CHANGES IN DIETARY RISK FACTORS OF COLON CANCER IN AFRICANS DURING URBANISATION

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TO ALL PATIENTS SUFFERING FROM CANCER
In loving memory of my Grandmother and Grandfather

" Your word is a lamp to guide me and a light for my path "

Psalms 119:105

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SUMMARY

Background

The prevalence and incidence rates of colorectal cancer are significant lower in black South Africans than those in white, coloured or Indian South Africans. The reason for this phenomenon is not known, but it is suspected that blacks may have a genetic protection against development of this cancer and/or that environmental and lifestyle-related risk factors for colorectal cancer in blacks have not reached levels where the disease is "precipitated" in genetically susceptible individuals.

Objectives

The objectives of this study were to review the literature on prevalence and incidence as well as risk factors for colorectal cancer, to measure the impact of urbanisation on changes in the diet-related risk factors in apparently healthy Africans, and to develop a hypothesis that may explain the low rates in Africans.

Setting

The Northwest Province of South Africa and the Nutrition Laboratory of the Department of Nutrition and Family Ecology of the Potchefstroom University for Christian Higher Education.

Methods

Study design and subjects:

The study was a cross-sectional epidemiological survey of 889 apparently healthy African volunteers recruited from rural and urban parts of the Province. The study formed part of the THUSA project (**T**ransition and **H**ealth during **U**rbanisation of **S**outh **A**fricans). The subjects, aged 15 to 65 years, were stratified into gender and 10-year interval age groups, as well as into 5 strata based on level of urbanisation: stratum 1 were subjects from deep rural areas; stratum 2 those living on farms; stratum 3 were living in squatter camps, and were mainly urban subjects which recently moved from rural areas; stratum 4 represented those urban subjects living in established

townships while stratum 5 consisted of professional urban dwellers such as doctors, nurses and teachers.

Methodology

Demographic and dietary information, as well as reproductive habits from women and bowel habits from selected subjects, were obtained during individual interview using validated and standardised questionnaires. Anthropometric measurements, clinical signs of malnutrition, oral temperature, blood pressure and two-hour glucose tolerance tests were performed by trained researchers and fieldworkers. Blood samples were collected and serum, citrated and EDTA plasma prepared within 3 hours and stored in aliquots, first at –20°C and later at –84°C, for later analyses of biochemical variables with standardised methods. Haematocrit and haemoglobin concentrations on EDTA blood were determined immediately, using a haematocrit centrifuge and capillary tubes and the method of Boehringer Mannheim respectively. All data were computerised and descriptive statistics, analyses of variance and Pearson correlation coefficients determined with the SAS® package by the Statistical Consultation Services of the PU vir CHO.

Results

The results clearly illustrated an effect of urbanisation on accepted risk factors for chronic diseases of lifestyle. For example, progressive increases in diastolic blood pressure and total serum cholesterol were observed. The results further indicated that although reported energy intakes did not increase with urbanisation, the sources of energy changed. Total protein intakes were adequate but urban subjects ate more animal protein foods and less plant proteins than rural subjects, mainly because of an increase in meat and chicken intake. The exception was egg intake. Except for the farm workers who reported very low egg intakes, mean intakes of men were approximately 30g per day and women 20g/day. No effect of urbanisation was seen. The percentage of energy provided by fat progressively increased across strata while the percentage of energy derived from carbohydrates decreased. Subjects in stratum 5 already took in >30% of their energy as fat

but still have relatively high carbohydrate intakes (>50% of energy). Dietary fibre intakes were low in all strata (mean intakes from 10.9 to 24.2g/day) and remained low with urbanisation. Sucrose intakes were also low (30-36g/day) and did not increase with urbanisation. Mean starch intakes decreased across strata from 286 to 224g/day in men and 238 to 193g/day in women. Urban men and women also ate more meat, chicken, fruit and vegetables. This resulted in higher intakes of micronutrients such as vitamins E, C, folate and iron by urban subjects. The exception was higher calcium intakes by rural subjects (farm workers).

Alcohol intakes were generally high in men. Dwellers of squatter camps reported the highest intakes. Patterns of reported intakes correlated with serum gamma glutamyl transferase values.

In addition to these diet-related risk factors for colorectal cancer, other risk factors such as BMI and adult height increased with urbanisation. Physical activity seemed not to change with urbanisation, while smoking habit actually declined. However, the number of young people smoking was high.

Discussion

Of the 13 accepted, probable or possible risk factors for colorectal cancer considered, eight in men and seven in women were related to low risk, while four in men and five in women were related to high risk. On the available information it was not possible to judge the effects or contribution of physical activity to risk.

Of the factors associated with low risk, six: red meat, starch and fat (total and saturated) intake, BMI in women, as well as adult height, changed with urbanisation in a direction to increase risk. However, of these six, three (red meat and starch intake and adult height), although changed, were still under cut-point levels where they present increased risk.

Of the five factors associated with an increased risk of colorectal cancer (low vegetable intake, smoking habit, high alcohol consumption by men, low dietary fibre intakes, and high BMI in women) three actually improved with

urbanisation, possibly conferring a lower risk. These were increases in vegetable and fibre intake and a decrease in smoking habits of urban men, especially those in stratum 5.

Because the contribution of a particular risk factor to total risk of colorectal cancer is not known, it is not possible to judge whether these changes will result in a lower overall risk. However, it is clear that with urbanisation, detrimental effects in some risk factors are not large enough to reach levels where they operate as risk factors, while in some instances, beneficial changes were observed in factors associated with high risk, possibly resulting in a lowering of risk.

Conclusion

In conclusion it is hypothesised that at this stage of the lifestyle and nutrition transition that black South Africans are experiencing during urbanisation, the effects on the risk factors for colorectal cancer are such that some factors associated with high risk in rural subjects are improved while detrimental effects on some factors associated with a low risk are not large enough to "push" these factors over a level where they will be associated with a high risk.

On balance it seems that at the present stage of urbanisation, changes in lifestyles and eating patterns generally are associated with lower risk of colorectal cancer. This is in contrast to some of the other chronic diseases of lifestyle such as hypertension and diabetes mellitus which already have emerged as a serious public health problem. It is possible that with further changes, especially further increase in meat and fat intake, the risk factor situation may change to such an extent that risk for colorectal cancer will increase. It is recommended that preventative measures such as promotion of a prudent, cancer-preventing diet should be targeted at black South Africans in transition.

OPSOMMING

Agtergrond

Die voorkoms en frekwensiesyfers van kolorektale kanker is betekenisvol laer in swart Suid-Afrikaners as in blanke, Kleurling of Indiese Suid-Afrikaners. Die rede vir hierdie verskynsel is onbekend, maar daar word vermoed dat swartes 'n genetiese beskerming teen die ontwikkeling van hierdie kanker het en/of dat omgewing- en lewenstylverwante risikofaktore vir kolorektale kanker in swartes nie die vlakke bereik het waar die siekte in genetiesvatbare individue "gepresipiteer" word nie.

Doelwitte

Die doelwitte van hierdie studie was om die literatuur oor die voorkoms en frekwensie asook die risikofaktore vir kolorektale kanker te hersien, om die impak van verstedeliking op veranderinge in die dieetverwante risikofaktore in oënskynlik gesonde swartes te bepaal en om 'n hipotese wat die lae voorkoms in swartes mag verklaar, te ontwikkel.

Opset

Die Noordwes-provinsie van Suid-Afrika en die Voedinglaboratorium van Departement Voeding en Gesinsekologie van die Potchefstroomse Universiteit vir Christelike Hoër Onderwys.

Metodes

Studie-ontwerp en proefpersone

Die studie was 'n deursnee epidemiologiese ondersoek van 889 oënskynlik gesonde swart vrywilligers gewerf uit landelike en stedelike dele van die Provinsie. Die studie het deel gevorm van die THUSA-projek (**T**ransition and **H**ealth during **U**rbanisation of **S**outh **A**fricans). Die proefpersone, 15 tot 65 jaar oud, is gestratifiseer in geslag- en 10-jaarinterval ouderdomsgroepe, asook in vyf strata gebaseer op vlak van verstedeliking: stratum 1 was proefpersone van diep landelike areas; stratum 2 die wat op plase woon;

stratum 3 het in plakkerskampe gewoon en was hoofsaaklik stedelike proefpersone wat onlangs vanaf landelike gebiede getrek het; stratum 4 het daardie stedelike proefpersone verteenwoordig wat gevestigde inwoners soos dokters, verpleegsters en onderwysers was.

Metodologie

Demografiese en dieetinligting, sowel as reproduktiewe gewoontes van vroue en ontlasgewoontes van geselekteerde proefpersone is gedurende individuele onderhoude verkry deur gebruik te maak van gevalideerde en gestandardiseerde vraelyste. Antropometriese afmetings, kliniese tekens van wanvoeding, liggaamstemperatuur, bloeddruk en twee-uurlikse glukoseverdraagsaamheidstoetse is deur opgeleide navorsers en veldwerkers uitgevoer. Bloedmonsters is versamel en serum, sitraat- en EDTA-plasma is binne drie ure berei en in deelvolumes by-20°C en later by -84°C opgeberg, vir latere ontledings van biochemiese veranderlikes met gestandardiseerde metodes. Hematokrit- en hemoglobienkonsentrasies van EDTA-bloed is onmiddelik bepaal deur gebruik te maak van 'n hematokritsentrifuge en kapillêre buisie en die metodes van Boehringer Mannheim respektiewelik. Alle data is gerekenariseer en beskrywende statistiek, variansieanalise en Pearsonkorrelasiekoëffisiënt met die SAS®-pakket deur die Statistiese Konsultasiediens van die PU vir CHO bepaal.

Resultate

Die resultate het duidelik die effek van verstedeliking op aanvaarde risikofaktore vir chroniese lewenstylsiektes geïllustreer. Byvoorbeeld, progressiewe stygings in diastoliese bloeddruk en totale serumcholesterol is waargeneem. Die resultate het verder aangedui dat hoewel gerapporteerde energie-innames nie met verstedeliking toegeneem het nie, het die energiebronne verander. Totale proteïeninname was voldoende maar stedelike proefpersone het meer dierlike proteïenvoedsels en minder plantproteïene as landelike proefpersone ingeneem, hoofsaaklik as gevolg van 'n toename in rooivleis- en hoender innames. Die uitsondering was eierinname. Buiten vir die plaaswerkers wat baie lae innames gerapporteer

het, was die gemiddelde eierinname van mans ongeveer 30g per dag en diè van vroue 20g per dag. Geen effek van verstedeliking is waargeneem nie. Die persentasie energie voorsien deur vet het progressief toegeneem oor strata, terwyl die persentasie energie verkry van koolhidrate afgeneem het. Proefpersone in stratum 5 het reeds meer as 30% van hul energie as vet ingeneem maar het steeds relatief hoë koolhidraatinnames gehad (meer as 50% van energie). Gemiddelde dieetveselinnames was laag in al die strata (tussen 10.9 en 24.2g/dag) en het laag gebly met verstedeliking. Sukrose-innames was ook laag (30-36g/dag) en het nie met verstedeliking toegeneem nie. Gemiddelde stylselinnames het oor strata van 286 tot 224g/dag in mans en 238 tot 193g/day in vroue afgeneem. Stedelike mans en vroue het ook meer vleis, hoender, vrugte en groente geëet. Dit het hoër innames van mikronutriënte soos vitamiene E, C, folaat en yster deur stedelike proefpersone tot gevolg gehad. Die uitsondering was hoër kalsiuminnames deur plattelandse proefpersone (plaaswerkers).

Alkoholinnames was oor die algemeen hoog in mans. Inwoners van plakkerskampe het die hoogste innames gerapporteer. Patrone van gerapporteerde innames het met serum-gamma-glutamieltransferase-konsentrasies gekorreleer.

Benewens hierdie dieetverwante-risiko faktore vir kolorektale kanker, het ander risikofaktore soos LMI en volwasse lengte met verstedeliking toegeneem. Fisiese aktiwiteit het nie met verstedeliking geblyk te verander nie, terwyl die rookgewoonte inderwaardheid afgeneem het. Die aantal jongmense wat gerook het, was egter hoog.

Bespreking

Van die 13 aanvaarde, waarskynlike of moontlike risikofaktore vir kolorektale kanker wat oorweeg is, het agt in mans en sewe in vroue met lae risiko verband gehou, terwyl vier in mans en vyf in vroue met hoë risiko verband gehou het. Dit was nie moontlik om die effekte of bydrae van fisiese aktiwiteit tot risiko uit die beskikbare inligting was te beoordeel nie.

Van die faktore geassosieer met lae risiko, het ses: die inname van rooivleis, stysel en vet (totale en versadigde), LMI in vroue, asook volwasse lengte met verstedeliking in die rigting van verhoogde risiko verander. Van hierdie ses, was drie egter (rooivleis- en styselinname en volwasse lengte), hoewel veranderd, steeds onder die afsnypuntvlakke waar hulle verhoogde risiko tot gevolg het.

Van die vyf faktore geassosieer met 'n verhoogde risiko van kolorektale kanker (lae inname van groente, rookgewoonte, hoë alkoholinname by mans, lae dieetveselinnames en hoë LMI in vroue) het drie inderwaarheid met verstedeliking verbeter, wat moontlik 'n laer risiko verleen. Hierdie was toenames in groente en veselinname en 'n afname in rookgewoontes van stedelike mans, veral diè in stratum 5.

Omdat die bydrae van 'n spesifieke risikofaktor tot totale risiko van kolorektale kanker onbekend is, is dit nie moontlik om te oordeel of hierdie veranderings tot 'n laer totale risiko sal lei nie. Dit is egter duidelik dat die nadelige effekte van sommige risikofaktore met verstedeliking nie groot genoeg is om vlakke te bereik waar hulle as risikofaktore funksioneer nie, terwyl voordelige veranderinge in faktore geassosieer met hoë risiko in sommige gevalle waargeneem is, wat moontlik 'n verlaging van risiko tot gevolg het.

Gevolgtrekking

Die hipotese word gevolglik gestel dat op hierdie stadium van die lewenstyl- en voedingtransisie wat swart Suid-Afrikaners tydens verstedeliking ervaar die effekte op die risikofaktore vir kolorektale kanker sodanig is dat sommige faktore geassosieer met hoë risiko in plattelanders verbeter, terwyl sommige faktore geassosieer met lae risiko nie groot genoeg is om hierdie faktore verby 'n vlak waar hulle met 'n hoë risiko geassosieer sal word, te "stoot" nie. Dit is in teenstelling met ander chroniese siektes van lewenstyl soos hipertensie en diabetes mellitus wat reeds as openbare gesondheids-probleme beskou kan word.

In geheel gesien blyk dit asof by die huidige vlak van verstedeliking, veranderinge in lewenstyl en eetpatrone oor die algemeen met 'n laer risiko van kolorektale kanker geassosieer word. Dit is moontlik dat met verdere veranderinge, veral as gevolg van 'n toename in vleis- en vetinname, die risikofaktor-situasie tot so 'n mate mag verander dat risiko vir kolorektale kanker mag verhoog. Dit word voorgestel dat voorkomende maatreëls, soos die bevordering van 'n omsigtige kanker-voorkomende dieet, by swart Suid-Afrikaners in transisie ingestel word.

LIST OF ABBREVIATIONS

α	Alpha
%	Percentage
β	Beta
↑	Increase
↓	Decrease
↔	No change
≥	Greater
°C	Degrees celcius
µg/dL	Microgram per decilitre
µl	Microlitres
µmol/l	Micromol per litre
A	Adequate
AAPC	Attenuated adenomatous polyposis coli
AICR	American institute for cancer research
ALP	Alanine phosphatase
ALT	Alanine transferase
ASIR	Age standardise incidence rates
BMI	Body mass index
Ca	Calcium
Cal/day	Calorie per day
CANSA	South African Cancer Association
CH_4	Methane
CHD	Coronary heart disease
CK	Creatinine kinase
cm	Centimetre
CO_2	Carbondioxide
CRUDE	Crude cancer incidence rate
CUM RISK	Cumulative risk
DAX	Discrete Analyser
DNA	Deoxyribonucleic acid
DPO	Dry Bean Producers Organisation
EDTA	Ethylene diamine tetra-acetic acid
EPA	Eicosapentaenoic acid
et al	Et alli
etc.	Etcetera
F	Female
FAO	Food and Agriculture Organisation
FAP	Familial adenomatous polyposis
FFQ	Food frequency questionnaire
FRD	Foundation for Research Development
g	Gram
g-	Gram negative
g/dl	Gram per decilitre
g+	Gram positive
GGT	Gamma gluthamyltransferase

GTT	Glucose tolerance test
H	High
H_2	Hydrogen
HDL	High density lipoprotein
HNPCC	Hereditary nonpolyposis colorectal cancer
i.e	Id est
IU/L	International units/litre
Kg	Kilogram
Kg/m^2	Kilogram per square metre
kJ	Kilojoule
L	Low
LD	Lactate dehydrogenase
LDL	Low density lipoprotein
LMI	Liggaamsmassa indeks
LR	Lifetime risk
Ltd	Limited
m	Metre
M	Male
M	Medium
mg	Milligram
mg/dL	Milligram per decilitre
MJ	Megajoule
ml	Millilitre
mm	Millimetre
mmHg	Millimetre mercury
mmol/l	Millimol per litre
MPC	Macromolecular protein complex
MRC	Medical Research Council
MUFAT	Mono-unsaturated fatty acid
N	Number
N	Normal
NCD	Clover and National Co-operative Dairies
NCD's	Non-communicable diseases
ng/ml	Nanogram per millilitre
NH_3	Ammonia
NIDDM	Non-insulin dependent diabetes mellitus
NJP	No judgement possible
NSAID	Non-steroid anti-inflammatory drugs
NSP	Non-starch polysaccharides
P/S	Polyunsaturated/saturated
pH	Acidity/alkalinity
Ph.D.	Philosophiae Doctor
pmol/l	Picomol per litre
PU vir CHO	Potchefstroomse Universiteit vir Christelike Hoër Onderwys
PUFA	Polyunsaturated fatty acid
RDA	Recommended daily allowance
RE	Retinol equivalents
rpm	Revelations per minute
RS_{1-3}	Resistant starch

S	Stratum
SANNS	South African National Nutrition Survey
SASA	South African Sugar Association
SCFA	Short chain fatty acid
SD	Standard deviation
TC	Total cholesterol
THUSA	Transition and Health during Urbanisation of South Africans
TNM	See text (p.11)
UC	Ulcerative colitis
UK	United Kingdom
USA	United States of America
VIGHOR	Vanderbijlpark Information Project on Gesondheid /Health
w	Women
WCRF	World cancer research fund
WHO	World Health Organisation
y	Year

CHAPTER 1

INTRODUCTION

1.1 Background

Globally, there is an increase in the non-communicable diseases (NCD's) (Murray & Lopez, 1990), also known as the chronic diseases of lifestyle related to overnutrition and "affluence" such as obesity, coronary heart disease (CHD), stroke, hypertension, non-insulin dependent diabetes mellitus (NIDDM) and the diet-related cancers (colon, breast and prostate) (Shetty & McPherson, 1997).

In South Africa, as in a number of other African countries, morbidity and mortality from many infectious diseases are decreasing while the burden from the NCD's is increasing. The survival of African infants in South Africa has improved tremendously to 97% and expectation of life at birth is now about 60 years (Kale, 1995). Although CHD and colon cancer remain very uncommon among Africans, mortality rates from hypertension, stroke and diabetes are much lower in whites than in coloured, Asian or African South Africans (Bradshaw *et al.* 1995).

There is agreement in the literature that this increase in NCD's in developing countries, is associated with the demographic, nutrition and epidemiologic transition during urbanisation (Omran, 1971; Popkin, 1994;). However, the reasons for the rapid increase in NCD's among indigenous and previously rural people during urbanisation are far from clear.

The NCD's are all diseases which develop over time and are "unmasked" or precipitated by a combination of environmental or "social" risk factors in genetically susceptible or "vulnerable" individuals (Zimmet *et al.* 1997). Many of these risk factors develop or change during urbanisation. The extremely high rates of some of these diseases in populations previously free from them, suggest either increased genetic vulnerability, or an inability to adapt to

rapid environmental changes during urbanisation. But urbanisation in South Africa can be seen as a two-way process. Migrant workers with rural family ties, as well as increased availability of the media, could influence lifestyles in rural areas. Very little is known about the risk factors for developing NCD's in Africans in rural and urban areas and of changes in these factors during economic development and acculturation.

