than white women (Jones *et al,* 2004) in addition to their low physical activity (Weinsier *et al,* 2002; Lovejoy *et al,* 2001), which could greatly increase their risk to become obese. Studies in the USA (Foster *et al,* 1999; Schiffman *et al,* 2000; Gannon *et al,* 2000) and South Africa (Kruger *et al,* 1994) indicated that black women lose weight more difficultly on a variety of weight loss programmes, suggesting that low resting metabolic rate may be a contributing factor in these results. Resting metabolic rate was not measured in South African black women, but there is not reason to believe that the situation would be different in them.

Dietary intakes

One of the puzzling aspects of obesity in South African black women is that they consume a low-fat diet, containing no more than 30% of fat (Vorster *et al*, 1997), which is expected to be associated with effective weight control (WHO, 1998). In a large number of studies high fat consumption was associated with obesity (Bray & Popkin, 1998), in contrast to the observation in South African black women. However, obesity was also observed in black women on a low fat diet in Santiago, Chile. Even with a fat intake as low as 22% of the energy intake,

obesity occurred in these women (Walker *et al,* 1990). In rural black women, for example Zulu women, it was found that with a low fat intake of 17% of total energy, 31.6% of the women were obese and 40% were overweight (De Villiers *et al,* 1988). These women had a high consumption of refined maize meal and low physical activity (O'Keefe, 1988). What should not be overlooked, however, is the high total energy intake of 10 150 kJ in those women (De Villiers *et al,* 1988), which is likely to be excessive to maintain ideal body weight (ADSA, 2000), especially if they would have a low metabolic rate and low physical activity. Similar high-energy intakes have been described in other groups of African women (Walker *et al,* 1990). Walker and Segal (1980) suggested that obesity in black women is caused by carbohydrate instead of fat. In the USA, it was observed that in white women, high fat intake was associated with obesity, while in blacks more women with appropriate fat intake were obese (Cook *et al,* 2000).

Studies that compared dietary intakes in black versus white women showed contradicting results. Some studies found that black women eat more and in particular, more fat than white women (Fitzgibbon *et al,* 2002; Kayrooz *et al,* 1998), while others found they eat the same amount (Lovejoy *et al,* 2001). Other studies indicated that black women eat less, even when under-reporting is taken into account (Kumanyika, 1987). The NHANES survey showed that the energy intake in young black females was higher than in whites, but after 21 years of age black women had lower energy intakes than white women. This suggests that the obesity problem in black women starts during adolescence (Schiffman *et al,* 2002).

In South Africa, the THUSA study found weak but significantly positive correlations between total fat and energy intakes with body mass index respectively (Kruger *et al,* 2002). The POWIRS study, a case-control study of 100 women, compared dietary intakes of obese and non-obese black women (Table 2). This study revealed that, after under-reporters had been excluded, the obese women consumed approximately 2000 kJ per day more than the non-

obese women. The obese women also had higher intakes of protein, especially animal protein, fat and carbohydrate than the non-obese women. There were significantly positive correlations between dietary intakes and obesity measures: energy intake ($r = 0.43$ for body mass index (BMI) and $r = 0.55$ for waist circumference (WC); $p < 0.05$), fat intake ($r = 0.49$ for BMI and $r = 0.65$ for WC; $p < 0.05$) and protein intake ($r = 0.57$ for BMI and $r = 0.67$ for WC; $p < 0.05$).

Table 2 ***Dietary intakes of normal-weight, overweight and obese black women of the POWIRS study (after exclusion of under-reporters)***

	Normal-weight N = 23		*Overweight N = 8*		*Obese N = 5*	
Nutrient	*Mean*	*SD*	*Mean*	*SD*	*Mean*	*SD*
Energy (kJ)	8419*	1874	8730	1531	10 591*	1148
Carbohydrate (CHO) (g)	257.2*	65.8	253.0	37.4	311.0*	49.6
Added sugar (g)	60.6	27.0	55.7	12.9	65.2	35.7
Dietary fibre (g)	16.5*	4.7	16.2	2.8	22.2*	3.8
Total protein (g)	69.4*	18.4	80.2	16.7	97.3*	15.6
Plant protein (g)	24.1*	7.2	23.8	4.9	31.0*	3.5
Animal protein (g)	40.4*	14.6	52.9	15.7	62.0*	14.8
Total fat (g)	66.3*	16.6	74.3	20.6	85.9*	14.1
Saturated fat (g)	21.1*	5.5	25.8	7.0	28.2*	4.5
Mono-unsaturated fat (g)	22.7*	6.1	24.7	7.7	29.6*	6.1
Poly-unsaturated fat (g)	15.8	4.8	16.5	5.8	19.2	5.9
Alcohol intake (g)	4.6	7.4	1.4	2.1	3.1	5.2
% Energy from CHO	55.1	5.0	52.9	5.6	53.6	5.8
% Energy from added sugar	12.2	4.4	11.0	2.9	10.2	5.0
% Energy from total protein	13.9*	1.7	15.6*	1.5	15.6*	1.4
% Energy from total fat	29.2	4.0	31.1	4.4	30.0	3.8
% Energy from alcohol	1.7	2.7	0.4	0.6	0.8	1.3

** Differ practical significantly ($d \geq 0.8$)*
Difference only indicated between normal-weight and obese women
SD = standard deviation

MacIntyre *et al.* (2002) indicated in the THUSA study that urban blacks have a higher fat intake contributed by meat instead of vegetable fats. A study in black South African diabetics also indicated higher animal protein consumption in urban compared to rural patients (Nthangeni *et al,* 2000). Traditionally, the African population were low meat consumers (Walker *et al,* 2001), but higher meat intake accompanied urbanisation (MacIntyre *et al,* 2002). It could be suggested that with urbanisation, meat is added to their usual high carbohydrate diet, and in this way, increase both the total energy, fat and protein intake of the diet, which could be expected to promote obesity. Meat intake is also perceived

by South African blacks to be associated with high socio-economic status (see 'cultural aspects'), which would encourage higher intakes.

Although the diets of the women in the POWIRS study seemed prudent due to a low percentage of fat (30%), the total amount of energy consumed was more than the assessed requirement (ADSA, 2000). It was concluded from the results that it would be important to focus on total energy restriction together with animal protein and fat restriction in order to effectively control obesity in black South African women. In a high-energy diet, the percentage contribution of a nutrient could be appropriate, but high if the total grams are calculated, for example 86 g fat (30%) and 97 g (15.6%) protein intake in the obese women, who consumed a 10 590 kJ diet.

If the dietary intakes of the urban black women in the POWIRS study are compared to those described in 1990 by Walker with 'healthy obesity', it is noticed that the obese women in the POWIRS study consumed 86 g fat daily, compared to 65 g in Walker's study. Mean total protein intake was 97 g in the POWIRS study, but only 73 g in Walker's study. The mean total energy intake in the POWIRS study was 1000 kJ higher than in Walker's study, 10 600 kJ compared to 9 600 kJ (Walker *et al,* 1990). This suggests deterioration of the diets, which could contribute to the adverse consequences of obesity, similar to the situation described in Santiago, Chile (Walker *et al,* 1990).

Socio-economic status

Most studies indicated that the prevalence of obesity is higher with lower socio-economic status (Jeffery & French, 1996; Hodge *et al,* 1996; De Spiegelaere *et al,* 1998; Olson, 1999), while some studies found no difference (Averett & Korenman, 1999). In South Africa, data on this subject are sparse, but the THUSA study indicated increased obesity risk with increasing household income. A higher income level was associated with a 1.5-fold increase in the risk to become obese (Kruger *et al,* 2002).

Education level showed an inverse relationship with obesity in some studies (Drenowski & Specter, 2004), while other studies showed the opposite (Martorell *et al,* 2001). In African-Americans, higher rates of obesity were found in spite of higher education, income and greater access to health-care and recreational facilities (Jefferson *et al,* 2002). In South Africa, education level was not associated with obesity in the THUSA study (Kruger *et al,* 2002). The SADHS (South African Demographic and Health Survey) indicated lower BMI with lower education in black women (Bourne *et al,* 2002). Similarly, in other developing countries, obesity rates seem to increase with higher education levels (Martorell *et al,* 2001).

On the other hand, South Africa is also a country with a large percentage poor people, where nearly two-thirds of the population have a household income below sustenance level and about 20% are obese (Health Systems Trust, 2004). Therefore, it seems that both low and high socio-economic status may contribute to obesity. With increasing socio-economic status, a more varied diet can be afforded and commonly leads to higher fat, animal protein and sugar intake (Drenowski & Popkin, 1997), contributing to weight gain. With lower socio-economic status and food insecurity, some other influential factors are suggested, discussed under 'food insecurity'.

According to Thompson (1994) food acts as a temporary balm to ease stress in poor women. He suggested that food is the remedy of choice for poor women of colour. One report indicated that obesity was six times more common in poor women than in women with a higher socio-economic status (Thompson, 1994). Therefore, economic deprivation may contribute to high obesity rates among women of lower income (Jefferey & French, 1996; Gibson, 2003). However, the relationship between poverty and obesity is probably influenced by a host of other factors.

Food insecurity

A large percentage of the South African population is still very poor (Health systems Trust, 2004), especially among the black population group (Bourne *et al,* 2002). Undernutrition and overnutrition are often found in the same household, where obese mothers have underweight children (Steyn *et al,* 1998). A positive association between food insecurity and obesity was found in a number of studies (Drenowski & Specter, 2004; Gibson, 2003; Olson, 1999; Oh & Hong, 2003; Adams *et al,* 2003). It was suggested that enough food, rather than food restriction, would be helpful in resolving the obesity problem in developing countries (Dietz, 1995).

In the Californian Women's Health Survey (8169 women) (Adams *et al,* 2003), obesity was found in 31.0% of the food-insecure women, compared to 16.2% in the food-secure women. In the same study, food insecurity without hunger was associated with an increased obesity risk in whites (odds ratio = 1.36) and other population groups (odds ratio = 1.47). Food insecurity with hunger was associated with increased obesity risk in Asians, Blacks and Hispanics (odds ratio = 2.81). According to this study, food insecurity was associated with a greater obesity risk in non-whites (Adams *et al,* 2003). Food insecurity was a strong independent predictor of weight gain in the report of Townsend *et al.* (2001). In mildly food-insecure women, overweight was 30% more likely to occur than in food-secure women (Townsend *et al,* 2001). Food insecurity (the fear that food will run out (Townsend *et al,* 2001)) shows a stronger association with obesity than food insufficiency or hunger (Alaimo *et al,* 2001).

Several mechanisms have been proposed to explain the relationship between food insecurity and obesity. Firstly, food-insecure individuals may be overweight because they can only afford to consume cheaper foods, which tend to be more energy-dense and could result in consumption of too much energy, leading to weight gain (Drenowski & Specter, 2004). Secondly, periods of insufficient food supplies could cause individuals to overeat when there is enough food, especially rich and palatable foods, resulting in increased overall energy intake, which

would cause weight gain (Alaimo *et al,* 2001; Townsend *et al,* 2001). Studies in humans and animals have shown a tendency to binge-eat when plentiful food supplies are available after a period of deprivation (Townsend *et al,* 2001; Olson, 1999). Thirdly, fluctuations in eating habits could result in the body becoming more efficient in energy utilisation, resulting in weight gain without consuming extra calories (Alaimo *et al,* 2001). Another possible mechanism is that due to the stress associated with food insecurity and poverty, sensible nutrition and health practices are simply not a priority (Jeffery & French, 1996).

Pregnancy

Pregnancy was suggested to be one of the contributors to higher obesity rates in black women, since they tend to have more children. The NHANES indicated some evidence of this (Kumanyika, 1987). Black women were reported to gain more weight with pregnancy than white women (Wolfe *et al,* 1999). In South Africa, the THUSA study did not find a correlation between parity and body mass index (Kruger *et al,* 2002). However, in a group of black female students in KwaZulu-Natal, the number of pregnancies showed a significantly positive relationship with body weight (Steyn *et al,* 2000). In the POWIRS study, pregnancy was considered as a cause of weight gain by some of the normal-weight and overweight women, but none of the obese women shared this experience.

Emotion-induced eating

Eating for reasons other than nutrition would undoubtedly increase the risk to become obese. A large amount of literature has explored the role of psychological factors in the development and maintenance of obesity. Emotion-induced eating has been suggested as an important factor in the etiology of obesity, where food intake is influenced by emotional states such as anxiety or depression, because eating serves as a coping tool in emotionally stressful situations (Striegel-Moore *et al,* 1999). Emotion-focused coping occurs more often in obese women (Laitinen *et al,* 2002). Women who eat for emotional reasons also tend to have higher preferences for sweet and fat rich foods

(Schiffman *et al*, 2000; Laitinen *et al*, 2002). Higher levels of emotion-induced eating in black girls compared to white girls were found in the USA (Striegel-Moore *et al*, 1999). The risk of binge-eating (also a form of emotional eating) was twice as high in black than in white women (Striegel-Moore *et al*, 2000[a]). Binge-eating was indentified as a significant problem in young adult black women in the USA (Striegel-Moore *et al*, 2000[b]). Stress-driven eating and drinking was associated with higher body mass index (BMI) and obesity, especially in women (Laitinen *et al*, 2002). It was also found that black women, currently subjected to higher socio-economic stressors, have higher levels of perceived stress than white women, which predisposes them to emotion-induced eating (Schiffman *et al*, 2000).

There are indications that the black South African population, particularly women, suffer from high levels of stress and psychopathology (Vorster *et al*, 2000). In a group of black female students in KwaZulu-Natal, 18% was moderately to severely depressed and 26% mildly depressed (Steyn *et al*, 2000). With rapid urbanisation, many black people are subjected to a process that may lead to social and cultural disruption, causing increased levels of stress (Seedat, 1983; Van Rooyen *et al*, 2000). This indicates a possible high risk for emotion-induced eating in black women.

Emotion-induced eating was assessed in 81 of the women in the POWIRS study. Differences between normal-weight and obese women were not evident, although under-reporting of the truth by the obese was suspected, as also suggested by Striegel-Moore *et al*. (1999). However, the presence of eating for other reasons than nutrition was clear. Fifty-three percent of the women reported eating when they were not hungry. Eating for a treat was particularly common, indicated by 81% of the women. Other reasons for eating included being worried (35%), bored (46%), mad (20%) and happy (72%). Fifty-five percent of the women reported overeating incidents. The women also indicated that they ate large amounts when plenty of food was available, felt guilty after eating too much and eating in response to stress. Other surveys in this population also indicated

eating when depressed, worried or upset (Mvo *et al*, 1999; Steyn, 2004; Puoane *et al*, 2004). The association between food, pleasure and comfort in South African black women was therefore recognised (Mvo *et al*, 1999; Puoane *et al*, 2004). Especially with urbanisation and the availability of highly attractive palatable foods, a high risk for emotion-induced eating in black South African women could be expected. Emotion-induced eating in a population already highly susceptible to obesity would be detrimental, and more research on this subject is suggested.

Cultural aspects

Attitudes towards weight control, knowledge and weight-related behaviour

A recent survey in urban black South African women indicated that a moderately overweight figure was preferred, which was associated with dignity, respect, confidence, beauty and wealth (Steyn, 2004). It has also been widely reported that black women tend to be more satisfied with their figures than white women (Gore, 1999; Baskin *et al*, 2001). Therefore, one of the suggested barriers to weight control in black women has been that the African culture has a different perception of obesity (Gore, 1999) and that it is not stigmatised like in the Western culture (Walker *et al*, 2001; Baskin *et al*, 2001).

Kruger *et al.* (1994) previously found that almost half of obese women did not want to lose weight, but that of the women who wanted to lose weight, 93.5% were obese. The majority of the women who wanted to lose weight preferred it for health reasons (72%). Bourne *et al.* (2002) indicated that a low percentage of obese women thought they were obese (15%). However, the POWIRS study showed that 87% of obese women and 81% of overweight women thought they should lose weight (Jonker *et al*, 2004).

A later study in the North-West Province by Kruger and van Aardt (1998) indicated a shift towards a more positive attitude regarding weight control. The POWIRS study found similar results and no practically significant differences in attitudes towards weight control occurred between normal-weight, overweight

and obese women. The mean total attitude score, which included attitudes towards obese people, thin people and weight control, was neutral. Although the attitude scores did not differ practical significantly between the body-weight groups, the following tendencies were observed: none of the women had a negative attitude towards thin people and weight control and the positive attitude towards thin people increased with increasing body mass index (BMI) (Jonker *et al*, 2004).

The finding that black women had a neutral attitude towards weight control could be positive, indicating that they could be motivated in the desired direction. The focus should be on clear information presented in a simple, culturally-sensitive way, to explain why obesity could be dangerous to their health. If a larger figure is preferred (Steyn, 2004), acceptable ranges could be explained to the women.

Lack of knowledge does not seem to be very important in weight control motivation (Walker *et al,* 1991; Kumanyika & Guilford-Davenport, 1993). In the POWIRS study, the obese women had the best knowledge about the relationship between obesity, nutrition and health, compared to the normal-weight and overweight women (Jonker *et al*, 2004).

Kumanyika and Guilford-Davenport (1993) mentioned that the perception that black women are not interested in weight control is too simplistic. In their study in African-American women, almost all the overweight women perceived themselves as being overweight, wanted to have a 'normal' weight, were aware of the obesity-related health consequences, had received weight loss advice by health professionals, had tried to lose weight in the past, and many were trying to lose weight at the time of the study. Results from the POWIRS study were consistent with these findings. However, ineffectiveness of weight control attempts suggested by Kumanyika and Guilford-Davenport (1993) were supported by the POWIRS study in black South African women. Barriers to effective weight control identified in the POWIRS study were overeating incidents and emotion-induced eating (Jonker *et al,* 2004).

A 1999 study in Mpumalanga, conducting in-depth interviews with rural black women, has revealed new insights on how obesity is experienced by black women. These women did not perceive obesity in a positive way at all, in fact, it was associated with very negative experiences (Ndlovo *et al,* 1999). Some quotes of the women were: 'A fat body is not nice, I hate obesity'; 'Obese women have a big problem'; 'The obese are found less attractive, seldom beautiful'; 'Obese women are not attractive to men'. The most frequent remark related to health: 'Obesity is a health risk'; 'A fat body is not all right because it causes many diseases'; 'An obese body is not healthy' (Ndlovo *et al,* 1999). Another report indicated that the black culture does not discriminate against obesity, but that discrimination is experienced from other population groups (Mvo *et al,* 1999). Therefore, it seems that despite social tolerance of obesity in the African culture, the woman with obesity have a very negative experience of her situation (Ndlovo *et al,* 1999). Even in a group of poor overweight women in a Cape Town squatter camp, the women expressed the wish to lose their excess weight (Mvo *et al,* 1999).

There seems to be a double standard in the cultural perception of obesity, where on the one hand black people value a larger body size (Steyn, 2004; Puoane *et al,* 2004), while on the other hand experiencing it negatively (Ndlovo *et al,* 1999). It may be that the cultural perception is that a larger body size is associated with respect, dignity and wealth, but once a person becomes obese and begins to experience weight-related physical pain and discomfort or disease (Ndlovo *et al,* 1999; Steyn, 2004), this perception changes. In other words, black people may think that a large body is good, but once they experience it, they realise the opposite. Another possible explanation for the observed double standard may be that a moderately large figure is viewed positively, but a very large (obese) figure is perceived negatively (Ndlovo *et al,* 1999; Baskin *et al,* 2001; Gore, 1999).

Due to the tolerance of obesity by the African culture (Walker *et al,* 2001), it seems that although black women in South Africa are not negative about weight control, there may not be enough motivation for them to lose weight. However, it

could be expected that with the increasing adoption of a Western culture, younger black women would in time adopt Western beauty standards and become more negative about obesity. A strong suggestion of this was already indicated in 1991, where body image and eating behaviour were assessed in black teenage girls in Soweto (Walker *et al,* 1991). A large percentage of the urban black girls were dieting (14%), equal to the percentage of white girls (15%) who dieted. Of the black girls, 68% was dissatisfied with the size of some body parts and 43% wanted certain body parts to be smaller (Walker *et al,* 1991). In a large survey in African-American girls, 61% were dieting (Emmons, 1992). In the USA, the prevalence of eating disorders such as anorexia and bulimia nervosa, thought to be associated with the stigma attached to obesity (Grundy, 1998) and previously thought to be rare in blacks, are increasing in this population (Yanovski, 2000; Striegel-Moore *et al,* 2000[a]). In a group of African-American women, small to medium body sizes were preferred because they viewed a large body size to be associated with negative social consequences (Liburd *et al,* 1999).

The role of food in the African culture

Another potential barrier to weight control in the African culture could be the value and meaning of food. In addition to being a source of nutrition, food is also a sign of warmth, acceptance and friendship (Puoane *et al,* 2004). Daily meat consumption indicates high socio-economic status, while consumption of only vegetables indicates low socio-economic status. Eating large portions of food sends a message that large amounts can be afforded. Africans use food for social occasions, celebrations, rituals, and as a way of welcoming people in their homes. Sweets, ice-cream and cakes are eaten on social and happy occasions, such as birthdays and weddings (Puoane *et al,* 2004). Mvo *et al.* (1999) indicated that food is highly valued because of a history of food insecurity and that it is unacceptable to voluntarily restrict nutrient intakes.

Low birth weight and genetic influences

One of the proposed mechanisms for higher rates of obesity in previously deprived populations is the so-called 'thrifty phenotype' hypothesis. According to this hypothesis, low birth weight predisposes an individual to develop degenerative diseases, such as obesity, NIDDM and cardiovascular disease (CVD) in adult life when exposed to affluence (Hales & Barker, 2001; Jacquet *et al*, 2003; Zimmet *et al*, 2001). It is proposed that *in utero* nutritional deprivation leads to 'programming' of the body to cope with the situation. This 'programming' becomes permanent and predisposes the individual to the mentioned diseases when exposed to abundant food supplies. A number of studies have found evidence to support this hypothesis (Hales & Barker, 2001). In South Africa, the incidence of low birth weight is 15% in the black population (Walker & Charlton, 1998), suggesting a high susceptibility to NCD, which might be a contributing factor to the high obesity rates. Involvement of genetic milieu in this matter has also been suggested in the 'thrifty genotype' hypothesis, proposing that in ancient times, people were hunter-gatherers and their bodies were genetically programmed to survive periods of shortages in foods supplies. To cope with this situation their bodies became insulin resistant. Some evidence for this hypothesis was indicated in Pima-Indians, Australian Aborigines and Pacific Islanders, all populations with high rates of obesity and NIDDM (Zimmet *et al*, 2001).

CONSEQUENCES OF OBESITY

Previous reports indicated a low frequency of obesity-related health consequences in black South African women (Walker *et al*, 1989; Walker *et al*, 1990). In 1998, the THUSA study (1854 subjects) (Vorster *et al*, 2000) assessed the effect of urbanisation on the health status of South African blacks. Body mass index correlated positively with diastolic blood pressure ($r = 0.21$, $p < 0.05$), serum triglycerides ($r = 0.3$, $p < 0.05$), fasting glucose ($r = 0.29$, $p < 0.05$), log fasting insulin ($r = 0.24$, $p < 0.05$) and negatively with HDL cholesterol ($r = -0.38$,

$p < 0.001$). High waist circumference was more strongly associated with NCD risk in black South African women than in white women (Kruger *et al*, 2001).

In 2003, the POWIRS study compared the metabolic profiles of obese with non-obese women. This study indicated higher levels of most of the known metabolic risk factors associated with obesity in the obese than the non-obese women (blood pressure, triglycerides, uric acid, fibrinogen, plasminogen-activator inhibitor-1 (PAI-1), C-reactive protein, fasting glucose, fasting insulin, insulin resistance as well as development of the metabolic syndrome and non-insulin-dependent diabetes mellitus) (Jonker *et al*, 2004). Table 3 compares the prevalence of different metabolic health-risk factors in normal-weight, overweight ansd obese women in this study. Unhealthy lipid profiles occurred with the same frequency in normal-weight, overweight and obese women, as previously observed in South African and USA blacks (Walker, 1998). The evidence of chronic subclinical inflammation indicated by increased levels of C-reactive protein, fibrinogen and possibly PAI-1 in the obese women, known to be predictors of development of cardiovascular disease and NIDDM (Haffner, 2003), may indicate considerable health risk in these women.