The high prevalence of these diseases demands that from a public health perspective, urgent preventative measures should be developed to reduce future human suffering and disability as well as socio-economic costs because of the premature morbidity and mortality. In a recent editorial Alexander Walker (Walker, in press) maintains that the recommendation for a more "prudent" (low-fat, high-fibre) diet, restriction of alcohol consumption, lessening of smoking practice and increases in physical activity are unlikely to be adopted by the majority of Africans in transition. Therefore, to develop appropriate, effective and sustainable intervention programmes, much more should be known about the changes in environmental risk factors and the response of Africans to these changes.

From the literature it seems that the pattern of emergence of these diseases are different in different developing countries. In India for example, NIDDM and coronary heart disease emerge early in life (Shetty, 1997). In South Africa, among the black population, obesity, hypertension and stroke seem to develop relative early in life (Walker, 1997), while coronary heart disease and colon cancer rates are still extremely low (Levy *et al.* 1994; Segal *et al.* 1991; Walker & Segal, 1997).

The low rates of colon cancer could mean that the African population may have a genetic protection against the disease, or that protective factors do not change with urbanisation and westernisation. It has been hypothesised (Vorster, 1996) that a dietary factor such as starch intake may be involved. It was argued that it is possible that despite increases in fat and animal protein intakes and decreases in dietary fibre intake, starch intakes may remain high

with urbanisation, because maize meal remains a dietary staple. Starch consists of several fractions, including rapidly digestible starch and α-amylase resistant starch. Resistant starch escapes digestion in the small intestine, but it is fermented in the large gut. Butyrate formed during the fermentation process protects the colon against carcinogenesis (Velazquez *et al.* 1996). Venter *et al.* (1990) have shown that maize porridge eaten cold or reheated develops more resistant starch. Maize porridge is the staple food in the African diet, and it is suspected that Africans often eat their maize porridge cold. Because of a lack of fuel and cooking constraints a large pot maize will be cooked and kept for more than a day, especially in rural areas. However, little is known about the amounts of starch intake in the African population.

1.2 The problem

The problem identified is that despite urbanisation and accompanied changes in lifestyle and eating patterns, the incidence and prevalence of colorectal cancer among black South Africans, compared to other ethnic groups in South Africa and globally, as well as with the incidence and prevalence of other NCD's, are very low. It is not known if this is due to a genetic protection in black South Africans, or if a dietary or other environmental factor that does not change with urbanisation, is responsible for these low rates of colorectal cancer.

1.3 Objectives of the study

The first objective of this study was to review the literature on the risk factors for colorectal cancer, with special emphasis on the dietary risk factors.

The second objective was to measure the impact of urbanisation on some of the risk factors of colorectal cancer in Africans, with special emphasis on the dietary risk factors.

The third objective was to develop a hypothesis that could explain the low rates of colorectal cancer in black South Africans.

This was possible in the THUSA (Transition and Health during Urbanisation of South Africans) project, a multidisciplinary study of the Nutrition Research Group of the PU vir CHO in which the influence of urbanisation on health and nutritional status of Africans in the Northwest Province is monitored.

1.4 Structure of the thesis

After this introductory chapter, a literature survey of colorectal cancer follows in chapter 2. The focus is on dietary risk factors and possible mechanisms through which these factors could influence the development of the disease.

In chapter 3 the objectives of the study are stated.

The methods used to measure the impact of urbanisation on the dietary risk factors of colorectal cancer are discussed in chapter 4. The results are given in chapter 5 and discussed in chapter 6, leading to the formulation of a hypothesis which tries to explain the slow emergence of colorectal cancer in black South Africans.

In chapter 7 the conclusions are followed by recommendations regarding the implementation of these results, as well as for further research to improve the hypothesis. The bibliography is given in chapter 8, and addendums in 9.

CHAPTER 2

LITERATURE SURVEY

2.1 INTRODUCTION

The major objectives of this study are to describe the risk factors of colorectal cancer in the African population of the Northwest Province of South Africa, and to show the impact of urbanisation on these risk factors. Therefore, in this chapter, the anatomy and physiology of the colon and rectum will be briefly reviewed to form a background and clarify terminology. Thereafter, colorectal cancer will be defined and classified, and possible causes and all known risk factors discussed. The prevalence and incidence of colorectal cancer in South African populations will then be given and the expected scenario for the future described. At the end of the chapter conclusions will be drawn that will be used in the development of a hypothesis regarding the impact of urbanisation of Africans on their risk for developing colorectal cancer.

2.2 ANATOMY AND PHYSIOLOGY OF THE COLON AND RECTUM

2.2.1 Anatomy of the large intestine

The large intestine is about 1,5 m long, extending form the distal end of the *ileum* to the anus (Williams *et al.* 1989). It differs in structure, size and arrangement from the small intestine. It has a greater calibre, and a more fixed position. Its longitudinal muscle is concentrated into three longitudinal *taenia coli.* The colonic wall is puckered into sacculations. Small adipose projections, *appendices epiploicae*, are scattered over the free surface of the whole colon except over the *caecum, vermiform appendix* and rectum.

The large intestine curves around the coils of the small intestine, commencing in the right *iliac* region as a dilated *caecum* (Williams *et*

al. 1989). The *caecum* leads to the *vermiform appendix* and colon. The colon ascends in the right lumbar and hypochondriac regions to the inferior aspect of the liver.

Here it bends (the right colic flexure) to the left with an antero-inferior convexity, loops across the abdomen as the transverse colon to the left hypochondriac region, where it curves again (the left colic flexure) to descend through the left. Here it forms a sinuous loop, the sigmoid colon, continuing along the lower posterior pelvic wall as the rectum and anal canal (Williams *et al.* 1989). The *caecum* lies in the right *iliac fossa*. It is a large *cul-de-sac* sac continuous with the ascending colon. Its average axial length is about 6 cm and its breadth about 7,5 cm (Williams *et al.* 1989).

The different parts of the colon will now be discussed briefly (reviewed by Williams *et al.* 1989).

The *ileocaecal* valve

The *ileocaecal* valve between the small and large intestine, consists of two flaps that project into the lumen of the large intestine. The valve not only prevents reflux from the caecum to the *ileum* but is probably also a sphincter regulating the passage of ileal contents into the *caecum* (Williams *et al.* 1989).

The ascending colon

The ascending colon is about 15 cm long and narrower than the caecum. It ascends to the inferior surface of the right lobe of the liver, where it makes a shallow depression. It turns abruptly forward and to the left at the right colic flexure. The right colic flexure is at the junction of the ascending and transverse colon. The latter turns down, forward and to the left.

The transverse colon

The transverse colon is about 50 cm long, extending from the right colic flexure in the right lumbar region, across into the left hypochondriac region.

Here it curves down and backwards below the spleen as the left colic flexure. The left colic flexure is above and on a more posterior plane than the right flexure at the junction of the transverse colon and descending colon in the left hypochondriac region.

The descending colon

The descending colon is about 25 cm in length, descending through the left hypochondriac and lumbar regions. First it follows the lower part of the lateral border of the left kidney and then descends in the angle between the *psoas major* and *quatratus lumborum* to the *iliac crest.* It then curves downwards and medially in front of the *illiacus* and *psoas major* to end in the sigmoid colon.

The sigmoid colon

The sigmoid colon begins at the pelvic inlet, forming a variable loop of about 40 cm and is normally in the lesser pelvis.

The rectum

The rectum is continuous with the sigmoid colon curving down and back, then downwards and finally down and forward to join the anal canal by passing through the pelvic diaphragm (Williams *et al.* 1989). The rectum is about 12 cm long, with the same diameter as the sigmoid colon, about 4cm in the empty state. The rectum has no sacculations, appendices or mesentery.

2.2.2 Histology of the large intestine

The histology of the colon and rectum is summarised in Table 2.1

Table 2.1 Histological features of the colon and rectum

Region	Muscularis externa	Submucosa	Muscularis mucosa	Lamina propria	Surface epithelium
Colon	Outer longitudinal Inner circular 3 Bands of taenia	No plicae	Two thin layers	No villi Tubular glands Large lymph nodes Thicker than in small intestine	Simple columnar Few goblet cells
Rectum	No taenia Much thicker than colon	Isolate lymph nodes Small veins	Not present Lamina propria Submucosa a merge here	Thicker than in colon	Stratified squamous longitudinal folds

Adapted from (Williams *et al.* 1989).

A major characteristic of the colon is the absence of villi (Martini, 1995). An abundance of goblet cells and intestinal glands are other characteristics. The glands are dominated by goblet cells (Martini, 1995). No enzymes are secreted by the mucosa. Mucus provides lubrication for faecal material when the latter becomes less moist and more compact. Friction or exposure to harsh chemicals stimulate mucous secretions.

2.2.3 Physiology of the large intestine

Absorption

The absorptive process in the colon is very important. Approximately 10 % of all absorption in the gastro-intestinal tract occurs in the colon (Martini, 1995; Guyton, 1991). Approximately 1,5 litre material enters the colon every day, and only 200 ml faeces are excreted. The large intestine (colon) also absorbs, apart from water, other substances such as short chain fatty acids, vitamins, urobilinogen, bile acids, bile salts, and toxins (Martini, 1995). Essentially all ions are absorbed. Ileal effluent contains principally saline solutions with some potassium bicarbonate (Cummings, 1997). The anions found in faeces comprise of short chain fatty acids, bicarbonate, chloride and traces of sulphate and phosphate. Of the cations existing, potassium, calcium and magnesium with some sodium can be found (Cummings, 1997). Short chain fatty acid transport is a very complex process. Several factors influence this transport by different mechanisms, which is still not yet clear (Van Engelhardt, 1995). Recent studies have shown that a combination of diffusion after protonation and

carrier-mediated SCFA anion exchanges with bicarbonate might explain the SCFA transport in the large intestine (Van Engelhardt, 1995).

The function of the *ileocaecal valve*

The *ileocaecal valve's* primary function is to prevent faecal material flowing back into the ileum (Guyton, 1991). The hormone gastrin, secreted from the stomach, increases ileal concentrations and helps relaxing the *ileocaecal valve* (Guyton, 1991). The prolonged stay of chyme in the ileum facilitates absorption. Strong reflexes from the caecum controls the degree of contraction of the ileocaecal valve, as well as the intensity of peristalsis in the terminal ileum (Guyton, 1991). When the caecum is distended, contraction of the ileocaecal valve is intensified, while ileal peristalsis is inhibited. This delays the emptying of additional chyme from the ileum (Guyton, 1991). The reflexes from the caecum to the ileocaecal valve and ileum are mediated by the mesenteric plexus in the gut wall itself and also through extrinsic nerves (Guyton, 1991).

Movements (haustrations) of the large intestine

Distinctive movements of the colon are distinguished, including mixing and mass movements. Circular constrictions combined with longitudinal muscle contractions cause the colon to bulge into sacs called haustrations. This movement provide a small amount of forward propulsion of the colonic material. New contractions occur in other areas nearby, providing a good mixing of faecal material. With this mixing all the faecal material is exposed to the surface of the epithelium.

Propulsion occurs by the analward movement of the haustral contractions and mass movements. Propulsion in the caecum and ascending colon results from slow haustral contractions (Guyton, 1991; Martini, 1995). After this, a part of the distal part of the constrictive ring in the transverse colon, loses its haustrations and contracts as a unit. This forces the faecal material down the colon until it reaches the rectum (Martini, 1995; Guyton, 1991).

Defecation

The rectum is usually empty of faeces. When a mass movement forces faeces into the rectum, a desire to defecate is initiated. The rectum contracts and the anal sphincters relax (Martini, 1995; Guyton, 1991).

2.3 COLORECTAL CANCER: DEFINITIONS

2.3.1 Definition of cancer

Several definitions of cancer are being used in the literature, but the main definition can be summarised as follows:

Cancer is a group of diseases all characterised by the abnormal growth of altered body cells (Koeffler *et al.* 1991). While cells divide and grow in an orderly fashion, cancer cells divide and grow out of control, disrupting normal body tissues and organ functions. During the first years of an individual's life, normal cells divide rapidly until full growth is attained. After this period, normal cells of almost all tissues divide only to replace and repair worn-out and scarred tissues. Cancer cells may compress, invade, and destroy normal tissue. Most types of cancer form a mass, called a *tumour*. If cells break away from the tumour, these cancer cells can be carried by the lymph system or bloodstream to other parts of the body. There they may form a colony tumour at their new location. This spreading of the tumour is called *metastasis*. Colon cancer, a common form of cancer, is a disease in which cancer cells are found in the tissues of the colon.

2.3.2 Classification of colorectal cancer

2.3.2.1 Stages of colorectal cancer

When cancer of the colon and rectum are diagnosed several other tests will point out whether the cancer has spread to other parts of the body. This is very important because it will point out what treatment should be imployed. Cancer of the colon can be staged by several methods. The two most common are TNM classification and Dukes staging. Dukes classification divides colon and rectal cancer into three stages, A, B and C.

The TNM classification classifies colon cancers in a more precise fashion (see Table 2.2).

"T" represents tumour. A cancer is placed in one of seven groups depending on the depth of invasion into the bowel wall. "N" represents lymph node. A tumour is given a "N" ranking according to whether the cancer has spread to the lymph nodes and the number of nodes involved. "M" relates to whether there is cancer spread to other distant organs from the colon and lymph nodes. The following stages are used for colon cancer:

Stage 0

This stage is a very early cancer. The cancer is found in the innermost lining of the colon.

Stage I

Also referred to as Dukes A, involves the second and third as well as the inside wall of the colon. The cancer however, has not spread to the outer wall of the colon or outside the colon , but is confined to the muscular lining of the intestinal wall.

Stage II

Also known as Dukes B. The cancer has spread outside the colon to adjacent tissue, but has not spread into the lymph nodes.

Stage III

Also known as Dukes C. At this stage, cancer has spread to nearby lymph nodes, but not to other parts of the body.

Stage IV

The cancer has spread to other parts of the body. This stage is referred to as Dukes D.

Table 2.2. TNM definitions of colorectal cancer*

Tx	Primary tumour cannot be assessed
T0	No evidence of primary tumour
Tis	Carcinoma in situ: intraepithelial or invasion of the lamina propria
T1	Tumour invades submucosa
T2	Tumour invades muscularis propria
T3	Tumour invades through the muscularis propria into the sub-serosa, or into the non-peritonealised pericolic tissues
T4	Tumour directly invades other organs or structures and/or perforates the visceral peritoneum
Regional lymphnodes (N)	
Nx	Regional nodes cannot be assessed
N0	No regional lymphnodes metastasis
N1	Metastasis in 1-3 pericolic lymph nodes
N2	Metastasis in 4 or more lymph nodes
N3	Metastasis in any lymph node along the course of a named vascular trunk and/or metastasis to apical node(s).
Distant metastasis (M)	
Mx	Presence of distant metastasis cannot be assessed
M0	No distant metastasis
M1	Distant metastasis

* Adapted from (Rubin & Faber, 1990; Smit, 1994).

2.4 POSSIBLE CAUSES AND PROTECTIVE FACTORS OF COLORECTAL CANCER

2.4.1 Environment, genes and cancer

It has been realised for many years that cancer has a genetic component. Genetic alterations that change proto-oncogenes into oncogenes activate and dysregulate normal genes and provide the cell with a growth advantage, usually in a dominant manner (Koeffler *et al.* 1991).

Loosing genetic information may also be oncogenic (Koeffler *et al.* 1991), in that a normal growth regulator that constrains cellular proliferation could be lost. Mutations of the *ras* and heterotrimetric G-proteins can activate the pathways that these proteins control. Activation of these pathways is thought to cause uncontrolled cell division (Koeffler *et al.* 1991).

Most cancers arise due to sporadic mutations that may occur as a result of spontaneous genetic events, environmental events or interaction between genetic and environmental factors (Claus, 1995). The term environment may indicate the physical environment but is often used to collectively describe risk factors other than genetic, such as diet and lifestyle. Little evidence exists that although the physical environment is deteriorating the cancer burden is increasing (Inskip, 1994). Only about 5 % of cancers are due to environmental factors and more than half of these are due to geographical properties of the environment, rather than man-made pollutants (Inskip, 1994).

Cancer of the large bowel (colon) is still relatively uncommon in Japan, but the incidence in US Japanese is currently higher than in both US Whites and Blacks (Muir, 1996). It is likely that this phenomenon in the US is due to the environment (Muir, 1996). The interplay between environmental factors and genetic predisposition is important in almost all diseases, especially in cancer (Bellacosa *et al.* 1996; Perera, 1996; Knudson, 1995; Burt, 1996; Alberts *et al.* 1996). A complex interaction exists between environmental/dietary factors and genetics, underlying the pathogenesis of colon carcinogenesis (Alberts *et al.* 1996). For colorectal cancer there has been much dispute about the relative contributions of hereditary and environmental factors in tumourigenesis (Peltomäki *et al.* 1993). Members of an individual family are likely to share similar environments (Peltomäki *et al.* 1993). Some kinds of common cancers such as breast cancer and colon cancer tend to run in families. Therefore, although diet and other environmental factors influence the development of cancer, susceptibility to this disease can be inherited (Marx, 1993; Perera, 1996).

2.4.2 Genetics: hereditary colon cancer syndromes

2.4.2.1 Introduction

From the above it is clear that a complex interaction between genetic susceptibility and environmental factors probably plays an important role in the development of colorectal cancer. In

this section, hereditary colon cancer syndromes will be briefly reviewed, after which the environmental factors thought to be important in the genesis of colorectal cancer will be discussed.

2.4.2.2 Familial adenomatous polyposis (FAP)

Hereditary colon cancer syndromes are inherited in a autosomal dominant pattern (Itzkowitz, 1995; Burt *et al.* 1995; Luce *et al.* 1996; Burt *et al.* 1991; Bellacosa *et al.* 1996; Simko & Zajac, 1996; Olschwang, 1994). FAP has an approximate incidence of 1 in 10 000 births per year (Stern, 1996; Burt *et al.* 1995). It is characterised by the development of hundreds and thousands of colonic adenomatous polyps (Burt *et al.* 1995; Stern, 1996; Potter, 1995). Colon cancer is inevitable if the colon is not removed. The average age at cancer diagnosis is 39 years (Itzkowitz, 1995; Burt *et al.* 1995; Knudson, 1995). FAP is classified as *Gardner Syndrome* when the intestinal findings are accompanied by certain benign extraintestinal growths, such as mandibular osteomas, skin cysts, dental abnormalities, desmoid tumours, etc. *Turcot Syndrome* refers to intestinal polyposis in combination with brain tumours. Some families with *Turcot Syndrome* exhibit colonic polyposis typical of FAP (Burt *et al.* 1995).

2.4.2.3 Attenuated adenomatous polyposis coli (AAPC)

This is a hereditary colon cancer characterised by multiple colonic adenomas, with less than 100 in number. It assumes a proximal predominance (Burt *et al.* 1995; Itzkowitz, 1995) and has a flat rather than polypoid growth pattern. The mean age of diagnosis of this syndrome is 55 years (Winawer *et al.* 1996; Han *et al.* 1996; Westergaard, 1996).

The mutated gene in AAPC is the APC gene (Lynch *et al.* 1997). APC is a tumour-suppressor gene (Burt *et al.* 1995; Stern, 1996;

Aaltonen *et al.* 1993; Itoh & Imai, 1996) and is involved in some manner with cell adhesion and cell communication (Potter, 1995; Burt *et al.* 1995).

Somatic mutations of this APC gene are an integral part of sporadic colorectal tumourigenesis (Stern, 1996; Itzkowitz, 1995, Senda *et al.* 1996; Cunningham & Dunlop, 1996).

2.4.2.4 Hereditary nonpolyposis colorectal cancer (HNPCC)

The colon cancers that arise in HNPCC individuals are marked by predominance in the proximal colon (Bellacosa *et al.* 1996; Smyrk, 1994; Lynch & Lynch, 1994; Vasen, 1994; Ponz de Leon, 1994) and at an average age at cancer of diagnosis of 48 years (Itzkowitz, 1995).

To date, four genes that appear to contribute to HNPCC have been discovered namely: *hHSH2(2p); hHMLH1 (3p); hPMS1 (2q); hPMS2 (7q)* (Luce *et al.* 1996; Jeon *et al.* 1996).

Mutations in *hMSH2* and *hMLH1* account for the vast majority of the cases in patients with HNPCC (Luce *et al.* 1996; Bellacosa *et al.* 1996; Jeon *et al.* 1996; Nystrom-Lahti *et al.* 1994; Bronner *et al.* 1994). HNPCC results from inherited mutations of mismatch repair genes. These genes normally repair mutations that occur during DNA replication. When these repair genes are mutated, replication error mutations persists (Burt *et al.* 1995).