Table 3 ***Frequencies of metabolic health-risk factors in black South African women (the POWIRS study)***

	Percentage of women		
	Normal-weight *n = 38* %	***Overweight*** *N = 25* %	***Obese*** *n = 37* %
Metabolic syndrome variable			
Waist circumference > 88 cm	0	8	70
Serum triglycerides ≥ 1.69 mmol/L	0	0	8
Serum total cholesterol > 5.5 mmol/L	5	20	8
Serum HDL cholesterol < 1.29 mmol/L	53	68	59
Serum LDL cholesterol ≥ 3.0 mmol/L	32	36	35
High blood pressure ≥ 130/85 mm Hg	26	52	57
Serum fasting glucose ≥ 6.1 mmol/L	0	4	14
Plasma fibrinogen ≥ 3.0 g/L	79	96	81
Plasma plasminogen activator inhibitor-1 > 7 U/ml	24	32	46
Serum C-reactive protein ≥ 1 mg/L < 3 mg/L	29	36	16
Serum C-reactive protein ≥ 3 mg/L	16	44	68
HOMA-insulin resistance index	5	28	43
Impaired glucose tolerance* (%)	3	4	30
Newly diagnosed diabetes mellitus* (%)	0	8	11
Percentage of women with the metabolic syndrome#	0	8	38

* Impaired glucose tolerance = 2h-post load glucose ≥ 7.8 mmol/L and < 11.1 mmol/L (ADA, 2002)
* Diabetes mellitus = fasting plasma glucose ≥ 7.0 mmol/L and 2h-post load glucose ≥ 11.1 mmol/L (ADA, 2002)
# Metabolic syndrome diagnosed according to the ATPIII definition (Grundy *et al*, 2004)

CONCLUSIONS AND RECOMMENDATIONS

Obesity in black South African women may be associated with increased risk for CVD and NIDDM. It seems that they are weight conscious and most obese women want to lose weight. Although the African culture seems to tolerate obesity, may prefer larger figures than whites and associate a large figure with certain positive attributes, it also seems that obesity is experienced negatively. This may indicate the beginning of a paradigm shift in the black population, once they begin to experience the adverse consequences of obesity.

The time may be ripe for weight control intervention in black South African women. However, more research is needed to properly understand the dynamics underlying obesity. Nevertheless, effective weight control programmes in black women and children have already been implemented in the USA (McNabb *et al,* 1997; Baskin *et al,* 2001; Sanders *et al,* 2002; Fitzgibbon *et al,* 2002). The desired approach was well-summarised by Badenhorst and Walker (1994): 'Nutrition professionals need to develop a comprehensive weight control programme, specifically designed for black women. It should be culturally-sensitive, practical, cost-effective and realistic for women with low education levels and geared to promote self responsibility in ensuring long-term weight control' (Badenhorst & Walker, 1994).

Studies indicated that obesity in black women starts in adolescence (Schiffman *et al,* 2000; Striegel-Moore *et al,* 1999) and that it is more difficult to maintain normal body weight once a woman gets married and becomes a mother (Ball *et al,* 2002). Therefore, intervention should start at a young age, even at primary school age (Badenhorst & Walker, 1994).

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CHAPTER 3

Obesity is associated with increased acute-phase proteins in black South African women – the POWIRS study

Jonker E (M.Sc), Vorster HH (D.Sc), Kruger A (Ph.D), Van Lieshout M (Ph.D), Underhay C (Ph.D)*
School for Physiology, Nutrition and Consumer Sciences, North-West University, Potchefstroom Campus, South Africa
** School for Biokinetics, Recreation and Sport Science, North-West University, Potchefstroom Campus, South Africa*

Summary

Introduction: One-third to half of South African black women is obese (Bourne *et al*, 2002). This study was performed to find baseline data to compare the health-risk profiles of obese and non-obese black women to develop a better understanding of the underlying mechanisms of obesity in these women, who are generally on a low-fat diet (MacIntyre *et al*, 2002).

Subjects and methods: A case-control comparative study with selected volunteers was performed in 100 urban black women of the North-West Province, South Africa, comparing the health-risk profiles of obese and non-obese women. Descriptive statistics, partial Spearman correlations and effects sizes were calculated. ***Results***: Metabolic syndrome components s-uric acid, s-fasting glucose, s-2h-post-load glucose, s-fasting insulin and the HOMA-insulin-resistance index were practical significantly higher in the obese than the normal-weight women. Systolic and diastolic blood pressure, s-triglycerides and s-gamma-glutamyl-transferase had higher levels indicative of practical significance in the obese compared to the normal-weight women. Evidence of an increased acute-phase response in the obese women was indicated by practical significantly higher s-CRP, p-fibrinogen and possibly p-plasminogen-activator-inhibitor-1 (PAI-1). After exclusion of dietary under-reporters, the obese women had practical significantly higher daily intakes of total energy, total carbohydrates, total protein, total fat, saturated fat and dietary fibre. ***Conclusions***: Since CRP, fibrinogen and PAI-1 in obese women may predict the development of diabetes

mellitus and cardiovascular disease, the results of this study may point to a large imminent health burden in these women. It is concluded that culturally-sensitive weight control programmes would be vital to improve the health status of black South African women.

s = serum; p = plasma

Key words: *Obesity, South Africa, black women, inflammatory, metabolic syndrome, health risk*

Introduction

Obesity has recently been declared by the World Health Organisation to be an epidemic (WHO, 1998). In the USA, two in three adults are overweight or obese, compared to less than one in four in the early 1960s (Manson & Bassuk, 2003). Results from the NHANES III survey showed that the age-adjusted prevalence of obesity in the USA 1999-2000 was 30.5% (Flegal *et al*, 2002). The medical consequences of obesity in many population groups are well known (Pi-Sunyer, 1993; Must *et al*, 1999), having the largest impact on the cardiovascular system (Grundy, 1998).

The black population of South Africa is currently experiencing a process of rapid urbanisation (Vorster, 2002) due to political changes and poor economic circumstances in rural areas (May, 1989; James *et al*, 2000). Rapid urbanisation is accompanied by demographic changes and a transition in dietary patterns, nutrient intakes and health, referred to as the epidemiological transition (Popkin, 1994). In developing countries like South Africa, undernutrition and overnutrition often coexist for a period of time, referred to as the 'protracted' transition. The problem with this phenomenon is that the fragile health budgets of these countries are challenged by a double burden of disease, where non-communicable diseases (NCD) due to overnutrition start to emerge, while infectious diseases and undernutrition are still a problem (Vorster *et al*, 1999). The HIV/AIDS epidemic puts an extra burden on this situation (Bourne *et al*, 2002). Although the complications of obesity would not be as serious as in the

case of HIV/AIDS or other infectious diseases, more long-term care would be needed and more people would be affected (Grundy, 1998).

Obesity in black South African women has reached a prevalence of double that of white women (Bourne *et al,* 2002; Vorster, 2002). Obesity was common in traditional (rural) African women (Walker *et al,* 1990), but the incidence of non-communicable diseases (NCD) was low (De Villiers *et al,* 1988). It was argued that they were protected from NCD by their traditional low fat diet (Walker *et al,* 1990). Since these women have become urbanised, their traditional diets have gradually been abandoned for the atherogenic diets of the Western culture, high in fat (especially saturated fat), animal protein and refined sugar and low in complex carbohydrates (Bourne *et al,* 2002; Puska *et al,* 2002; Popkin, 1999). In other aspects their diets have become more nutritionally adequate and varied (MacIntyre *et al,* 2002).

The development of obesity, contributing factors and consequences, remain a puzzle amongst black South African women who are often food-insecure, may have undernourished children (Vorster *et al*, 1997) and do not show the coronary heart disease risk profiles normally associated with obesity (Walker *et al,* 1990). They often follow a low-fat, high-carbohydrate diet (MacIntyre *et al,* 2002), recommended for weight control (WHO, 1998) and optimal health (Vorster *et al,* 2001). The aim of this study was to compare the health-risk profiles of obese and non-obese urban black South African women.

Methods

Subjects, exclusion criteria, study design and ethics

The POWIRS study (Profiles of Women with the Insulin Resistance Syndrome) had a case-control design with selected volunteers. The subjects were 100 urbanised black women of the North-West Province, South Africa. Ages ranged from 19 to 55 years and body mass indices (BMI) from 19 to 50 kg/m^2. Exclusion criteria were pregnancy, lactation, being on medication, being HIV positive, signs of acute infection or temperatures above 37 °C. Women with an education level

lower than grade 7 were excluded because responses to psychological questionnaires (results not reported here) required a certain level of education.

The women were divided into three body mass index (BMI) groups, according to the WHO body-weight classification (Must *et al*, 1999): a normal-weight group (BMI 19.0 - 24.9 kg/m^2; 38 subjects); an overweight group (BMI 25.0 - 29.9 kg/m^2; 25 subjects) and an obese group (BMI >= 30 kg/m^2; 37 subjects).

The study was approved by the Ethics Comittee of the North-West University (Ethics no 03M03). All subjects signed an informed consent form at recruitment. As part of the ethics protocol, subjects immediately received feedback about their individual health status. The employer of the subjects was informed about trends observed.

Order of experimental procedures

The experimental procedure was performed at the Metabolic Unit of the North-West University. Women arrived in groups of ten at the Metabolic Unit in the late afternoon, when the procedures started. After consent forms were signed, anthropometric measurements were taken, with the exception of weight and height. All subjects received the same supper and fasted from 22:00, drinking only tap water. The women spent the night at the Metabolic Unit.

The next morning at 06:00, weight and height were measured. A registered nurse took the women's blood pressures, examined them for clinical signs of malnutrition and took their body temperatures. Fasting blood samples were taken, after which a glucose solution was taken in order to perform an oral glucose-tolerance test (OGTT), followed by repeated blood sampling. The women stayed in bed untill the OGTT was finished.

During and after the OGTT, questionnaires were completed to obtain demographic, dietary intake and physical activity data, and psychological responses. Trained Tswana interviewers assisted in completion of the questionnaires. All the questionnaires were validated for this population group

(Kruger, 2000; MacIntyre *et al,* 2001[a]; MacIntyre *et al,* 2001[b]; Venter *et al,* 2000). After completion of all procedures, the subjects received lunch, could go home, and their travelling expenses were covered.

Anthropometric measurements

Trained anthropometrists measured weight, height, waist circumference and abdominal saggital diameter. Subjects were weighed without shoes in light night wear to the nearest 0.1 kg on a portable electronic scale (Precision Health® scale, A & D Company, Japan). Height was measured to the nearest 0.5 cm with a stadiometer (Invicta, IP 1465, UK) in the standing position, without shoes and with the head in the Frankfort plane position.

Waist circumference (WC) and abdominal saggital diameter were measured to the nearast 0.5 cm using a flexible, but inelastic 7 mm wide steel tape (Holtain®). Waist circumference was measured at the midpoint between the lower rib margin and iliac crest. Abdominal saggital diameter was measured as the largest distance between the roundest part of the abdomen up to the corresponding point on the back, in a horizontal level, with the subject standing.

Blood pressure and clinical examination

A registered nurse measured the blood pressures with a sphygmomanometer (Tycos®) in the supine position. Subjects were examined for clinical signs of malnutrition by assessing their nails, eyes, hair, skin and thyroid. Their oral temperatures were measured using a clinical thermometer.

Blood sample collection

A registered nurse took the blood samples from the *vena cephalica,* using a sterile butterfly (21G) infusion set and syringes (Johnson & Johnson, 21G, 19 mm). After the blood was allowed to clot in glass tubes, serum was prepared by centrifugation of the blood at 3000 rpm for 15 minutes. The serum samples were divided into 1 ml aliquots and frozen at – 84 °C. Citrated blood samples (0.5 ml 0.1 mol/L citrate, pH 4.5 – 4.8, plus 4.5 ml venous blood) were prepared by

centrifugation at 3000 rpm for 10 minutes to separate the platelet-free plasma. The citrated plasma was divided into 1 ml aliquots and frozen at –84 °C.

Oral glucose-tolerance test (OGTT)

After the fasting blood sample was taken, the subjects took a glucose solution containing 75 g glucose (Medicolab® glucose power, Amalgan) to perform a glucose-tolerance test. The glucose solution was taken within 15 minutes. Blood samples were taken at 30, 60, 90 and 120 minutes after the glucose load. The butterfly system was kept in the vein during the 2 hours of the OGTT. The system was kept viable with a heparin solution (2.5 units heparin, 5000 m/ml in 5 ml sterile saline).

Biochemical analyses

The following serum metabolites were measured using the Vitros Chemistry System® (Johnson & Johnson Co, USA) with a colorimetric method: total protein, albumin, gamma-glutamyl-transferase (GGT), uric acid, triglycerides, total cholesterol and HDL cholesterol. LDL cholesterol was calculated using the Friedewald equation (Friedewald *et al,* 1972). Plasma glucose was measured using the hexokinase method from the Chemical Pathology Laboratory, University of Pretoria. Plasma fibrinogen was measured using the Clauss method (Instrumentation Laboratory, with ACL 200 Automated Coagulation Laboratory, Milan, Italy). Plasma plasminogen activator inhibitor-1 (PAI-1) was measured with the Chromogenic assay kit (Spectrolyse®/pL PAI, biopool cat 101 201, Sweden) and Labsystems Multiskan Ascent (USA). Plasma insulin was measured with the enzyme immuno assay (Biosource Europe SA, Belgium). Serum C-reactive protein (CRP) was measured with a high-sensitivity C-reactive protein kit (Immage® Immunochemistry Systems and Beckman Coulter, Inc.)

Calculation of indices

The HOMA-insulin-resistance index was calculated as (fasting insulin in uU/ml) x fasting glucose in mmol/L) / 22.5 (Matthews *et al,* 1985). The body mass index

(BMI) was calculated by the formula body weight in kg / height in meter^2 (Must *et al,* 1999).

Demographic information

A demographic questionnaire, developed and tested for the Tswana population (Kruger, 2000), was used to collect data such as age, education, income, smoking and drinking habits. Trained Tswana field workers assisted in completion of the questionnaires.

Habitual dietary intakes

Habitual dietary intakes were measured by a quantitative food frequency questionnaire, developed and validated for Tswana subjects (MacIntyre *et al,* 2001[a]; MacIntyre *et al,* 2001[b]). Books with photographs of typical foods and portion sizes commonly used by this population (Venter *et al,* 2000) assisted trained Tswana field workers in collecting the data. The food data were coded and analysed by the Food Finder® computer programme of the Medical Research Council (MRC), based on the South African Food Composition Tables (Langenhoven, 1991), to obtain habitual dietary intakes. Dietary under-reporters were identified by energy intake / basal metabolic rate < 1.2 (Goldberg *et al,* 1991).

Diagnosis of the metabolic syndrome

The metabolic syndrome was diagnosed using the National Cholesterol Education Program Adult Treatment Panel III (ATPIII) criteria (Grundy *et al,* 2004): Blood pressure ≥ 130/85 mm Hg, waist circumference > 88 cm, HDL cholesterol < 1.29 mmol/L, triglycerides ≥ 1.69 mmol/L and fasting glucose ≥ 6.1 mmol/L. The metabolic syndrome was diagnosed when three or more of these criteria were present in an individual.

Statistical analyses

Statistical analyses were performed, using the SPSS 11.0 for Windows and STATISTICA 6.0 packages. Descriptive statistics were performed for all variables, calculating means, standard deviations and 95% confidence intervals.

Partial Spearman correlation coefficients were calculated, adjusting for age. Age-adjusted analysis of covariance was used to determine significant differences between the BMI groups. Effect sizes were calculated to indicate practical significant differences between means, because the sample was not truly random and consisted of volunteers (Ellis & Steyn, 2003). A medium effect size ($d \geq 0.5$) indicated possible practical significance and a large effect size ($d \geq 0.8$) practical significance. Practical significance is a measure that indicates a difference large enough to have an effect in practice. The difference indicated by the effect size is independent of units and sample size and also relate to the spread of the data. A natural way to comment on practical significance is to divide the difference between the two means under consideration by the standard deviation (Ellis & Steyn, 2003).

Results

Table 1 ***Characteristics of the study groups***

	Normal-weight group *N = 38*	***Overweight group*** *N = 25*	***Obese group*** *N = 37*
Mean age (years)	28.5 ± 7.6	30.8 ± 8.5	34.3 ± 9.1
Mean body mass index (BMI) (kg/m^2)	22.0 ± 1.5	27.2 ± 1.3	34.6 ± 4.2

Table 2 ***Frequencies of the metabolic syndrome* in normal-weight, overweight and obese women***

	Normal-weight *N = 38*		***Overweight*** *N = 25*		***Obese*** *N = 38*	
	N	%	*N*	%	*N*	%
With metabolic syndrome	0	0	2	8	14	38
Without metabolic syndrome	38	100	23	92	23	62

Prevalence of the metabolic syndrome (Table 2)

None of the women in the normal-weight group, 8% of the women in the overweight group and 38% of the women in the obese group had the metabolic syndrome.

Means of metabolic syndrome components (Table 3)

Although not always indicating practical significance, levels of most metabolic syndrome components increased gradually from the normal-weight towards the obese group. Serum-albumin and s-protein did not differ practical significantly between the three groups of women. BMI and waist circumference differed practical significantly between all three body-weight groups.

Table 3 ***Age-adjusted means of metabolic syndrome components***

	Normal-weight *N = 38*		***Overweight*** *N = 25*		***Obese*** *N = 37*	
	Mean	***95% CI***	***Mean***	***95% CI***	***Mean***	***95% CI***
Variable						
Age (years)	28.5 a*b*	26.0-31.0	30.8 a*c*	27.3-34.3	34.3 b*c(	31.3-37.4
BMI (kg/m^2)	22.0 a*b*	21.5-22.5	27.2 a*c*	26.6-27.7	34.6 b*c*	33.2-36.0
Waist circumference (cm)	70.4 a*b*	68.7-72.2	80.0 a*c*	77.4-82.7	93.9 b*c*	90.2-97.5
Systolic blood pressure (mm Hg)	123.2 ab	118.1-128.3	132.9 ab	126.7-139.1	131.0 a	125.7-136.2
Diastolic blood pressure (mm Hg)	75.8 a	72.2-79.4	78.5	74.1-82.9	80.8 a	77.0-84.5
s-Total cholesterol (mmol/L)	4.2	3.91-4.5	4.4	4.1-4.8	4.1	3.8-4.4
s-HDL cholesterol (mmol/L)	1.3	1.2-1.4	1.2	1.1-1.3	1.2	1.1-1.3
s-LDL cholesterol (mmol/L)	2.8	2.5-3.0	3.1	2.7-3.4	2.7	2.5-3.0
s-Triglycerides (mmol/L)	0.6 a	0.5 – 0.7	0.7	0.5-0.8	0.8 a	0.7-0.9
s-Uric acid (mmol/L)	269.9 a*	244.9-294.9	282.1 b	251.8-312.5	327.0 a*b	301.3-352.6
p-Plasma fibrinogen (g/L)	3.4 a*b	3.1-3.8	4.1 b	3.7-4.5	4.2 a*	3.9-4.6
p-PAI-1 (u/ml)	4.5 a	3.0-5.9	5.2 b	3.2-7.2	7.4 ab	5.9-9.0
s-Total protein (g/L)	87.4	84.2-90.5	90.5	86.7-94.4	86.7	83.4-89.9
s-Albumin (g/L)	42.3	41.2-43.5	41.5	39.9-43.1	40.6	39.4-41.8
White blood cells (10e3u/ml)	5.2	4.6-5.8	5.1	4.4-5.8	5.8	5.2-6.4
s-GGT (u/L)	28.6 a	22.0-35.2	36.0	26.9-45.0	41.2 a	34.3-48.1
s-C-reactive protein (mg/L)	1.9 a*	0.6-3.2	3.2 b	1.6-4.8	6.0 a*b	4.7-7.2
s-Fasting free fatty acids (mmol/L)	0.5	0.4-0.6	0.5	0.3-0.6	0.6	0.5-0.7
s-Fasting glucose (mmol/L)	4.9 a*b	4.7-5.0	5.2 b	4.9-5.4	5.3 a*	5.1-5.5
s-2h-Post-load glucose (mmol/L)	6.0 a*	5.4-6.6	6.3 b	5.5-7.1	7.3 a*b	6.6-7.9
s-Fasting insulin (pmol/L)	76.3 a*	63.1-89.6	93.8	75.6-111.9	110.4 a*	96.5-124.4
HOMA-insulin-resistance index	2.4 a*b	1.9-2.8	3.1 c	2.5-3.7	3.7 a*bc	3.3-4.2

CI = confidence interval
Variables with the same symbol may differ practical significantly (d ≥ 0.5)
*Variables with the same symbol marked with * differ practical significantly (d ≥ 0.8)*
s = serum; p = plasma

Normal-weight versus obese women

Levels of s-uric acid, s-fasting glucose, s-2h-post-load glucose, s-fasting insulin, the HOMA-insulin-resistance index, s-CRP and p-fibrinogen were practical significantly higher in the obese than the normal-weight women. Mean systolic and diastolic blood pressure, s-triglycerides, s-GGT and p-PAI-1 were higher in

the obese compared to the normal-weight women, indicative of practical significance.

Normal-weight versus overweight and overweight versus obese women

Mean systolic blood pressure, p-fibrinogen, s-fasting glucose, and the HOMA-insulin resistance index were higher indicative of practical significance in the overweight than the normal-weight women. Mean serum-uric acid, p-PAI-1, s-CRP, s-2h-post-load glucose and the HOMA-insulin-resistance index were higher indicative of practical significance in the obese than the overweight women.

Association of obesity with metabolic syndrome components (Table 4)

Obesity measures correlated positively with most metabolic syndrome components, i.e. blood pressure, s-triglycerides, s-uric acid, s-GGT, p-fibrinogen, p-PAI-1, s-fasting glucose, s-fasting insulin and the HOMA-insulin-resistance index. Except for s-CRP, serum-fasting insulin and the HOMA-insulin-resistance index showed the strongest relationships with obesity measures, compared to other variables.

Table 4 ***Partial Spearman correlation coefficients of BMI, waist circumference, and abdominal saggital diameter with metabolic syndrome components***

Metabolic syndrome variable	***BMI***	***Waist circumference***	***Abdominal diameter***
	r	*r*	*r*
Systolic blood pressure	*NS*	*NS*	*NS*
Diastolic blood pressure	0.34	0.41*	0.41
s-HDL cholesterol	*NS*	- 0.22	- 0.23
s-Triglycerides	0.31	0.38*	0.35
s-Uric acid	0.32	0.34	0.27
s-GGT	0.31	0.35	0.33
s-C-reactive protein	0.60*	0.54*	0.55*
p-Fibrinogen	0.29	0.23	0.26
p-PAI-1	0.31	0.31	0.26
White blood cells	*NS*	*NS*	*NS*
s-Fasting glucose	0.36*	0.37*	0.40*
s-Fasting insulin	0.46*	0.56*	0.47*
s-2h-post-load glucose	0.32	0.34	0.33
s-2h-post-load insulin	0.28	0.36	0.32
s-2h-post-load free fatty acids	*NS*	*NS*	*NS*
HOMA-insulin-resistance index	0.52*	0.62*	0.53*
Body mass index	*NS*	0.88*	0.88*
Waist circumference	0.86*	*NS*	0.95*
Abdominal saggital diameter	0.88*	*NS*	*NS*

*$p < 0.05$, exept those marked with *;* ** $p < 0.0001$*

NS = non-significant

Association of acute-phase proteins with obesity measures (Table 4)

Serum-CRP showed a strong positive correlation with measures of obesity, the strongest correlation with BMI ($r = 0.60$, $p < 0.0001$), followed by abdominal saggital diameter ($r = 0.55$ $p < 0.0001$) and then waist circumference ($r = 0.54$, $p < 0.0001$). Abdominal saggital diameter is considered as a more accurate surrogate measure of visceral fat than waist circumference (Pouliot *et al,* 1994). Plasma-fibrinogen showed a strongly positive correlation with BMI and less strongly with waist circumference and abdominal saggital diameter. Serum-CRP and p-fibrinogen correlated strongly positive with each other. Plasma-PAI-1 showed a strong and equally positive correlation with BMI and waist circumference and a slightly weaker correlation with abdominal saggital diameter. The acute-phase proteins s-CRP, p-fibrinogen and p-PAI-1 showed the strongest correlations with obesity measures.

Dietary intakes (Table 5)

It seemed that a large number of women probably under-reported dietary intakes (67%). Therefore, results are shown for the total group as well as under-reporters. Results are discussed only for the non-under-reporters. Dietary intakes did not differ practical significantly between the three groups of women when under-reporters were included in the analyses.

Normal-weight versus obese women

Most nutrient intakes were practical significantly higher in the obese than the normal-weight women, such as total energy, total carbohydrates, total protein, plant and animal protein, percentage total protein, total fat, saturated and mono-unsaturated fat and dietary fibre.

Normal-weight versus overweight women and overweight versus obese women

The overweight and obese women had practical significantly higher intakes of animal protein and percentage of total protein than the normal-weight women. The obese women had practical significantly higher intakes than the overweight

women of total energy, total carbohydrates, total protein, animal protein and dietary fibre.