The first human mismatch gene to be cloned was *hMSH2*, the homologue of yeast *MSH2* and *E.Coli mutS* (Bellacosa *et al.* 1996; Jass *et al.*1994). The second human mismatch gene, *hMLH1* displays a striking homology to yeast *MLH1* and *E.Coli mutL* (Bellacosa *et al.* 1996; Jeon *et al.* 1996).

In a study by Jeon *et al.* (1996) two families were identified with identical genetic lesions resulting from a single base deletion at codon 705 in the *hMSH2* gene (Jeon *et al.* 1996). A similar mutation was absent in one individual, which may suggest that a second gene or environmental factors may play an important role in the manifestation of HNPCC (Jeon *et al.* 1996; Han *et al.* 1996).

In a much larger population of cases, possibly the majority, inherited factors confer susceptibility and interact with environmental factors to determine expression of polyps and cancer (Burt, 1996). In some cases environmental exposures alone give rise to colonic neoplasm's (Burt, 1996).

It is likely that pro-carcinogens are present in the diet, and undergo modification and activation during transit through the stomach, small intestine and colon (Tsiotou *et al.* 1995), and influence mutation processes in these genes.

2.4.2.5 Familial colorectal cancer

Large kindred studies have shown that there is an inherited susceptibility to colonic neoplasm's (Itzkowitz, 1995). Syndromic colorectal cancers account for ± 5 % of all the colorectal cancers in the USA. Another 95 % are "sporadic". It is estimated that about half of the "sporadic" colon cancers may have an underlying genetic susceptibility (Itzkowitz, 1995). A family history of one or more first degree relatives with colorectal cancer places an asymptomatic person in a high risk group (Westergaard, 1996).

2.4.2.6 Inflammatory bowel disease

The exact cause of increased risk of developing colorectal cancer in inflammatory bowel disease is still not clear (Choi & Kim, 1995). Evidence supporting the importance of chronic

inflammation in colon carcinogenesis comes from studies examining the effect of anti-inflammatory agents in affecting the development of colorectal cancer. A variety of nonsteroidal anti-inflammatory agents, including aspirin, inodmethacin, sulindac, and piroxicam, have been found to inhibit the growth of tumours in the colon of rodents treated with carcinogens (Choi & Kim, 1995).

- **Ulcerative colitis**

  There is a link between low levels of faecal short chain fatty acids and the occurrence of ulcerative colitis (Segal *et al.* 1995)

  A hypothesis stating that ulcerative colitis (UC) is due to exogenous or to endogenous starvational factors limiting fatty-acid oxidation in colonocytes, has been formulated (Roedinger, 1995). Mucosal energy deficiency may play a role in the pathogenesis of inflammatory bowel conditions (Scheppach *et al.* 1995).

  The risk of developing colorectal cancer starts only 8-10 years after the onset of ulcerative colitis (Bansal & Sonnenberg, 1996; Kroser *et al.* 1997). Bansal & Sonnenberg (1996) found that cancer associated with inflammatory bowel disease occurred at a younger age than the regular type of cancer affecting the non-inflamed mucosa.

- **Crohn's disease**

  The association between Crohn's disease and colorectal cancer risk has been controversial (Ribeiro *et al.* 1996). Populations highly at risk are patients with extensive (18-fold increase risk), long-standing unresected colon disease, patients with colonic and ileocolonic Crohn's disease (Ribeiro *et al.* 1996; Austoker, 1995; Kroser *et al.* 1997). The absolute number of cancer cases reported in association with Crohn's disease, nevertheless, remains much smaller than that seen with ulcerative colitis (Choi & Kim, 1995).

  This may be a reflection of less available bowel at risk in Crohn's

disease (Choi & Kim, 1995).

2.4.3 The role of intestinal bacteria and fermentation

2.4.3.1 Colonic bacteria

Dietary non-starch polysaccharides (NSP), a small proportion of dietary starch, as well as small amounts of dietary and secreted proteins, lipids and other substances are poorly digested by host enzymes in the small intestine. These substances reach the colon and are metabolised by the bacteria present in the large intestine (Cummings, 1997 ;Young, 1996).

In healthy humans, the colonic microflora is dominated by bacteria (Topping, 1996). The types of bacteria in the gut can be divided on the basis of their oxygen-tolerance and requirements for carbon dioxide into strictly aerobic *facultative*, *micro-aerophilic* and strictly *anaerobic* genera (see Table 2.3).

Strictly aerobic organisms are bacteria which will only grow in the presence of oxygen and die in its absence and are present in the mouth and nose but not in the colon. Facultative organisms use oxidative pathways to generate energy in the presence of oxygen radicals produced during oxidative metabolism. In consequence they die in the presence of high oxygen levels. They grow best in the presence of high concentrations of CO_2. Strictly anaerobic organisms are not able to use oxygen or to carry out oxidative metabolism.

The role of bacteria in the genesis or protection against colorectal cancer, is probably related to the ability of bacteria to ferment undigested dietary substrates, (NSP, starch, protein, lipid), intestinal cells, secretions and also biliary secretions (De Schrijver, 1996), that reach the colon, through effects of the fermentation products on colonic mucosa.

In addition to the formation of fermentation products with putative beneficial effects (see 2.4.3.2), bacteria forms a major part of faecal mass.

Growth and proliferation of bacteria will therefore contribute to stool bulk,

defecation frequency, prevention of constipation and less exposure of colonic epithelium to possible harmful agents. Bacteria could, through this mechanism, protect to colon against cancer genesis.

Table 2.3. The main genera of bacteria in the human digestive tract*

ORGANISM	DESCRIPTION	AREA
Strictly aerobic		
Pseudomonas	g-, rods	
Bacillus	g+, spore-forming rods	
Neisseria	g-	Mouth
Staphylococcus	g+	Nose
Micro-aerophyllic		
Lactobacillus	g+	all GI tract
Streptococcus	g+	all GI tract
Enterococcus	g+	colon
Camphylobacter	g-	pathogen
Facultative		
Escherichia	g-	Colon
Klebsiella	g-	Colon
Proteus	g-	Colon
Salmonella	g-	Pathogen
Shigella	g-	Pathogen
Strictly anaerobic		
Bacteriodes	g-	all GI tract
Actinomyces	g+	mouth
Bifidobacterium	g+	colon
Eubacterium	g+	colon
Propionibacterium,	g+	colon
Fusobacterium	g-	all GI tract
Veillonella	g-	all GI tract
Peptostreptococcus	g+	colon
Clostridium	g+	colon

* Adapted from: (Davis *et al.* 1990)

2.4.3.2 Colonic fermentation

Colonic fermentation can be defined as the non-digestibility of dietary fibre, which is fermented to produce gas and certain short chain fatty acids (Cummings, 1997).

The products of fermentation in the large bowel are likely to have a number of direct and indirect effects. There is convincing evidence that the products of carbohydrate fermentation protect against colorectal cancer. It has been suggested that low intakes of carbohydrate cause butyrate starvation in the colonic mucosa and that this could lead to several diseases such as inflammatory bowel disease and colorectal cancer (Bansal & Sonnenberg, 1996). It has been shown that countries with a low consumption of cereals present high rates of colon cancer (Giacosa *et al.* 1993).

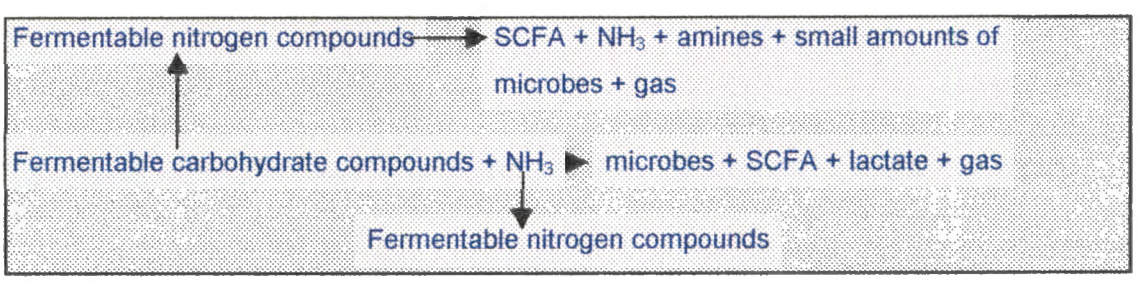
Fermentation of undigested dietary carbohydrates in the colon can be divided into the following steps:

- hydrolysis of polysaccharides to oligosaccharides (extracellular enzymes are used)
- further hydrolysis to simple sugars (cell bound enzymes are used)
- simple sugars are fermented by usual pathways to short chain fatty acids (SCFA).

The fermentation process by which compounds are bacterially dissimilated in the *caecum* is complex, and leads to partial or complete decomposition (Eastwood, 1992; Gibson *et al.* 1995). In a typical diet an estimated 70 % of dietary NSP is broken down by colonic flora (Klurfeld, 1992). The *Bacteroides genus* are the most versatile polysaccharide utilisers (Gibson *et al.* 1995). Many of the bacteria are not able to degrade polymerised carbohydrates and grow by crossfeeding on fragments produced by primary polysaccharide degraders (Gibson *et al.* 1995). This feeding is an important characteristic and maintains the diversity of the ecosystem and variety of fermentation end products produced (Gibson *et al.* 1995).

The gut flora has two major requirements namely, *energy* and, *nitrogen*.

Fermentable carbohydrates provide the main energy, while the nitrogen source is provided by ammonia (De Schrijver, 1996). The fermentation process can be summarised as follows:



Adapted from (De Schrijver, 1996).

Table 2.4 The major end products of fermentation of various species of human gut bacteria*

ORGANISM	ACID PRODUCTS	GAS PRODUCTION
Facultative anaerobes		
Streptococcus spp.	lactic, acetic, formic	-
Enterobacteria	lactic, acetic, formic	CO_2, H_2
Micrococci	lactic, acetic, formic	CO_2
Lactobacilli	lactic	-
Strictly anaerobic		
Bacteriodes	Acetic, propionic, butyric	CO_2, H_2
Fusiforms	acetic, butyric	CO_2, H_2
Bifidobacteria	acetic, lactic	-
Eubacteria	little acid	H_2
Propionibacteria	acetic, propionic	CO_2(little)
Veillonella	little acetic, propionic	H_2
Clostridia	acetic, propionic, butyric, caprioc, lactic, valeric	CO_2, H_2

*Adapted from:(Davis *et al.* 1990)

The SCFA, acetic, propionic and butyric acids are produced by this fermentation process as well as CO_2 and CH_4 (Muir *et al.* 1995; Kaaks & Riboli, 1995; Young, 1996; Baghurst *et al.* 1996; Hill, 1995; Nordgaard *et al.* 1995; Young, 1996). As shown in Table 2.4 the three main SCFA's (acetate, propionate, and butyrate) stimulate colonic sodium and fluid absorption and exert proliferative effects on the colonocyte (Scheppach, 1994; Topping, 1996; Mitsuoka, 1996).

Although the physiological effects of the SCFA's are far from sorted out, the end products of fermentation have been related to several conditions such as cholesterol and glucose concentration, gallstones, inflammatory diseases and cancer of the colon (De Schrijver, 1996).

Acetate increases colonic blood flow and enhances ileal motility, whereas butyrate is the preferred energy substrate for the colonocyte (Young, 1996) and a very potent differentiating agent in cell cultures (Scheppach, 1994; Clausen & Mortensen, 1995). Furthermore, butyrate is purported to have typical antineoplastic effects (Van Munster & Nagengast, 1993; Young, 1996). Butyrate production from carbohydrate fermentation is thought to be a major mechanism through which dietary NSP and resistant starch may protect against colorectal cancer (Vorster, 1997). In a study conducted by Segal *et*

al. (1995) it was found that Africans had significantly higher faecal SCFA concentrations than whites. The high concentrations of *faecal* SCFA's in this African group could protect them against chronic bowel diseases and such as inflammatory bowel disease, colon cancer. This could explain why rural South African blacks with a traditional lifestyle have a low prevalence of these diseases (Levy *et al.* 1994).

A number of factors can influence the fermentation of substrates by gut bacteria (Gibson *et al.* 1995; De Schrijver, 1996). Nutrient supply will often become limited due to the vast range of microbial species. The physio-chemical environment of the large intestine, including pH, substrate availability, colonic anatomy and oxygen may also affect fermentation (Gibson *et al.* 1995). Shorter transit times results in an inefficient fermentation process (Cummings, 1997).

Dietary manipulation (sodium sulphate, oligosaccharides, etc.) affect the growth of certain bacterial species (Gibson *et al.* 1995). *Bifidobacteria* exert some beneficial properties. Their numbers are much higher than other species. They produce strong acids, thus lowering the pH and exert an anti-microbial effect. Also, they bind toxic NH_3 with H^+ to produce NH_4^+, which in effect lowers blood ammonia levels. Another characteristic is the production of B-vitamins, and certain components of *bifidobacteria* promotes immunological attack against malignant cells (Gibson *et al.* 1995).

2.4.4 The causative and protective role of dietary factors

2.4.4.1 Introduction

It appears that the major environmental determinants of colorectal cancer risk may be dietary in origin. Several dietary components have been implicated as possible factors in the aetiology of colorectal cancer (Kroser *et al.* 1997). Major factors such as overall energy intake, fat content of the diet, starch, fibre and micronutrient consumption may all play a very significant role in the genesis of or protection against colorectal cancer. In this section the role of dietary factors will be reviewed with emphasis on the possible mechanisms by

which these factors may influence colorectal cancer.

2.4.4.2 Dietary starch

Starch is the principal carbohydrate produced by plants during photosynthesis (Guzman-Maldonado & Paredes-Lopez, 1995). Starch provides a source of numerous mono-, di-, and oligosaccharides. Amylose, one of the two glucose polymers of starch is a linear polymer to which glucose residues are linked by α-glycosidic links. Amylopectin, the second glucose polymer has a more complex structure, consisting of a branched polymer of α(1-4)-links in the linear segments and α(1-6) links at the branch points (Baghurst *et al.* 1996). Starch occurs as granules, and inside these granules are crystalline regions that consists of amylopectin (Baghurst *et al.* 1996). When starch containing foods are heated the crystalline regions "melt" (Baghurst *et al.* 1996). At lower temperatures starch gelatinises (in the presence of water) and the granular structure disappears. In this form, starch is rapidly digested by α-amylase in the small intestine to maltose and glucose. But this gel is unstable and a crystal forms by a process referred to as retrogradation, in which gelatinised starch looses some of its water. Non-retrograded starch will be broken down or digested by α-amylase in the small intestine, but a fraction, called resistant starch, is resistant to amylolytic degradation (Baghurst *et al.* 1996; Reddy, 1995; Englyst *et al.* 1992).

Resistant starch (RS) can be subdivided into three fractions:

- RS_1 - starch which is resistant because it is in a physically inaccessible form, found in partly milled grains
- RS_2 - resistant starch which is resistant to enzyme digestion, such as fractions found in raw potato and banana
- RS_3 - the most resistant starch fraction, such as found in green bananas/old bread, cooked and cooled potatoes

(Van Munster & Nagengast, 1993; Englyst *et al.* 1992).

The ratios of SCFA produced during fermentation depend on the nature of the carbohydrate being used as the substrate (Cummings, 1997). When starch is fermented the ratio of SCFA formed is ; acetate (62): butyrate (15): propionate (23) compared to other carbohydrates such as pectin (80:12:8) (Cummings, 1997). Larger amounts of butyrate are produced when starch is fermented (Cummings, 1997). Starch fermentation therefore provides relatively more butyrate than fermentation of other carbohydrates. Butyrate is known to protect against growth and proliferation of cancerous cells (Clausen *et al.* 1991).The fermentability of starch by anaerobic microflora in the colon is thought to be the main mechanism through which high resistant starch intakes protect against colorectal cancer (Baghurst *et al.* 1996).

2.4.4.3 Dietary fibre (Non-starch polysaccharides or NSP)

There are some controversies about the role of dietary fibre (NSP) in the aetiology of colon cancer in humans (Baghurst *et al.* 1996). Studies of diet and colon cancer relationships found decreased risks associated with a high consumption of fruit and vegetables (Baghurst *et al.* 1996; Young, 1996) and also with unprocessed cereals (Baghurst *et al.* 1996). The colonic mucosa is vulnerable during starvation and malnutrition, as luminal fuels make such a great contribution to energy metabolism (Scheppach, 1994). A lack of luminal nutrients may impair the morphology (atrophy, colitis) and function (sodium absorption) of colonic epithelial cells (Scheppach, 1994).

Several plausible mechanisms by which NSP may protect against the development of colon cancer exist: Some will be discussed briefly here:

- ***Secondary bile acids***

The main components of bile acids are cholic and chenodeoxycholic acids. When metabolised by bacterial enzymes, carcinogenic secondary bile acids, lithocholic and deoxycholic acids, thought to be carcinogenic (Young, 1996), are formed (Baghurst *et al.* 1996). Therefore, NSP, by sequestering bile acids and preventing formation of secondary bile acids, may protect against colon carcinogenesis (Young, 1996). Since high-fat low-NSP intakes, as seen in a typical western diet, increase bile production (Burkitt, 1980), the

apparent effects of NSP on bile acid metabolism may be overwhelmed by the effects of fat in these diets (Baghurst *et al.* 1996). By lowering the pH of the colonic contents the conversion of primary to secondary bile acids could be reduced.

- ***Stoolbulk and transit times***

The fermentation process results in an increase in bacterial mass and therefore of stoolbulk (Kaaks & Riboli, 1995; Young, 1996; Nordgaard *et al.* 1995). High NSP diets *per* se will also increase stoolbulk (Baghurst *et al.* 1996) and decrease intestinal transit time (Cummings *et al.* 1992). Soluble NSP produce viscous solutions that delay gastric emptying and absorption from the small intestine. However, these substances are very accessible to bacterial enzymes and are very rapidly fermented in the proximal colon, contributing to increased bacterial mass. Insoluble NSP have much less effect on the viscosity of the gut contents and tend to accelerate rather than delay small bowel transit time and decrease total transit time. The resultant increased volumes and shorter transit times are associated with less exposure of the colonic mucosa to possible carcinogens, and therefore to a decreased risk of colon cancer. In a study by Levy *et al.* (1994), no difference in stool weight of blacks and whites was found, but the faecal pH was higher in whites (Levy *et al.* 1994).

- ***Butyric acid***

As mentioned in 2.4.3.2 and 2.4.4.2, butyric acid is produced in the colon of mammals as a result of microbial fermentation of dietary fibre, (NSP) starch and proteins (Velazquez *et al.* 1996). Butyric acid inhibit the growth and proliferation of cancerous cells (Clausen *et al.* 1991). It also modifies the expression of several oncogenes in a favourable way (Van Munster & Nagengast, 1993). Both NSP and starch are fermentable, and are excellent sources of butyric acid (Baghurst *et al.* 1996; Velazquez *et al.* 1996).

2.4.4.4 Dietary fat

Dietary fat has received considerable attention as a possible risk factor in the aetiology of colon cancer (Reddy, 1993). Evidence suggest that diets high in

animal fat may increase the risk of colon cancer (Potter, 1996; Potter, 1993, Reddy, 1995; Silvester & Cummings, 1995; Giovannucci *et al.* 1994; Weisburger, 1991; Iscovich *et al.* 1992). These diets are however also high in meat, which is an independent risk factor for colon cancer (see 2.4.4.11).

Nevertheless, populations with high incidences of colon cancer are characterised by consumption of high dietary fat and energy, which may be risk factors in the absence of protective factors such as fibre (Reddy, 1993). It is thought that the marked international differences in the incidence and mortality of colon cancer and increased risk in populations migrating from rural to urban areas may be because of differences in fat intake (Reddy, 1995; Giovannucci & Willett, 1994).

Reddy and co-workers (1995) have shown that the dietary fats, corn oil, safflower oil, and beef tallow increase the concentration of faecal secondary bile acids i.e. deoxycholic acid and lithocholic acid as well as fatty acids. Populations at high risk for colon cancer excrete high levels of secondary bile acids (Reddy, 1995). These secondary bile acids induce cell proliferation and act as promoters for colon cancer (see 2.4.4.3). Fat increases bile acid production, ultimately increasing the exposure of the bowel mucosa to the toxic, trophic and promoting effects of bile acids (Potter, 1996; Reddy, 1995).

Other aspects of the colonic milieu, including bacterial species, bacterial enzyme production and transit time, will change with a higher meat/fat intake and in turn will alter the production and concentration of secondary bile acids (Potter, 1993). These will increase cell damage and repair and cause changes in gut mucosa (Potter, 1993). Overall risk of cancer might be regarded as the sum, over long periods, of the moment-to-moment results of these cascades - set against a background of genetic susceptibility (Potter, 1993).

Tumour induction can be divided into two distinct stages; initiation and promotion, the latter being reversible. During the initiation of carcinogenesis the genetic information of the cell is being altered, whereas promotion leads

to the expression of genetic changes and malignancy which involves loss of control over cellular proliferation (Reddy, 1993). Diet may enhance these processes at certain stages. Animals fed a diet containing a high amount of fat had a greater incidence of colon tumours than did rats fed a low-fat diet (Reddy, 1993).

The effect of dietary cholesterol on colon cancer is still unclear. There appears to be an inverse relationship between serum and plasma cholesterol levels, and the risk for colon cancer (Broitman *et al.* 1993).

2.4.4.5 Animal protein

High rates of colorectal cancer have been observed in populations consuming diets high in meat and fat, low in vegetables, starch and NSP (Bingham, 1996). The factors in red meat responsible for the development of colorectal cancer are not clear yet, but carcinogens may be created during the high-temperature cooking of animal protein (Harvard report on cancer prevention, 1996). A diet rich in animal protein contains large amounts of sulphur-rich amino acids, whose metabolism generates hydrogen sulphate. This must be detoxified with methyl groups, leaving less available for DNA repair (Oettle, 1996). Red meat may be important in the initiation of colorectal cancer development rather than the promotion of it (Bingham, 1996). Bingham *et al.* (1996) showed increased endogenous production of *N* - nitroso compounds and precursors from increased red meat consumption. These may be relevant to the aetiology of colorectal cancer.

2.4.4.6 Calcium

Another dietary component found to reduce colon cancer risk is a high intake of calcium (Alberts *et al.* 1996; Kampman *et al.* 1994(a)). Calcium may reduce colon cancer risk by forming insoluble soaps with ionised fatty acids and secondary bile acids in the colon lumen (Giovannucci & Willett, 1994; Govers *et al.* 1996). This could be involved in regulating colonic epithelial proliferation by decreasing luminal levels of fatty acids and bile acids (Kroser *et al.* 1997). In a study by Lupton *et al.* (1996) calcium supplementation

decreased the proportion of primary bile acid, chenodeoxycholate.

2.4.4.7 Selenium

Selenium is an essential trace element and functions as an antioxidant in the body. It is a cofactor for glutathione peroxidase, which participates in preventing free radical damage to tissues, a likely factor in carcinogenesis (Kroser *et al.* 1997; Fleet, 1997). Dietary supplementation with selenium can inhibit the development of colon cancer (Reddy *et al.* 1997). Compounds which generate oxygen-containing free radicals alter DNA and is critical in the carcinogenic process. The antioxidants, selenium, Vitamin A, C and E protect against this oxidative damage (Butterworth & Bendich, 1991).

Several hypotheses have been proposed to explain the procarcinogenic effects of low selenium levels and chemoprotective effects of high selenium intake (Fleet, 1997). Selenium may inhibit protein synthesis or specific enzymes within cancerous cells and may stimulate apoptosis, leading to slower tumour growth (Fleet, 1997). No definite studies examining the relationship of the chemoprotective effect for selenium in humans exist.

2.4.4.8 β-Carotene

The National Cancer Institute emphasises the intakes of antioxidants such as β-carotene, vitamin E, vitamin C and selenium by consuming vegetables and fruits (Ziegler & Subar, 1991). β-Carotene, a precursor of vitamin A, is a plausible protective agent against cancer. Diets high in yellow and dark green vegetables and fruits are β-carotene rich which might reduce cancer risk (Ziegler & Subar, 1991; Gerster, 1996).

2.4.4.9 Vitamin E

Vitamin E, found in many vegetables, possibly inhibits the development of cancer through its antioxidant effects. There is weak evidence that the association between intake of vegetables and cancer risk are due to their vitamin E content (Knekt, 1991).

2.4.4.10 Zinc

Zinc is also a trace element, and is found in red meat. If the concentration of zinc in blood is high the risk of certain cancers (breast and colon) rises (Kroser *et al.* 1997). The mechanism by which zinc may influence cancer development is not known.

2.4.4.11 Vitamin D

Low serum levels of vitamin D have been associated with an increased risk for colon cancer (Sadava *et al.* 1996; Tangrea *et al.* 1997). In a study by Sadava *et al.* (1996) four week old mice were fed a diet either containing or lacking this vitamin for 3 weeks. Results showed that vitamin D deficiency significantly altered colonic but not duodenal epithelial cells (Sadava *et al.* 1996). In another study (Pritchard *et al.* 1996) it was found that vitamin D may reduce the risk of large bowel cancer and particularly rectal cancer. According to Kroser *et al.* (1997) colorectal cancer rate increases with increasing distance from the equator. This correlates with the decreasing intensity of sunlight (Kroser *et al.* 1997).

2.4.4.12 Summary: dietary factors

From the above it seems that several dietary factors (nutrients) may either promote or protect against colon cancer genesis, through a variety of possible mechanisms. The promotive factors are thought to be animal protein (meat) and fat, while starch, NSP, calcium and antioxidants seem to be protective. Therefore, diets high in fat and animal protein foods, low in fibre (NSP) and starch (provided by unprocessed cereals and grains) will be associated with an increased risk of colon cancer. Diet as a risk factor will be further explained in section 2.5.2.

2.5 RISK FACTORS FOR COLORECTAL CANCER

2.5.1 Introduction

Risk factors are factors that cause individuals of a population or group to be vulnerable to a specific, unpleasant, or unhealthful event. Several kinds of risk factors exist. In the physical environment there are toxins, drugs and infectious agents that may contribute to diseases. Disruption of the family environment such as the loss of a loved one is part of the social environment which may increase risk of some diseases. Behaviour is also a known risk factor. People's lifestyles are directly associated with their risk of developing certain diseases such as eating habits and smoking habits and physical inactivity. The inherited factors can not be excluded, since a person's tendency to develop a particular disease is greatly affected by the genetic background of that specific family member.

Exposure to risk factors for chronic diseases take place over a period of time. Chronic diseases therefore have long latency periods. Risk factors are used to predict the occurrence of disease. In this study, the impact of urbanisation on some of the risk factors for colorectal cancer which is rare among black South Africans but not in whites, have been examined.

The mechanisms through which some known risk and protective factors may influence development of colorectal cancer, have been discussed in 2.4. In this section, the epidemiological and clinical evidence that these factors are associated with risk of colorectal cancer, will briefly be reviewed. Emphasis is placed on those dietary risk and protective factors that could be measured in this study.

Table 2.5 gives known conditions associated with colorectal cancer, while Table 2.6 summarises studies which examined the relationship between diet and risk of colorectal cancer.

Table 2.5 The following conditions are associated with high risk for colorectal cancer:

FACTORS	REFERENCE
Age > 50 years	Winawer *et al.* 1996; Han *et al.* 1996; Bingham & Cummings, 1997
Familial polyposis coli	Itzkowitz, 1995; Ferrante, 1996; Knudson, 1995
First degree relative with colorectal cancer	Ferrante, 1996; Gelfand, 1996
Flat adenoma syndrome	Van't Hof *et al.* 1995
HNPCC (Lynch syndrome I)	Itzkowitz, 1995; Lee, 1992
History of colorectal neoplasia	Han *et al.* 1996
Turcot syndrome	Piper & Carr-Locke, 1992;
Ulcerative colitis (inflammatory bowel disease; Crohn's disease)	Bansal & Sonnenberg, 1996; Austoker, 1995)
History of adenomatous colonic polyps	Winawer *et al.* 1996
Familial adenomatous polyposis (FAP) (Gardner syndrome)	Winawer *et al.* 1996
Attenuated adenomatous polyposis coli (AAPC)	Winawer *et al.* 1996

2.5.2 Dietary risk factors

A large number of studies have examined the relationship between diet and risk of colorectal cancer. In Table 2.6, some of the more recent studies are summarised. These studies include longitudinal and cross-sectional epidemiological studies, studies in which populations with different rates of colorectal cancer are compared, as well as clinical intervention studies in both humans and animal models (mainly rats).

In addition to studies in which different foods and nutrients of the total diet have been related to colon cancer risk, effects of protein-rich foods, dietary fibre, NSP, starch, resistant starch, vegetables and fruits, dairy products, calcium, selenium, EPA, alcohol as well as conditions such as obesity and consumption have been examined.

It is not always possible to compare findings of different studies. Differences in study design, period of intervention, quality of dietary control and intake measurements, as well as end points measured, often complicate interpretation of data and comparison of results.

Nevertheless, from Table 2.6 it seems that there is evidence that intakes of dietary protein (especially meat), total fat, alcohol and possibly total energy,

may increase risk of colorectal cancer, while starch, RS, calcium, dietary fibre, NSP and foods rich in these (dairy products, unprocessed cereals, fruits and vegetables) have protective effects.

Alcohol

Excessive consumption of alcoholic beverages might be associated with an increased risk for cancer (Tomatis, 1990; Goldbohm *et al.* 1994). Associations have been observed between the frequency of cancer of the rectum and beer consumption (Goldbohm *et al.* 1994; Meyer & White, 1993; Newcomb *et al.* 1993). No association have been shown with cancer of the colon, except in the study by Newcomb *et al.* (1993) where liquor and colon cancer were related (Newcomb *et al.* 1993).

2.5.3 Other "environmental or behavioural" risk factors

2.5.3.1 Physical inactivity

Low levels of physical activity increases the risk of developing certain cancers such as colon, breast, and prostate (Walker, 1995). Total energy intake relative to energy requirement is related to occurrence of colon cancer (Giovannucci & Willet, 1994). Populations with high incidence of colon cancer are characterised by consumption of high dietary fat and calories which may be risk factors in the absence of factors that are protective (Reddy, 1993). In a study by Giovannucci *et al.* (1996), body mass index was associated directly with risk of large adenomas in the distal colon for BMI $\geq$ 29. Women with both a high body mass index (BMI) and high waist-hip-ratio (WHR) were at greater risk of large colon adenoma (Giovannucci *et al.* 1996). It is not known if exercise (activity) has a direct effect or indirect because of energy balance and obesity.

2.5.3.2 Non steroid anti-inflammatory drugs

There appears to be an association with the regular use of nonsteroidal anti-inflammatory drugs (NSAID) and reduced mortality from colon cancer (Müller *et al.* 1994).

Table 2.6 Reported associations between diet and risk of colon cancer

Diets/Foods/ Nutrients? Conditions examined	Nutrients/ Condition	Study design	Findings/effects	Author(s)
Total diet	Fat, meat, fibre Foods: Red-meat, beef, lamb, fat-vegetables, pork, poultry	Cohort (follow-up) men dentist, optometrist, osteopaths, podiatrist, pharmacists, veterinarians	High consumption of red meat increases the risk of colon cancer (distal). Fibre doesn't. Fat from dairy, poultry + vegetables inversely associated. Beef, pork - most strongly associated, lamb, red meat intake and colon cancer have an association. Fat consumption per se doesn't increase risk.	Giovannucci *et al.* 1994
	Protein=55g; fat= 52g; CHO=265g, fibre=13,5g	Epidemiological. Urban whites & Africans	Mean faecal pH values were lower in Africans. Faecal SCFA concentration are much higher, faecal pH is relatively lower.	Segal *et al.* 1993
	Western diet + wheat, oat or corn bran supplement	Epidemiological. 48 Women	All sources of dietary fibre increased the amount of faecal fat excreted. Wheat bran ↓ DAG and DAG containing lauric-, mysteric-, palmitic, stearic,-linoleic acid. Corn bran decreased the DAG containing stearic acid.	Reddy *et al.* 1994
	Alcohol, dietary fibre:cereal fibre, fruit fibre, vegetable fibre, calcium	Epidemiological. 1985-1989	No indication of an association between colon cancer and fat or protein consumption or dietary vitamins	Meyer & White, 1993
	Total energy intake, fat, protein, carbohydrate, dietary fibre, β-carotene, retinol, Vit. C	Epidemiological. Argentina	Carbohydrates are the most important factor driving the total energy effect. Other nutrients make little contribution to risk. There is a positive association between total energy intake and colon cancer risk. Fibre decreases this risk.	Iscovich *et al.* 1992
	Food constituents were analysed by a dietician	Epidemiological. 13 Males with previously sigmoid colon cancer surgery. 14 Males as controls with normal colon	Faeces of colon cancer patients were abundant in colon cancer promotors. Appeared unrelated to food constituents. Microflora play a key role in carcinogenesis by producing promoters	Kanazawa *et al.* 1996
	Effect of diet diversity	Epidemiological. Western New York	Total diversity was positively related to colon cancer risk	McCann *et al.* 1994
	70 Foods and beverages	Epidemiological. Southern Italy	The local Mediterranean dietary pattern could explain the low risk of colorectal cancer	Centonze *et al.* 1995
	Fat, alcohol, sucrose, fibre, vegetables, other vitamins, meats, poultry, breads, sweets and yoghurt	Epidemiological. Los Angeles	Crude fibre was protective in the ascending colon but not even weakly protective in the distal colon	Peters *et al.* 1992

Table 2.6 (Continued)

Diets/Foods/ Nutrients? Conditions examined	Nutrients/ Condition	Study design	Findings/effects	Author(s)
Protein-rich foods	Test meals: Cheese Small/large fried beef stake Large steak with RS	Clinical 5 Subjects with ileostomies - basal diet fed 5 days	Poor digestibility of meat doesn't explain the association between meat intake and large bowel cancer risk. Relation of meat intake and large bowel cancer may reflect higher protein intakes in meat eaters or may be explained by other mechanisms.	Silvester & Cummings, 1995
	Meat, poultry, seafood, dairy products and eggs.	Epidemiological. Adelaide case-control	High consumption of red meat-weakly associated with increased risk for colon cancer. High intakes of liver, seafood and eggs appeared to be risk factors for females but not for males, high intake of poultry increased risk for colon cancer for males. Egg consumption – stronger for cancer of the proximal colon, maybe dietary cholesterol plays a role in the aetiology of colon cancer	Steinmetz & Potter, 1993 (b)
Dietary fibre: Humans	Low fibre wheat, whole meal wheat, whole grain rye	Clinical. Different types of bread. 12 Finish women	The type of bread significantly affects the concentration of co-carcinogenic and co-mutagenic free lithocholic and deoxycholic acids by changing modes of conjugation in the gut	Korpela *et al.* 1992
	Fibre groups: wheat, oat or corn bran supplements. In the form of muffins	Clinical intervention. 78 Women	Wheat bran ↓ faecal deoxycholic and lithocholic acid, 12-ketolithocholic acid and neutral sterols and the activity of all bacterial enzymes. Oat bran had no effect on secondary bile acids and 7α-dehydroxylase, lithocholic acid, and cholesterol levels and ↓ deoxycholic acid coprostanol, cholestenone, nitroreductase and azoreductase levels.	Reddy *et al.* 1992
	Wheat bran fibre cereal + Ca supplements	Clinical. Men and women who had a complete colonoscopy	High dose wheat bran fibre and Ca^{2+} supplements are associated with reductions in both total and secondary faecal bile acid concentrations and excretion rates in patients with resected colon adenomas. This study supports the hypothesis that a high intake of wheat fibre and Ca may reduce the risk of colorectal cancer and cancer, by reducing the concentration of faecal bile acids.	Alberts *et al.* 1996
	Whole grain intake	Meta-analyses. All case-control.	Whole grain intake is not associated with reduced risk of colon cancer.	Jacobs *et al.* 1996
	Dietary fibre + fermentation to SCFA's	Epidemiological	Butyrate production to total SCFA's was lower in patients with colonic cancer and adenomas compared with healthy subjects	Clausen *et al.* 1991
	Sodium butyrate and fibre supplementation	Clinical. Human colonic tumour cell lines	Sodium butyrate is produced during fermentation of fibre. The observation that this fatty can induce apoptosis could in part explain why a diet in fibre appears to be protective against colon cancer	Hague *et al.* 1993

Table 2.6 (Continued)

Diets/Foods/ Nutrients? Conditions examined	Nutrients/ Condition	Study design	Findings/effects	Author(s)
Dietary fibre: Animals	Elemental diet and diet with 30% mixed fibre	Clinical study. Rats fed elemented diet or elemented diet with fibre	Dietary fibre consumption increases substrate oxidation by isolated colonocytes but not distal small intestinal enterocytes	Marsman & McBurney, 1995
	Experimental diets. Fibre free diet, elemented diet + fibre	Clinical. Rat colonocytes	Fibre increases in vitro colonocyte proliferation, the unchanged oxygen uptake rate indicates that there was no concurrent increase in energy expenditure	Marsman & McBurney, 1996
	Dietary fibre - pulverised chow + wheat bran	Clinical. Rats	Shortening of the transit time through the caecum and colon with increased faecal volume and suppression of fat absorption participate in the inhibitory action of wheat bran on carcinogenesis	Munakata *et al.* 1995
NSP	Nonstarch polysaccharides (NSP)	Epidemiological.	Diets high in NSP are associated with stool weights of 150g/day and should reduce the risk of bowel cancer.	Cummings *et al.* 1992
Starch	Starch fermentation and bile acid metabolism	Epidemiological	Formation of secondary bile acid by bacteria is inhibited when starch is simultaneously fermented, an effect that is mainly, but not completely explained by reduction of pH	Christl *et al.* 1995
Resistant starch	High and low RS diets, healthy subjects 3 meals for 1 day	Clinical	RS may have effects comparable with those of some fermentable dietary fibres	Muir *et al.* 1995
	Diets high in RS or low RS for 3 weeks	Clinical. 11 Subjects	RS significantly attenuates the accumulation of potentially harmful by-products of protein fermentation	Birkett *et al.* 1996
	Low RS diets, with or without cholesterol, to which bile acid binding resin cholestyramine was added (reference). High RS diets	Clinical. Rats	Serum cholesterol lowering effect of high RS diets may be explained by an increased influx of neutral steroids and bile acids into the caecum, and increased faecal excretion of bile acids, and/or by an altered intestinal bile acid profile.	Verbeek *et al.* 1995

Table 2.6 (continued)

Diets/Foods/ Nutrients? Conditions examined	Nutrients/ Condition	Study design	Findings/effects	Author(s)
Vegetables and fruits	Vegetables - ↓ risk, fibre not only for this association. Meat - ↑ risk, but not solely by its fat content	Epidemiological	Variety of links between dietary epidemiology and physiology of colorectal cancer	Potter, 1996
	Vegetable and animal products	Epidemiological. Dutch case-control	This study supports the preventative potential of a high-vegetable diet in colon cancer risk. Play an important role in women who consumes a diet high in red meat	Kampman *et al.* 1995
	Vegetable, fruits	Epidemiological. Uruguay	A reduction in risk for total vegetable, fruit intake and lettuce, apple and banana intake was observed. The strongest protection was observed for banana intake	Deneo-Pellegrini *et al.* 1996
	Vegetables + fruits	Epidemiological. Adelaide case-control	High intakes of onions, legumes, cabbage, green leafy vegetables and raw fruit were inversely associated with colon cancer in this population based study. Consumption of potatoes appeared to be a positive risk factor	Steinmetz & Potter, 1993 (a)
Dairy foods (products)	Calcium, Vitamin D, Diary foods	Cohort. Same as above	No important association of Ca intake and occurrence of colorectal adenomas. Slight support for Vit. D intake protection.	Kampman *et al.* 1994 (a)
	Fermented diary products: Buttermilk, yoghurt, curds & kefir Hard cheese: Gouda Unfermented diary products Whole fat milk, slim/low fat milk, custards, evaporated milk and porridge	Epidemiological case-control. Netherlands	Do not support the hypothesis that an increased intake of commercially available, commonly used fermented diary products or dietary calcium decreases the risk of colon cancer	Kampman *et al.* 1994 (b)
Calcium	Supplemented subjects with 2000-3000 mg Ca for 16 weeks	Clinical. 22 Subjects	Calcium protects against colon cancer. Calcium decreased chenodeoxycholic acid in bile. The concentration of primary bile acids in faecal water decreased after 16 weeks after Ca supplementation.	Lupton *et al.* 1996
Selenium	Selenium levels	Epidemiological.	Selenium levels are lower in subjects affected with colon cancer, there are differences in Se levels between the healthy and neoplastic tissues.	Caroli *et al.* 1994
	Serum selenium	Epidemiological	No trend could be detected toward a protective effect of selenium for colonic benign or malignant tumours	Nelson *et al.* 1995
EPA	Eicosapentaenoic acid supplementation	Clinical. Ten healthy subjects, EPA supplementation	Dietary supplementation with EPA reduces breath H_2 excretion without apparently impairing overall colonic carbohydrate fermentation	Thompson & Spiller, 1995

Table 2.6 (continued)

Diets/Foods/ Nutrients? Conditions examined	Nutrients/ Condition	Study design	Findings/effects	Author(s)
Alcohol	Alcoholic beverages, beer, red wine, white wine, sherry, liqueur, gin, brandy, whisky.	Cohort. 120 852 men and women	Consumption of beer particularly is associated with an increased risk for rectal cancer than colon cancer	Goldbohm *et al.* 1994
	Beer, wine, liquor	Epidemiological. Wisconsin USA	Beer was associated with rectal cancer. Wine consumption was inversely associated with these cancers. High intakes of alcohol was associated with a increased risk of large bowel cancer	Newcomb *et al.* 1993
Obesity	Obesity	Clinical. Endoscopic examination of 13 057 nurses in USA	Inverse association exists between physical activity and the occurrence of colon cancer, obesity associated with elevated risk of large adenomas.	Giovannucci *et al.* 1996
Constipation	Constipation	Clinical. 35 Patients with severe constipation	Constipation is a disorder of defecation rather than an impairment of colonic motility	Karasick & Ehrlich, 1996
Cooking methods	Key nutrients. Mode of cooking	Epidemiological	Dietary factors play a key role in causation and prevention of colon cancer. 25% fat + 25 g fibre recommended	Wynder *et al.* 1992

* Dietary fibre : Term used to describe total fibre or cell-wall material in plant foods not digested by human enzymes
Dietary fibre = NSP + lignin (FAO/WHO, 1998)

** NSP : Non-starch polysaccharides

*** RS : Resistant starch. Starch that is not absorbed in the small intestine of healthy humans (FAO/WHO, 1998)

2.6 PREVALENCE AND INCIDENCE OF COLORECTAL CANCER

2.6.1 COLORECTAL CANCER IN SOUTH AFRICANS

2.6.1.1 The South African population structure

At present mortality rates in South Africa are stratified according to population groups. The different populations will be briefly discussed.