Table 5 ***Dietary intakes of the non-under-reporters***

Nutrient	***Total group*** *N = 33* *Mean ± SD*	***Normal-weight*** *N = 23* *Mean ± SD*	***Overweight*** *N = 8* *Mean ± SD*	***Obese*** *N = 5* *Mean ± SD*
Energy (kJ)	8790 ± 1839	8419 b ± 1874	8730 a ± 1531	10 591 ab ± 1148
95% CI	8168.0 – 9412.0	7609.0 – 9320.0	7450.0 – 10 010.0	9166.0 – 12 016.0
Carbohydrate (CHO) (g)	263.7 ± 60.5	257.2 b ± 65.8	253.0 a ± 37.4	311.0 ab ± 49.6
95% CI	243.2 – 284.2	228.7 - 285.6	221.7 – 284.2	249.5 – 372.6
Added sugar (g)	60.2 ± 25.4	60.6 ± 27.0	55.7 ± 12.9	65.2 ± 35.7
95% CI	51.6 – 68.8	49.0 – 72.3	44.9 – 66.5	20.9 –109.6
Dietary fibre (g)	17.2 ± 4.6	16.5 b ± 4.7	16.2 a ± 2.8	22.2 ab ± 3.8
95% CI	15.7 – 18.7	14.5 – 18.5	13.8 – 18.5	17.5 – 26.9
Total protein (g)	75.6 ± 19.9	69.4 b ± 18.4	80.2 a ± 16.7	97.3 ab ± 15.6
95% CI	68.9 – 82.4	61.4 – 77.3	66.2 – 94.1	78.0 – 116.6
Plant protein (g)	25.0 ± 6.7	24.1 a ± 7.2	23.8 ± 4.9	31.0 a ± 3.5
95% CI	22.8 – 27.3	21.0 – 27.2	19.7 – 27.9	26.6 – 35.4
Animal protein (g)	46.1 ± 16.6	40.4 ab ± 14.6	52.9 ac ± 15.7	62.0 bc ± 14.8
95% CI	40.5 – 51.8	34.0 – 46.7	39.7 – 66.0	43.6 – 80.4
Total fat (g)	70.8 ± 18.2	66.3 a ± 16.6	74.3 ± 20.6	85.9 a ± 14.1
95% CI	64.7 – 77.0	59.1 – 73.5	57.1 – 91.5	68.4 – 103.5
Saturated fat (g)	23.1 ± 6.3	21.1 a ± 5.5	25.8 ± 7.0	28.2 a ± 4.5
95% CI	21.0 – 25.3	18.7 – 23.5	19.9 – 31.7	22.6 – 33.8
Mono-unsaturated fat (g)	24.1 ± 6.7	22.7 a ± 6.1	24.7 ± 7.7	29.6 a ± 6.1
95% CI	21.8 – 26.4	20.0 – 25.3	18.3 – 31.1	22.1 – 37.2
Poly-unsaturated fat (g)	16.4 ± 5.1	15.8 ± 4.8	16.5 ± 5.8	19.2 ± 5.9
95% CI	14.7 – 18.2	13.7 – 17.9	11.7 – 21.3	11.9 – 26.5
Alcohol (g)	3.7 ± 6.3	4.6 ± 7.4	1.4 ± 2.1	3.1 ± 5.2
95% CI	1.6 – 5.9	1.4 – 7.8	0.3 – 3.2	3.4 – 9.7
% Energy from (CHO)	54.4 ± 5.2	55.1 ± 5.0	52.9 ± 5.6	53.6 ± 5.8
95% CI	52.6 – 56.1	52.9 – 57.2	48.2 – 57.6	46.4 – 60.8
% Energy from added sugar	11.7 ± 4.2	12.2 ± 4.4	11.0 ± 2.9	10.2 ± 5.0
95% CI	10.3 – 13.1	10.3 – 14.1	8.6 – 13.5	4.0 – 16.3
% Energy from total protein	14.5 ± 1.7	13.9 ab ± 1.7	15.6 a ± 1.5	15.6 b ± 1.4
95% CI	13.9 – 15.1	13.2 – 14.7	14.3 – 16.8	13.9 – 17.3
% Energy from total fat	29.7 ± 4.0	29.2 ± 4.0	31.1 ± 4.4	30.0 ± 3.8
95% CI	28.4 – 31.1	27.4 – 30.9	27.4 – 34.7	25.4 – 34.7
% Energy from alcohol	1.3 ± 2.3	1.7 ± 2.7	0.4 ± 0.6	0.8 ± 1.3
95% CI	0.5 – 2.1	0.5 – 2.9	0.1 – 0.9	0.9 – 2.4

Variables with the same symbol differ practical significantly (d ≥ 0.8)
SD = standard deviation

Table 5 (cont.) ***Dietary intakes of the total group (under-rerporters included)***

Nutrient	*Total group* *N = 100* *Mean ± SD*	*Normal-weight* *N = 38* *Mean ± SD*	*Overweight* *N = 25* *Mean ± SD*	*Obese* *N = 37* *Mean ± SD*
Energy (kJ)	7151.3 ± 1939.9	7352.1 ± 2007.4	6903.0 ± 1729.6	7113.0 ± 2031.2
95% CI	6766.4 – 7536.3	6692.2 – 8011.9	6189.1 – 7617.0	6435.7 – 7790.2
Carbohydrate (CHO) (g)	211.3 ± 63.0	223.7 ± 67.4	203.1 ± 50.2	204.0 ± 65.7
95% CI	198.7 – 223.8	201.5 – 245.8	182.4 – 223.8	182.1 – 225.9
Added sugar (g)	50.0 ± 22.4	55.0 ± 24.2	49.5 ± 20.9	45.2 ± 21.0
95% CI	45.6 – 54.5	47.1 – 63.0	40.9 – 58.1	38.2 – 52.2
Dietary fibre (g)	14.0 ± 4.7	14.1 ± 4.9	13.3 ± 3.7	14.5 ± 5.1
95% CI	13.1 – 15.0	12.5 – 15.7	11.7 – 14.8	12.8 – 16.2
Total protein (g)	60.4 ± 19.4	60.5 ± 18.8	60.7 ± 19.9	60.2 ± 20.3
95% CI	56.6 – 64.3	54.3 – 66.7	52.5 – 68.9	53.4 – 67.0
Plant protein (g)	20.1 ± 6.7	20.4 ± 7.6	18.7 ± 6.1	20.6 ± 6.2
95% CI	18.7 – 21.4	17.9 – 22.9	16.2 – 21.3	18.6 – 22.7
Animal protein (g)	36.3 ± 15.4	35.3 ± 13.4	38.4 ± 17.0	35.7 ± 16.5
95% CI	33.2 – 39.3	30.9 – 39.8	31.4 – 45.4	30.2 – 41.2
Total fat (g)	59.3 ± 17.2	59.0 ± 16.2	58.3 ± 18.1	60.3 ± 17.9
95% CI	55.9 – 62.7	53.7 – 64.3	50.8 – 65.8	54.3 – 66.3
Saturated fat (g)	18.9 ± 6.1	18.8 ± 5.5	19.2 ± 7.1	18.8 ± 6.3
95% CI	17.7 – 20.1	17.0 – 20.6	16.3 – 22.1	16.7 – 20.9
Mono-unsaturated fat (g)	20.0 ± 6.2	20.1 ± 5.9	19.6 ± 6.6	20.2 ± 6.5
95% CI	18.8 – 21.2	18.1 – 22.0	16.9 – 22.3	18.0 – 22.4
Poly-unsaturated fat (g)	14.4 ± 4.5	14.2 ± 4.5	13.8 ± 4.2	15.0 ± 4.8
95% CI	13.5 – 15.3	12.7 – 15.7	12.1 – 15.5	13.4 – 16.6
Alcohol (g)	2.6 ± 4.8	3.4 ± 6.3	1.3 ± 2.2	2.6 ± 4.1
95% CI	1.6 – 3.5	1.3 – 5.4	0.3 – 2.2	1.2 – 4.0
% Energy from (CHO)	53.9 ± 4.9	54.8 ± 4.4	53.6 ± 6.0	53.1 ± 4.5
95% CI	52.9 – 54.8	53.3 – 56.2	51.1 – 56.1	51.6 – 54.6
% Energy from added sugar	11.9 ± 4.2	12.9 ± 4.8	12.3 ± 4.6	10.7 ± 3.0
95% CI	11.1 – 12.8	11.3 – 14.5	10.4 – 14.2	9.7 – 11.7
% Energy from total protein	14.3 ± 1.9	13.9 ± 1.6	14.7 ± 2.0	14.3 ± 2.0
95% CI	13.9 – 14.7	13.4 – 14.5	13.9 – 15.6	13.7 – 15.0
% Energy from total fat	30.8 ± 3.8	29.9 ± 3.7	31.1 ± 4.1	31.5 ± 3.7
95% CI	30.0 – 31.5	28.7 – 31.1	29.4 – 32.8	30.3 – 32.8
% Energy from alcohol	1.0 ± 1.9	1.3 ± 2.4	0.6 ± 1.1	1.0 ± 1.6
95% CI	0.6 – 1.4	0.5 – 2.1	0.1 – 1.0	0.5 – 1.5

Evaluation of dietary intakes

From Table 5 it appears that intakes of carbohydrates, protein and fat were within the ranges of dietary guidelines for all three groups of women (carbohydrates 50-55%, protein 10-20% and fat ≤ 30%) (ADSA, 2000). The daily dietary fibre recommendation of 20 g (ADSA, 2000) per day was met by the obese (22 g) and almost by the normal-weight and overweight women (16.5 and 16.2 g respectively). Reported alcohol intake was very low, 1.7% of total energy in the normal-weight women and 0.4 and 0.8% in the overweight and obese women respectively. The percentages of sugar was 12.2, 11.0 and 10.2% of total energy, respectively, and therefore slightly higher in the normal-weight than the

obese women. The recommendation of 10% of total energy allowed from sugar (ADSA, 2000) was slightly exceeded by all the groups.

When under-reporters were excluded, the total energy intakes of all three groups of women might be excessive (8419, 8730 and 10 591 kJ respectively). The average ideal body weight for a woman in this study was 60 kg (BMI = 24.0 kg/m^2), which means that the daily energy requirement would be 7500 kJ for an inactive woman (125 kJ/kg ideal body weight/day) (ADSA, 2000).

Dietary intakes did not correlate with or predict development of the metabolic syndrome or increased acute-phase proteins in a logistic regression model (results not shown).

Association of obesity with dietary intakes (Table 6)

Strong positive correlations were found for BMI and waist circumference with intakes of total energy, total protein, animal protein, total fat and saturated fat, when underreporters were excluded. Abdominal saggital diameter showed a slightly stronger correlation with the mentioned nutrients than waist circumference.

Table 6 ***Partial Spearman correlation coefficients for nutrient intakes with BMI and waist circumference (non-under-reporters) (n = 33)***

	BMI	***Waist circumference***	***Abdominal saggital diameter***
Nutrient	***r***	***r***	***r***
Total energy (kJ)	0.43	0.55	0.57
Total protein (g)	0.57	0.67	0.71
Total protein, % of total energy	0.51	0.53	0.55
Animal protein (g)	0.59	0.67	0.68
Total fat (g)	0.49	0.65	0.68
Total fat, % of total energy		0.43	0.44
Saturated fat (g)	0.58	0.68	0.69
Saturated fat, % of total energy	0.37	0.42	0.40

$p < 0.05$

Discussion

The aim of the POWIRS study was to compare the health-risk profiles of obese and non-obese urban black women of the North-West Province, South Africa. The salient observations of the study were the prevalence of the metabolic syndrome (diagnosed by ATPIII criteria; Grundy *et al,* 2004) in 38% of the obese compared to none of the normal-weight women and 8% of the overweight women. The metabolic syndrome is defined as a cluster of cardiovascular risk factors and is associated with increased risk of non-insulin-dependent diabetes mellitus (NIDDM) and cardiovascular disease (CVD) (Grundy *et al,* 2004). The finding that the metabolic syndrome occurred predominantly in the obese women is consistent with findings in other populations, describing the metabolic syndrome to be associated with obesity (Vega, 2001). Practical significantly higher levels of the metabolic syndrome components s-uric acid, s-fasting glucose, s-2h-post-load glucose, s-fasting insulin, the HOMA-insulin-resistance index, s-C-reactive protein and p-fibrinogen were found in the obese compared to the normal-weight women. Mean systolic and diastolic blood pressure, s-triglycerides, s-gamma-glutamyl-transferase and p-plasminogen activator inhibitor-1 (PAI-1) were higher, indicative of practical significance in the obese than the normal-weight women. Of particular interest was the occurrence of increased levels of acute-phase proteins (s-CRP, p-fibrinogen and possibly p-PAI-1) in the obese women, indicating that in these women obesity was asssociated with an acute phase-response. When dietary under-reporters had been excluded, the obese women had practical significantly higher daily intakes of macronutrients and dietary fibre than the normal-weight women. For most variables, including nutrient intakes, overweight women had levels intermediary to those of the normal-weight and obese women, except for total serum protein, albumin and cholesterol, which did not increase with BMI. Therefore, overweight probably represents a transitional phase from normal weight towards obesity.

The increased levels of acute-phase proteins, s-CRP, p-fibrinogen and possibly p-PAI-1, observed in the obese women in this study are particularly important.

Recent interest has centred on the role of subclinical inflammation in obesity (Yudkin, 2003; Das, 2000). It is suggested that cytokines, such as IL-6 and TNF-alpha, is released by adipose tissue, especially visceral adipose tissue (Yudkin, 2003), which stimulates the production of acute-phase proteins, such as CRP and fibrinogen by the liver (Haffner, 2003). Therefore, obesity was recently described as a state of chronic subclinical inflammation (Yudkin, 2003; Das, 2000). PAI-1 is also a product released by visceral adipose tissue (Janaud-Delenne *et al*, 1998) and CRP increases its expression (Devaraj *et al*, 2003). In prospective studies, CRP has been an independent predictor of myocardial infarction, stroke, and non-insulin-dependent diabetes mellitus (NIDDM) (Ridker, 2003; Ridker *et al*, 2003). Both fibrinogen and PAI-1 have also been associated with the development of cardiovascular disease (CVD) (Janaud-Delenne *et al*, 1998; Tracy, 2003) and NIDDM (Haffner, 2003).

In this study, s-CRP was strongly associated with obesity ($r = 0.60$, $p < 0.0001$), as measured by body mass index, as well as abdominal obesity, measured by high waist circumference and abdominal saggital diameter ($r = 0.54$ and 0.55 respectively, $p < 0.0001$). Similar results were found in the NHANES survey (Visser *et al*, 1999), the IRAS study (Festa *et al*, 2000), the Healthy Women Study (Barinas-Mitchell *et al*, 2001) and the Women's Health Study (Ridker, 2003). BMI was also the best independent predictor of CRP in this study. NHANES found CRP to be strongly associated with all measures of obesity (Visser *et al*, 1999). The correlation between CRP and BMI was stronger in this study ($r = 0.60$) than in other studies, with correlation coefficients (r) ranging from 0.3 to 0.43 (Festa *et al*, 2000; Pannacciulli *et al*, 2001; Barinas-Mitchell *et al*, 2001; Haffner, 2003). It seemed that BMI (general obesity) had a slightly stronger association with CRP than waist circumference (abdominal obesity). Some other studies found that abdominal obesity (waist circumference) was slightly stronger associated with CRP than BMI (Festa *et al*, 2000; Pannacciulli *et al*, 2001; Haffner, 2003), although another study supported our results (Barinas-Mitchell *et al*, 2001). Nevertheless, CRP seemed to be strongly associated with obesity, whether it was general or abdominal obesity.

Levels of CRP rise acutely with systemic infection. However, in asymptomatic individuals CRP levels showed only weak and inconsistent correlations with antibodies against some common pathogens. Moreover, percentage of body fat as measured by DEXA showed a correlation coefficient of 0.85 with CRP (Yudkin, 2003).

The USA Centres for Disease Control and Prevention / American Heart Association Working Group has endorsed the use of CRP in risk-screening in clinical practice (Pearson *et al,* 2003). It is a stable and sensitive marker of subclinical inflammation, with high independent predictive value for development of diabetes, myocardial infarction and stroke (Ridker, 2003). Cut-off values for CVD risk are: CRP < 1 mg/L = low risk, 1 - < 3 mg/L = moderate risk and ≥ 3 mg/L = high risk (Pearson *et al,* 2003). According to these cut-off values, the obese women in our study had an extremely high risk (CRP = 6.0 mg/L), the overweight women a high risk (CRP = 3.2 mg/L) and the normal-weight women a moderate risk (CRP = 1.9 mg/L) for CVD.

Consistent with findings in other populations, higher fibrinogen levels were found in the obese than the normal-weight women (Raynaud *et al,* 2000). A level of 3 g/L is associated with high cardiovascular risk (James *et al*, 2000). Levels above this cut-off value were also found in the normal-weight women, as also observed in the THUSA study (James *et al,* 2000). It has been described that Africans have higher levels of fibrinogen (Vorster *et al,* 1998). Results from the THUSA study suggested that urbanisation probably contributes to higher fibrinogen levels in this population, which is possibly involved in the observed higher stroke incidence (James *et al,* 2000).

The main proposed source of CRP in healthy individuals is excess adipose tissue (Després, 2003; Yudkin, 2003). However, in our study CRP levels were also increased in the normal-weight women. Higher CRP levels in black than in white people have been described (Barinas-Mitchell *et al,* 2001), but the levels seen in our women have reached high-risk levels. The reason for the high CRP levels in

the normal-weight women is uncertain. It is not known whether urbanisation also contributes to higher CRP levels in this population, as observed for fibrinogen. In previous studies, low-grade inflammation was linked to psychosocial stress (Hjemdahl, 2002), which might have been a factor in the women, since high levels of stress and psychological symptomatology were previously described in this population (Vorster *et al,* 2000; Steyn *et al,* 2000).

Obesity seems to exaggerate the health-risk status, as reflected in higher levels of insulin resistance and development of the metabolic syndrome and NIDDM in the obese women. Obesity may increase the health risk in three ways. Firstly, obesity directly contributes to higher CRP levels due to excess fat tissue (Das, 2000). Secondly, obesity leads to insulin resistance (Grundy *et al,* 2002), which on its own may lead to increased CRP and fibrinogen production (Haffner, 2003). Thirdly, obesity leads to multiple risk factors, which may injure the arterial wall and elicit an inflammatory reponse. Proof for the latter hypothesis has been indicated in the finding that drugs that decrease metabolic risk factors also decrease CRP levels (Grundy, 2003).

Despite prudent dietary intakes, the women in this study were obese. However, if one looks at the total daily energy intakes, a different picture is obtained. The total energy intakes of the women increased with increasing BMI, being 10 590 kJ in the obese and 8 420 kJ in the normal-weight women when underreporters were excluded. The obese women were older than the normal-weight women (mean age 34.3 *vs* 28.5 years), yet they consumed much more calories, while the opposite would be desirable since metabolic rate decreases with age (Grundy, 1998). On average the ideal body weight for a woman in this study was about 60 kg (BMI = 24 kg/m^2) and she would need 7500 kilojoules per day (125 kJ/kg/day) if she would be inactive (ADSA, 2000). Low energy expenditure, including low metabolic rate, has been described in African women (O'Keefe *et al,* 1988; Weinsier *et al,* 2002; Jones *et al,* 2004). Therefore, the obese women might have consumed approximately 3000 kJ more than needed to maintain

ideal body weight and approximately 2000 kJ more than the normal-weight women.

Obesity in black women with low fat intake has been described previously (De Villiers *et al,* 1988; Walker & Segal, 1980). In Santiago, Chile, severely obese women were found with fat intakes of 22-31% of energy (Walker *et al,* 1990). Walker and Segal (1980) described carbohydrate-induced obesity, proposing that in black women, excess carbohydrate instead of fat leads to obesity. Another study comparing dietary intakes of white and black women in the USA, indicated that more black women who were overweight had appropriate fat intake, contrary to the situation in white women, where high fat intakes were positively associated with obesity (Cook *et al,* 2000). The THUSA study found a weaker correlation between BMI with fat and energy intakes respectively ($r = 0.05$, $p \leq 0.05$ and $r = 0.05$, $p \leq 0.05$) (Kruger *et al,* 2002) than this study ($r = 0.49$ and 0.43, $p < 0.05$ for fat and energy respectively).

It was suspected that a high percentage of the women in this study under-reported their dietary intakes (67%). This could be expected, since the group consisted of 62% overweight and obese women and under-reporting is a known phenomenon in these people (Ballard-Barbash *et al,* 1996). The food frequency questionnaire employed in this study also tends to under-report intakes in this population (MacIntyre *et al,* 2002). Other aspects associated with under-reporting are dieting and weight consciousness, which might have been a factor in our subjects. Under-reporting could also be associated with socially desirable responses, where foods perceived as bad for health would be under-reported (Livingstone & Black, 2003). Foods high in fat and sugar in particular, are important under-reported items (Macdiarmid *et al,* 1998). Under-reporting as high as 71% in the overweight population has been reported previously in the literature (Mennen *et al,* 2000). Although the number of subjects was small after the under-reporters had been excluded, the dietary intakes of the non-under-reporters were very similar to those reported for urbanised black women in the THUSA study (James *et al,* 2000).

The results of this study suggest that obesity in this group of black South African women does not lead to increased serum total cholesterol levels like in white populations, consistent with results in USA blacks (Pereira *et al,* 2002; Walker, 1998). Another South African study supported our results, where black female students with a high prevalence of obesity had low rates of hyperlipidaemia (Steyn *et al,* 2000). However, it seemed that obesity leads to increased levels of acute-phase proteins (s-CRP, p-fibrinogen and possibly p-PAI-1) in black South African women, indicating a state of chronic low-grade inflammation. Since CRP, fibrinogen and PAI-1 are known to predict the development of NIDDM, myocardial infarction and stroke (Haffner, 2003; Ridker, 2003), obesity seems to carry considerable health risk in these women. This study may have discovered a possible underlying determinant of the high emergence of NIDDM and stroke in African women, if the results could be reproduced in a larger number of women. Further, obesity seemed to result in abnormal glucose homeostasis and development of the metabolic syndrome in these women, possibly with a similar profile seen in African-Americans (Ford *et al,* 2002). Since CRP, PAI-1, fibrinogen and metabolic syndrome components could be decreased by weight loss and exercise (Church *et al,* 2002; Abrams, 2003; Janaud-Delenne *et al,* 1998; Rauramaa & Vaisanen, 1999), appropriate lifestyle changes could considerably decrease the health risk in these women.

The strengths of this study were the case-control design, enabling comparison of the health-risk profiles of normal-weight, overweight, and obese women. The limitations of this study were the high percentage of underreporters in the dietary analysis. Further, cross-sectional studies cannot give results on the mechanisms involved in obesity. However, a follow-up study of these women within a few years would be of great value.

Conclusions

In a group of urban black women of the North-West Province, South Africa, obesity was associated with increased acute-phase proteins, abnormal glucose homeostasis and development of the metabolic syndrome. Obesity did not have an obvious adverse effect on serum total cholesterol levels. The results may point to a serious imminent health burden in urbanised black South African women and therefore obesity in this population should be prevented and treated. More research is needed to explore the mechanisms leading to obesity to enable health authorities to develop culturally-sensitive weight control programmes.

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CHAPTER 4

Insulin resistance, the metabolic syndrome and acute-phase proteins in black South African women – the POWIRS study

Jonker E (M.Sc), Vorster HH (D.Sc), Kruger A (Ph.D), Van Lieshout M (Ph.D), Underhay C (Ph.D)*
School for Physiology, Nutrition and Consumer Sciences, North-West University, Potchefstroom Campus, South Africa
** School for Biokinetics, Recreation and Sport Science, North-West University, Potchefstroom Campus, South Africa*

Summary

Introduction: The aim of this study was to describe the metabolic syndrome in black South African women and to explore the association of acute-phase proteins with the metabolic syndrome and insulin resistance.

Subjects and methods: A case-control study with 100 apparently healthy, non-pregnant, urban black women of the North-West Province, South Africa, was performed. The subjects were recruited as selected volunteers. Descriptive statistics, partial Spearman correlations, effects sizes and odds ratios were calculated. ***Results:*** Women with the metabolic syndrome were characterised by higher age, body mass index (BMI), s-2h post-load glucose, s-fasting insulin, s-2h-post-load insulin, the HOMA-insulin resistance index and possibly s-gamma-glutamyl-transferase and s-uric acid. The mean acute-phase protein s-C-reactive protein (CRP) were higher, indicative of practical significance in women with the metabolic syndrome compared to those without the syndrome. Other acute-phase proteins, p-fibrinogen and p-plasminogen activator inhibitor-1 (PAI-1) were not associated with the metabolic syndrome. With increasing HOMA-insulin-resistance index, the women had higher BMI, waist circumference, systolic and diastolic blood pressure, s-uric acid, s-gamma-glutamyl-transferase (GGT), p-PAI-1, s-CRP, s-fasting glucose, s-2h-post-load glucose, s-fasting insulin, s-2h-post-load insulin, 2h-post-load free fatty acids, haemoglobin,

haematocrit and white blood cells and lower s-HDL cholesterol. The acute-phase proteins s-CRP and p-PAI-1 were positively associated with the HOMA-insulin-resistance index, but no association was found with p-fibrinogen.