South Africa's population is heterogeneous and multinational. Each major population group, namely, blacks, whites, coloureds and Indians has its own cultural heritage and language. After 1948, strict laws were passed to enforce total segregation (so called apartheid) and to minimize social contact between people of different colours and races.

International efforts and protests by many local South Africans moved the government toward modifying "apartheid" policies, abolishing many of the laws restricting blacks, coloureds and Indians. A majority of African or black South Africans resided in homelands, namely the Transkei, Lebowa, Gazankulu, Venda, Bophuthatswana, Quaqua, KwaZulu and Ciskei. In the late 1970's and 1980's, four of these homelands got their independence and became separate republics. The four independent homelands were reabsorbed by South Africa following the 1994 democratic elections. The Republic of South Africa was divided into 9 Provinces, namely; Northern Transvaal, Mpumalanga, Gauteng, Northwest, Free State, Kwazulu/Natal, and Eastern Cape, Northern Cape and Western Cape.

The people of South Africa are categorised into four groups: blacks (Africans), the largest group; whites, coloureds and Asians (mainly Indians). The 1995 mid year estimated census indicated that the distribution of the different population (ethnic) groups are as follows:

Population	Percentage
Blacks (Africans)	**76%**
Whites (Europeans)	**13%**
Coloureds (Mixed origin)	**9%**
Asians (mainly Indian)	**3%**

Central Statistical Services: Census, 1996.

2.6.1.2 Prevalence and incidence of colorectal cancer in South Africa

In recent years there have been major decreases in mortality rates from the two cardiovascular diseases, but this was not the case for cancer (Walker, 1995; Walker & Segal, 1997). Mortality rates from cancer are high and even increasing as with the incidence rates of some cancers (Walker, 1995). The low incidence of adenomatous polyps in the African population suggests that there may be an alternative route for developing colorectal cancer (van't Hof *et al.* 1995).

Numbers and incidence

Table 2.7 indicates that a total of 1034 males and 1025 females were reported with colorectal cancer in 1992 (Sitas, 1997). In white males the lifetime risk was 1 in 40 and in black males 1 in 357 (Sitas, 1997). Colorectal cancer ranked 12th in black males and 3rd in white males. It was the most common cancer in Asian males and 8th most common in coloured males. In females, it was the 7th most common in blacks, 2nd most common in whites, 3rd in coloured and 4th in Asians (Sitas, 1997).

Table 2.7. Summary statistics for colorectal cancer in South Africa for the year 1992.

	N	%	CRUDE	ASIR	CUM RISK	LR
African female	202	2.01	1.38	2.12	0.26	385
African male	203	2.24	1.37	2.31	0.28	357
Coloured female	70	4.61	4.08	6.28	0.72	139
Coloured male	54	3.76	3.30	6.53	0.81	123
White female	576	5.41	22.40	16.50	1.94	52
White male	587	4.75	23.06	21.02	2.50	40
Asian female	32	5.21	6.31	8.78	1.06	94
Asian male	43	10.02	8.63	12.83	1.40	71
Female	1025	4.08	5.27	7.25	0.86	116
Male	1034	3.99	5.32	9.18	1.10	91

(Addapted from Sitas, 1997)

N - Number of cases, 1992
ASIR - Age standardise incidence rates, World standard/100 000
CUM RISK - Cumulative risk, expressed as one in x number of people
LR - Lifetime risk
CRUDE - Crude cancer incidence rate/100 000
% - Percentage of cancer cases per group

Table 2.8 summarises the top 20 causes of death in South Africa during 1990 (Bradshaw *et al.* 1995)

Table 2.8 The top 20 causes of death in South Africa, 1990. (total deaths = 199 537)

CONDITION	*PERCENTAGE*
Sign/symptoms/ill-defined	23.33
Other respiratory	9.58
Cerebrovascular	7.35
Certain perinatal	6.22
Ischaemic heart disease	4.86
Pulmonary circulation	4.76
Tuberculosis	4.61
Digestive cancer	3.99
Intestinal infections	3.85
Other violence	3.82
Other digestive	2.91
Endocrine/metabolism	2.90
Respiratory cancer	2.02
Genitourinary cancer	1.84
Nervous system	1.82
Other bacterial	1.73
Urinary system	1.52
Hypertensive disease	1.50
Homicide	1.49
Nutritional deficiency	1.19

(Bradshaw *et al.* 1995)

Nearly 4 % of the total deaths are from digestive cancers in South Africa (Bradshaw *et al.* 1995). It must be emphasised that this is the total for all the digestive cancers and not for colorectal cancer alone.

2.6.2 COLORECTAL CANCER GLOBALLY

Globally, about 6 million deaths in 1990 were the result of cancer, nearly half of this were men (Murray & Lopez, 1996). By 1990, there were already 50 % more cancer deaths in less developed countries compared with developed countries (Murray & Lopez, 1996). Of this, 472 000 deaths globally, are the result of colorectal cancer (Murray & Lopez, 1996). Tables 2.8 to 2.14 give incidence and prevalence figures for colorectal cancer in males and females in established market economies, in Sub-Saharan Africa as well as globally.

Table 2.9 Incidence & prevalence for colorectal cancer in established market economies, for males, 1990.

	Incidence		Prevalence	
Age group	N (10^3)	Rate (100 000)	N (10^3)	Rate (100 000)
0-4	0	0.0	0	0.1
5-14	0	0.0	0	0.2
15-44	8	4.2	31	17.0
45-59	43	65.3	175	265.2
60+	208	343.8	809	1336.6
All ages	259	66.4	1016	260.2

Table 2.10 Incidence & prevalence for colorectal cancer in established market economies, for females, 1990.

	Incidence		Prevalence	
Age group	N (10^3)	Rate (100 000)	N (10^3)	Rate (100 000)
0-4	0	0.0	0	0.0
5-14	0	0.0	0	0.0
15-44	7	4.1	30	17.0
45-59	33	48.4	134	197.2
60+	200	236.0	746	882.2
All ages	240	58.9	910	223.4

Table 2.11 Incidence & prevalence for colorectal cancer in Sub-Sharan Africa, for males, 1990.

	Incidence		Prevalence	
Age group	N (10^3)	Rate (100 000)	N (10^3)	Rate (100 000)
0-4	0	0.0	0	0.1
5-14	0	0.1	0	0.3
15-44	2	1.6	5	5.2
45-59	2	11.2	7	36.3
60+	8	78.2	26	244.0
All ages	12	4.9	39	15.3

Table 2.12 Incidence & prevalence for colorectal cancer in Sub-Sharan Africa, for females, 1990.

	Incidence		Prevalence	
Age group	N (10^3)	Rate (100 000)	N (10^3)	Rate (100 000)
0-4	0	0.0	0	0.0
5-14	0	0.1	0	0.2
15-44	1	1.4	5	4.5
45-59	2	8.0	6	26.0
60+	8	64.7	25	194.6
All ages	12	4.5	35	13.7

Table 2.13 World incidence & prevalence for colorectal cancer, for females, 1990.

	Incidence		Prevalence	
Age group	N (10^3)	Rate (100 000)	N (10^3)	Rate (100 000)
0-4	0	0.1	0	0.1
5-14	1	0.2	3	0.6
15-44	37	3.0	136	10.9
45-59	102	32.5	388	124.2
60+	375	171.3	1389	634.8
All ages	515	19.4	1917	72.2

Table 2.14. World incidence & prevalence for colorectal cancer, for males, 1990.

	Incidence		Prevalence	
Age group	N (10^3)	Rate (100 000)	N (10^3)	Rate (100 000)
0-4	0	0.1	0	0.1
5-14	1	0.2	4	0.7
15-44	30	2.5	112	9.4
45-59	85	27.4	324	104.3
60+	362	134.5	1294	480.6
All ages	478	18.3	1734	66.4

The incidence measures the occurrence of new cases of colorectal cancer in 1990. The estimated number (N) of new cases are shown in the first column of each table as well as the estimated incidence rate expressed per 100 000 population per year. It is also very important to estimate the amount of colorectal cancer actually present in each region in 1990 (Murray & Lopez, 1996). From these tables (2.8-2.14) it is clear that for both men and women, colon cancer incidence and prevalence in Sub-Saharan Africa are substancially lower than in the established market economies. In men the global prevalence of colon cancer is 1917 per 100 000. The established market economies contribute approximately 53% to this burden, and Sub-Saharan Africa only 2%.

CHAPTER 3

3.1 OBJECTIVES OF THE STUDY

It is clear that South Africans are entering a new health era. Diseases of lifestyle are emerging in our black population as a result of a changing physical and social environment. The environment changes during urbanisation. The literature review has indicated that many cancers arise due to environmental events which act on the genetic properties of a person. Certain genes have been known to contribute to inherited cancers such as the APC gene, *hMSH2*, *hMLH1*, *hPMS1* and *hPMS2*. During the process of urbanisation these genes may be activated by a change in the social or physical environment. The genetic - environmental interaction in the development of colorectal cancer has been discussed in the literature review. It became clear that dietary factors, because of effects on colonic fermentation, butyrate production and microbial mass, faecal mass and transit times, binding of bile acids, and inhibition of the fermentation of secondary bile acids may play an important role in colorectal carcinogenesis. There is convincing evidence that regular physical activity and vegetable intake protect against colorectal cancer. There is also evidence that fibre, NSP, starch, RS, dairy products, selenium as well as EPA, may protect against colorectal cancer, while protein-rich foods, alcohol, obesity, constipation and some high-temperature, high-fat cooking methods may be promotive (WCRF, 1997).

Rural people moving from deep rural areas and farms to big cities have an inevitable change in their diet. However, these changes in dietary and nutrient intakes related to colorectal cancer have not been well described for Africans in the Northwest Province in South Africa. Therefore, the objective of this study was to describe the changes in dietary patterns and nutrient intakes of Africans in this Province in order to explore the possibility that a dietary factor known to be related to colorectal cancer risk may be responsible for the non-emergence of this disease in urbanising Africans. It

is possible that protective factors do not change or are enhanced, or that promotive factors are still low in urban populations.

As stated in chapter 1 (1.3) the major objective of this study was to measure the impact of urbanisation on some of the known dietary risk factors of colorectal cancer in order to develop a hypothesis that might explain the low rates in Africans. The model and study chosen, was a cross-sectional epidemiological survey of adult Africans (15 to 65 years of age), stratified for level of urbanisation. Subjects at different levels of urbanisation were compared. The levels of urbanisation were based on the places/areas in which subjects lived at the time of the study and are defined in chapter 4. A longitudinal design in which risk factor changes are monitored over time of subjects that have moved from rural to urban areas, is probably the most ideal design to study the problem, but could take years since risk factors develop over time. Because socio-economic factors such as personal and household income, level of education, number of people living in the household, household food security and medical history were measured, it was thought that a cross-sectional study could provide valid information on the effect of urbanisation and changing lifestyles on dietary patterns and nutrient intakes. The methods employed in this study are given in chapter 4.

CHAPTER 4

METHODS

4.1 INTRODUCTION

Because this study formed part of a larger epidemiological survey (the THUSA study), not all measurements were done by the researcher. Therefore, the group of colleagues or fieldworkers who were mainly responsible for the measurements of which the results are used in this dissertation, will be mentioned in the discussion of each method. For the purpose of this dissertation, the results obtained from the first 889 of the intended 2 500 subjects of the THUSA study are used. The researcher took responsibility for the blood samples, the "colon questionnaire", collection of faeces samples and preparation of cells from these. However, the results of the research on the faecal samples are not reported in this dissertation, and since the methods are confidential, they will not be given here.

4.2 DESIGN OF THE STUDY

This study formed part of the long-term THUSA project in which the prevalence and severity of known risk factors for NCD's and the determinants of these risk factors in Africans of the Northwest Province at different levels of urbanisation are monitored. A cross-sectional representative sample of apparently healthy individuals from the African population was recruited and stratified for gender, age and stratum (level of urbanisation) into 50 different cells (see Figure 4.1).

4.3 SELECTION OF SUBJECTS

4.3.1 Number of subjects (sample size)

Based on the largest variation in (vitamin A and C) intakes reported in the VIGHOR study for subjects on a typical western diet (Vorster *et al.* 1995), it was calculated that at least 50 subjects will have to be included per cell to show significant differences between cells in nutrient intakes of subjects eating a wide variety of foods. It is therefore possible that in subjects following a more limited diet with less variety and smaller variation in intakes, smaller numbers will show significant differences. However, because it was expected that the diet of urban Africans may be very similar to the typical western diet, it was decided to include 50 subjects per cell in the original study design.

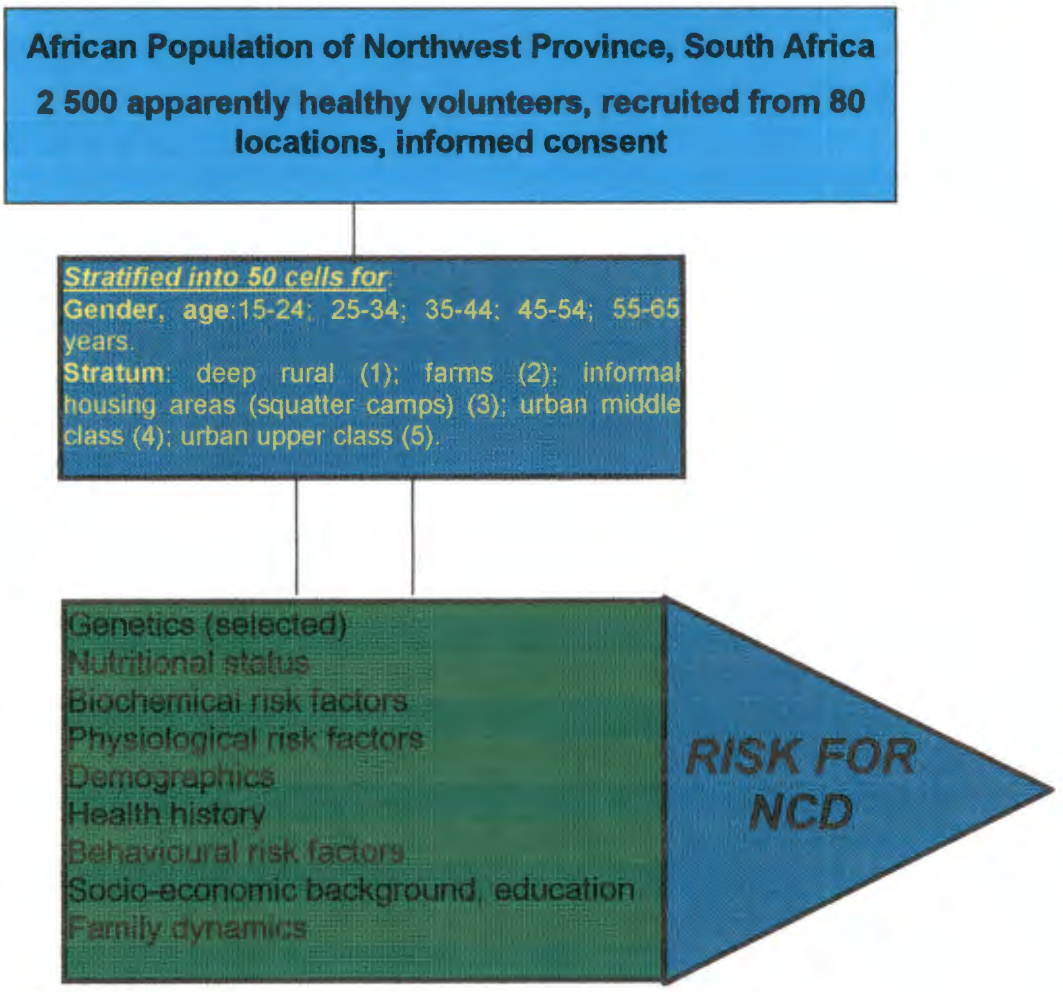


Figure 4.1 Design of the THUSA study

4.3.2 Age ranges

NCD's are diseases of adulthood but may start developing in childhood. It was therefore decided to include subjects from 15 to 65 years of age in the sample. Older persons were not included because of the pronounced effect of age on most of the NCD's. Subjects were grouped into 5 age groups (15-24.9; 25-34.9; 35-44.9; 45-54.9; 55-65 years) as indicated in figure 4.1.

4.3.3 Randomisation and representativeness

Because of several logistic reasons selection of a total randomised sample of Africans in the Northwest Province was not possible. With the help of the Biostatistic Consultation Services of the PU vir CHO, a model was designed to recruit a representative sample. The Northwest Province was divided into 4 quarters and 5 centres in each quarter were randomly chosen. Subjects were recruited from 4 different locations in or around each centre. The recruitment of 2500 subjects therefore will be from 80 different locations around 20 randomly chosen centres throughout the Province. The requirements for each centre were that a temporary field laboratory could be set up centrally for all 4 different locations, and that suitable accommodation for a team of 20 researchers was available. Furthermore, the 5 centres within each quarter of the Province should have given access to subjects from all 5 strata or levels of urbanisation. In the part of the study reported here, 889 subjects were recruited from 18 different locations.

4.3.4 Criteria for defining the strata

Stratum 1: Rural people

Usually, rural depicts pastoral or agricultural circumstances. In this study, rural Africans were defined as people who were living under the authority of a traditional

tribal head (chief, captain or headman) in and around traditional African villages, mostly without running water and electricity.

Stratum 2: Farm workers

These subjects worked on commercial farms, mostly owned by white farmers, and lived in brick or mud houses clustered together on the different farms. Running water and electricity were available at some, but not all farms.

Stratum 3: Squatters

These subjects were from the informal housing areas (squatter camps) near larger towns and lived in temporary houses made from corrugated iron and other materials (makuku's). Electricity and communal water taps were available in some of these areas.

Stratum 4: Urban, "middle class"

These subjects were from established townships or locations found adjacent to all towns and cities in South Africa. The subjects lived in their own brick houses and most had running water and electricity.

Stratum 5: Urban, "upper-class"

Subjects for this stratum were recruited from professional people (nurses, teachers, doctors, governmental workers, politicians, etc.) and lived either in the established townships or in formerly "white" residential areas in towns and cities.

4.3.5 Exclusion criteria

The sample consisted of "apparently healthy" subjects. Therefore, the following subjects were not included in the study:

- Pregnant and lactating women

- Subjects known to suffer from any disease (infectious or NCD's)
- Subjects using any form of chronic medication (many subjects known to be hypertensive, were excluded because of this criterium)
- Subjects younger than 15 and older than 65 years (identification documents were used to obtain correct age)
- Inebriated subjects
- Subjects suffering from epilepsy and mental diseases or defects.

4.4 ETHICAL CONSIDERATIONS

The study was approved by the Ethics Committee of the PU vir CHO (ethics number: HHK4M5-95). Subjects had to sign an informed consent form giving permission to draw blood samples. In many instances, especially in strata 1 to 3, subjects were illiterate and signed with a cross.

4.5 ORGANISATIONAL PROCEDURES

- After a location (centre) has been chosen, two researchers visited the area and identified a suitable person, from the Department of Health Regional Offices, clinics, schools, the Tribal Office, or a community leader to help with the organisation of the field visits and recruitment of subjects.

- The necessary permission to do the study was obtained from the relevant authorities (tribal chief, mayor, clinic head, hospital matron, secondary school headmaster, or employer of subjects).

- The local organiser in each centre helped to identify the four locations around the centre where subjects were recruited and studied. This person arranged a meeting between the researchers and the community during which the objectives and methods of the study were explained and questions answered. The local organiser also arranged that the subjects voluntarily fasted for 8-12 hours and that they bring their identification documents on the day of the study. In the case

of schoolchildren, teachers were asked to distribute informed consent forms among volunteers to take home for parental approval and signatures.

- Each local organiser received detailed written instructions (Addendum 1) to assist in the recruitment of subjects. This person also received copies of a brochure (Addendum 2) motivating and explaining the study, with detailed information, requirements and instructions for distribution among possible subjects.

- The local organiser advised the researchers on reimbursement of subjects. It was clearly stated that subjects will not be paid to participate, except those subjects who had to keep 7 day weighed dietary records and collect 24 hour urine for the dietary validation study. These subjects also received a packet of dry beans, donated by the Dry Bean Producers Organisation. In one rural area (4 locations) subjects were given clothing (donated by an international relief organisation) and in another 8 locations subjects received a packet of fruit after completion of the study.

- To avoid recruitment of ill subjects, subjects were not recruited from community or hospital clinic attendees. Hospital nurses, clinic sisters and administrative staff from the Department of Health were, however, recruited as part of subjects in stratum 5.

- During the field study, a temporary field laboratory was established in each centre for processing of blood samples and measurements of haematocrit and haemoglobin.

- The researchers stayed at the chosen centre and travelled daily to each location with the necessary equipment and ice to keep the samples at 4°C. If the location was too far from the centre to process the samples within 3-6 hours, centrifuges

(with generators where necessary) were taken to the location for processing the samples in the field.

- On the morning of each field visit, the study was explained in detail to all subjects intending to participate, in their home language. Subjects were then asked to sign (signature or cross for illiterate subjects) the informed consent form (Addendum 3). Schoolchildren had to hand in consent forms signed by a parent or guardian.

- Each subject then received a "green card" (Addendum 4) with their name and subject number, that had to be filled in (or ticked off) by the researchers at each of the following "stations" where information, measurements and samples were collected:

Station 1: Demographic questionnaire (Addendum 5).

Station 2: Dietary questionnaire (Addendum 6)

Station 3: Anthropometric measurements (Addendum 4)

Station 4: Clinical signs of malnutrition, blood pressure, oral temperature and blood samples (Addendum 4).

Station 5: Urine sample.

Station 6: Two hour glucose tolerance tests (Addendum 4).

Station 7: Reproductive questionnaire (only women, Addendum 7).

Station 8: Cardiovascular reactivity (stress test). Two out of every 3 subjects were asked to participate in measurements of cardiovascular reactivity and hormonal responses after a stress test (dynamometer). Subjects with systolic blood pressure > 170 mmHg and diastolic pressure > 110 mmHg were excluded.

Station 9: Colon questionnaire for selected subjects (Addendum 8).

4.6 DIETARY INTAKES

A quantitative food frequency questionnaire (FFQ) was developed by Ms. U. MacIntyre as part of her Ph.D. studies. A pilot study was conducted to assess the types of foods usually eaten by Africans in the Northwest. The FFQ (Addendum 6) was validated by comparing reported intakes of a subsample of 100 subjects with seven day weighed records and 24 hour urinary nitrogen excretions (MacIntyre *et al.* 1998). Books of photographs of three different portion sizes of the foods most frequently eaten, were developed. Five Setswana speaking fieldworkers were trained to use the FFQ together with these books, food models, food containers and package materials, as well as household utensils to measure habitual intakes. This was done in individual interviews with each subject. The questionnaire also included questions to evaluate level of acculturation, meal frequency, alcohol intakes, snacking habits and where meals are usually eaten. The reported food intakes were coded by an experienced dietician and analysed with a computer programme based on the South African Food Composition Tables (Langenhoven *et al.* 1991).

4.7 DEMOGRAPHIC, MEDICAL, REPRODUCTIVE AND SOCIO-ECONOMIC INFORMATION

Several members of the Nutrition Research Group used a questionnaire (Addendum 5) during individual interviews with subjects to obtain information on age, home language, medical history, prevalence of NCD's in family members, smoking habits, alcohol intake, own and family income, housing, ownership of property, occupation, education and activity levels. Additional interviews were held with all female subjects to obtain information on reproductive issues (addendum 7) such as age of menarche, first parity, and menopause, type and period of contraceptive usage, number of children, breast feeding history and family history of breast cancer. Subjects who volunteered a stool sample in the latter part of the study were

interviewed by the researcher to gain information on bowel habits, using the questionnaire given as Addendum 8.

4.8 ANTHROPOMETRIC MEASUREMENTS

Anthropometric measurements were made by Ms. A. Coetzer and Mrs. S. James as part of their post-graduate studies. The information was coded on the anthropometric data sheet (Addendum 10).

- Weight was measured to the nearest 0,5 kg with a portable electric balance (Precision Health Scale, A & D Company, Japan). Subjects were barefoot and wore only light indoor clothing.

- Height was measured to the nearest 0,5 mm using a plastic anthropometer (Invicta, IP 1465, UK). Subjects were barefoot or wore only socks.

- Waist and hip circumferences were measured with an unstretchable measuring tape around the smallest and widest parts of the waist and hips respectively.

- The triceps and subscapular skinfolds were measured with a skinfold calliper (John Bull, British Indicators, Ltd) in triplicate, and mean values calculated and reported.

4.9 CLINICAL EXAMINATIONS

Two specially trained nursing sisters (Mrs. C. Lessing and Mrs. A. Kruger) examined the thyroid, hair, skin, tongue and eyes of subjects for any signs of malnutrition. They also took oral temperatures and blood pressures while subjects were seated for at least 10 minutes. The researcher assisted with blood pressure measurements. A sphygmomanometer (Tycos®; USA) with adjustable cuffs of different sizes was used. Systolic and diastolic blood pressure were recorded as the first and fifth

Korotkoff sounds. The lowest blood pressure recording of the two or more measurements was used.

4.10 GLUCOSE TOLERANCE TEST (GTT'S)

Two hour oral GGT's were performed on each subject (by Mrs. M. Van Staden as part of her Ph.D. study) after blood samples were taken. A load of 50g glucose (Alpha® Glucose Powder; Allied Pharmaceuticals) dissolved in 200ml water per subject was used. In addition to serum glucose determined later in the laboratory, glucose concentrations of blood obtained by a finger prick before and after 120 minutes of the test were measured in the field in order to give immediate feedback to subjects. A glucometer (Ames Glucometer®G_x; Miles, USA)) and glucose strips (Glucostix®, Bayer Diagnostics; Ames, UK) were used.

4.11 BLOOD SAMPLES

- Blood samples were drawn from the *vena cepahalica* using a sterile butterfly infusion set (Johnson & Johnson, 21G, 19 mm) and syringes. Serum and plasma were prepared by the researcher and Ms. H. Burger. For the preparation of serum, 50 ml of blood was allowed to clot in glass tubes, centrifuged at 3000 rmp for 15 minutes. For each subject 30 x 1 ml aliquots were frozen at -20°C for 1-4 days and then at -84°C in the laboratory. Citrated blood samples (0,5 ml 1 mol/l citrate, pH 4,5 to 4,8, plus 4,5 ml venous blood) were centrifuged in plastic tubes at 300 rpm for 10 minutes. The citrated plasmas were divided into 5 aliquots, stored at -20°C for 1-4 days and then at -84°C. Five ml of blood was transferred to EDTA glass tubes. Blood cells and EDTA plasma were separated by centrifugation (3000 rpm for 10 minutes) and aliquots of plasma and cells stored separately, first at -20°C for 1 to 4 days and then at -84°C.

- The butterfly system was kept in the vein of those subjects participating in the cardiovascular reactivity test for blood sampling and serum preparation during

and after the stress test. The system was kept viable with a solution of 3 units heparin per 5 ml saline.

- For DNA analyses, whole blood of each subject was pipetted in four drops (4 x 200 µl) onto blotting paper, dried and sealed in separate envelopes.

4.12 BIOCHEMICAL ANALYSES

Table 4.1 gives the different variables determined and methods used in the analyses of the blood, serum and plasma samples. The normal ranges for adults with the appropriate references are also given in Table 1. Haemoglobin and haematocrit of whole blood were determined in the field by the researcher and Dr. FJ. Veldman, using the colorimetric method from Boehringer Mannheim (Germany) and a haematocrit centrifuge (Hettich Zentrifugen, Haematocrit 24 D-78532, Tuttlingen) respectively.

The serum analyses (DAX: discrete analyzer, Technikon DAX 48) were done in the laboratory of the Department of Chemical Pathology of the University of Pretoria. This laboratory makes use of the Thistle and Murex quality control methods.

TABLE 4.1: Blood, serum and plasma analyses.

VARIABLE	*METHOD AND SUPPLIER OF REAGENTS*	*NORMAL RANGE*
BLOOD:		
Haematocrit	Capillary tube (Marienfeld, Germany) and haematocrit centrifuge (Hettich Zentrifugen 24, D-78532, Tuttlingen)	Men: 39-49% Women: 33-44%
Haemoglobin	Colorimetric (Boehringer Mannheim)	Men: 14-18 g/dl Women: 12-16 g/dl
SERUM:	DAX Profile (Discrete analyzer."Technikon DAX 48"	
Sodium		137-144 mmol/L
Potassium		3.6-4.7 mmol/L
Chloride		98-108 mmol/L
CO_2-content		23-29 mmol/L
Anion gap		7-14 mmol/L
Urea		3.6-7.8 mmol/L
Urate		0.31-0.47 mmol/L
Creatinine		81-114 mmol/L
Phosphate		0.6-1.4 mmol/L
Calcium		2.20-2.60 mmol/L
Corrected calcium		2.20-2.60 mmol/L
Magnesium		0.70-0.89 mmol/L
Total protein		66-79 g/L
Albumin		39-50 g/L
Globulin		
Bilirubin : T		4-30 µmol/l
Bilirubin : D		4-30 µmol/l
ALP:		0-4 IU/L
GGT:		40-104 IU/L
ALT:		12-43 IU/L
LD:		60-200 IU/L
CK: Creatine kinase		45-165 IU/L
Total cholesterol		3.40-5.40 mmol/L
Triglyceride		< 2 mmol/L
HDL-cholesterol		>1 mmol/L
LDL-cholesterol		< 3.5 mmol/L
Osmolarity (mmol/l)		275-295
Glucose		3.5-6.2 mmol/L
Amylase		34-126
Iron		10-30 µmol/l
Ferritin		> 8,5 µmol/l
Pyridoxalphoshate		20-80 mmol/l
Vitamin B_{12}		118-716 pmol/l
Homocystein		-
Folic acid		2-10 ng/ml
Red cell folate		> 272 µmol/l
Transferrin		2-3.2g/l
Vitamin A		30-65 µg/dL
Retinol binding protein		3-6 mg/dL
PLASMA		
Fibrinogen (g/l)	Ratnoff & Menzies, & Clauss	200-400mg/dL
Macromolecular protein complex (MPC)		-

4.13 STATISTICAL ANALYSES

The collective data was computerised and cleaned by the researcher and three colleagues from the Nutrition Research Group (Mr. J. Jerling, Ms. H. Burger and Mrs. M. Van Staden). Descriptive statistics as well as analyses of variance and calculation of correlation coefficients were done by the Statistical Consultation Service of the Potchefstroom University using the SAS® computerised programme (SAS® User's Guide, 1985).

CHAPTER 5

RESULTS

5.1 INTRODUCTION

In this chapter, the results of the impact of urbanisation on the diet-related and some lifestyle-related risk factors for colorectal cancer are reported. The initial calculation indicated that 2500 subjects will be needed in the sample to show significant differences in variables known to have a large variation between subjects. The results of this first phase of the THUSA study include dietary intakes of 890 subjects and biochemical profiles of 845, and should therefore be regarded as preliminary. In the descriptive statistics given here as Figures to illustrate the effect of urbanisation, median values and box plots representing values between the 25th and 75th percentiles are used. These graphs therefore, only show directions of changes and not significant differences. In the results of nutrient intakes, starch, meat and vegetable intakes, significant differences between strata in the same age and gender groups were calculated. To facilitate interpretation of urbanisation effects, means for men and women of the different strata (age groups combined) were calculated, without correcting for number of subjects in each age group. This data is therefore only used to obtain a measure of the effect of urbanisation on a particular variable.

5.2 SUBJECT DETAILS

Table 5.1 Description of subjects

AGE (y)	TOTAL	MEN					TOTAL	WOMEN				
		S1	S2	S3	S4	S5		S1	S2	S3	S4	S5
15-24.9	95	27	13	11	9	35	104	29	13	15	13	34
25-34.9	71	11	15	8	19	18	138	31	13	19	48	27
35-44.9	65	19	13	5	15	13	124	30	14	24	27	29
45-54.9	68	15	21	8	18	6	91	33	14	11	22	11
55-64.9	42	16	9	5	11	1	47	16	6	10	15	0
TOTAL	**341**						**504**					

S1: Stratum 1, deep rural
S2: Stratum 2, farms
S3: Stratum 3, squatter camps
S4: Stratum 4, established township: urban middle class
S5: Stratum 5, township, towns, cities: urban upper class (professional people)

In Table 5.1 the total distribution of the subjects (n = 845 according to age and stratum is given.)

5.3 EFFECTS OF URBANISATION

Because the objective of this study was to examine the **effect of urbanisation** on risk factors for colorectal cancer in Africans, the **effect of urbanisation** on diastolic blood pressure and total serum cholesterol are shown in Figures 5.1 and 5.2. Throughout the results and discussion chapters, differences in each age group of men and women between the strata 1 to 5 are equated to an "effect of urbanisation".

5.3.1 Diastolic blood pressure

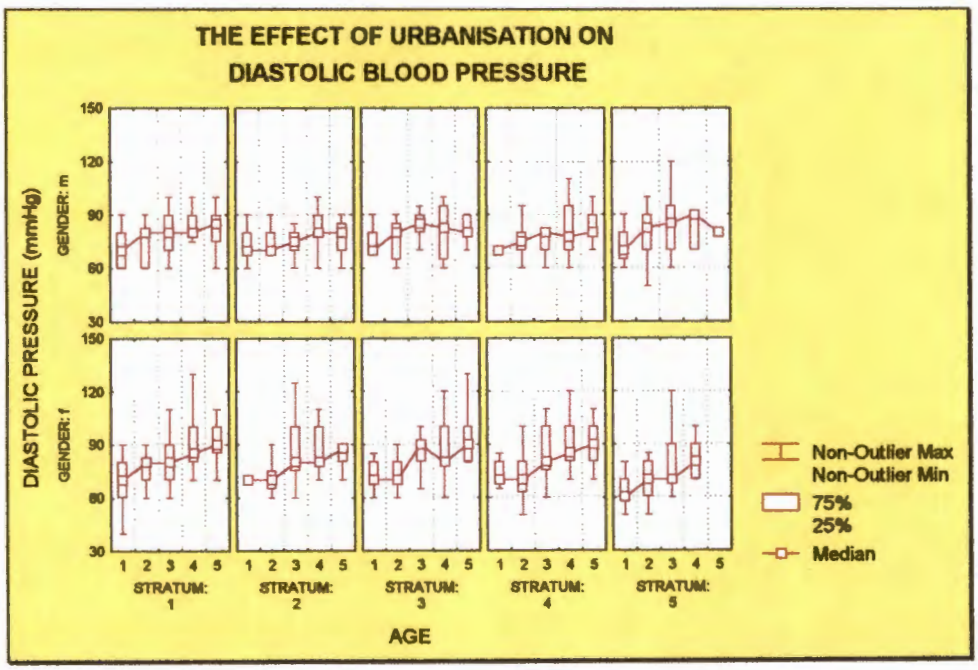


Figure 5.1 **Median diastolic blood pressure (mmHg) of men and women of the different age groups in each of stratum.**

In Figure 5.1 the trend for diastolic blood pressure to increase with urbanisation is clear for men and women in all age groups.

5.3.2 Total cholesterol

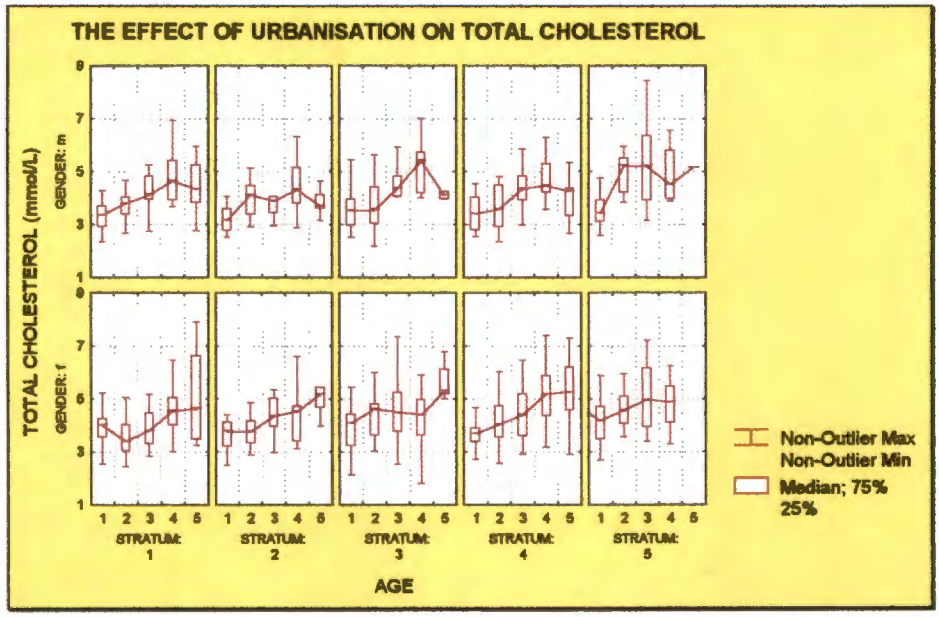


Figure 5.2 **Median TC of men and women of the different age groups of strata 1 to 5.**

Figure 5.2 clearly shows that total serum cholesterol increased progressively with urbanisation in men and women of all age groups. It is noteworthy that young (15-25 years) men and women in strata 4 and 5 had higher median cholesterol levels than older men and women (55-65 years) from the rural strata. These changes in diastolic blood pressure and TC show that in this sample of Africans from the Northwest, expected changes associated with urbanisation and acculturation of developing populations (Grossman & Rosenthal, 1993) are present. In the next section, the impact of urbanisation, on known dietary risk factors for colorectal cancer will be reported.

5.4 MACRONUTRIENT INTAKES

Tables 5.2 and 5.3 give the reported mean and standard deviation (SD) of macronutrient intakes of women and men of the different age groups for strata 1 to 5.