Conclusions: The acute-phase protein CRP is possibly associated with the metabolic syndrome in this group of women. CRP and PAI-1 was positively associated with insulin resistance. Inflammation may be a possible underlying determinant of the high rates of hypertension, diabetes mellitus and stroke in this population.

s= serum; p = plasma

Key words: *Insulin resistance, metabolic syndrome, inflammatory, South Africa, black women*

Introduction

Due to a process of rapid urbanisation in the black population of South Africa, accompanied by an increasingly atherogenic diet and decreasing physical activity (Bourne *et al,* 2002), non-communicable diseases (NCD) such as obesity, stroke, hypertension and non-insulin-dependent diabetes mellitus (NIDDM), previously rare in this population (Walker, 1995), are now reaching high rates (Lambert *et al,* 2001; Vorster, 2002). The pattern of NCD-emergence in this population seems to differ from those in other population groups. For example, coronary heart disease is still very rare, while the incidence of stroke is already higher than in the white population (Vorster, 2002). Mortality rates from NIDDM and hypertension also affect a larger proportion of the black than the white population (Punyadeera *et al,* 2001). Furthermore, one-third to one half of the black women is obese, up to double the prevalence in white women (Bourne *et al,* 2002; Vorster, 2002). Vorster (2002) suggested that a different set of NCD risk factors may be interacting in this population.

The metabolic syndrome, defined as a clustering of cardiovascular disease (CVD) risk factors, has recently been identified in this population (Kruger *et al,* 2004). The components of the syndrome include increased adiposity, especially visceral adiposity, high blood pressure, dyslipidaemia, abnormal glucose homeostasis (glucose intolerance, insulin resistance or diabetes), a

prothrombotic and a proinflammatory state (Grundy *et al,* 2004). Ethnic differences occur in the presentation of the syndrome (Pereira *et al,* 2002; Ford *et al,* 2003). Therefore, there is a need to describe the metabolic syndrome in black South Africans, especially due to the proposed unique set of risk factors.

The prevalence of the metabolic syndrome in Westernised societies is approximately 25%. Black people have a higher prevalence than whites. NHANES III indicated a prevalence of 21.6% in African-Americans and a 57% higher prevalence in African-American women than in men (Ford *et al,* 2002). In South Africa, Kruger *et al.* (2004) described the metabolic syndrome in 17% of black women and 13% in black men. The syndrome is associated with increased risk for CVD and NIDDM (Grundy *et al,* 2004).

The association of inflammation with obesity, CVD and NIDDM has recently been extensively documented (Tracy, 2003). Inflammation is also considered to be part of the metabolic syndrome. C-reactive protein (CRP), a sensitive marker of inflammation, was the strongest independent predictor for the development of NIDDM, myocardial infarction and stroke in prospective studies (Haffner, 2003; Ridker, 2003).

The prevalence of NIDDM and hypertension in black South African women is higher than in white women (Punyadeera *et al,* 2001), as also observed in African-American women (Campbell *et al,* 2002). Insulin resistance, the underlying determinant of NIDDM (Young-Hyman *et al,* 2001), was associated with the acute-phase proteins, fibrinogen, plasminogen activator inhibitor-1 (PAI-1) and CRP in other populations (Festa *et al,* 2000; Haffner, 2003). In previous studies in South African black women, the development of insulin resistance seemed to follow a different pattern from those observed in Caucasians (Kruger, 2000). Therefore, the aim of this study was to investigate whether the acute-phase proteins CRP, fibrinogen and PAI-1, are associated with insulin resistance and the metabolic syndrome in a group of urban black South African women.

Methods

Subjects, exclusion criteria, study design and ethics

The POWIRS study (Profiles of Women with the Insulin Resistance Syndrome) had a case-control design with selected volunteers. The subjects were 100 urbanised black women of the North-West Province, South Africa. Ages ranged from 19 to 55 years and body mass indices (BMI) from 19 to 50 kg/m^2. Exclusion criteria were pregnancy, lactation, being on medication, being HIV positive, signs of acute infection or temperatures above 37 °C. Women with an education level lower than grade 7 were excluded because responses to psychological questionnaires (results not reported here) required a certain level of education.

The study was approved by the Ethics Comittee of the North-West University (Ethics no 03M03). All subjects signed an informed consent form at recruitment. As part of the ethics protocol, subjects immediately received feedback about their individual health status. The employer of the subjects was informed about trends observed.

Order of experimental procedures

The experimental procedure was performed at the Metabolic Unit of the North-West University. Women arrived in groups of ten at the Metabolic Unit in the late afternoon, when the procedures started. After consent forms were signed, anthropometric measurements were taken, with the exception of weight and height. All subjects received the same supper and fasted from 22:00, drinking only tap water. The women spent the night at the Metabolic Unit.

The next morning at 06:00, weight and height were measured. A registered nurse took the women's blood pressures, examined them for clinical signs of malnutrition and took their body temperatures. Fasting blood samples were taken, after which a glucose solution was taken in order to perform an oral glucose-tolerance test (OGTT), followed by repeated blood sampling. The women stayed in bed untill the OGTT was finished.

During and after the OGTT, questionnaires were completed to obtain demographic, dietary intake and physical activity data, and psychological responses. Trained Tswana interviewers assisted in completion of the questionnaires. All questionnaires were validated for this population group (Kruger, 2000; MacIntyre *et al,* 2001[a]; MacIntyre *et al,* 2001[b]; Venter *et al,* 2000). After completion of all procedures, the subjects received lunch, could go home and their travelling expenses were covered.

Anthropometric measurements

Weight, height, waist circumference and abdominal saggital diameter, were measured by trained anthropometrists. Subjects were weighed without shoes in light night wear to the nearest 0.1 kg on a portable electronic scale (Precision Health® scale, A & D Company, Japan). Height was measured to the nearest 0.5 cm with a stadiometer (Invicta, IP 1465, UK) in the standing position, without shoes and with head in the Frankfort plane position.

Waist circumference (WC) and abdominal saggital diameter were measured to the nearest 0.5 cm using a flexible, but inelastic 7 mm wide steel tape (Holtain®). Waist circumference was measured at the midpoint between the lower rib margin and iliac crest. Abdominal saggital diameter was measured as the largest distance between the roundest part of the abdomen up to the corresponding point on the back, in a horizontal level, with the subject standing.

Blood pressure and clinical examination

A registered nurse measured the blood pressures with a sphygmomanometer (Tycos®) in the supine position. Subjects were examined for clinical signs of malnutrition by assessing their nails, eyes, hair, skin and thyroid. Their oral temperatures were taken using a clinical thermometer.

Blood sample collection

A registered nurse took the blood samples from the *vena cephalica,* using a sterile butterfly (21G) infusion set and syringes (Johnson & Johnson, 21G, 19 mm). A full blood count was performed immediately after the samples were

taken. After the blood was allowed to clot in glass tubes, serum was prepared by centrifugation of the blood at 3000 rpm for 15 minutes. The serum samples were divided into 1 ml aliquots and frozen at – 84 °C. Citrated blood samples (0.5 ml 0.1 mol/L citrate, pH 4.5 – 4.8, plus 4.5 ml venous blood) were prepared by centrifugation at 3000 rpm for 10 minutes to separate the platelet-free plasma. The citrated plasma was divided into 1 ml aliquots and frozen at –84 °C.

Oral glucose tolerance test (OGTT)

After the fasting blood sample was taken, the subjects took a glucose solution containing 75 g glucose (Medicolab® glucose power, Amalgan) to perform a glucose-tolerance test (OGTT). The solution was taken within 15 minutes. Blood samples were taken at 30, 60, 90 and 120 minutes after the glucose load. The butterfly system was kept in the vein during the 2 hours of the OGTT. The system was kept viable with a heparin solution (2.5 units heparin, 5000 m/ml in 5 ml sterile saline).

Biochemical analyses

A full blood count was performed, using the Beckman Coulter (Inc.) counter to obtain the red blood cell count, white blood cell count, haemoglobin, and haematocrit. The following serum metabolites were measured using the Vitros Chemistry System® (Johnson & Johnson Co, USA) with a colorimetric method: total protein, albumin, gamma glutamyl-transferase (GGT), uric acid, triglycerides, total cholesterol and HDL cholesterol. LDL cholesterol was calculated using the Friedewald equation (Friedewald *et al,* 1972). Plasma glucose was measured using the hexokinase method from the Chemical Pathology Laboratory, University of Pretoria. Plasma fibrinogen was measured using the Clauss method (Instrumentation Laboratory, Italy) with ACL 200 Automated Coagulation Laboratory. Plasma PAI-1 was measured with the Chromogenic assay kit (Spectrolyse®/pL PAI, biopool cat 101 201, Sweden) and Labsystems Multiskan Ascent (USA). Plasma insulin was measured with the enzyme immuno assay (Biosource Europe SA, Belgium). Plasma free fatty acids were analysed with a half micro-optimised enzymatic colorimetric assay

(Roche®). Serum C-reactive protein (CRP) was measured with a high-sensitivity C-reactive protein kit (Immage® Immunochemistry Systems and Beckman Coulter, Inc.).

Calculation of indices

The HOMA-insulin-resistance index was calculated as (fasting insulin (uU/ml) x fasting glucose (mmol/L)) / 22.5 (Matthews *et al,* 1985). The body mass index (BMI) were calculated by the formula body weight in kg / height meter^2 (Must *et al,* 1999).

Demographic information

A demographic questionnaire, developed and tested for the Tswana population (Kruger, 2000), was used to collect data such as age, education, income, smoking and drinking habits. Trained Tswana field workers assisted in the completion of the questionnaires.

Habitual dietary intakes

Habitual dietary intakes were measured by a quantitative food frequency questionnaire, developed and validated for Tswana subjects (MacIntyre *et al,* 2001[a]; MacIntyre *et al,* 2001[b]). Books with photographs of typical foods and portion sizes commonly used by this population (Venter *et al,* 2000) assisted trained Tswana field workers in collecting the data. The food data were coded and analysed by the Food Finder® computer programme of the Medical Research Council (MRC), based on the South African Food Composition Tables (Langenhoven, 1991), to obtain habitual dietary intakes. Dietary under-reporters were identified by energy intake / basal metabolic rate < 1.2 (Goldberg *et al,* 1991).

Diagnosis of the metabolic syndrome

The metabolic syndrome was diagnosed using the National Cholesterol Education Program Adult Treatment Panel III (ATPIII) criteria (Grundy *et al,* 2004): Blood pressure ≥ 130/85 mm Hg, waist circumference > 88 cm, HDL cholesterol < 1.29 mmol/L, triglycerides ≥ 1.69 mmol/L and fasting glucose ≥ 6.1

mmol/L. Three or more of these criteria had to be present in an individual for diagnosis of the metabolic syndrome.

Statistical analyses

Statistical analyses were performed using the Statistica 6.0 package. The subjects were divided into two groups, i.e. women with and without the metabolic syndrome, and means and 95 % confidence intervals were calculated. Since age differered between the two groups of women, analyses were adjusted for age.

Since the sample consisted of volunteers and was therefore not a truly random sample, effect sizes were calculated to assess practical significant differences between the means of variables (Ellis & Steyn, 2003). A medium effect size ($d \geq 0.5$) indicated possible practical significance and a large effect size ($d \geq 0.8$) practical significance. Practical significance is a measure that indicates a difference large enough to have an effect in practice. The difference indicated by the effect size is independent of units and sample size and also relate to the spread of the data. A natural way to comment on pratical significance is to divide the difference between the two means under consideration by the standard deviation (Ellis & Steyn, 2003). Odds ratios were calculated to determine the relative risks and best independent predictors (logistic regression models) of increased p-fibrinogen, s-CRP and p-PAI-1 respectively.

The women were also divided into HOMA-insulin-resistance quartiles and the same statistical analyses as mentioned above were performed, also adjusting for age. The first quartiles ranged from HOMA-insulin-resistance values 0.8 - 2.1 (24 women), the second quartile 2.2 – 2.6 (28 women). The third quartile included HOMA values of 2.7 - 3.6 (23 women) and the fourth quartile 3.8 – 10.2 (25 women). The fourth quartile was considered as the insulin-resistant quartile, with HOMA-insulin-resistance levels consistent with those described by Ascaso *et al*, (2003).

Results

Comparison of variables in women with and without the metabolic syndrome (Table 1)

The women with the metabolic syndrome were older than those without the syndrome (35.6 years *versus* 30.4 years). They were also heavier (BMI = 34.9 *versus* 26.6 kg/m^2) and had a larger waist circumference (97.8 *versus* 78.6 cm). Therefore, they were also more abdominally obese. Women with the metabolic syndrome were characterised by practical significantly higher levels of systolic and diastolic blood pressure, s-triglycerides, s-fasting glucose, s-2h-post-load glucose, s-fasting insulin, s-2h-post-load insulin, the HOMA-insulin-resistance index and lower s-HDL cholesterol. Mean serum-GGT, s-uric acid, s-CRP and 2h-post-load free fatty acids were higher, indicative of practical significance in the women with compared to those without the metabolic syndrome.

Serum-CRP were higher, indicative of practical significance in women with the metabolic syndrome than in those without the syndrome (6.5 *versus* 3.4 mg/L). Fibrinogen and PAI-1 levels were not practical significantly different between women with and without the metabolic syndrome.

Dietary intakes

Dietary intakes could not be compared between the women with and without the metabolic syndrome, since only two subjects remained in the metabolic syndrome group after under-reporters had been excluded.

Comparison of metabolic syndrome variables and acute-phase proteins by HOMA-insulin-resistance quartiles (Table 2)

The following metabolic syndrome variables had practical significantly higher levels in the highest quartile compared to the lowest HOMA-insulin-resistance quartile: BMI, waist circumference, diastolic blood pressure, s-uric acid, s-fasting glucose, s-2h-post load glucose, s-fasting insulin, s-2h-post-load insulin, and s-2h-post-load free fatty acids. Systolic blood pressure, s-triglycerides, s-albumin, s-GGT, haemoglobin, haematocrit and 2h-post-load free fatty acids had higher

levels indicative of practical significance in the highest compared to the lowest HOMA-insulin-resistance quartile. Serum-HDL cholesterol showed lower levels indicative of practical significance in the highest compared to the lowest insulin-resistant quartile.

Levels of the acute-phase protein s-CRP and possibly p-PAI-1 and white blood cells were higher indicative of practical significance, in the highest compared to the lowest HOMA-insulin-resistance quartile. Plasma-fibrinogen did not show a difference indicating practical significance between the highest and lowest HOMA-insulin-resistance quartiles.

Table 1 ***Means of metabolic syndrome variables for women with and without the metabolic syndrome (age-adjusted)***

Metabolic syndrome variable	***Without metabolic syndrome N = 16*** *Mean (95% CI)*	***With metabolic syndrome N = 84*** *Mean (95% CI)*
Age (years)	30.4 ** (28.6-32.3)	35.6 ** (31.2-40.0)
BMI (kg / m^2)	26.6 ** (25.4-27.9)	34.9 ** (31.8-38.1)
Waist circumference (cm)	78.6 ** (76.2-81.0)	97.8 ** (91.5-104.1)
Systolic blood pressure (mm Hg)	126.6 ** (123.2-129.9)	144.6 ** (136.1-153.2)
Diastolic blood pressure (mm Hg)	76.3 ** (74.0-78.6)	91.7 ** (85.1-97.0)
s-Total cholesterol (mmol/L)	4.3 * (4.1-4.6)	3.9 * (3.4-4.4)
s-HDL cholesterol (mmol/L)	1.3 ** (1.2-1.4)	1.0 ** (0.8-1.1)
s-LDL cholesterol (mmol/L)	2.8 (2.7-3.0)	2.7 (2.3-3.2)
s-Triglycerides (mmol/L)	0.6 ** (0.6-0.7)	0.9 ** (0.7-1.1)
s-Uric acid (umol/L)	286.8 * (270.6-303.1)	323.7 * (281.9-365.4)
s-Total protein (g/L)	87.8 (85.6-90.1)	89.1 (83.4-94.9)
s-Gamma-glutamyl-transferase (GGT) (U/L)	32.2 * (27.6-36.7)	44.6 * (33.0-56.3)
p-Fibrinogen (g/L)	3.9 (3.6-4.1)	3.6 (2.9-4.2)
p-Plasminogen activator inhibitor-1 (PAI-1) (U/ml)	5.6 (4.6-6.6)	6.1 (3.4-8.7)
s-Albumin (g/L)	41.4 (40.5-42.3)	41.2 (38.9-43.4)
s-C-reactive protein (mg/L)	3.4 * (2.4-4.4)	6.5 * (3.9-9.0)
Haemoglobin (g/dL)	12.3 (12.0-12.6)	12.2 (11.4-13.0)
Haematocrit (%)	35.2 (33.9-36.4)	35.4 (32.2-38.5)
Red blood cells (10e6u/L)	4.0 (3.9-4.1)	4.1 (3.8-4.4)
s-Fasting glucose (mmol/L)	4.9 ** (4.8-5.0)	5.4 ** (5.1-5.6)
s-2h-Post-load glucose (mmol/L)	6.1 ** (5.8-6.5)	7.8 ** (6.9-8.6)
s-Fasting free fatty acids (FFA) (mmol/L)	0.51 (0.45-0.57)	0.57 (0.42-0.72)
s-2h-Post-load free fatty acids (mmol/L)	0.03 * (0.02-0.03)	0.04 * (0.03-0.05)
s-Fasting insulin (pmol/L)	84.8 ** (76.2-93.4)	139.8 ** (117.7-161.9)
s-2h-Post-load insulin (pmol/L)	386.9 ** (312.9-460.9)	691.2 ** (500.7-881.6)
HOMA-insulin-resistance index	2.7 ** (2.4-3.0)	4.7 ** (4.0-5.5)
White blood cells (10e3u/L)	5.3 (4.9-5.7)	5.9 (4.8-6.9)

** Means may differ practical significantly (d ≥ 0.5)*
*** Means differ practical significantly (d ≥ 0.8)*

Table 2 ***Means of metabolic syndrome variables divided into HOMA-insulin-resistance quartiles***

	HOMA-insulin-resistance quartiles			
	Quartile 1 *0.8-2.1* *N = 24*	*Quartile 2* *2.2-2.6* *N = 28*	*Quartile 3* *2.7-3.7* *N = 23*	*Quartile 4* *3.8-10.2* *N = 25*
Metabolic syndrome variable	*Mean (95% CI)*	*Mean (95% CI)*	*Mean (95% CI)*	*Mean (95% CI)*
Age (years)	31.5 (28.1-34.9)	28.1 (24.6-31.6)	30.5 (27.1-33.9)	34.5 (31.1-37.9)
Body mass index (BMI) (kg/m^2)	24.2 a*cd (21.8-26.5)	26.9 b*c (24.7-29.2)	28.1 de (25.8-30.5)	32.1a*b*e (29.6-34.5)
Waist circumference (cm)	72.3 a*b*e (67.9-76.8)	78.3 c*ef (74.0-82.7)	83.0 a*d*f (78.5-87.5)	91.8 a*b*c*d* (87.1-96.5)
Systolic blood pressure (mm Hg)	123.6 ab (116.9-130.3)	129.7 (123.2-136.1)	131.8 b (125.1-138.5)	130.9 a (123.9-137.8)
Diastolic blood pressure (mm Hg)	71.6 a*b*c (67.1-76.1)	78.5 cd (74.1-82.8)	79.7a* (75.2-84.2)	83.4 b*d (78.7-88.1)
s-Total cholesterol (mmol/L)	4.5 ab (4.1-4.9)	4.0 a (3.7-4.4)	4.3 (3.9-4.7)	4.0 b (3.7-4.4)
s-HDL cholesterol (mmol/L)	1.4 abc (1.2-1.5)	1.2 b (1.1-1.4)	1.2 c (1.1-1.4)	1.2 a (1.0-1.3)
s-LDL cholesterol (mmol/L)	3.0 (2.7-3.4)	2.7 (2.3-3.0)	2.9 (2.6-3.3)	2.7 (2.3-3.1)
s-Triglycerides (mmol/L)	0.6 a (0.5-0.8)	0.6 b (0.4-0.7)	0.7 (0.6-0.9)	0.8 ab (0.6-0.9)
s-Uric acid (umol/L)	265.8 a* (236.4-295.3)	291.6 b (263.0-320.1)	281.3 c (251.8-299.6)	330.3 a*bc (300.0-361.0)
s-Total protein (g/L)	84.8 (80.7-89.0)	90.2 (86.1-94.2)	88.9 (84.7-93.0)	87.9 (83.6-92.2)
s-GGT (U/L)	25.7 a*c* (17.6-33.9)	29.3 b* (21.4-37.2)	44.6 a*b* (36.4-52.8)	36.2 c* (27.7-44.7)
p-Fibrinogen (g/L)	3.7 (3.3-4.2)	3.7 (3.2-4.1)	4.1 (3.6-4.5)	3.9 (3.4-4.3)
p-PAI-1 (U/ml)	4.8 b* (2.9-6.7)	4.3 a (2.5-6.1)	6.1 (4.3-8.0)	7.6 ab* (5.6-9.5)
s-Albumin (g/L)	39.6 (37.8-40.9)	42.3 (40.8-43.9)	42.0 (40.5-43.6)	41.7 (40.0-43.3)
s-C-reactive protein (mg/L)	2.2 a*b (0.3-4.0)	3.3 c (1.5-5.0)	4.2 b (2.4-6.1)	5.6 a*c (3.7-7.5)
Haemoglobin (g/dL)	11.8 a (11.2-12.4)	12.2 (11.7-12.9)	12.4 (11.9-13.0)	12.6 a (12.0-13.2)
Haematocrit (%)	33.8 a (31.6-36.1)	34.6 (32.4-36.8)	36.0 (33.8-38.3)	36.4 a (34.1-38.7)
Red blood cells (10e6u/L)	3.9 a (3.7-4.1)	3.9 b (3.7-4.2)	4.2 ab (4.0-4.4)	4.1 (3.9-4.3)
s-Fasting glucose (mmol/L)	4.7 a*d (4.5-4.9)	4.8 b*e (4.6-5.0)	5.0 c*d*e (4.9-5.2)	5.4 a*b*c* (5.2-5.6)
s-2h-Post-load glucose (mmol/L)	5.5 a*b*d (4.9-6.1)	6.2 cd (5.6-6.8)	6.6 a* (6.0-7.2)	7.1 b*c (6.5-7.8)
s-Fasting FFA (mmol/L)	0.53 (0.42-0.64)	0.52 (0.41-0.63)	0.49 (0.38-0.60)	0.53 (0.41-0.64)
s-2h-Post-load FFA (mmol/L)	0.02 a* (0.02-0.03)	0.02 b (0.02-0.03)	0.02 c (0.02-0.03)	0.04 a*bc (0.03-0.04)
s-Fasting insulin (pmol/L)	52.9 a*b* (42.4-63.5)	77.9 c*e (67.6-88.1)	94.2 a*d*e (83.6-104.8)	147.6 a*b*c*d* (136.6-158.7)
s-2h-Post-load insulin (pmol/L)	248. 3 a*c (114.1-382.5)	371.9 b* (241.8-502.1)	479.0 cd (344.7-613.3)	626.5 a*b*d (486.7-766.4)
HOMA-insulin-resistance index	1.6 a*b*e (1.3-1.9)	2.4 c*ef (2.1-2.7)	3.0 a*d*f (2.7-3.3)	5.0 b*c*d* (4.7-5.4)
White blood cells (10e3u/ml)	4.7 ab (3.9-5.4)	5.3 (4.6-6.0)	5.5 b (4.8-6.3)	5.9 a (5.2-6.7)

Means with the same symbol may differ practical significantly (d ≥ 0.5)
*Means with the same symbol marked with * differ practical significantly (d ≥ 0.8)*

Dietary intakes

Dietary intakes correlated with insulin resistance, but the relationship was dependent on obesity (results not shown). In the normal-weight group, no relationship was found between the HOMA-insulin-resistance index levels and dietary intakes.

Association of acute-phase proteins with metabolic syndrome variables (Table 3)

Serum-CRP was positively associated with all metabolic syndrome variables, showing the strongest correlation with BMI ($r = 0.60$, $p < 0.0001$), waist circumference ($r = 0.54$, $p < 0.0001$) and abdominal saggital diameter ($r = 0.55$, p

< 0.0001). Serum-CRP also correlated positively with s-GGT, p-fibrinogen, white blood cells, s-2h-post-load glucose, s-fasting insulin and the HOMA-insulin-resistance index.

Plasma-fibrinogen was positively associated with BMI, waist circumference, s-GGT, s-fasting glucose, s-2h-post-load glucose and s-CRP. Plasma-PAI-1 showed a positive relationship with BMI, waist circumference, s-fasting insulin and the HOMA-insulin-resistance index. All correlation coefficients were significant at $p < 0.05$. The best correlates of s-CRP, p-fibrinogen, p-PAI-1, s-fasting insulin and the HOMA-insulin-resistance index were obesity measures (BMI, waist circumference and abdominal saggital diameter).