Table 5.2 **Mean and standard deviation of reported macronutrient intakes for women of the different age groups for strata 1-5.**

Age 15-24.9 n=108	Stratum									
Nutrient	**1**		**2**		**3**		**4**		**5**	
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
Energy (kJ)	8925	1703	6914	1600	7917	2013	8918	3330	8513	2477
Total protein (g)	65.8	13.6	57.2	15.2	57.8	15.8	69.2	23.2	66.7	20.8
Plant protein (g)	35.4*	9.5	25.2	9.6	27.8	8.2	31.3	13.9	25.3*	7.9
Animal protein (g)	30.2*	11.2	31.9	11.8	29.9	12.0	37.7	15.7	41.2*	15.2
Total fat (g)	59.8	20.6	53.1	23.2	64.9	24.0	74.8	30.3	76.1	24.6
Cholesterol (mg)	283.6	137.1	275.2	153.1	294.1	216.2	302.7	155.1	332.3	164.4
Total carbohydrate (g)	334.1	91.2	240.3	61.6	272.5	71.1	296.9	125.8	271.5	87.6
Fibre (g)	18.9	7.5	11.9	4.4	16.0	5.7	20.2	10.4	16.0	5.8
Alcohol (g)	1.9	8.5	0.03	0.09	0.2	0.8	0	0	0.11	0.34

* **means with the same symbol differ significantly (ANOVA for means and standard deviations, Tukey tests for honest significance, p<0,05))**

Age 25-34.9 n=151	Stratum									
Nutrient	**1**		**2**		**3**		**4**		**5**	
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
Energy (kJ)	7917	2766	8762	2290	8732	3328	8819	2792	8791	2938
Total protein (g)	59.38	19.8	65.66	16.5	62.8	24.3	69.4	19.9	70.5	22.1
Plant protein (g)	32.6	13.7	35.4*	11.6	32.1#	13.4	30.8	11.0	27.7*#	8.9
Animal protein (g)	26.8	12.5	30.1	10.7	30.5	14.8	38.4	15.8	42.7	17.8
Total fat (g)	50.6	20.1	53.8	25.2	58.4	30.0	67.6	26.3	73.0	30.0
Cholesterol (mg)	250.5	170.0	224.5	150.2	251.7	158.8	336.5	186.9	318.4	148.8
Total carbohydrate (g)	301.6	117.6	328.1	97.1	332.0	130.5	301.8	107.3	288.5	104.8
Fibre (g)	16.7	7.2	15.5	5.5	17.7	7.1	19.0	7.0	18.6	7.5
Alcohol (g)	1.45	4.3	7.8	15.1	0.9	2.3	3.9	10.7	2.0	6.3

*,# **means with the same symbol differ significantly (ANOVA for means and standard deviations, Tukey tests for honest significance, p<0,05)**

Table 5.2

Age 35-44.9 n=127	Stratum									
Nutrient	**1**		**2**		**3**		**4**		**5**	
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
Energy (kJ)	7378	2727	7527	3236	7129	2283	7816	2136	8092	2945
Total protein (g)	52.3	16.0	57.2*	26.2	58.2	21.9	62.5	17.4	71.2*	23.2
Plant protein (g)	30.0	12.9	28.6	13.1	23.6	8.8	27.8	8.8	25.1	11.1
Animal protein (g)	22.2	9.9	28.6	16.9	34.5	20.1	34.6	14.5	46.0	16.8
Total fat (g)	44.9	18.4	47.7	21.9	51.0	23.4	62.2	19.4	68.6	26.5
Cholesterol (mg)	236.7	170.2	201.1	163.7	327.6	253.3	298.3	198.6	374.2	177.4
Total carbohydrate (g)	290.3	135.8	280.8	136.3	234.4	99.6	261.3	94.4	253.1	110.9
Fibre (g)	16.1	8.0	12.8	6.2	12.6	5.6	16.8	6.7	17.9	7.2
Alcohol (g)	1.1	5.1	5.2	10.5	10.6	32.5	2.8	10.1	2.4	8.7

* **means with the same symbol differ significantly (ANOVA for means and standard deviations, Tukey tests for honest significance, p<0,05)**

Table 5.2 (continued)

Age 45-54.9 n=103	Stratum									
Nutrient	1		2		3		4		5	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Energy (kJ)	8756	2585	6618	3356	7960	3504	7003	2134	8153	2972
Total protein (g)	67.9	21.6	47.9	25.6	63.3	26.9	55.5	15.8	69.6	24.2
Plant protein (g)	35.2	11.5	25.4	16.0	26.7	10.1	27.2	12.1	26.5	15.3
Animal protein (g)	32.6	15.8	22.5*	14.0	36.6	21.6	28.3#	10.6	42.9*#	13.5
Total fat (g)	56.9*	23.9	32.9	20.8	60.6	31.9	48.5#	14.2	70.1*#	29.0
Cholesterol (mg)	285.5	185.4	137.2	83.0	394.4	247.9	268.6	151.8	313.5	149.6
Total carbohydrate (g)	320.6	108.9	270.9	138.0	255.0	107.9	250.6	92.6	259.6	99.6
Fibre (g)	18.8	7.4	10.9	6.7	16.2	4.3	15.9	6.9	19.3	11.1
Alcohol (g)	6.0	13.7	4.7	13.8	10.9	15.4	3.3	7.7	0.3	0.57

*,# means with the same symbol differ significantly (ANOVA for means and standard deviations, Tukey tests for honest significance, p<0,05)

Age 55-64.9 n=163	Stratum									
Nutrient	1		2		3		4		5	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Energy (kJ)	7371	2576	7199	2213	8374	2854	6341	2195	10954	1255
Total protein (g)	58.5	20.1	58.7	18.9	62.4	23.4	49.4	13.8	84.8	5.5
Plant protein (g)	28.8	12.8	26.1	12.8	34.7	13.3	23.6	10.0	36.1	4.1
Animal protein (g)	29.8	14.1	32.5	13.2	27.6	13.9	25.7	9.8	48.5	9.4
Total fat (g)	49.6	21.3	44.0*	14.4	54.1#	19.9	44.5&	14.2	94.2*#&	17.9
Cholesterol (mg)	305.1	145.0	215.3	114.2	206.8	114.2	224.3	140.7	413.7	53.9
Total carbohydrate (g)	265.4	105.1	273.8	108.7	306.1	124.9	232.3	98.5	354.3	61.1
Fibre (g)	16.5	6.7	11.7	4.2	17.0	5.5	12.9	5.3	24.2	2.7
Alcohol (g)	3.0	9.2	2.6	3.8	7.9	20.1	0.1	0.4	1.6	2.7

*,#,& means with the same symbol differ significantly (ANOVA for means and standard deviations, Tukey tests for honest significance, p<0,05)

Table 5.3 **Mean and standard deviation of reported macronutrient intakes for men of the different age groups for strata 1 to 5.**

Age 15-24.9 n=110	Stratum									
Nutrient	1		2		3		4		5	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Energy (kJ)	9379	2998	7361	2855	8589	3227	10991	3201	10100	3500
Total protein (g)	76.5	26.0	58.9	23.6	69.8	25.5	88.3	23.4	80.2	28.8
Plant protein (g)	38.8	17.1	27.7	10.6	30.8	12.2	38.2	15.8	35.8	12.7
Animal protein (g)	37.6	15.0	31.1	15.9	38.8	17.5	49.8	12.6	44.3	22.4
Total fat (g)	66.1	17.6	49.0#	20.5	66.0	31.4	87.4*	27.9	82.4#	33.8
Cholesterol (mg)	354.9	140.3	262.4	206.5	390.9	232.6	515.9	268.6	430.7	308.8
Total carbohydrate (g)	335.6	130.2	271.3	121.6	289.2	121.6	364.5	124.1	335.9	121.9
Fibre (g)	23.5*	11.8	12.6*#&	4.8	14.2	6.9	23.4#	18.5	21.9&	3.8
Alcohol (g)	2.1	3.6	2.3	5.8	5.3	7.9	6.5	19.2	3.5	6.3

*,#,& means with the same symbol differ significantly (ANOVA for means and standard deviations, Tukey tests for honest significance, p<0,05)

Table 5.3 (continued)

Age 25-34.9 n=70	Stratum									
Nutrient	**1**		**2**		**3**		**4**		**5**	
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
Energy (kJ)	7634	2923	8270	3564	10479	2443	9507	2220	8745	2198
Total protein (g)	63.6	26.6	64.5	24.7	78.7	29.3	76.7	20.2	78.0	16.8
Plant protein (g)	29.5	14.9	33.3	15.6	35.1	15.7	34.5	13.7	29.8	9.8
Animal protein (g)	33.9	19.2	31.2	13.6	43.5	18.5	41.9	18.7	48.1	15.9
Total fat (g)	54.1	22.4	48.0*	20.7	81.6	38.9	74.1	20.3	78.9*	29.0
Cholesterol (mg)	256.1	203.3	258.7	102.6	435.3	197.3	354.7	205.5	365.4	175.9
Total carbohydrate (g)	264.0	106.4	304.5	149.7	328.0	91.4	305.5	106.4	289.6	83.6
Fibre (g)	15.7	9.3	14.5	7.1	18.9	9.5	17.1	6.2	18.1	6.9
Alcohol (g)	5.1	9.8	12.7	16.6	21.9	28.5	12.8	18.9	21.2	21.7

* means with the same symbol differ significantly (ANOVA for means and standard deviations, Tukey tests for honest significance, p<0,05)

Age 35-44.9 n=58	Stratum									
Nutrient	**1**		**2**		**3**		**4**		**5**	
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
Energy (kJ)	10377	3191	8727	2604	8769	3592	10524	21298	8794	2712
Total protein (g)	76.4	25.1	61.6	17.5	67.1	18.2	77.6	23.5	72.5	23.2
Plant protein (g)	41.3	16.1	37.8*	16.6	27.4	16.0	33.6	9.8	27.0*	9.0
Animal protein (g)	35.1	17.2	23.7*	14.0	39.4	23.4	43.8	21.6	45.4*	16.0
Total fat (g)	58.6	18.9	37.5	19.9	63.3	22.7	79.5	28.5	75.5	27.7
Cholesterol (mg)	275.1	116.8	197.1*	173.6	400.0	212.6	382.8	165.4	452.1*	218.3
Total carbohydrate (g)	376.2	140.1	361.8	147.4	240.9	112.7	333.7	103.2	261.7	77.6
Fibre (g)	21.3	9.8	14.9	5.9	13.0	4.9	17.1	5.4	15.7	6.8
Alcohol (g)	20.6	30.2	12.2	11.3	38.4	53.1	24.7	18.9	12.2	16.4

* means with the same symbol differ significantly (ANOVA for means and standard deviations, Tukey tests for honest significance, p<0,05)

Age 45-54.9 n=61	Stratum									
Nutrient	**1**		**2**		**3**		**4**		**5**	
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
Energy (kJ)	8962	3204	9303	3144	7252	2627	9007	2300	8329	3268
Total protein (g)	70.2	27.4	66.7	24.5	52.1	18.3	64.3	17.4	73.6	28.4
Plant protein (g)	32.4	13.0	35.8	18.2	26.8	9.6	35.6	9.7	26.8	12.5
Animal protein (g)	37.8	20.2	30.9	12.9	25.3	12.1	28.6	13.7	46.9	20.1
Total fat (g)	61.5	27.4	45.1	17.4	42.9	17.8	55.4	22.2	71.5	24.3
Cholesterol (mg)	366.2	204.2	258.7	214.6	260.0	238.6	237.1	136.0	489.1	207.6
Total carbohydrate (g)	300.2	104.8	365.5	131.8	252.6	93.8	328.8	91.5	250.2	116.4
Fibre (g)	17.6	8.6	14.2	6.7	12.8	3.9	18.1	5.6	16.5	6.4
Alcohol (g)	15.5	24.5	16.4	26.8	20.0	23.6	13.9	16.3	7.1	9.9

*,#,& means with the same symbol differ significantly (ANOVA for means and standard deviations, Tukey tests for honest significance, p<0,05)

Table 5.3 (continued)

Age 55-64.9 n=39	Stratum									
Nutrient	**1**		**2**		**3**		**4**		**5**	
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
Energy (kJ)	8649	3254	7617	2668	8271	4622	9366	3426	8055	2984
Total protein (g)	69.6	30.6	57.0	21.7	65.5	33.4	68.0	26.9	69.1	16.6
Plant protein (g)	37.3	15.1	30.7	13.2	33.3	24.4	34.2	14.1	29.2	14.1
Animal protein (g)	32.2	20.6	26.3	9.9	31.9	12.7	33.8	17.2	39.9	2.5
Total fat (g)	53.0	26.0	37.6	15.6	49.3	20.0	61.1	37.7	66.2	19.9
Cholesterol (mg)	306.3	189.0	164.9	38.1	351.5	330.2	341.0	225.0	386.5	208.6
Total carbohydrate (g)	323.4	120.2	294.0	110.4	293.9	206.0	314.5	123.9	264.3	127.0
Fibre (g)	20.5	11.9	14.1	6.5	16.3	10.4	16.0	8.2	21.5	10.6
Alcohol (g)										

Means with the same symbol differ significantly (ANOVA for means and standard deviations, Tukey tests for honest significance, p<0,05)

5.4.1 Changes in energy intake

Table 5.2 shows that in women reported mean energy intakes of the 15-24.9 year age group were similar in rural and urban groups but that the farm workers and squatter camp dwellers (strata 2 and 3) had lower intakes. In the age group 25-34.9 years, rural women reported an intake of ±1000kJ less than the other groups. The urban women of stratum 5 of the 35-44.9 year age group had the highest intake, while the rural women of stratum 1 of the 45-54.9 year group reported the highest intake. In the women aged 55-64.9 years, the highest intakes were reported by strata 3 and 5. Although the lowest intakes were found in the farm groups, no consistent increases in energy intakes with urbanisation in women were observed.

In men (Table 5.3) there were also no significant differences in energy intake with urbanisation. Therefore, despite the changes observed in BMI (see Table 5.22) especially in women, the same stepwise increases in energy intake with urbanisation were not found.

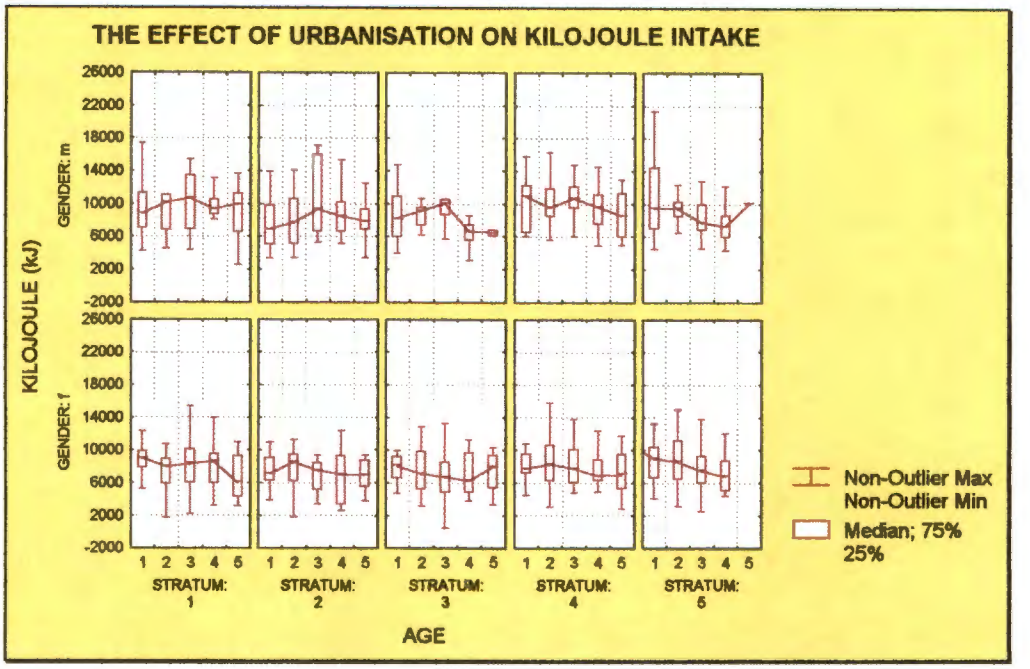


Figure 5.3 **Median total energy intake of men and women of the different age groups of strata 1-5.**

The reported intakes of energy are also illustrated in Figure 5.3. In both men and women, no significant changes with urbanisation were observed. The trends in women of an increase in BMI and decrease in energy intake with urbanisation, may reflect a decrease in physical activity. However, it is known that especially in obese subjects, energy intake is often underreported (Lafay *et al.* 1997; Briefel *et al.* 1997). Mean energy intakes of both men and women compare well with results summarised by Vorster *et al.* (1997) and SANNS (1995) from published South African literature. For example the 24-hour recall method showed the total energy intake for men and women to be 6 400 (women) and 8 500 (men) (SANNS, 1995), and from the food frequency questionnaire method 8900 (women) and 12000 (men) kilojoules.

5.4.2 Protein intake

Table 5.2 shows that the young women (age 15-24.9 years) of all strata took in similar amounts of total protein. However, in the rural women protein from plant foods exceeded those from animal derived foods (35.4 versus 30.3 g/day) while in all other strata there was a progressive decrease in plant and an increase in animal protein intakes. This change in plant/animal protein ratio was also seen in the other age groups as well as a significant increase

in total protein intake in most groups. A similar pattern was observed in the men, with the exception that rural men often had higher total protein intakes than the farm workers and squatters.

These increases in total and animal protein intakes were not reflected in total serum protein and albumin, possibly because in all groups daily protein requirements were met (Figures 5.4 and 5.5), and total serum protein and albumin were within normal ranges of 66-79 g/L and 39-50 g/L respectively (DAX, 1996).

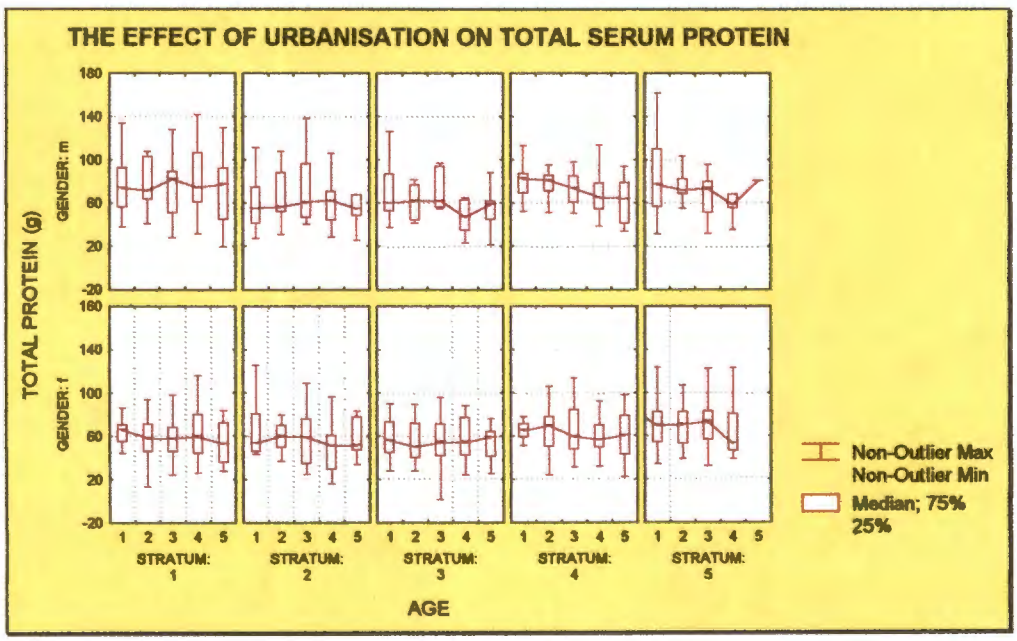


Figure 5.4 **Median total serum protein of men and women of the different age groups of strata 1 - 5.**

Figures 5.4 and 5.5 reflect the adequate protein status of the participants. Proteins with a shorter half-life, such as retinol binding protein are a better indication of protein status (Vorster *et al.* 1994) but were unfortunately not measured in this study. Nevertheless, these figures illustrate that in all strata and in all age groups, dietary protein intakes probably met requirements.

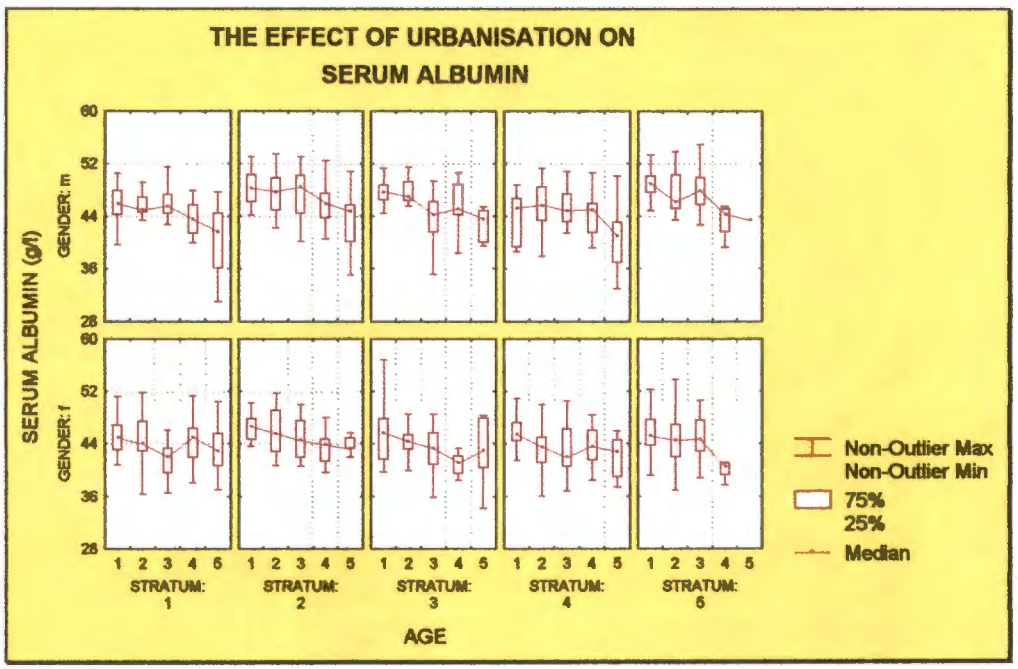


Figure 5.5 **Median total serum albumin of men and women of the different age groups of strata 1 - 5.**

5.4.3 Total fat intake

Mean total fat intakes of women (Table 5.2) and men (Table 5.3) for the different age groups and strata, clearly indicate a progressive increase with urbanisation. Again, the exceptions were men and women from stratum 2 (farm workers) who mostly had lower intakes than stratum 1. With the exception of women aged 45-64.9 (stratum 4) groups from strata 4 and 5 had the highest fat intakes. The lowest mean intake in women was 44.9 g/day in the rural women aged 35-44.9 years, while the highest of 94.2 g/day was observed in the older women of stratum 5. In men, an intake of 37.5 and 37.6 g/day in the 35-44.9 and 55-64.9 year groups of the farm workers were found, while the highest intake of 87.4 g/day was seen in stratum 4 of the 15-24.9 year old group.