Table 3 ***Partial Spearman correlation coefficients of metabolic syndrome variables with acute-phase proteins, insulin and HOMA-insulin-resistance index***

	CRP	***Fibrinogen***	***PAI-1***	***Fasting insulin***	***HOMA-insulin-resistance index***
	r	*r*	*r*	*r*	*r*
Metabolic syndrome variable					
Body mass index	0.60*	0.29	0.31	0.46*	0.52*
Waist circumference	0.54*	0.23	0.31	0.56*	0.62*
Abdominal saggital diameter	0.55*	0.26	0.26	0.47*	0.53*
Systolic blood pressure	0.24	*NS*	*NS*	*NS*	*NS*
Diastolic blood pressure	0.30	*NS*	*NS*	*NS*	0.26
s-HDL cholesterol	*NS*	*NS*	*NS*	-0.22	-0.25
s-Triglycerides	*NS*	*NS*	*NS*	0.24	0.30
s-Uric acid	*NS*	*NS*	*NS*	0.36*	0.32
s-GGT	0.35	0.25	*NS*	*NS*	*NS*
p-Fibrinogen	0.36		*NS*	*NS*	*NS*
p-PAI-1	*NS*	*NS*		0.21	0.22
White blood cells	0.28	*NS*	*NS*	*NS*	
s-Fasting glucose	*NS*	0.24	*NS*	*NS*	0.41*
s-Fasting insulin	0.22	*NS*	0.21		0.97*
s-2h-post-load glucose	0.28	0.21	*NS*	*NS*	0.26
s-2h-post-load insulin	0.28	*NS*	*NS*	*NS*	0.36
s-2h-post-load free FFA	*NS*	*NS*	*NS*	*NS*	0.25
HOMA-insulin-resistance index	0.25	*NS*	0.22	0.97*	
s-C-reactive protein		0.36	*NS*	0.22	0.25

Analysis adjusted for age
*$p < 0.05$: all correlations, except those marked with **
** $p < 0.0001$)*
NS = non-significant

Predictors of increased acute-phase protein levels (Table 4)

Serum-CRP

The highest relative risk for s-CRP ≥ 3 mg/L was contributed by BMI ≥ 30 kg/m^2 (odds ratio = 6.4) and waist circumference > 88 cm (odds ratio = 6.1). In a

logistic regression model with waist circumference, s-triglycerides, s-GGT, BMI, systolic and diastolic blood pressure, p-fibrinogen, p-PAI-1, the HOMA-insulin-resistance index and age as dependent variables, BMI was the best independent predictor of s-CRP (odds ratio = 2.1, p = 0.03, 95% CI 1.1-3.9).

Plasma-fibrinogen

Serum-GGT ≥ 38 U/ml showed the highest relative risk for a p-fibrinogen level of ≥ 3 g/L (odds ratio = 3.7), followed by s-CRP ≥ 3 mg/L (odds ratio = 2.6). Serum-GGT was also the best independent correlate of p-fibrinogen in the logistic regression model (odds ratio = 1.1, p = 0.02, 95% CI 1.1-1.2).

Serum-GGT correlated positively with the following metabolic syndrome variables: body mass index ($r = 0.31$, $p < 0.05$), waist circumference ($r = 0.35$, $p < 0.05$), abdominal saggital diameter ($r = 0.33$, $p < 0.05$), systolic blood pressure ($r = 0.25$, $p < 0.05$), diastolic blood pressure ($r = 0.30$, $p < 0.05$), s-total cholesterol ($r = 0.21$, $p < 0.05$), s-LDL cholesterol ($r = 0.22$, $p < 0.05$), s-triglycerides ($r = 0.29$, $p < 0.05$), s-uric acid ($r = 0.21$, $p < 0.05$), p-fibrinogen ($r = 0.25$, $p < 0.05$) and s-fasting glucose ($r = 0.25$, $p < 0.05$).

Plasma-PAI-1

BMI ≥ 30 kg/m^2 contributed the highest relative risk for high p-PAI-1 levels (> 7 U/ml) (Ranby *et al,* 1990) (odds ratio = 2.3), followed by s-triglycerides ≥ 1.69 mmol/L (odds ratio = 2.0). In the logistic regression model, no independent predictors of p-PAI-1 were found.

Predictors of the metabolic syndrome (Table 4)

A waist circumference > 88 cm contributed the highest relative risk for development of the metabolic syndrome (odds ratio = 71.0), followed by s-fasting glucose ≥ 6.1 mmol/L (odds ratio = 42.0) and systolic blood pressure ≥ 130 mm Hg (odds ratio = 30.0). Both the HOMA-insulin-resistance index and BMI had odds ratios of 16.5, and s-CRP an odds ratio of 10.9, and therefore also contributed high relative risks. Plasma-fibrinogen and p-PAI-1 contributed lower

and insignificant relative risks for development of the metabolic syndrome (odds ratios = 0.41 and 1.0 respectively).

In a logistic regression model with BMI, waist circumference, systolic and diastolic blood pressure, s-triglycerides, s-HDL cholesterol, s-GGT, s-CRP, the HOMA-insulin-resistance index, s-fasting insulin, s-fasting glucose and age as dependent variables, diastolic blood pressure was the best independent predictor of development of the metabolic syndrome (odds ratio = 1.3, p = 0.006, 95% CI 1.1-1.5), followed by s-GGT (odds ratio = 1.1, p = 0.04, 95% CI 1.0-1.1). Serum-CRP, p-fibrinogen and p-PAI-1 were not predictors of the metabolic syndrome in the regression model.

Table 4 ***Relative risks for development of the metabolic syndrome and increased acute-phase protein levels***
Odds ratios with metabolic syndrome, s-CRP, p-fibrinogen and p-PAI-1 as dependent variables

	Metabolic syndrome		***s-CRP ≥ 3 mg/L***		***p-Fibrinogen ≥ 3 g/L***		***p-PAI-1 > 7 U/ml***	
Independent variable	***Odds ratio***	***95% CI***	***Odds ratio***	***95% CI***	***Odds ratio***	***95% CI***	***Odds ratio***	***95% CI***
Waist circumference > 88 cm	71.0	8.6-584.6	6.1	2.3-16.5	0.8	0.2-2.4	1.7	0.7-4.2
BMI ≥ 30 kg/m^2	16.5	3.5-78.8	6.4	2.6-15.8	0.9	0.3-2.7	2.3	1.0-5.1
s-Fasting glucose ≥ 6.1 mmol/L	42.0	4.5-396.5						
Systolic blood pressure ≥ 130 mm Hg	30.0	3.8-240.6	4.1	1.8-9.7	2.2	0.6-7.4	1.2	0.5-2.8
Diastolic blood pressure ≥ 85 mm Hg	18.5	5.0-68.4	3.5	1.3-9.5	0.8	0.2-2.6	1.1	0.4-3.1
s-Triglycerides ≥ 1.69 mmol/L	6.0	0.4-101.6	0.4	0.4-0.6	0.9	0.8-0.9	2.0	0.1-32.5
s-HDL cholesterol < 1.29 mmol/L	12.4	1.6-98.9						
HOMA-insulin resistance index ≥ 3.8	16.5	4.5-60.4	4.0	1.5-10.9	1.3	0.3-5.0	1.8	0.7-4.6
s-C-reactive protein ≥ 3 mg/L	10.9	2.3-51.6			2.6	0.8-8.7	1.0	0.4-2.2
p-Fibrinogen ≥ 3 g/L	0.4	0.1-1.53	2.6	0.8-8.7			1.5	0.5-5.2
p-PAI-1 > 7 U/ml	1.0	0.3-3.1	1.0	0.4-2.2	1.5	0.5-5.2		
s-GGT ≥ 38 U/ml	1.8	0.8-4.3	2.8	1.2-6.8	3.7	0.8-17.4		

Discussion

Apart from higher levels of diagnostic metabolic syndrome variables as recommended by ATPIII, women with the metabolic syndrome were also older and had higher levels of s-GGT, s-uric acid, the HOMA-insulin-resistance index, s-fasting insulin and the acute-phase protein s-C-reactive protein (CRP).

Consistent with findings in other populations (Vega, 2001), women with the metabolic syndrome were generally obese (BMI ≥ 30 kg/m^2), with a mean body mass index of 35 kg/m^2. Other acute-phase proteins p-fibrinogen and p-plasminogen activator inhibitor-1 (PAI-1), were not associated with the metabolic syndrome in this study. White blood cells, also involved in the acute-phase response, were not associated with the metabolic syndrome. Women with higher levels of the HOMA-insulin-resistance index had higher BMIs and waist circumferences and higher levels of systolic and diastolic blood pressure, s-triglycerides, s-GGT, haemoglobin, haematocrit, white blood cells, s-uric acid, s-CRP, p-PAI-1, s-fasting glucose, s-2h-post-load glucose, s-fasting insulin, s-2h-post-load insulin, and s-2h-post load free fatty acids. Lower levels of s-HDL cholesterol occurred with increasing levels of the HOMA-insulin-resistance index.

Chronic subclinical inflammation has been recognised as part of the metabolic syndrome (Grundy *et al,* 2004). In particular, high CRP levels were found to be common in individuals with the syndrome (Abrams, 2003). This study showed that CRP may be associated with the metabolic syndrome in a group of black South African women. Serum-CRP levels in the women with the metabolic syndrome were had higher levels indicative of practical significance than those without the syndrome. The high inter-individual variation of CRP (Pearson *et al,* 2003) may explain why s-CRP levels did not reach practically significant differences between women with compared to those without the metabolic syndrome. The Women's Health Study (27 939 women) showed that CRP adds important prognostic information to the risk of coronary events at all levels of severity of the metabolic syndrome (Ridker *et al,* 2003). The Women's Health Study indicated that CRP levels ≥ 3 mg/L were associated with higher incidence of coronary events in women with, as well as without the metabolic syndrome (Ridker *et al,* 2003). A CRP level between 1 and 3 mg/L was associated with moderate CVD risk and levels higher than 3 mg/L with high risk. In this study, the women *without* the metabolic syndrome had an average CRP level of 3.4 mg/L and therefore high CVD risk. In prospective studies (Haffner, 2003; Ridker,

2003) CRP has been an independent predictor of NIDDM and CVD. It showed a stronger association with cardiovascular events than LDL cholesterol (Ridker, 2003). The finding that acute myocardial infarction occured in 63% of women with normal lipid profiles (O'Neal *et al,* 1997) suggested a need to look beyond classical risk factors to properly manage CVD (Després, 2003). Therefore, our results may indicate that it could be sensible to focus more on CRP than only the presence of the metabolic syndrome and classic CVD risk factors in health-risk assessment in black South African women.

Insulin resistance, also a CVD and NIDDM predictor (Lebovitz, 2001; Perseghin *et al,* 2003; Erkelens, 2001), possibly due to its involvement in inflammation (Dandon & Aljada, 2002; Hanley *et al,* 2002), was associated with the metabolic syndrome in 75% of the women, while 14% of the women had insulin resistance, but not the metabolic syndrome, according to the ATPIII criteria. Of these 14 women, 10 (71%) would have the metabolic syndrome if insulin resistance were added as a diagnostic criterium for the metabolic syndrome. Accordingly, when the HOMA-insulin-resistance index was included in the definition of the metabolic syndrome, 26% of the women in this study would have the syndrome, whereas the ATPIII definition diagnosed only 16%. Since NIDDM is common in black South African women (Punyadeera *et al,* 2001), and are preceded by insulin resistance (Erkelens, 2001), it seems sensible to include insulin resistance in the definition of the metabolic syndrome in this population and rather call it the insulin-resistance syndrome.

Although fibrinogen was not associated with the metabolic syndrome or insulin resistance, high CVD-risk levels (≥3.0 g/L) (James *et al,* 2000) occurred in the women whether or not they had the metabolic syndrome or insulin resistance. Fibrinogen was positively associated with insulin resistance in other studies (Yudkin, 2003; Raynaud *et al,* 2000) and therefore our result was unexpected. However, PAI-1 was positively associated with insulin resistance in this study, consistent with previous findings in other populations (Haffner, 2003). CRP was previously found to increase expression of PAI-1 by endothelial cells (Devaraj *et*

al, 2003), but in this study no correlation was found between PAI-1 and CRP. In the Women's Health Study, PAI-1 and fibrinogen predicted development of NIDDM (Haffner, 2003).

The lack of association with p-fibrinogen with the metabolic syndrome and insulin resistance could be explained by the occurrence of high p-fibrinogen levels in all body-weight groups. It seemed that whether or not these women had the metabolic syndrome, they may have considerable CVD risk, having both high-risk CRP and fibrinogen levels. It is not known what causes the high fibrinogen levels in black South Africans. The high fibrinogen levels (≥ 3 g/L) correlated best with the liver enzyme GGT, which was positively associated with a large number of metabolic syndrome variables (systolic and diastolic blood pressure, BMI, waist circumference, abdominal saggital diameter, total cholesterol, LDL cholesterol, triglycerides, uric acid, fibrinogen and fasting glucose), consistent with other studies (Lee *et al,* 2001). In other studies, GGT was found to independently predict future development of NIDDM and CVD (Lee *et al,* 2003).

It is hypothesised that adipose tissue, especially visceral adipose tissue, secrete cytokines such as IL-6 and TNF-alpha, shown to interfere with insulin receptor signalling and leading to insulin resistance (Pannacciuli *et al,* 2001). Further, these cytokines, mainly IL-6, stimulate the production of acute-phase proteins, such as fibrinogen and CRP (Yudkin, 2003). It has been known for some time that in type 1 diabetes, the beta cells of the pancreas are destroyed by inflammation (Hohmeier *et al,* 2003). The use of salicylates for the treatment of diabetes was first reported a century ago and the glucose-lowering action of aspirin was confirmed in the 1950s. More recently, it was shown that high-dose salicylates improved insulin sensitivity (Marette, 2003). This clearly indicates a direct association between inflammation and diabetes.

Blood pressure was positively associated with CRP in this study, but it did not show correlations with fibrinogen or PAI-1. Hypertension was also associated with inflammation in larger studies, e.g. the Women's Health Study and it has

been proposed that hypertension is an inflammatory condition. The Womens' Health Study (20 525 women) showed that hypertension is more likely to develop in individuals with high CRP levels, possibly through the effect of inflammation on the arterial wall itself (Sowers *et al,* 2004; Sesso *et al,* 2003).

High levels of CRP could promote arterial inflammation by interacting directly with endothelial cells or other cells in the arterial wall. In turn, this could promote vasoconstriction, leukocyte adherence, platelet activation, LDL oxidation and thrombosis. High CRP levels may upregulate angiotensin receptors and enhance PAI-1 expression by endothelial cells. Both of these changes could increase blood pressure and promote atherosclerosis (Grundy, 2003)

NIDDM and hypertension are common in black South African women and have reached higher rates than in white women (Punyadeera *et al,* 2001). The same situation is found in the USA (Campbell *et al,* 2002). The results of this study, showed high levels of acute-phase proteins in these women, which may be a possible underlying determinant of the high levels of insulin resistance, NIDDM hypertension and stroke rates in this population. Insulin resistance by itself may add to increased CRP levels in two ways. Firstly, insulin itself is an anti-inflammatory hormone, regulating the production of acute-phase proteins. During insulin resistance, it may have the opposite effect and increases production of acute-phase proteins such as CRP and fibrinogen (Haffner, 2003; Grundy, 2003). Secondly, high concentrations of insulin, which generally accompany insulin resistance, cause injury of the arterial wall (Okouch *et al,* 2002; Steinberg & Baron, 2002), which may elicit an inflammatory response (increased CRP) as a protective mechanism (Tracy, 2003).

The rarity of coronary heart disease in black South Africans has been the subject of much speculation. Low levels of homocysteine and favourable lipid profiles have been suggested as protective factors (Vorster, 2002). However, Mollentze *et al.* (1995) speculated that it might just be a matter of time before the disease would emerge, due to an incubation period of about 30-40 years. In the USA, all

forms of CVD occur at higher rates in blacks than in whites (Cooper, 1999; Gillum, 1996; Geronimus *et al,* 1996), where it had also been rare previously (Walker, 1999). The results of our study may present evidence to support the speculation of Mollentze *et al.* (1995), due to the well-researched role of inflammation in atherosclerosis (Tracy, 2003).

Conclusions

In a group of urban black South African women of the North-West Province, South Africa, CRP was positively associated with insulin resistance and may be associated with the metabolic syndrome. Fibrinogen was not associated with the metabolic syndrome or insulin resistance. PAI-1 was not associated with the metabolic syndrome, but it was positively associated with insulin resistance. CRP and fibrinogen reached high CVD-risk levels in women both with and without the metabolic syndrome. Inclusion of CRP in health-risk assessment or the metabolic syndrome definition may be sensible in this population.

Inflammation may be an important underlying determinant of the high levels of NIDDM, hypertension and stroke observed in this population. Therefore, inflammation may be an important NCD risk factor in black South African women. Weight loss through diet control and exercise was shown by Campbell and White (2002) to prevent approximately 58% of insulin-resistant patients from progressing to NIDDM. A weight loss of only 5-7% was necessary to show these changes. Since obesity seemed to be an important determinant of increased acute-phase proteins and insulin resistance, weight control and prevention of obesity would be essential in this population.

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CHAPTER 5

Weight-related attitudes, knowledge and behaviour of black South African women – the POWIRS study

Jonker E (M.Sc), Vorster HH (D.Sc), Kruger A (Ph.D)
School for Physiology, Nutrition, and Consumer Sciences, North-West University, Potchefstroom Campus, South Africa

SUMMARY

Introduction: The high obesity rates in black South African women (Bourne *et al,* 2002) and concern about the consequences thereof, have motivated an investigation of the underlying determinants of obesity.

Subjects and methods: The weight-related attitudes, knowledge and behaviour of 38 normal-weight, 25 overweight and 37 obese urban black South African women of the North-West Province, South Africa were compared. The women were selected as apparently healthy, non-pregnant volunteers.

Results: The women seemed conscious about weight control and were interested in losing weight. Nearly half of the women had a positive attitude towards thinness. Most overweight and obese women thought they should lose weight and have tried to do so at some time. The obese women had better knowledge about the relationship between obesity, nutrition and health than the normal-weight women. An alarmingly high percentage (75%) of all the women have gained weight since age 18 years, while only 12% decreased and 6% kept their weight constant. A low success rate with weight loss in the long term seemed common, especially in the overweight and obese women. Overeating incidents and emotion-induced eating were identified as barriers to successful weight control. ***Conclusions:*** Eating for other reasons than nutrition, especially emotion-induced eating may be a strong potential risk factor for the development of obesity and a factor in the maintenance thereof in black South African women. Weight control programmes should empower women to identify their personal

barriers and reasons for eating other than nutrition and develop appropriate coping skills and social support networks.

Key words: *obesity, black women, South Africa, weight-related behaviour, attitude, knowledge, emotion-induced eating*

INTRODUCTION

Obesity in black South African women has reached high rates (Bourne *et al,* 2002). The prevalence of obesity in these women is double than those in white women (Walker, 1998). There is concern about the high obesity rates, due to the known health consequences thereof, such as diabetes, hypertension and cardiovascular disease (Hodge & Zimmet, 1994; Grundy 1998).

Little is known about weight-related attitudes, knowledge and behaviour in black South African women and also whether there are differences in these factors between obese and non-obese women. The POWIRS study (Profiles of Women with the Insulin Resistance Syndrome) compared these factors in normal- weight, overweight and obese women to investigate the underlying mechanisms acting in obesity. Furthermore, the incidence of emotion-induced eating was assessed, which was shown to be associated with obesity in other populations (Striegel-Moore *et al,* 1999).

METHODS

Subjects, exclusion criteria, study design and ethics

The POWIRS study (Profiles of Women with the Insulin Resistance Syndrome) had a case-control design with selected volunteers. The subjects were 100 urbanised black women of the North-West Province, South Africa. Ages ranged from 19 to 55 years and body mass indices (BMI) from 19 to 50 kg/m^2. Exclusion criteria were pregnancy, lactation, being on medication, being HIV positive, signs of acute infection or temperatures above 37 °C. Women with an education level lower than grade 7 were excluded because responses to psychological questionnaires (results not reported here) required a certain level of education.

The subjects were divided into three BMI groups, according to the WHO body-weight classification (Must *et al,* 1999): a normal-weight group (BMI 19.0 - 24.9 kg/m^2; 38 subjects), an overweight group (BMI 25.0 - 29.9 kg/m^2; 25 subjects) and an obese group (BMI ≥ 30 kg/m^2; 37 subjects).

The study was approved by the Ethics Comittee of the North-West University (Ethics no 03M03). All subjects signed an informed consent form at recruitment.

Anthropometric measurements

Weight and height were measured by trained anthropometrists. Subjects were weighed without shoes in light night wear to the nearest 0.1 kg on a portable electronic scale (Precision Health® scale, A & D Company, Japan). Height was measured to the nearest 0.5 cm with a stadiometer (Invicta, IP 1465, UK) in the standing position, without shoes and with head in the Frankfort plane position.

Attitudes towards weight control and knowledge of the relationship between obesity, nutrition and health

Attitudes towards weight control and knowledge of the relationship between obesity, nutrition and health were assessed, using the attitude and knowledge scales of Kruger and Van Aardt (1998).

Emotion-induced eating and weight-related behaviour

Emotion-induced eating was assessed using the scale of Striegel-Moore *et al.* (1999). The weight-related behaviour questionnaire was developed and validated for the POWIRS study. A question on food insecurity was included, because this may influence eating behaviour (Mvo *et al,* 1999; Alaimo *et al,* 2001). A qualified dietician (EJ) completed the questionnaires by interviews.

Statistical analyses

The results were analysed with the STATISTICA 6.0 package. The attitude and knowledge scores were calculated according to the method of Kruger and Van Aardt (1998). A higher score indicated a more positive attitude and better knowledge. The percentages of women with a positive, neutral and negative

attitude were also calculated. For the weight-related behaviour and emotion-induced eating results, the percentages of women who responded to the 'sometimes' and 'often or always' categories were calculated (Balentine *et al,* 1991).

Analysis of covariance and calculation of effect sizes were used to determine practical significant differences between means in the attitude and knowledge scales. A medium effect size ($d \geq 0.5$) indicated possible practical significance and a large effect size ($d \geq 0.8$) practical significance. For the weight-related behaviour and emotion-induced eating scale, a medium effect size was indicated by $\omega \geq 0.3$ and a large effect size by $\omega \geq 0.5$ (Ellis & Steyn, 2003) for associations of the responses with body weight.

RESULTS

Attitude towards obese people, thin people and weight control (Table 1)

The total attitude score towards weight control, which included attitudes towards obese people, thin people and weight control was remarkably similar in the normal-weight, overweight and obese women. Certain tendencies were observed, although not indicating practical significance. The total attitude score was in the neutral range, but closer to positive than negative (score = 65%). In the subscales, the highest score was towards thin people and were in the positive range (score = 70%). The score towards thin people was higher and therefore more positive than the score towards obese people.

The percentage of women that were positive about thin people as well as weight control increased with increasing BMI. Therefore, the higher the BMI of the woman, the more positive she was about thinness and weight control. About two-thirds of the total group of women was neutral towards the total attitude scale. About 60% of the women were neutral towards obese and thin people respectively. However, in the obese women, a smaller percentage of women were neutral towards thin people (47%) than the normal-weight and overweight women, since more of the obese women were positive towards thin people. A

small percentage of the women were negative towards obese people, with an equal distribution throughout the body weight groups. None of the women were negative towards the total scale, thin people and weight control.

Table 1 ***Attitude towards weight control and knowledge about the relationship between obesity, health and nutrition***

Scale	Negative 0-35 ***Total group*** *N = 100* *Mean ± SD*	Neutral 36-70 ***Normal weight*** *N = 38* *Mean ± SD*	Positive 71-100 ***Overweight*** *N = 25* *Mean ± SD*	 ***Obese*** *N = 37* *Mean ± SD*
Attitude towards weight control				
Total attitude score	64.9 ± 9.9	64.0 ± 9.2	64.3 ± 10.0	66.3 ± 10.6
Attitude towards obese people	62.0 ± 17.4	61.5 ± 16.7	60.8 ± 17.9	63.5 ± 18.0
Attitude towards thin people	70.0 ± 17.4	68.2 ± 16.6	68.3 ± 19.3	72.9 ± 16.8
Attitude towards weight control	64.7 ± 9.9	64.0 ± 8.0	65.5 ± 10.9	64.8 ± 11.0
Knowledge of obesity, health and nutrition				
Total knowledge score	64.4 ± 15.8	60.2 ± 16.0 a	64.3 ± 16.5	68.8 ± 14.4 a
Causes of obesity	61.0 ± 22.8	55.8 ± 25.5 a	68.0 ± 20.0 a	61.7 ± 20.7
Relationship of obesity with health	70.7± 20.7	63.5 ± 19.3 a*	67.9 ± 31.9 b	79.8 ± 18.2 a*b
Treatment of obesity	60.5 ± 21.9	59.8 ± 22.7	58.3 ± 23.3	62.8 ± 20.4

	Percentage response			
	Total group	***Normal weight***	***Overweight***	***Obese***
	N = 100	*N = 38*	*N = 25*	*N = 37*
Total attitude score				
Positive attitude	31.7	31.6	32.0	31.6
Negative attitude	0.0	0.0	0.0	0.0
Neutral attitude	68.3	68.4	68.0	68.4
Obese people				
Positive attitude	31.7	34.2	28.0	31.6
Negative attitude	8.9	10.5	8.0	7.9
Neutral attitude	59.4	55.3	64.0	60.5
Thin people				
Positive attitude	41.6	31.6	40.0	52.6
Negative attitude	0.0	0.0	0.0	0.0
Neutral attitude	58.4	68.4	60.0	47.4
Weight control				
Positive attitude	32.7	23.7	36.0	39.5
Negative attitude	0.0	0.0	0.0	0.0
Neutral attitude	67.3	76.3	64.0	60.5

Variables with the same symbol may differ practical significantly (d ≥ 0.5)
*Variables with the same symbol marked with * differ practical significantly (d ≥ 0.8)*

Knowledge about the relationship between obesity, nutrition and health (Table 1)

The obese women had a higher total knowledge score indicative of practical significance than the normal-weight women, with the overweight women having a

score intermediate to those of the normal-weight and obese women. The obese women had a slightly non-significantly higher score on the causes of obesity than the normal-weight women, while the overweight women had a higher score indicative of practical significance, than the normal-weight women. In the subscale of the relationship between obesity and health, the obese women had a practical significanlty higher score than both the normal and overweight women. In the treatment of obesity subscale, the obese women had slightly better knowledge than both the normal-weight and overweight women. The highest score in the subscales was obtained for the relationship between obesity and health, which was obtained by the obese women. The score of 80% indicates very good knowledge about this subject.

Weight-related behaviour (Table 2)

Factors influencing weight

High percentages of the women indicated that they engaged in overeating, stress-related eating and drinking and felt guilty after eating too much. The responses were similar in all the body-weight groups, although possible under-reporting by the obese was not excluded. Self-reported food insecurity in both childhood and adulthood were higher, indicative of practical significance, in the obese than the normal-weight women.

Weight consciousness

Indicative of practical significance, it was observed that with increasing BMI, more women had been on weight reducing diets before, thought that they should lose weight and were currently trying to lose weight. Almost half of the women indicated that they weigh themselves regularly, most of them once a month, with more than half of these women being obese.

Reasons for weight loss

The reasons most often reported for weight loss results were exercise and eating less. Eating less as a reason for weight loss was reported with equal frequency by the normal-weight and obese women, but more normal-weight than

overweight or obese women, indicative of practical significance, reported exercise to result in weight loss,

Reasons for weight gain

Eating too much and too little exercise were the reasons most often reported to result in weight gain. A higher percentage, indicative of practical significance, of the obese women than the normal-weight or overweight women reported that eating too much caused them to gain weight.

Weight reducing methods

Eating less and exercise were the most often reported methods used in weight reducing attempts. Eating less was reported more often, indicative of practical significance, by the obese women. Meal skipping was frequent in the overweight and obese women and low in the normal-weight women. Following of weight reducing diets has also been more common in the overweight and obese women compared to the normal-weight women.

Weight change during lifetime

An alarmingly high percentage of the total group of women (75%) reported weight gain since age 18, with equal distribution throughout the body-weight groups. A small percentage (6%) of the women had kept their weights constant during their lifetime, also with equal distribution throughout the body-weight groups. A small percentage (12%) of the women were able to lose weight during their lifetime and they were significantly more often in the normal-weight group than in the overweight and obese groups.

It seemed that weight loss results during the past year had been more succesful than those during their lifetime. Only 8% of the obese women had lost weight during their lifetime, but 24% had lost weight during the past year. The same tendency has been observed in the overweight women. In the normal-weight women, the same number of women who had lost weight during their lifetime had lost weight during the past year. Therefore, it seemed that the normal-weight

women were more successful in weight reducing attempts in the long term than overweight and obese women.

Of the overweight and obese women, 84% thought they should lose weight. Of the normal-weight women, 12% thought they should lose weight and an equal percentage of them were currently trying to lose weight. The percentages of the overweight and obese women that were currently trying to lose weight were lower than those reported that they thought they should lose weight.

Table 2 ***Weight-related behaviour results of the women***

Question	***All*** *N = 100*	***NW*** *N = 38*	***OW*** *N = 25*	***OB*** *N = 37*
	Percentage response to 'sometimes', 'often' and' always'			
Factors influencing body weight				
When plenty of food is available, I eat as much as possible	59.0	59.4	56.0	60.5
I feel guilty after I have eaten too much	66.3	62.1	72.0	68.4
I eat a lot of food at once and then I feel uncomfortable	54.5	52.6	64.0	50.0
In my childhood (< 18 y) there were times when we didn't have enough food in the home	36.3	26.0	28.0	55.0
In my adulthood (≥ 18 y) there were times when we didn't have enough food in the home	15.0	13.0	8.0	24.0
Weight consciousness				
I think I should lose weight				
No	34.7	26.0	16.0	13.2
Yes	65.3	12.0 a*b*	81.0 a*	86.8 b*
I have been on a diet for weight reduction	33.6	15.8 ab	44.0 a	44.8 b
I am trying to lose weight at the moment				
No	47.5	27.0	40.0	29.0
Yes	52.5	11.0	60.0	71.1
Please indicate how often you weigh yourself				
Daily	0.0	0.0	0.0	0.0
1-6 x / week	5.0	2.6	8.0	5.3
1x / 2 weeks	5.0	5.3	8.0	2.6
1x / month	41.6	36.8	32.0	52.6
1 x / 2-3 months	18.8	23.7	24.0	10.5
1 x / 4-6 months	8.9	10.5	16.0	2.6
< 1 x / 6 months	9.9	13.2	0.0	13.2
Never	10.9	7.9	12.0	13.2
Current weight reducing methods				
Eating less	39.6	26.3 a	36.0	55.3 a
Exercise	39.6	36.8	40.0	42.1
Not snacking between meals	11.9	10.5	12.0	13.2
Skipping meals	16.8	5.3 ab	24.0 a	23.7 b
Fasting	10.9	7.9	20.0	7.9
Diet plan for weight loss	11.9	5.3 ab	16.0 a	15.8 b
Drinking more water	2.0	0.0	8.0	0.0
Tables or other preparations	5.9	2.6	8.0	7.9

Table 2 (continued) ***Weight-related behaviour results of the women***

Question	***All*** *N = 100*	***NW*** *N = 38*	***OW*** *N = 25*	***OB*** *N = 37*
	Percentage response to 'sometimes', 'often' and' always'			
Reasons for own weight gain				
Eating too much	21.8	13.2 a	16.0 b	34.2 ab
Eating 'wrongly'	7.0	0.0	8.0	13.2
Too little exercise	15.0	7.9	8.0	13.2
'Growing up'	8.9	7.9	16.0	5.3
Ageing	11.9	13.2	8.0	13.2
Inherited	5.0	2.6	0.0	10.5
Pregnancy	11.0	13.2	16.0	0.0
Increased exercise	4.0	5.3	8.0	0.0
Birth control	7.0	0.0	8.0	13.2
Don't know	15.9	21.1	12.0	13.2
Laziness / relaxing / free of worries	4.0	5.3	4.0	0.0
Other	12.0	10.5	16.0	7.9
More happy	8.0	7.9	16.0	2.6
Reasons for own weight loss				
Exercise	6.8	10.5 ab	0.0 a	2.6 b
Eating less	7.6	5.3	0.0	5.3
Stress / worrying	1.0	2.6	0.0	0.0
Pregnancy	2.0	2.6	0.0	2.6
Food type / healthy eating	3.8	2.6	4.0	0.0
Stopping birth control	1.0	0.0	0.0	2.6
Total	22.2	23.6	4.0	13.1
Weight cycling				
Weight change since age 18				
Constant	6.0	5.3	4.2	7.9
Increasing	75.0	68.4	83.0	76.3
Decreasing	11.9	21.1 ab	4.2 a	7.9 b
Varies, ending larger	1.0	2.6	0.0	0.0
Varies, ending smaller	1.0	0.0	0.0	2.6
Varies, ending the same	2.0	0.0	4.2	2.6
Don't know	3.0	2.6	4.2	2.6
Weight cycling in the past year				
Constant	39.6	50.0 a	48.0 b	23.7 ab
Increasing	18.8	15.8	20.0	21.1
Decreasing	17.8	15.8	12.0	23.7
Varies, ending large	4.0	0.0	8.0	5.3
Varies, ending smaller	3.0	0.0	8.0	2.6
Varies, ending the same	16.8	18.4	4.0 a	23.7 a
Weight cycling in the last 6 months				
Constant	36.6	44.7	48.0	21.1
Increasing	28.7	26.3	32.0	29.0
Decreasing	34.7	29.0	20.0	50.0

All = total group; NW = normal weight; OW = overweight; OB = obese
Variables with the same symbol may differ practical significantly ($\omega \geq 0.3$)
*Variables with the same symbol marked with * differ practical significantly ($\omega \geq 0.5$)*

Emotion-induced eating

Responses to the emotion-induced eating scale were strikingly similar for the three body-weight groups, although under-reporting by the obese was suspected. The most common response was eating for a treat (81%), followed by being

happy (72%), eating between meals even if not hungry (53%) and being bored (46%). Binge-eating occurred in 38% of the women, eating when worried in 35%, eating when sad in 32% and eating when mad in 20%. Stress-related eating and drinking occurred in 36.7% of the women, with equal distribution throughout the body-weight groups. Therefore, these results indicate a high frequency of emotion-induced eating in this group of black women, independent of body weight.

Table 3 ***Emotion-induced eating scale results***

	Percentage of women with 'sometimes' and 'usually or always' responses			
	Total group *N = 100*	***Normal weight*** *N = 38*	***Overweight*** *N = 25*	***Obese*** *N = 37*
Age *(Mean ± SD)*	31.2 ± 8.7	28.5 ± 7.6	30.8 ± 8.5	34.3 ± 9.1
Question				
When I am worried I eat more	35.0	33.3	43.8	31.5
I eat when I am mad	20.0	16.6	12.6	27.3
When I do something well I give myself a treat (food)	81.0	86.7	81.3	77.1
When I am sad I eat more	32.0	34.5	31.3	28.5
When I am happy I eat more	72.0	73.3	62.6	74.3
When I am bored I eat more	46.0	66.7	37.5	31.5
I eat between meals even when I am not hungry	53.0	53.3	56.3	51.4
I eat a large amount of food in a short period of time, feeling that the eating gets out of control (can't stop eating)	38.0	34.5	37.6	40.0
I try to make myself feel better by eating or drinking in a stressful situation	36.7	36.9	36.0	36.8

DISCUSSION

Attitude towards weight control did not differ practical significantly between the normal-weight, overweight and obese women. However, a tendency was observed that with increasing BMI, more women were positive about thin people. A large percentage (almost two-thirds) of the women were neutral towards weight control, but none was negative. A small percentage was negative towards obese people, with no differences between the body-weight groups. More obese women than normal-weight or overweight women were positive towards thin people, but the percentage of women that were positive towards obese people was equal throughout the body-weight groups. None of the women were

negative towards thin people. Therefore, it seemed that many women admired a thin body image and were not negative about weight control. The high neutral response may be explained by the finding that the African culture apparently does not judge people according to their appearance (Gore, 1999), which may explain the apparent tolerance of obesity in this culture.

The finding that black women were neutral towards weight control could be positive, indicating that they could be motivated in the desired direction. The focus should be on clear information, presented in a simple culturally-sensitive way, to explain why obesity could be dangerous to their health (Jonker *et al,* 2004). If a larger figure is preferred (Steyn, 2004), acceptable ranges could be explained to the women.

An alarmingly 75% of the women have gained weight since adulthood (≥18 years), with equal distribution throughout the body-weight groups. This is in contrast with the situation in traditional Africans (Walker, 1998). More overweight and obese than normal-weight women thought they should lose weight, have been on weight reducing diets before and indicated that they were currently trying to lose weight. However, it seems that they were not succesful in losing weight in the long term. These results are strongly supported by the findings of Kumanyika and Guilford-Davenport (1993) in African-American women.

It seemed that more normal-weight than overweight and obese women had success with weight control in the long term. The proportion of overweight and obese women who indicated being interested in losing weight were lower than the proportion who indicated that they were at some time trying to lose weight. However, the percentage of normal-weight women that thought they should lose weight was equal to those who engaged in weight reducing attempts. It also seemed that exercise as a weight reducing method was more successful in the normal-weight women than in the overweight and obese women.

It has been suggested that lack of health and nutrition knowledge could be related to obesity (McArthur *et al,* 2001). This study may suggest that knowledge is not a strong influencial factor, since the obese women had better knowledge than the overweight and normal-weight women. Indications in this direction were also given by Walker *et al.* (1991) in South African adolescents and Kumanyika and Guilford-Davenport (1993) in African-American women. However, we suggest that knowledge need to get a new focus, extending basic nutrition and health knowledge to self-awareness, identification of personal barriers, non-nutritive reasons for eating and vulnerable moments, and how to make sensible choices in vulnerable times. Therefore, health and nutrition professionals are encouraged to work together with professionals from behavioural sciences to improve the effectiveness of weight control efforts of women in the community.

A high frequency of overeating and emotion-induced eating, as well as a high frequency of guilt feelings after eating too much, was indicated in this study. Overeating and eating for other reasons than nutrition, such as stress, when happy or bored, will represent a barrier to success in weight control. It has been suggested that emotional stress leads to increased fat and sugar intake (Korkeila *et al,* 1998; Laitinen *et al,* 2002; Schiffman *et al,* 2000). Emotion-induced eating may be a potential strong risk factor for the development of obesity in this population. In African-American girls, higher scores on the emotional eating scale were found than in white girls (Striegel-Moore *et al,* 1999) and binge-eating occurred twice as often in black than in white women (Fitzgibbon *et al,* 1998; Striegel-Moore *et al,* 2000). The role of eating as a coping mechanism has also been described previously in black women (Thompson, 1994). Stress and emotion-induced eating was associated with higher BMI and weight gain in other populations (Striegel-Moore *et al,* 1999; Korkeila *et al,* 1998), and has occured more often in women (Laitinen *et al,* 2002; Striegel-Moore *et al,* 1999). Previous reports in black South African women indicated high levels of stress and depression (Vorster *et al,* 2000; Steyn *et al,* 2000), which may increase the risk of emotion-induced eating. Other reports (Mvo *et al,* 1999; Puoane *et al,* 2004;

Steyn, 2004) also indicated that black women eat when worried, upset and depressed. Therefore, obtaining of coping skills would be valuable to achieve better weight control results.

The role of food for uses other than nutrition seems to be part of the African culture (Puoane *et al,* 2004). Mvo *et al.* (1999) indicated that food is highly valued against a background of deprivation. The use of food to give pleasure and comfort (Mvo *et al,* 1999) and being a sign of warmth, acceptance and friendship (Puoane *et al,* 2004), have been recognised before in this population. Therefore, emotion-induced eating seems to be a practice that fits in well in the African culture.

The association of food insecurity with obesity has been described (Olson, 1999; Drenowski & Specter, 2004). In this study, the obese women reported twice as often as the normal-weight women that they had experienced food insecurity in their childhood (54% versus 26%) and adulthood (24% versus 13%). This may be another barrier to successful weight control. In a group of overweight food-insecure women in a Cape Town squatter camp, it was indicated that the idea to voluntarily restrict food intake seemed unacceptable, although these women expressed the wish to lose their excess weight (Mvo *et al,* 1999). Thompson (1994) suggested that food acts as a balm to ease stress in poor women. It is also hypothesised that food-insecure people eat more when plenty of food become available (Alaimo *et al,* 2001). Furthermore, weight control and healthy eating may not be a priority in poor or food-insecure people (Sengwana & Puoane, 2004; Jeffery & French, 1996). Therefore, the association between food insecurity and obesity seem to depend on many factors. Even after food insecurity has resolved, women may still continue the compensative habits acquired in their food-insecure days (Kumanyika, 1987).

It seems that the normal-weight women, who were also younger, were more successful in their weight reducing attempts than the obese women. These results may suggest lack of certain psychological strenghts in the obese women.

Other results from our study indicated poorer psychological well-being in the obese than the normal-weight women (Schutte *et al,* 2004). It may also be that the larger the women get, the more they tend to give up trying to lose weight. Some indication in this direction is revealed in the report of Ndlovo *et al.* (1999). However, the normal-weight women also gained weight with age and engaged in overeating and emotion-induced eating, but since they were younger, it might not yet have been enough to result in obesity. Since BMI increased with increasing age in this study, as well as in a larger study in this population (Puoane *et al,* 2002), it could be predicted in view of their eating behaviour that in time, the normal-weight women may also become obese.

A host of barriers that may impair attempts to reduce weight in black South African women could be suggested. These barriers include: low metabolic rate (Jones *et al,* 2004), lack of emotional support (Laitinen *et al,* 2002), the high value of food in the African culture (Puoane *et al,* 2004), especially against a background of food insecurity; poverty, past and present food insecurity (Mvo *et al,* 1999; Drenowski & Specter, 2004; Kumanyika, 1987), overeating, emotion-induced eating, the availability of a variety of highly palatable foods rich in sugar and fat (Golay & Bobbioni, 1997; Popkin, 1998) and lack of certain psychological strengths to practise effective stimulus control and cope in emotional and stressful situations. Because the African culture apparently does not judge people according to their appearance (Gore, 1999) and may therefore be tolerant of obesity, the obese woman who experience her obesity negatively (Ndlovo *et al,* 1999) may not receive the support and motivation she needs from her family members, when trying to lose weight. Lack of emotional support was indicated as the most important factor leading to stress-related eating in women (Laitinen *et al,* 2002). It will be important that these barriers are addressed in weight-control programmes.

It is tempting to speculate that the occurrence of underweight children and obese mothers in the same household may be a result of overeating and emotion-induced eating in the mothers, which exhaust the food budget and result in the

children being underfed. An indication of this was suggested in the report of Mvo *et al.* (1999): 'You see people having up to three dishes in front of them. One is for herself, the other one is for the child, and the last one is for herself again'.

CONCLUSIONS

From this study it appears that both non-obese and obese black South African women are weight conscious, positive about thinness and try to lose weight. However, a low success rate with weight reducing attempts was suggested. The high percentage of women who gained weight (75%) since 18 years of age is a matter of great concern. Overeating incidents and emotion-induced eating were identified as barriers in achieving long-term weight loss. Emotion-induced eating and eating for other reasons than nutrition may be a potentially strong risk factor for the development of obesity and a factor in the maintenance thereof in this population. It seems that basic health and nutrition knowledge about obesity is not be a very important factor in poor weight control.

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CHAPTER 6

Summary, conclusions and recommendations

This chapter summarises the most important findings of the POWIRS study and discusses conclusions and recommendations.

6.1 Health-risk factors in obese compared to non-obese urban black South African women

The metabolic syndrome, known to be associated with increased risk for NIDDM and CVD, was diagnosed in 38% of the obese women, none of the normal-weight women and 8% of the overweight women. Variables associated with the metabolic syndrome s-uric acid, s-fasting glucose, 2h-post-load glucose, s-fasting insulin, the HOMA-insulin-resistance index, s-C-reactive protein (CRP) and s-fibrinogen had practical significantly higher levels in the obese than the normal-weight women. Systolic and diastolic blood pressure, s-triglycerides, s-gamma-glutamyl-transferase (GGT) and p-plasminogen activator inhibitor-1 (PAI-1) had higher levels indicative of practical significance in the obese than the normal-weight women. Therefore, the results indicate that obesity may lead to increased health-risk factors in this group of women. The metabolic syndrome in these women was characterised by higher body mass index, age, the HOMA-insulin-resistance index and possibly s-uric acid, s-gamma-glutamyl transferase (GGT) and the acute-phase protein s-C-reactive protein.

6.2 Association of acute-phase proteins with obesity, insulin resistance and the metabolic syndrome

Levels of the acute phase proteins s-CRP, p-fibrinogen and possibly p-PAI-1 were practical significantly higher in the obese than the normal-weight women. Serum-CRP correlated positively with BMI ($r = 0.60$, $p < 0.0001$), waist circumference ($r = 0.54$, $p < 0.0001$) and abdominal saggital diameter ($r = 0.55$, $p < 0.0001$).

Serum-CRP was possibly associated with the metabolic syndrome in this study, since women with the metabolic syndrome had higher levels indicative of practical significance than women without the syndrome. The high inter-individual variation of CRP-levels (Pearson *et al,* 2003) may explain why the difference between women with compared to those without the metabolic syndrome has not reached practical significance. The results indicate that it may be sensible to include CRP measurement in health-risk assessment in this population, since it has been shown in prospective studies that CRP adds prognostic information to the risk of coronary incidents in women with, as well as without the metabolic syndrome. It is also a stronger predictor of CVD and NIDDM than individual metabolic syndrome components (Ridker, 2003). Guidelines for cut-off values to indentify patients at risk have been published (Pearson *et al,* 2003).

With increasing HOMA-insulin-resistance index, levels of s-CRP, p-PAI-1, white blood cells, haemoglobin and haematocrit increased, consistent with findings in other populations (Haffner, 2003; Barbieri *et al,* 2001). Serum-CRP and p-PAI-1 had correlation coefficients of $r = 0.25$ ($p < 0.05$) and $r = 0.22$ ($p < 0.05$) respectively, with the HOMA-insulin-resistance index. The Women's Health Study showed that CRP independently predicts the development of CVD and NIDDM (Ridker, 2003; Haffner, 2003) and that PAI-1 and fibrinogen are also predictors of NIDDM and CVD (Haffner, 2003). In view of these findings, inflammation may be an important NCD risk factor in this population and obesity a risk factor for an increased acute-phase response.

Black women seem to be predisposed to develop insulin resistance, since higher levels of fasting insulin and insulin resistance occurs in lean black children compared to white children (Caprio, 2002; Schuster *et al,* 1998). It may be sensible to screen black females in adolescence for risk factors such as insulin resistance and increased blood pressure and CRP concentrations.

The normal-weight women also presented with high-risk levels of fibrinogen and CRP. The reason for this is not known. A suggested factor involved in an

increased inflammatory response in other studies was psychosocial stress (Hjemdahl, 2002). Previous studies found high levels of stress and psychopathology in urban black South African women (Vorster *et al,* 2000; Steyn *et al,* 2000). It may also be speculated that the high levels of acute-phase proteins in the normal-weight women may be related to early nutritional deprivation (Hales & Barker, 2001). It is not known whether low birth weight was a factor in the normal-weight women, but 26% of them indicated that they had experienced food insecurity during their childhood. Studies in animals indicated deficient liver development with low protein diets, which could have implications for hepatic acute-phase protein turnover (Hales & Barker, 2001). Hales and Barker (2001) speculated that this could fit in with their hypothesis that early nutritional deprivation predisposes individuals to develop diabetes and CVD and adult life, since the association between inflammation and diabetes is now well-recognised.

6.3 Dietary intakes

For the first time in this population, it was indicated that the obese women had practical significantly higher daily intakes of total energy, total carbohydrates, total protein, plant and animal protein, total fat, saturated and monounsaturated fat and dietary fibre than the normal-weight women, after exclusion of under-reporters. Since the overweight and obese groups were small after under-reporters had been excluded, it is suggested that the nutritional analyses are repeated in a larger overweight and obese group.

Previous studies have failed to indicate higher macronutrient intakes to be associated with obesity. Except for under-reporting, the role of socially desirable answers might have been a factor (Macdiarmid *et al,* 1998; Mennen *et al,* 2000), as well as emotion-induced eating, where food consumed in emotional situations would probably not be reported in a nutritional questionnaire (Laitinen *et al,* 2002). In particular, foods rich in sugar and fat are important under-reported items, especially in obese women (Macdiarmid *et al,* 1998). It may be sensible to quantify, at least in part, food consumed for emotional and other reasons, together with the food frequency questionnaire.

The author suggests that the interviewers of the nutritional questionnaires are trained to perform some cross-control checks. In particular, subjects should be asked about second portions and food eaten away from home. In the accompanying dietary questionnaire (see Addendum 5, p. 132), the women reported regular eating of 'take-away' food (results not shown) and it is not sure if all of this was reported in the food frequency questionnaire.

The high caloric intakes may be the result of large portion sizes, common in the African population (Venter *et al*, 2000), especially stiff maize meal porridge that is eaten as a staple, which is a more concentrated source of carbohydrates and energy per volume than other starches, such as rice or pasta (Food Composition Table, 1991; Food Quantities Manual, 1991) and may therefore promote overconsumption of energy.

6.4 Physical activity

The physical activity results of this study were unexpected. The Global Physical Activity Questionnaire (GPAQ)) of the WHO was used for the first time in this population. The previous questionnaire developed by Kruger *et al.* (2002) indicated that lower physical activity was associated with obesity, consistent with other studies (King *et al*, 2001; Trost *et al*, 2001). However, a study in Australian women also did not find a difference in physical activity levels between weight gainers and weight maintainers (Ball *et al*, 2002), which may indicate that it is not impossible that our results were in the right direction. The sitting or resting activities of the women in our study were strikingly similar, while the overweight and obese women indicated more time spent at some activities than the normal-weight women. Therefore, it was suspected that the obese women tended to over-report both the duration and intensity of the activities. In the GPAQ (see Addendum 7, p. 151) subjects had to indicate the time they usually spent at a certain type of physical activity, without any time categories to select from. In this way the time assessments are likely to be inaccurate, since they are 'guessed'. Further, they had to assess for themselves whether an activity in their lives were of moderate or vigorous intensity, since the activities indicated as examples in the questionnaire often did not apply to their own lives. It is

suggested that the physical activity questionnaire of Kruger *et al.* (2000) should be used in this population.

Although it may be desirable to measure physical activity in kilojoules expended, it may not give accurate results in an overweight and obese population. The formula suggested for calculation of energy expenditure is based on body weight (Ainsworth, 1995) and this would implicate that a fatter person have a higher metabolic rate than a leaner person. This is unlikely to be the case, since fat tissue does not increase metabolic rate (Mahan & Arlin, 1994; Escott-Stump, 1992) and obese women may also tend to move slower. Furthermore, the lower resting metabolic rate in black women (Jones *et al,* 2004) may lead to an overestimation of energy expenditure. Therefore, measuring of physical activity in time units or with a score as previously performed (Kruger *et al,* 2000) seems more appropriate in this population.

6.5 Attitudes, knowledge and weight-related behaviour

The attitudes towards weight control did not differ practical significantly between normal-weight, overweight and obese women. The total attitude score was neutral, but closer to positive than negative (score = 65%). Although not indicative of practical significance, a tendency was observed that women with a higher BMI were more positive towards thin people and the obese women had an overall positive attitude towards thin people. The high percentage of women that were neutral towards obese people, thin people and weight control, may be explained by the findings that Africans apparently do not judge people according to their appearance and that obesity may be differently defined in the African culture than in the Western culture, where it is highly stigmatised (Gore, 1999; Baskin *et al,* 2001). The condition of obesity, however, seems not to be regarded with favour. In an excellent report by Ndlovo *et al.* (1999) on black South African women's perceptions of obesity, it was clear that the majority of the women had very negative perceptions about obesity.

Obese women had better knowledge, indicative of practical significance, about the relationship between obesity, nutrition and health than the normal-weight

women. Especially in the subscale of the relationship between obesity and health, the obese women scored practical significantly higher than both the overweight and obese women. The score of 80% obtained by the obese women indicated very good knowledge about this subject. However, the questions included in the knowledge scale were limited and did not include everything necessary to know for successful weight control. Nevertheless, these results indicate that good knowledge is not necessarily associated with proper weight control, consistent with suggestions in African-American women (Kumanyika & Guilford-Davenport, 1993) and South African adolescents (Walker *et al,* 1991).

The women also seemed conscious about weight control, were interested in losing weight and have at some time tried to lose weight. However, it seems that the overweight and obese women were not very successful in the long term, while the normal-weight women seemed to have achieved better results and a higher percentage of them were apparently motivated. These results raise the question as to which factors in the normal-weight women motivate or enable them to have more success, which is absent or less functional in the obese women. Since the obese women apparently had poorer psychological health than the normal-weight women (Schutte *et al,* 2004), it may be suggested that psychological strengths are involved.

In the weight behaviour questionnaire, the aim of the question 'I think I should lose weight' was to assess whether the women would realise the need for weight loss, implicating health reasons. It would be of value to include the question 'Do you *want* to lose weight?' in the future, since there may be a difference between what they want to do and what they think they should do. For example, in a study in African-American women, they preferred small to medium body sizes, but they thought that medium to large body sizes were the healthiest (Liburd *et al,* 1999).

6.6 *Barriers to successful weight control and potential risk factors for development of obesity*

Overeating incidents and emotion-induced eating were identified as potential risk factors for obesity and barriers to succesful weight control. In all three groups of women, overeating, binge-eating, stress-related eating and eating associated with some emotions, such as being happy, bored, worried or mad, were common. No differences occurred between the normal-weight, overweight and obese women in this respect. However, under-reporting of the truth by the obese women was suspected. The fact that all the women have gained weight with age, suggests that these factors would be detrimental to all the women and present a barrier to success with weight reducing attempts as well as a potential risk factor for weight gain.

Since food has a central role in the African culture other than simply nutrition (Puoane *et al,* 2004), emotion-induced eating may be particularly important in black women. In African-American girls, higher emotion-induced eating scores were obtained than in white girls and the risk of binge-eating (also a form of emotion-induced eating) was twice as high in black than in white women in the USA (Striegel-Moore *et al,* 2000). It is recommended that emotion-induced eating is further researched in this population, since this may represent a strong risk factor for the development of obesity and a factor in the maintenance thereof (Striegel-Moore *et al,* 1999).

In the same way that nutrient intakes are under-reported by obese persons, it was suspected that the obese women under-reported in the emotion-induced eating scale. The questions asked were quite direct, which may have offended the obese woman if she was sensitive about this issue (Striegel-Moore *et al,* 1999).

Especially with urbanisation and the availability of highly palatable, high energy fat and sweet foods, this environment may encourage eating for other reasons than nutrition, which may be detrimental to these women, especially in view of their apparent low energy expenditure.

6.7 Recommendations for weight control programmes

Development of culturally-sensitive weight control programmes seems mandatory at this stage. Some recommendations from this study are discussed next.

Portion size control seems important in this population. In the Food-based dietary guidelines, the guideline 'enjoy a variety of foods' (Vorster *et al,* 2001) may promote overeating in the African culture, since food is highly valued (Mvo *et al,* 1999; Puoane *et al,* 2004). Therefore, it may be considered to include the words 'in moderation' in this guideline and specify moderation, for example by using a 'plate model' (Hanekom, 2002), allowing only *one* plate of food and no second servings. The guideline 'meat, fish, chicken, milk and eggs can be eaten every day' may also need specification for the same reason, but also because meat is particularly highly valued in the African culture (Puoane *et al,* 2004) black people may eat more than desirable.

The topic of emotion-induced eating and eating for other reasons than nutrition (e.g. as a remedy or coping tool in stressful situations) should be addressed in the programmes, as well as obtaining of certain psychological strengths or coping skills. The value of food against a history of food insecurity (Mvo *et al,* 1999; Kumanyika, 1987) should be dealt with appropriately, so that the women do not unconsciously 'live in the past'. If food insecurity is currently experienced, effective coping skills should be obtained. If food is used for other purposes, as mentioned above, this would not only promote obesity, but also decrease the family's available food supplies and money and in this way increase the stress associated with poor economic circumstances.

Implementation of low glycemic index foods (GI) should be encouraged, since it is associated with better weight control (Brand Miller *et al,* 2002) and glycemic control (Willet *et al,* 2002) and seem to help prevent development of degenerative diseases (Augustin *et al,* 2002). It may also increase satiety and decrease food cravings, which would be particularly important with regards to emotion-induced eating. Furthermore, low GI foods are part of the African

culture, for example dry beans (Walker, 1995). Healthy eating could become an exciting new 'culture' or trend in the black population, where traditional foods with the focus on low GI, could be combined with foods from the Western culture, such as meat, with more vegetables and fruit. It may be sensible to restrict fruit juice and drinks containing sugar, which have no satiety value and therefore easily lead to overconsumption of energy.

Black women should be educated to obtain knowledge about how energy balance in the body works – the 'energy in and energy out' principle. Physical activity should be an integral part of the programmes to increase energy expenditure (Baskin *et al*, 2001). It could be performed in groups, for example culturally-sensitive aerobic dances.

Development of self-efficacy seems important. Women need to be guided and empowered to identify their own barriers and vulnerable situations and obtain skills to cope effectively in those situations, so that they do not give in to stimuli to eat for reasons other than nutrition. They should also learn that food used on social occasions and celebrations contribute to daily nutrition. Skills on how to enjoy these occasions without exceeding their required daily energy needs would be helpful.

It needs to be explored whether one programme focusing on optimum nutrition or a separate weight control programme is needed. Some of the factors hampering healthy choices associated with obesity may also be important factors in good nutrition and health overall. Poor choices may therefore lead to both obesity and poor health.

From numerous studies showing a relationship between lifestyle and disease (Puska *et al*, 2002), developing countries could prevent a disease epidemic by learning from those experiences. If the behavioural change phase of the nutrition transition (Popkin, 1999) could be entered sooner, before many people's lives and the countries' health resources become burdened by degenerative diseases, this could have an enormously positive impact in developing countries.

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ADDENDUM

Acknowledgements

Table 1.1 ***The POWIRS study co-workers and agreement to use the results***

Role / analysis	*Co-workers*	*Signature*
Initiating, planning, and funding	Prof HH Vorster	
	Prof MP Wissing	
Manager	Dr AE Schutte	
Drawing of blood samples, clinical examination, oral temperatures, blood pressure measurement	Sr CM Lessing	
Anthropometry	Dr C Underhay	
	Miss SS Slabbert	
Demographics Dietary intakes Attitude and knowledge scale Weight-related behaviour	Miss E Jonker	
Physical activity	Miss M Opperman	
Serum lipids, nutritional status markers, full blood count, uric acid, gamma-glutamyl-transferase (GGT), plasma fibrinogen and plasminogen activator inhibitor-1	Mrs Z Pieterse	
	Miss E Jonker	
	Prof W Oosthuizen	
C-reactive protein	Dr AE Schutte	
Oral glucose tolerance test, serum glucose, insulin, pro-insulin, C-peptides, free fatty acids	Dr A van der Merwe	
	Mr W Towers	
	Mr M ~~Alessandri~~ ALESSANDRINI	
Leptin, endothelin, adiponectin	Dr AE Schutte	Not relevant to this thesis
	Mr R Schutte	
Genetic analyses	Dr AC Olckers	Not relevant to this thesis
	Prof PJ Pretorius	
Psychological responses	Miss E Botha	Not relevant to this thesis
	Mr M Tamane	
Data manager	Dr M van Lieshout	
Statistics	Dr SM Ellis	

ADDENDUM 2

INFORMATION SHEET

THE POWIRS PROJECT

Dear Participant,

Welcome to the Metabolic Unit of the Potchefstroom University !

Thank you for participating in the POWIRS project. With your help it will be possible to create culturally sensitive programs to prevent health problems like obesity, diabetes, hypertension and stroke in the black community of South Africa.

WHAT IS THE POWIRS PROJECT ?

POWIRS stands for the ***P***rofiles of ***O***bese ***W***omen with the ***I***nsulin ***R***esistance ***S***yndrome.

The aim of this project is to determine risk factors for the development of the insulin resistance syndrome, a condition which develops in obese people. The insulin resistance syndrome is a condition characterized by high blood pressure, high blood sugar levels and high blood fat levels. Diabetes, heart disease and stroke develop frequently in people suffering from the insulin resistance syndrome.

WHY IS THE POWIRS PROJECT IMPORTANT ?

Obesity is a common problem in black women, especially in South Africa, where about 50% of these women are obese. Since obesity leads to the insulin resistance syndrome and eventually to diseases like diabetes, heart disease and stroke, we are concerned about the effect of obesity on the health of black women in South Africa.

In the POWIRS project we want to determine the factors leading to obesity and the insulin resistance syndrome in black South African women. The results of the project will enable The Department of Health and Developmental Social Welfare to develop culturally sensitive programs to prevent the development of obesity and the insulin resistance syndrome in black women.

WHAT WILL BE MEASURED IN THE POWIRS PROJECT ?

Each person who participates in the project will complete a number of quiestionnaires to obtain information on:

Demographic background, eg income, education, area of living
Lifestyle habits, eg, physical activity, smoking, alcohol consumptoin
Medical background
Eating and drinking habits
Knowledge, attitude and behaviour about health, beauty and weight
Weight history

Mental health (psychological quiestionnaires)

A physical examination will be done to check for signs of malnutrition and to measure:

Weight, height, waist and hip circumferences and skinfold thickness

Temperature (orally)

Blood pressure

Blood samples will be taken to measure indicators of health, nutrition and disease, eg blood sugar, cholesterol, blood fats.

The results of all these measurements will give an indication of the health and nutrition status of the individual as well as his risks for diseases like stroke and sugar diabetes.

HOW COULD YOU BENEFIT FROM THE PROJECT ?

A large number of indicators of nutrition and health will be measured, informing you about your health status and risk factors for stroke, sugar diabetes and overweight. We will give feedback on the results to you. If a problem is discovered, nutritional and / or social counseling will be given or you will be referred to a medical doctor if necessary, for treatment and prevention of development of a disease.
Each participant will receive an amount of R100.00.

HOW WILL YOU BE INVOLVED ?

On arrival at the Metabolic Unit, you will receive a number and card and we will show you your room. The procedures of the project will be explained to you and you will sign a consent form that you are willing to participate voluntarily in the project. Please take note that you may quit from the project any time you need to. All information collected from you as well as the results of the measurements will be kept strictly confidental. Please feel free to ask any questions at any time of the project.

The program for the evening will be as follows:

Your skinfold measurements will be taken and you will complete psychological and demographic questionnaires. At 19:00 supper will be served. ***Please do not eat anything else after supper. Only pure water may be taken***. The reason for this is that you must be fasted for the blood tests to be done in the morning. The program continues the following morning. Please sleep before 23:00, because you will be wakened early.

Program for tomorrow morning:

Lady number 1 will be wakened at 6:00. She will give an urine sample. Then she will be weighed and her height, waist and hip circumferences and blood pressure measured. After this she may go back to her bed, and then the next lady will be wakened for the same procedures. Back in bed, the blood samples will be taken by a registered sister. Blood samples are taken with a very thin needle, so you will hardly feel it. This needle will be kept in your arm for two hours and will be removed after the last blood sample is being taken. This will not be painfull, you won't feel the needle while it is in your arm.

Four blood samples will be taken, *one* sample every 30 minutes. It will be finished after *two hours*. After the first blood sample has been taken, you will receive a sugar drink. The other *three* blood samples to be taken will test how your body reacts to sugar. The sugar drink will also replace fluid lost by the body because of the blood taken. The amount of blood taken will be about 100 ml, which is far less than the amount usually given at blood donating, where they take 500 ml. Your body will immediately start to make new blood to replenish the blood taken out. We shall help your body to do so by giving you breakfast after the last blood sample has been taken.

During the 2 hours of the sugar test, you will complete questionnaires, asking you about your demographic background, lifestyle, eating habits, attitudes and knowledge about health, beauty and weight as well as questions on your weight history. The research team members will assist you in completing these questionnaires.

After breakfast, you will complete another psychologic questionnaire. Thereafter lunch will be served for the hungry ones, after which you will be taken back home.

Thank you very much for your participation !

We hope you will enjoy the time with us !

THE RESEARCH TEAM

PROGRAM FOR THE POWIRS PROJECT

Program for the evening:

1. Explanation of procedures and signing of consent forms
2. Measurement of skinfolds
3. Completion of psychological and demographic questionnaires
4. Supper at 19:00
5. ***NOTHING MAY BE EATEN OR DRUNK AFTER SUPPER. ONLY PURE WATER IS ALLOWED***
6. Sleep before 23:00

Program for the next morning:

1. Lady number 1 wakened at 6:00
2. Urine sample, weighing, measurement of height, waist and hip circumferences and blood pressure.
   Lady 2 wakened for the same procedures. Then lady 3 and so on.
3. Back to bed
4. Blood sample taken
5. Sugar drink taken
6. Three more blood samples taken, every 30 minutes.

7. Inbetween completion of questionnairses on physical activity, eating habits, attitude, knowledge on health, beauty and weight and weight history will be done.
8. Breakfast
9. Completion of psychological questionnaires
10. Lunch
11. Departure

ADDENDUM 3

RECRUITMENT AND INFORMED CONSENT FORM

FEEDBACK FORM

POWIRS PROJECT

RECRUITMENT AND INFORMED CONSENT FORM

Title of the project: The profiles of obese women suffering from the insulin resistance syndrome

Ethics Committee no: 03M03

Name: _____ **Subject number:** _____

Adress: _____

Tel no: _____

Age: _____

Are you pregnant ?_____

Are you breastfeeding ? _____

Do you suffer from diabetes ? hypertension ? other disease ?

_____

INFORMED CONSENT

I, the undersigned _____
(full names in print), have read the details of the project or have listened to the oral explanation thereof, and declare that I understand it. I have had the opportunity to discuss relevant aspects with the researcher and declare that I voluntarily participate in the project. I hereby give consent to participate in the project.

_____
Signature of participant

Witnesses:

1. _____ 2._____

Signed at _____ on _____

POWIRS PROJECT: FEEDBACK FORM

Subject number: _____

BLOOD PRESSURE: _____ mm Hg

Normal ☐
Low ☐
High ☐

BLOOD IRON STATUS:

Haemoglobin: ____ g/dl

Normal ☐
Low ☐
High ☐

Haematokrit: _____ %

Normal ☐
Low ☐
High ☐

Please consult your doctor:

Yes ☐
No ☐

We will inform you of all the other results in a few month's time.

THE RESEARCH TEAM

ADDENDUM 4

DEMOGRAPHIC QUESTIONNAIRE

Demographic and lifestyle questionnaire

All information given in this questionnaire is confidential

1. Subject number		
2. Date		
3. Age:		
4. Age	20 – 24	(1)
	25 – 29	(2)
	30 – 34	(3)
	35 – 39	(4)
	40 – 44	(5)
	45 +	(6)

5. First language (please mark the correct block with a X)	Tswana	(1)
	Afrikaans	(2)
	English	(3)
	Sotho	(4)
	Xhosa	(5)
	Zulu	(6)
	Other	(7)
	Specify other	(8)

6. Second language	Tswana	(1)
	Afrikaans	(2)
	English	(3)
	Sotho	(4)
	Xhosa	(5)
	Zulu	(6)
	Other	(7)
	Specify other	(8)

7. What is your marital status?	Never married	(1)
	Married	(2)
	Divorced / separated	(3)
	Widowed	(4)
	Cohabiting	(5)

8. What is your highest qualification ?	Matric	(1)
	Diploma	(2)
	Degree	(3)
	Postdegree	(4)

9. What is your occupation ?	
10. Do you work: shifts, eg night shifts	(1)
office hours (± 8 hours per day)	(2)
mornings / part time (± 5 hours per day)	(3)

11. Are you pregnant ?	No (1)	Yes (2)
12. Are you breastfeeding ?	No (1)	Yes (2)
13. When did you have your last menstrual period (please give the date of the first day of your last period):	D D M	M Y Y

Do you suffer from any of the following ?		
14. Hypertension	No (1)	Yes (2)
15. Diabetes	No (1)	Yes (2)
16. Stroke	No (1)	Yes (2)
17. Heart disease	No (1)	Yes (2)
18. Gout	No (1)	Yes (2)
19. Arthritis	No (1)	Yes (2)
20. Malaria	No (1)	Yes (2)
21. TB	No (1)	Yes (2)
22. Sexually transmitted disease	No (1)	Yes (2)
23. Head injury (previously)	No (1)	Yes (2)

Does anyone in your family suffer from:			
24. Hypertension	No (1)	Yes (2)	Uncertain (3)
25. Diabetes	No (1)	Yes (2)	Uncertain (3)
26. Stroke	No (1)	Yes (2)	Uncertain (3)
27. Heart disease	No (1)	Yes (2)	Uncertain (3)
28. Gout	No (1)	Yes (2)	Uncertain (3)
29. Arthritis	No (1)	Yes (2)	Uncertain (3)
30. Malaria	No (1)	Yes (2)	Uncertain (3)
31. TB	No (1)	Yes (2)	Uncertain (3)
32. Sexually transmitted disease	No (1)	Yes (2)	Uncertain (3)

33. Do you take any medication ?	No (1)	Yes (2)

34. If yes, please list medication:

_____

_____

_____

_____

35. Do you take birth control tablets ?	No (1)	Yes (2)
36. Do you get a birth control injection ?	No (1)	Yes (2)
37. Have you ever been on anti-depressants ?	No (1)	Yes (2)
38. Have you ever been hospitalized for any psychiatric illness (eg depression, anxiety, panic attacks) ?	No (1)	Yes (2)
39. How many pregnancies did you have ? _____		
40. Are your parents still alive ?		
Mother	No (1)	Yes (2)
Father	No (1)	Yes (2)
41. If your mother has died, what were the cause of death ? _____		
42. If your father has died, what were the cause of death ? _____		
43. Do you have other sources of income other than your job?	No (1)	Yes (2)
44. If yes, please name the source of income: _____		
45. Give an indication of your income per month (mark the correct block with a X):		
R 1 000 – R 2 0000		(1)
R 2 000 – R 3 000		(2)
R 3 000 – R 4 000		(3)
R 4 000 – R 5 000		(4)
> R 5 000		(5)
46. Does your work offer any benefits ?	No (1)	Yes (2)
47. If yes, please mark the benefits:		
Pension		(1)
Medical aid		(2)
Housing		(3)
Car		(4)
Allowance for car / housing / medical aid		(5)
Food (please list the foods)		(6)

48. Do you own property ?	No (1)	Yes (2)

49. If yes, what type of property, eg house, flat ? _____

50. How many people live in your house (give the number of people):	Number
Children under 11 years	(1)
Children under 18 years	(2)
Adults (children older than 18 years,grand parents, brothers, sisters, wives, husbands, etc)	(3)

51. Do they contribute to your household costs?	No (1)	Yes (2)
52. In what way: food / money	Food (1)	Money (2)

53. If money, how much money? _____

54. Please name the members of your household:

Member	*Age*	*Education*	*Present job*
(1)	(2)	(3)	(4)
(5)	(6)	(7)	(8)
(9)	(10)	(11)	(12)
(13)	(14)	(15)	(16)
(17)	(18)	(19)	(20)
(21)	(22)	(23)	(24)
(25)	(26)	(27)	(28)
(29)	(30)	(31)	(32)
(33)	(34)	(35)	(36)

55. In what type of area do you live (please tick the appropriate block with a X):

Township	(1)
Squatter camp	(2)
Traditional White area	(3)
Traditional Indian area	(4)
Other	(5)
Specify other	(6)

56. How long have you been living here (years)? _____		
57. Where have you been living before:		
Township		(1)
Squatter camp		(2)
Traditional white area		(3)
Traditional Indian area		(4)
Other		(5)
Specify other		(6)
58. What type of house do you live in:		
Brick house		(1)
Informal housing (eg. shack / mokuku)		(2)
Other		(3)
Specify other		(4)
59. Do your stay away from your family when you work and go home only on weekends or holidays ?	No (1)	Yes (2)
60. If yes, where do you stay during when you work ? _____		
61. Do you smoke ?	No (1)	Yes (2)
If yes, mark what you smoke and indicate the amount per day / week	Type of smoking	Amount per day / week
62. Cigarettes	(1)	(2)
63. Cigars	(1)	(2)
64. Tobacco (zoll)	(1)	(2)
65. Snuff	(1)	(2)
66. Pipe	(1)	(2)
67. Other, please specify: _____	(1)	(2)
68. For how long are you smoking (years) ? _____		
69. If you don't smoke at the moment, have you been smoking regularly before ?	No (1)	Yes (2)
70. If yes, for how long have you been smoking before ? _____		
71. Do you use alcohol ?	No (1)	Yes (2)

72. If yes, mark the type of alcohol you use:	
Traditional beer (homemade)	(1)
Tlokwe	(2)
Beer (commercial)	(3)
Spirits	(4)
Wine	(5)
Liqueur	(6)

Try to tell the amount of alcohol you use per day / per week:		Per day	Per week
73. Traditional beer (home made)	glass	(1)	(2)
74. Tlokwe	box	(1)	(2)
75. Beer (commercial)	quart / tin / dumpy	(1)	(2)
76. Spirits	tot / bottle	(1)	(2)
77. Wine	glass / bottle	(1)	(2)
78. Liqueur	glass / bottle	(1)	(2)

79. In my *childhood* (< 18 y) there were times when we didn't have enough food in the house:

	Never (1)	Seldom (2)	Sometimes (3)	Often (4)	Always (5)

80. In my *adulthood* (> 18 y) there were times when we didn't have enough food in the house:

	Never (1)	Seldom (2)	Sometimes (3)	Often (4)	Always (5)

ADDENDUM 5

DIETARY QUESTIONNAIRE

FOOD FREQUENCY QUESTIONNAIRE

DIETARY QUESTIONNAIRE

All information given in this questionnaire is confidential

1. Subject number : _____ 2. Date: _____
3. Interviewer: _____

4. What are your favourite foods ? _____
5. What foods do you dislike ? _____

Do you *usually* eat :

Breakfast (6)	No (1)	Yes (2)
Lunch (7)	No (1)	Yes (2)
Supper (8)	No (1)	Yes (2)
Snacks / food between meals (9)	No (1)	Yes (2)

10. Do you usually use margarine / oil / fat in food preparation ?

NO (1)	YES (2)

11. Are there any foods to which you are allergic or intolerant (foods that make you ill) to, eg milk or peanuts ?

NO (1)	YES (2)

12. If YES, please specify type of food: _____

13. Please indicate *how often you use the following food types.* (One choice per type of food is sufficient):

	Times per day	Times per week	Times per month	Seldom/ never
Dining hall food, prepared by a food service				
Cafeteria / canteen food				
Take away food *				
Home prepared food, prepared by myself				
Home prepared food, prepared by someone else				

* *Take away foods* are any prepared food bought at a cafeteria, café, kiosk, fast food restaurant, eg Wimpy / Chicken Licken / Kentucky or food stand.)

14. What type of take away food do you usually buy (eg hamburger, chips, fish, meat pie, pap and meat, pizza)?

_____

15. How do your eating pattern differ during week ends ? _____

_____

PART II FOOD FREQUENCY QUESTIONNAIRE

INSTRUCTIONS: Circle the subject's answer. Fill in the amount and times eaten in the appropriate columns.

SUBJECT NO: .....

I shall now ask you about the type and the amount of food you have been eating in the last few months. Please tell if you eat the food, how much you eat and how often you eat it. We shall start with maize meal porridge.

Do you eat maize meal porridge? YES 1 NO 2
If YES, what type do you have at home now?

Brand name: .....
Don't know 2
Grind self 3

If brand name given, do you usually use this brand?
YES 1 NO 2 DON'T KNOW 3
Where do you get your maize meal from? (May answer more than one)

Shop	1
Employer	2
Harvest and grind self	3
Other – specify	4
Don't know	5

FOR OFFICE USE

FOOD	DESCRIPTION	Amount	TIMES EATEN				CODE	AMOUNT/ DAY
			Per day	Per week	Per month	Seldom Never		
Maize meal porridge	Stiff ('pap')						e4225 4250	
Maize meal porridge	Soft ('slap pap')						e4225 4250	
Do you pour milk on your soft porridge? YES 1 NO 2 If YES, what type of milk (whole fresh, sour, 2 %, fat free, milk blend)? ..... INSTRUCTION: Show subject examples.								
If YES, how much milk?								
Do you pour sugar on your soft porridge? YES 1 NO 2								
If YES, how much sugar?							9012	
Maize meal porridge	Crumbly (phutu)						e4225 4250	
Ting								
Mabella Coarse Fine Rice	Stiff						4082	
Mabella Coarse Fine Rice	Soft						4082	

FOOD	DESCRIPTION	Amount	TIMES EATEN				CODE	AMOUNT/ DAY
			Per day	Per week	Per month	Seldom Never		
Do you pour milk on your mabella porridge? YES 1 NO 2 If YES, what type of milk (whole fresh, sour, 2 %, fat free, milk blend)? ..... INSTRUCTION: Show subject examples.								
If YES, how much milk?								
Do you pour sugar on your mabella? YES 1 NO 2								
If YES, how much sugar?							9012	
Oats							4032	
Do you pour milk on your oats? YES 1 NO 2 If YES, what type of milk (whole fresh, sour, 2 %, fat free, milk blend)? ..... INSTRUCTION: Show subject examples.								
If YES, how much milk?								
Do you pour sugar on your oats? YES 1 NO 2								
If YES, how much sugar?							9012	
Breakfast cereals	Brand names of cereals at home now: Don't know						4036	
Do you pour milk on your cereal? YES 1 NO 2 If YES, what type of milk (whole fresh, sour, 2 %, fat free, milk blend)? ..... INSTRUCTION: Show subject examples.								
If YES, how much milk?								
Do you pour sugar on your cereal? YES 1 NO 2								
If YES, how much sugar?							9012	
Samp	Bought Self ground with fat without fat						4043	
Samp and beans							A014	
	Are the amounts of samp and beans the same as in the picture? YES NO If NO, do you use more beans than in the picture or less? MORE LESS							
Samp and peanuts							A013	
	Are the amount of samp and peanuts the same as in the picture? YES NO If NO, do you use more peanuts than in the picture or less? MORE LESS							
Rice	White Brown Maize rice						4040 4134 4043	
Pastas	Macaroni Spaghetti Other						4062	

FOOD	DESCRIPTION	Amount	TIMES EATEN				CODE	AMOUNT/ DAY
			Per day	Per week	Per month	Seldom Never		
You are being very helpful. Can I now ask you about meat? CHICKEN, MEAT, FISH How many times per day/week do you eat meat, fish or chicken? .....X/day .....X/week								
Chicken:	Boiled, nothing added						1521	
	Fried: in butter/crumbs Not coated						1634 1520	
	Roasted, grilled						1520	
	Stewed						1520	
	What vegetables are in the stew?							
	Don't know							
Do you eat chicken skin? ALWAYS 1 SOMETIMES 2 NEVER 3								
Chicken bones stew							A003	
Chicken feet	How do you cook it?						A004 1609	
Chicken offal	How do you cook it?						1610	
Where do you get your MEAT from? (May answer more than 1) Shop, supermarket, spaza Employer Slaughter own Gift Other specify: Do not eat red meat								1 2 3 4 5 6
Red meat:	How do you like meat? With fat Fat trimmed							
Beef	Fried – with bone							
	Fried – without bone							
	Stewed – with bone						A001	
	Stewed – without bone						A001	
	Grilled – with bone							
	Grilled – without bone							
	Minced						1585	
Mutton	Fried – with bone						1522	
	Fried – without bone						1571	
	Stewed – with bone						1511	
	Stewed – without bone						1511	
	Grilled – with bone							
	Grilled – without bone							
	Minced						1662	

FOOD	DESCRIPTION	Amount	TIMES EATEN				CODE	AMOUNT/ DAY
			Per day	Per week	Per month	Seldom Never		
Pork	Fried – with bone							
	Fried – without bone							
	Stewed – with bone							
	Stewed – without bone							
	Grilled – with bone							
	Grilled – without bone							
Beef Offal	Intestines: boiled, nothing added						161	
	Stewed with vegetables							
	Tripe						1546	
	Heart						1565	
	Lungs							
	Liver						1515	
	Kidneys						1518	
	Other specify:							
What vegetables are usually put into meat stews?								
Wors sausage	Fried Grilled						1526	
Bacon							1501	
Cold meats	Polony						1514	
	Ham						1564	
	Viennas						1531	
	Other specify:							
Canned meat	Bully beef						1535	
	Other specify:							
Meat pie	Home made Bought						1548	
Hamburger	Home made Bought						A015	
Dried beans, peas, lentils (10)	How do you prepare them?							
Soya products e.g. Toppers	Brands at home now Don't know..... Show examples						3527	
Pilchards in tomato chilli brine	Whole						2557	
	Mashed with fried onion						A005	
Fried fish	With batter/ crumbs						2523	
	Without batter/crumbs						2509	
Other canned fish	Tuna							
	Pickled fish Other:						2562	
Fish cakes	Home made (describe) Frozen Bought						2531	

FOOD	DESCRIPTION	Amount	TIMES EATEN				CODE	AMOUNT/ DAY
			Per day	Per week	Per month	Seldom Never		
Eggs	Boiled poached Scrambled Fried						1001 1025 1003	
WE NOW COME TO VEGETABLES AND FRUIT How many times per day/week do you eat vegetables? .....X/day .....X/week								
Cabbage	How do you cook cabbage?							
	Boiled, nothing added							8066
	Boiled with potato and onion and fat							A006
	Fried, nothing added							A007
	Boiled, then fried with potato, onion							A006
	Other:							
	Don't know							
Spinach / morogo / other green leafy	How do you cook spinach?							
	Boiled, nothing added						8071	
	Boiled fat added						8209	
	Boiled with – onion, tomato & fat						A011	
	-onion, tomato & potato						8212	
	- with peanuts							
	Other:							
	Don't know							
Tomato and onion 'gravy'	Home made - with fat - without fat						 A012 A016	
	Canned (Is this the amount of pap you eat? How much more or less?)						8221	
Pumpkin	How do you cook pumpkin?							
	Cooked in fat & sugar						A010	
	Boiled, little sugar and fat						A009	
	Other:							
	Don't know							

FOOD	DESCRIPTION	Amount	TIMES EATEN				CODE	AMOUNT/ DAY
			Per day	Per week	Per month	Seldom Never		
Carrots	How do you cook carrots?							
	Boiled, sugar & fat						8129	
	With potato/ onion						A008	
	Raw, salad Chakalaka						8015 A025	
	Other:							
	Don't know							
Mealies / Sweet corn	How do you eat mealies? On cob –with fat -without fat						8033	
	Off cob –with fat -without fat						8261	
Beetroot salad	Home made Bought						8005	
Potatoes	How do you cook potatoes?							
	Boiled/baked - with skin						8046	
	- without skin						8045	
	Mashed						8187	
	Roasted						8189	
	French fries						8048	
	Salad						8236	
	Other:							
Sweet potatoes	How do you cook sweet potatoes?							
	Boiled/baked - with skin						8057	
	- without skin						8214	
	Mashed						8058	
	Other:							
	Don/t know							
Salad vegetables	Raw tomato						8059	
	Lettuce						8031	
	Cucumber						8025	
Other vegetables specify:								
FRUIT: Do you like fruit? YES NO How many times per day/week do you eat fruit? .....x/day .....x/week								

FOOD	DESCRIPTION	Amount	TIMES EATEN				CODE	AMOUNT/ DAY
			Per day	Per week	Per month	Seldom Never		
Apples/Pears	Fresh						7001	
Pears	Fresh Canned						7053 7054	
Bananas							7009	
Oranges / naartjies							7031	
Grapes							7020	
Peaches	Fresh Canned						7036 7038	
Apricots	Fresh Canned						7003 7004	
Mangoes	Fresh						7026	
Guavas	Fresh Canned						7021 7023	
If subject eats canned fruit: Do you have custard with canned fruit? YES 1 NO 2								
Custard	Home made Ultramel						0004	
Wild fruit / berries	Stamvrugte Noen-noem Klappers Maroelas Nastergals Other – specify						7070	
Dried fruit:	Types:							
Other fruit:								
BREAD AND BREAD SPREADS								
Bread Bread rolls	White						4001	
	Brown						4002	
	Whole wheat						4003	
Do you spread anything on the bread? ALWAYS 1 SOMETIMES 2 NEVER 3 If YES, what do you spread?								
Margarine	What brand do you have at home now? ..... Don't know Show examples						6508 6521	
Butter	What brand do you have at home now? ..... Home made Don't know						6502	

FOOD	DESCRIPTION	Amount	TIMES EATEN				CODE	AMOUNT/ DAY
			Per day	Per week	Per month	Seldom Never		
Peanut butter							6509	
Jam/syrup/ honey							9008	
Marmite/Fray Bentos etc.							9501	
Fish/meat paste							1512	
Cheese	Type:						0010	
Atchar							3004	
Polony							1514	
Other spreads: specify								
Dumpling							4001	
Vetkoek							4057	
Provita, crackers etc.								
FATS: What fats do you use and where do you use them?								
Margarine	Where used: on bread							
	with vegetables** Number of spoons ..... /number in family							
Butter	on bread with vegetables** Number of spoons ..... /number in family							
Holsum / vegetable fat	Where used: Number of spoons ..... /number in family						6508	
Oil	Where used: Number of spoons ..... /number in family						6510	
Dripping	Where used: Number of spoons ..... /number in family							
Mixed fat (makhuru)	Where used: Number of spoons ..... /number in family							

FOOD	DESCRIPTION	Amount	TIMES EATEN				CODE	AMOUNT/ DAY
			Per day	Per week	Per month	Seldom Never		
Lard	Where used: Number of spoons ..... /number in family						6520	
Mayonnaise/ salad dressing	Number of spoons ..... /number in family						6573	
Cream	Fresh/Long life /canned Orley whip						6503	
DRINKS:								
Tea							9514	
Sugar/cup tea							9012	
Milk / cup tea	What type of milk do you use in tea?							
	Fresh / long life whole						0006	
	Fresh / long life 2%							
	Fresh / long life fat free						0072	
	Whole milk powder Brand						0009	
	Skimmed milk powder Brand						0008	
	Milk blend Brand						0068	
	Whitener Brand						0039	
	Condensed milk						0002	
	Evaporated milk						0003	
	None							
Coffee								
Sugar / cup coffee							9012	
Milk / cup coffee	What type of milk do you use in coffee?							
	Fresh / long life whole						0006	
	Fresh / long life 2 %							
	Fresh / long life fat free						0072	
	Whole milk powder Brand						0009	
	Skimmed milk powder Brand						0008	
	Milk blend Brand						0068	
	Whitener Brand						0039	

FOOD	DESCRIPTION	Amount	TIMES EATEN				CODE	AMOUNT/ DAY
			Per day	Per week	Per month	Seldom Never		
	Condensed milk						0002	
	Evaporated milk						0003	
	None							
Milk as such	What type of milk do you drink as such?							
	Fresh / long life whole						0006	
	Fresh / long life 2 %							
	Fresh / long life fat free						0072	
	Sour / Maas						0006	
	Buttermilk						0001	
	Whole milk powder Brand						0006	
	Skimmed milk powder Brand						0072	
	Milk blend Brand						0068	
Milk drinks Brand	Nestle Milo Other						0023	
Yoghurt	Drinking yoghurt Thick yoghurt						0044 0020	
Squash	Sweeto SixO Oros/Lecol - with sugar - artificial sweetner Kool Aid Other						9013 9013 9002 9013 9002	
Fruit juice	Fresh/Liquifruit/Ceres Tropica Concentrates e.g. Halls Nectars Flavour							
Fizzy drinks Coke, Fanta	Sweetened Diet						9001 9013	
Mageu/Motog o							9562	
Home brew							9516	
Tlokwe							9516	
Beer							9506	
Spirits							9510	
Wine red							9508	
Wine white							9518	
Liqueur							9517	
Other: specify								

FOOD	DESCRIPTION	Amount	TIMES EATEN				CODE	AMOUNT/ DAY
			Per day	Per week	Per month	Seldom Never		
SNACKS AND SWEETS:								
Potato crisps							4275	
Cheese curls Niknaks etc.							4067	
Peanuts	Raw Roasted						6001 6007	
Raisins							7022	
Peanuts and raisins								
Chocolates	Name						9024	
Candies	Sugars, gums, hard sweets						9009	
Sweets	Toffees, fudge, caramels						9014	
Biscuits	Type							
Cakes & tarts	Type							
Scones							4029	
Rusks							4160	
Savouries	Sausage rolls Samoosas Biscuits e.g. Bacon kips Other						1534 4196 4162	
PUDDINGS:								
Canned fruit	Type							
Jelly							9004	
Custard	Homemade Ultramel						0004	
Baked pudding							4181	
Instant pudding							4066	
Ice cream Sorbet							6507 6516	
Other: specify								
SAUCES / GRAVIES / CONDIMENTS:								
Atchar							3004	
Tomato sauce Worcester sauce							3027	
Chutney							9524	
Pickles							8176	
Packet soups							3046	
Others:								
INSECTS:								
Locusts								
Mopani worms								
Others:								

FOOD	DESCRIPTION	Amount	TIMES EATEN				CODE	AMOUNT/ DAY
			Per day	Per week	Per month	Seldom Never		
WILD BIRDS OR ANIMALS (hunted in rural areas or on farms)								
MISCELLANEOUS: Please mention any other foods used more than once/two weeks which we have not talked about:								

Use of salt

Vitamin and mineral supplements

1. What type of salt do you use? Fine=1, coarse=2			1	2
2. Do you add salt to food during cooking?		Always=1	Sometimes=2	Never=3
3. Do you add salt to food at the table?		Always=1	Sometimes=2	Never=3
4. Do you eat salty foods (chips/salted peanuts)?		Often=1	Sometimes=2	Never=3

5. Do you take any vitamin tablets or syrup other than those supplied by the clinic?	
If yes, specify:	

ADDENDUM 6

OBESITY ATTITUDE SCALE

OBESITY KNOWLEDGE SCALE

WEIGHT-RELATED BEHAVIOUR QUESTIONNAIRE

EMOTION-INDUCED EATING SCALE

Obesity attitude scale

In this questionnaire you'll find statements on issues regarding health, beauty and weight.

All information given in this questionnaire is confidential

1. Subject number: _____ 2. Date: _____
3. Interviewer: _____

Please mark the correct block with a X, indicating whether you agree, disagree or are uncertain, about the following statements.

STATEMENT	Agree (1)	Uncertain (2)	Disagree (3)
4. Fat people have more friends			
5. Children do not like their mothers to be fat			
6. The clothes of fat people do not fit well			
7. Fat people cannot work hard			
8. Fat people are people who eat too much			
9. Thin people are sick people			
10. Thin women get jobs easier			
11. Foods for reducing diets are cheaper			
12. Thin people can wear more fashionable clothes			
13. Thin women are beautiful			
14. When one eats less to lose weight, one feels hungry all day			
15. Men prefer fat women			
16. Foods for reducing diets are tasty			
17. Fat people feel more unhappy			
18. People who eat healthy foods, are thin			
19. If one exercises daily, one feels healthy			
20. When one eats less to lose weight, one always wants to eat something tasty			
21. It is difficult to lose weight			
22. Fat women are well-cared for by their husbands			
23. If one loses weight, one feels proud			
24. I enjoy bodily exercise			
25. If one loses weight, one looks unattractive with loose skin			

Obesity knowledge scale

In this questionnaire you'll find statements on issues regarding health and weight.

All information given in this questionnaire is confidential

1. Subject number: _____ 2. Date: _____

3. Interviewer: _____

Please mark the correct block with a X, choosing 'true', 'false' or 'don't know' for each of the following statements.

STATEMENT	True (1)	False (2)	Don't know (3)
4. If one eats in the evening, one is likely to get fatter (1)			
5. Fat on meat does not make one fatter (2)			
6. Lots of sugar in tea make one fatter (3)			
7. Lots of oil and fat in food make one fatter (4)			
8. When one drinks a lot of water, one eats less (5)			
9. Thin people get more sugar diabetes than fat people (6)			
10. More fat people than thin people suffer from high blood Pressure			
11. Thin people get tired easier than fat people			
12. Fat people with high blood pressure will be more healthy when they lose weight			
13. When one eats a lot of fat in food, one feels well (comfortable)			
14. Fat people with sugar diabetes get more healthy when they lose weight			
15. Thin women do not get pregnant easily			
16. One can eat a lot of raw vegetables with each meal and one will not get fatter			
17. One can eat a large portion of fatty meat with each meal and one will not get fatter			
18. Dry beans contain more fat than meat			
19. Coffee creamer (Cremora) contains less fat than skim milk powder			
20. One can buy pills to make one eat less food			
21. Most of the fat in chicken is in the white flesh			
22. Dry beans can be taken instead of meat as a building food			

Weight-related Behaviour questionnaire

In this questionnaire you'll find statements on issues regarding your weight.

All information given in the questionnaire is confidential

1. Subject number: _____ 2. Date: _____

3. Interviewer: _____

Please answer the following questions by indicating your choice with a X in the appropriate block:

4. When plenty of food is available, I eat as much as possible					
	Never (1)	Seldom (2)	Sometimes (3)	Often (4)	Always (5)
5. I feel guilty after I have eaten too much					
	Never (1)	Seldom (2)	Sometimes (3)	Often (4)	Always (5)
6. I try to make myself feel better by eating or drinking in a stressful situation					
	Never (1)	Seldom (2)	Sometimes (3)	Often (4)	Always (5)
7. I think I should lose weight				No (1)	Yes (2)
8. I have been on a diet for weight loss					
	Never (1)	Seldom (2)	Sometimes (3)	Often (4)	Always (5)
9. I am trying to lose weight at the moment				No (1)	Yes (2)
10. If you are trying to lose weight at the moment, please indicate by which method(s): Please mark the correct block(s) with a X (more than one choice is acceptable):					
Eating less				(1)	
Exercise				(2)	
Not snacking between meals				(3)	
Skipping meals				(4)	
Fasting (stay without foods for a long period)				(5)	
Diet plan for weight loss				(6)	
Other				(7)	
Please specify other: _____ (8)					
11. I eat a lot of food at once and then I feel uncomfortabe					
	Never (1)	Seldom (2)	Sometimes (3)	Often (4)	Always (5)
12. I eat a large amount of food in a short period of time, associated with a feeling that the eating gets out of Control					
	Never (1)	Seldom (2)	Sometimes (3)	Often (4)	Always (5)

13. Please indicate how often you weigh yourself :

Daily (1)	1 - 6 x per week (2)	1 x per 2 weeks (3)	1 x per month (4)	1 x per 2-3 months (5)	1 x per 4-6 months (6)	< 1 x per 6 months (7)	Never (8)

14. Please indicate your weight and / or dress size at your different ages (NOT DURING PREGNANCY): (Mark the correct blocks with a X)

Age	*Weight (kg)*	*Dress size*	*Don't know*	*For office use only:*
18				1 = constant
20				2 = increase
25				3 = decrease
30				4 = varies, ending larger
35				5 = varies, ending smaller
40				
45				
50				

15. If you have gained weight (clothing size became larger), what do you think the reason could be for that ? _____

16. During the last 6 months, my clothes began to fit: (more than one choice acceptable)

Looser (1)	More tight (2)	Fit the same (3)

17. Please complete the following table by indicating your most comfortable clothing size during 2002 in the beginning of the year, winter, spring and end of the year. Mark the correct block with a X:

Clothing size	*Beginning of 2002*	*Winter*	*Spring*	*End of 2002*	*For office use only:*
32 (8)					Please include q17 when processing
34 (10)					
36 (12)					5 = decrease
38 (14)					6 = increase
40 (16)					7 = decrease
42 (18)					8 = cycling, ending with the same weight
44 (20)					
46 (22)					9 = cycling, ending with a higher weight
48 (24)					
50 (26)					10 = cycling, ending with a lower weight
52 (28)					

18. What are your most comfortable clothing size at the moment ? _____

EMOTION-INDUCED EATING SCALE

Please mark the correct block with a X, choosing 'neer or almost never', 'sometimes' or 'usually or always' for each of the following statements.

Statement	*Never or almost never (1)*	*Sometimes (2)*	*Usually or always (3)*
1. When I am worried I eat more			
2. I eat when I am mad			
3. When I do something well I give myself a treat (food)			
4. When I am sad I eat more			
5. When I am happy I eat more			
6. When I am bored I eat more			
7. I eat between meals even when I am not hungry			

ADDENDUM 7

PHYSICAL ACTIVITY QUESTIONNAIRE

Physical activity questionnaire

Subject number: ..... Date: ..... Interviewer: .....

	SECTION 9A: PHYSICAL ACTIVITY (World Health Organisation STEPwise Programme. Global Physical Activity Questionnaire (GPAQ)) *(Now I would like to ask you a few questions about your diet and other habits.)*		
	Occupation-related Physical Activity (paid or unpaid work): *When answering the following questions, think back over the past year and consider a usual week:*		
43A	Do you work mostly in the household?	YES ..... NO	➢46
43B	Does your work involve mostly sitting or standing still or walking for short periods (less than 10 minutes at a time)?	YES ..... NO	
44A	How many days during one week, if any, does your work involve **vigorous activities** like heavy lifting, digging, or heavy constructions work, chopping wood, ploughing, cutting sugar cane, carrying heavy wood (forestry), etc.?	DAYS_____ If "0 days" ➢45A	
44B	On an average day, how much time **during work** do you spend doing vigorous activity for at least 10 minutes at a time?	_____ HOURS _____ MINUTES	
45A	How many days during one week, if any, does your work involve **moderate activities** like brisk walking, carrying light loads, carrying water, scrubbing floors or hanging washing, sweeping yard (outdoors), planting or harvesting crops such as maize, tending animals, walking carrying load on head, gathering and breaking kindling, picking fruit?	DAYS_____ If "0 days" ➢46	
45B	On an average day, how much time **during work** do you spend doing moderate activity for at least 10 minutes at a time?	_____ HOURS _____ MINUTES	
	Travel-related Physical Activity: *Other than activities that you've already mentioned, I would like to ask you about the way you travel to and from places (work, market, church, etc).*		
46	How many days during one week, if any, do you walk or use a bicycle for travel for at least 10 minutes at a time?	DAYS_____ If "0 days" ➢48A	
47	On an average day, how much time do you spend walking or using a bicycle for travel	_____ HOURS _____ MINUTES	
	Non-work related and leisure time Physical Activity: *I would like to ask you about activities other than you've already mentioned, such as recreation, sport and leisure time activities.*		
48A	Does your non-work or leisure time involve mostly sitting, standing or walking for less than 10 minutes at a time?	YES ..... NO	➢..
48B	How many days during one week, if any, do you do vigorous activities like heavy lifting, digging, or strenuous sports, as part of your non-work or leisure time for at least 10 minutes at a time?	DAYS_____ If "0 days" ➢49A	
48C	On an average day, how much time do you spend doing vigorous activity for at least 10 minutes at a time?	_____ HOURS _____ MINUTES	
49A	How many days during one week, if any, do you do moderate activities like brisk walking, bicycling or swimming, as part of your non-work or leisure time?	DAYS_____ If "0 days" ➢50	
49B	On an average day, how much time do you spend doing moderate activity for at least 10 minutes at a time?	_____ HOURS _____ MINUTES	
	Sitting / Resting Activity: *I would like to ask you about the time that you spent sitting or resting, not including sleeping, **in the past 7 days**. This may include time sitting at a desk, visiting friends, reading, or sitting down to watch television.*		
50.	On an average day, how much time do you spend sitting or resting?	_____ HOURS _____ MINUTES	