The mean percentages of energy that fat contributed to the total energy intakes of the different groups are shown in Table 5.4.

Table 5.4 Mean fat intakes expressed as percentage of total energy intake.

STRATUM GENDER	AGE GROUPS IN YEARS					
	15-24.9	25-34.9	35-44.9	45-54.9	55-64.9	Total (Mean) *
S1						
Men	26.8	26.9	21.5	26.0	23.3	24.9±2.4
Women	25.1	24.3	23.1	24.7	25.6	24.6±0.9
S2						
Men	25.3	22.0	16.3	18.4	18.8	20.2±3.5
Women	29.2	23.3	23.9	18.9	23.2	23.7±3.7
S3						
Men	29.2	29.6	27.4	22.5	22.7	26.3±3.5
Women	31.2	25.4	27.2	28.9	24.5	27.4±2.7
S4						
Men	30.2	29.6	28.7	23.4	24.8	27.4±3.0
Women	31.9	29.1	30.2	26.3	26.7	28.8±2.4
S5						
Men	31.0	34.3	32.6	32.6	31.2	32.3±1.3
Women	34.0	31.6	32.2	32.7	32.7	32.6±0.9

* Means calculated from means without correcting for number.

The gradual increase of fat intake as percentage of total energy across the 5 strata is very marked. The farm workers had the lowest intake. In the "upper class" urban men and women (stratum 5), fat contributed more than 30% of total energy, values higher than the usual recommendation of less than 30% to protect against chronic diseases of lifestyle (James, 1997).

The mean P/S ratio's of the diet are shown in Table 5.5. The farm workers and "upper class" urban diet had the lowest P/S ratio (0.69 and 0.62 for men and women respectively). The diet of the rural subjects had the highest P/S ratio (0.90 and 0.85 for men and women respectively). The gradual decrease in the P/S ratio with urbanisation (with the exception of the farm workers) indicates that the increase in fat intake is mainly an increase in saturated fat. A P/S ratio recommended to protect against chronic diseases of ≥1.0 (James, 1993), was not reached by any of the sub-groups.

Table 5.5 **Mean P/S ratios* of the diet of the men and women for the different age groups and strata.**

	AGE GROUPS IN YEARS					
STRATUM GENDER	15-24.9	25-34.9	35-44.9	45-54.9	55-64.9	Total (Mean)
S1						
Men	0.83±0.21	0.89±0.36	0.93±0.53	0.86±0.38	0.98±0.42	0.90±0.06
Women	0.76±0.33	0.89[a]±0.34	0.91[b]±0.37	0.85[a]±0.32	0.85±0.35	0.85±0.06
S2						
Men	0.77±0.38	0.69±0.37	0.79±0.51	0.55[d]±0.23	0.63±0.28	0.69±0.10
Women	0.56±0.21	0.76±0.35	0.68±0.27	0.66±0.27	0.46±0.11	0.62±0.11
S3						
Men	0.81±0.36	0.67±0.21	0.89±0.51	0.70±0.22	0.99±0.46	0.81±0.13
Women	0.77±0.20	0.83±0.21	0.79±0.34	0.80±0.20	0.76±0.21	0.79±0.03
S4						
Men	0.71±0.14	0.85±0.32	0.85±0.47	0.94[d]±0.39	0.74±0.18	0.82±0.09
Women	0.75±0.20	0.80±0.29	0.74±0.27	0.80±0.27	0.84±0.41	0.79±0.04
S5						
Men	0.72±0.72	0.63±0.20	0.61±0.16	0.82±0.25	0.61±0.08	0.61±0.22
Women	0.67±0.22	0.63[a]±0.26	0.64[b]±0.20	0.55[a]±0.16	0.80±0.43	0.66±0.08

* : P/S ratio: Ratio of polyunsaturated/saturated fat

a,b,c : Means with the same symbol differs significantly ($p<0.05$)

5.4.4 Carbohydrate intake

5.4.4.1 Total carbohydrate

The total mean carbohydrate and dietary fibre intakes in g/day for men and women of the different age and strata groups are given in Tables 5.2 and 5.3. Total carbohydrate expressed as a percentage of total energy intake and dietary fibre as gram per megajoule (1000 kJ) are shown for the different age groups and strata in Table 5.6. From this table it is clear that the percentage of total energy provided by carbohydrate progressively decreased in both men and women from strata 1 to 5, with the exception of the highest percentage intakes seen in the farm workers. In all groups except one instance (35-44.9 year old men of stratum 3), mean carbohydrate intakes exceeded 50% of total energy and values as high as 65 to 71% were observed.

5.4.4.2 Dietary fibre

Dietary fibre intakes varied from 1.48 to 2.37 g/MJ. In diets of 8000 to 9000 kJ, an intake of 25 to 35 grams is recommended (Garrow & James, 1993). This means approximately 3.1 to 3.9 g/MJ. Therefore, it seems that the dietary fibre intake of these subjects were relatively low. However, fibre intakes did not decrease with urbanisation. In men and women of all age

groups, the farm workers had the lowest intakes. Intakes in strata 4 and 5 (urban groups) were often higher than in corresponding gender and age groups of strata 1 to 3. The calculated mean intake for the most rural group of men and women (stratum 1) is 2.19±0.24 and 2.16±0.05 g/MJ/day respectively. For the most urban groups (stratum 5) these values were 2.14±0.33 and 2.15±0.17 g/MJ/day respectively.

These results suggests that despite urbanisation and increases in total fat and animal protein, carbohydrate intakes remain high and fibre intakes remain relatively low. The high percentage energy from carbohydrate found in the farm workers and their low fibre intakes are probably because of the high intakes of refined maize meal. If total amounts of dietary fibre is considered the amount that will reach the colon for fermentation, - Table 5.2 and 5.3 indicate that only one group of women (55-64.9 year olds in stratum 5) had an intake of 20 grams or more, and that only 6 of the 25 groups of men, of 15-64.9 years, reached this minimum recommended amount.

Table 5.6 Total carbohydrate intake expressed as percentage of total energy intake and dietary fibre as gram per megajoule (1000kJ).

	AGE GROUPS IN YEARS					
STRATUM GENDER	**15-24.9**	**25-34.9**	**35-44.9**	**45-54.9**	**55-64.9**	**Total (Mean)**
S1						
Men	60.8*	58.8	61.6	56.9	63.6	60.3±2.6
	2.51#	2.06	2.05	1.96	2.37	2.19±0.24
Women	63.6	64.8	66.9	62.2	61.2	63.7±2.2
	2.12	2.11	2.18	2.15	2.24	2.16±0.05
S2						
Men	62.7	62.6	70.5	66.8	65.6	65.6±3.3
	1.71	1.75	1.71	1.53	1.85	1.71±0.12
Women	59.1	63.7	63.4	69.6	64.7	64.1±3.8
	1.72	1.77	1.70	1.64	1.63	1.69±0.06
S3						
Men	57.2	53.2	46.7	59.2	60.4	55.3±5.5
	1.65	1.80	1.48	1.76	1.97	1.73±0.18
Women	58.5	64.6	55.9	54.5	62.1	59.1±4.2
	2.02	2.03	1.77	2.04	2.03	1.98±0.12
S4						
Men	56.4	54.6	53.9	62.1	57.1	56.8±3.2
	2.13	1.80	1.62	2.01	1.71	1.85±0.21
Women	56.6	58.2	56.8	60.8	62.3	58.9±2.5
	2.27	2.15	2.15	2.27	2.03	2.17±0.10
S5						
Men	56.5	56.3	50.6	51.1	55.8	54.1±2.9
	2.17	2.07	1.79	1.98	2.67	2.14±0.33
Women	54.2	55.8	53.2	54.1	55.0	54.5±1.0
	1.88	2.12	2.21	2.34	2.21	2.15±0.17

* Total carbohydrate expressed as a percentage of total energy

\# Dietary fibre expressed as g per megajoule (1000kJ).

5.4.4.3 Sugar intake

The South African Food Tables (Langenhoven *et al.* 1991) give values for added sugar (sucrose), but not for other sugars. Figure 5.6 illustrates median sucrose intakes of men and women of the different age groups in each stratum.

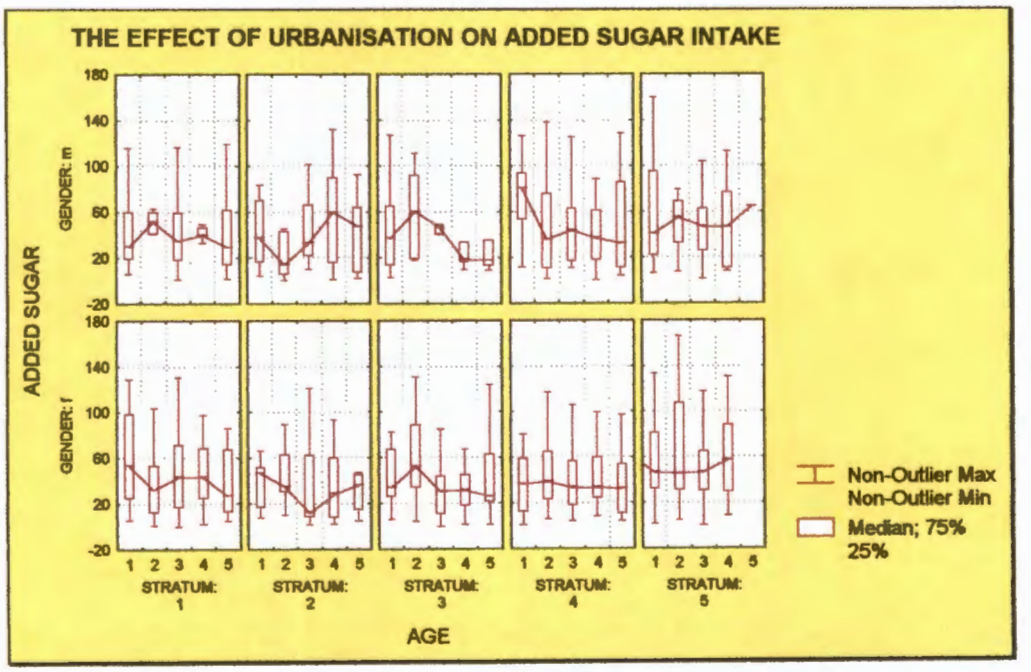


Figure 5.6 Median added sugar of men and women of the different age groups of strata 1 - 5.

The figure shows no constant increase across strata or noticeable differences between age groups. Mean intakes of men and women for the different strata were: S1 - 29.6 and 36.2; S2 - 36.3 and 33.4; S3 - 30.9 and 33.6; S4 - 34.2 and 29.2; S5 - 26.3 and 30.6 g per day (data not shown in Tables 5.2 and 5.3).

5.4.4.4 *Starch intakes*

Table 5.7 **Calculated mean starch intakes in g/day of men and women of the different age groups and strata.**

	Stratum 1		Stratum 2		Stratum 3		Stratum 4		Stratum 5	
AGE	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
15-24.9										
M	275	122	279	211	233	131	228	97	271	121
W	257	72	227	107	217	66	232	88	202	58
25-34.9										
M	310	212	269	156	211	30	258	88	212	67
W	249	125	247	106	228	113	222	92	204	77
35-44.9										
M	294	117	365	184	257	142	278	130	195	57
W	245	112	239	110	179	91	195	73	188	90
45-54.9										
M	266	113	272	128	192	53	273	100	180	79
W	241	89	230	136	215	99	187	66	176	91
55-64.9										
M	287	152	247	127	185	112	228	86	261	0
W	199	101	204	91	213	82	181	78	-	-

* : Starch calculated as Total carbohydrate - (fibre + sucrose)
SD : Standard deviation
M : Men
W : Women

Total mean for stratum: gram starch/day

	Stratum 1	Stratum 2	Stratum 3	Stratum 4	Stratum 5
Men	286	286	216	253	224
Women	238	229	210	203	193

The starch content of South African foods is not known. The carbohydrate content given in the Food Tables (Langenhoven *et al.* 1991) is calculated as the difference between total energy of the food and energy provided by analysed fat and protein, using a value of 17kJ per gram carbohydrate. To examine the influence of urbanisation on starch intake, starch therefore had to be calculated as the difference between total carbohydrate and fibre plus sucrose. The values given in Table 5.7 should therefore be seen only as an approximation of starch intakes.

The total amount of starch is considered, and not the percentage contribution to energy, since it is accepted that the total starch intake will probably be proportional to the amount that will reach the colon because of resistance to digestive enzymes (Cummings *et al.* 1997).

In the colon the resistant starch is fermented, together with the NSP (dietary fibre). Table 5.7 show that although the differences were not large, gradual decreases in mean starch intakes with urbanisation were observed. The two rural groups of men (rural and farm workers) had calculated starch intakes of 286 g/day, while the two urban strata (4 and 5) had intakes of 253 and 224 g/day. In women the corresponding figures were 238 and 229 g/day for rural women and 203 and 193 g/day for urban women. The dwellers in the squatter camps had relatively low intakes (216 and 210 g/day). It therefore seems as if total starch intakes decrease with urbanisation.

5.4.4.6 Alcohol intakes

Alcohol is not normally regarded as a nutrient. However, it does provide energy and is known as a risk factor for colorectal cancer (Goldbohm *et al.* 1994). The effect of urbanisation on alcohol intakes will therefore be considered with the other macronutrients. Mean alcohol intakes are given in Tables 5.2 and 5.3 and are summarised in Table 5.8. The alcohol intake is expressed as gram intake per day after all reported alcoholic beverages were converted, based on their alcohol content (MacIntyre, 1998).

Table 5.8 Summary of mean alcohol (g/day) intakes.

	AGE GROUPS IN YEARS					
STRATUM GENDER	**15-24.9**	**25-34.9**	**35-44.9**	**45-54.9**	**55-64.9**	**Total (Mean)**
S1						
Men	2.1	5.1	20.6	15.5	20.5	15.4±7.3
Women	1.9	1.5	1.1	6.0	3.0	2.9±2.2
S2						
Men	2.3	12.7	12.2	16.4	14.1	13.9±1.9
Women	0.03	7.8	5.2	4.7	2.6	5.1±2.1
S3						
Men	5.3	21.9	38.4	20.0	16.3	24.2±9.8
Women	0.2	0.9	10.6	10.9	7.9	7.6±4.7
S4						
Men	6.5	12.8	24.7	13.9	16.0	16.9±5.4
Women	0	3.9	2.8	3.3	0.1	2.5±1.7
S5						
Men	3.5	21.2	12.2	7.1	21.5	15.5±7.1
Women	0.1	2.0	2.4	0.3	1.6	1.6±0.9

Table 5.8 shows that reported alcohol intakes of the 15-24.9 year old groups were substantially lower than those of the other age groups. Therefore, the mean total intakes for men and women across strata were calculated for the age group 24.9-64.9 years. Women reported much lower intakes than men. It seems that men in stratum 3 had the highest mean intake (24.2 g/day) while differences between the strata for women were small.

GAMMA GLUTHAMYL TRANSFERASE (GGT)

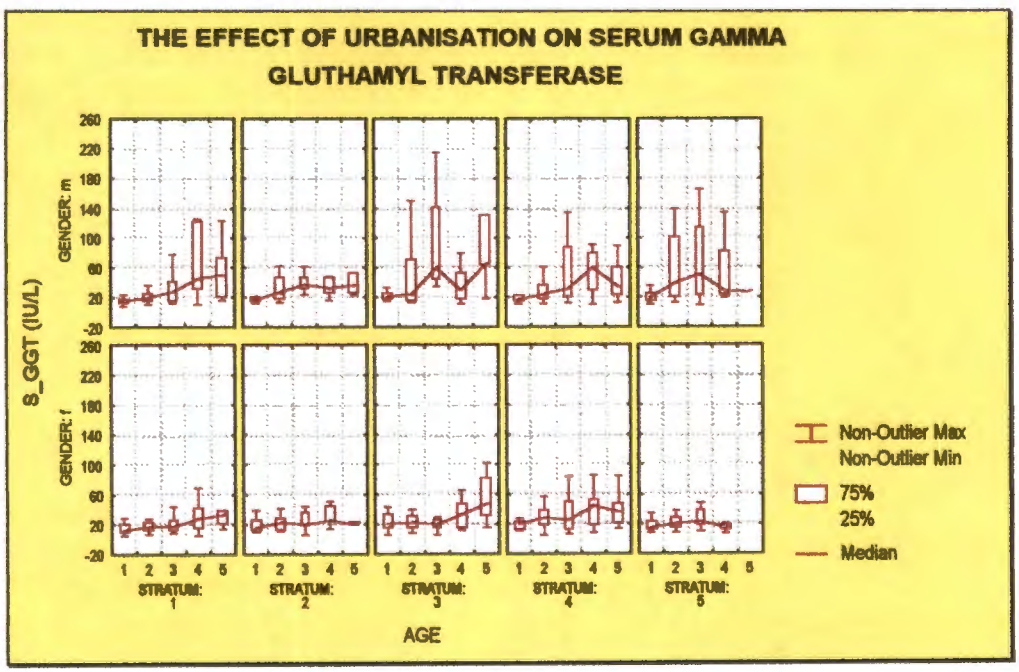


Figure 5.7 **Median gamma gluthamyl transferase of men and women of the different age groups of strata 1 - 5.**

The liver enzyme, gamma gluthamyl transferase (GGT) is used as a proxy for alcohol intake. In Figure 5.7 it can be seen that GGT values of men were higher than those of women, and that men in stratum 3 of the age groups used to calculate mean intake for men indeed had the highest median GGT values. The figure further shows that in young men and all the groups of women (except the 55-64.9 year old group) GGT increased across the strata - indicating that with urbanisation young men, and women up to 55 years of age, increase their alcohol intake.

The figure further indicates that median GGT levels in many groups of men were above the 12 to 44 IU/L (DAX, 1996) regarded as normal. Clearly, the men in this population have a high alcohol intake.

5.5 MICRONUTRIENT INTAKES

5.5.1 Introduction

According to the literature (WCRF & AICR, 1997) it is possible that the carotenoids decrease colorectal cancer risk, while vitamin C, D, E and folate may decrease risk. For these vitamins, however, there is insufficient evidence of their role and more research is needed. The same report concluded that there is possibly no relationship of calcium and selenium with risk, and that iron may increase risk. Despite these uncertainties, the impact of urbanisation on the intake of some of these micronutrients will be reported in this section.

The mean and standard deviations of vitamin and mineral intakes of women and men stratified for age and stratum, are given in Tables 5.9 to 5.12.

Table 5.9 Mean and standard deviation of reported vitamin intakes for women

Age 15-24.9 n=108	Stratum									
Nutrient	1		2		3		4		5	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Vitamin A (RE)	819.8	811.8	920.0	753.8	847.1	526.1	895.8	567.8	1015.6	895.8
Vitamin E (mg)	9.62	3.96	7.24	3.38	11.16	6.20	11.00	4.54	10.85	4.39
Thiamine (mg)	1.23	0.27	0.95	0.35	1.06	0.25	1.17	0.46	1.06	0.33
Riboflavin (mg)	1.60	0.51	1.6	0.75	1.31	0.50	1.45	0.46	1.56	0.57
Niacin (mg)	13.98	3.87	9.43$^{*\#}$	5.72	14.03	4.29	16.27$^{*\#}$	5.33	15.28$^{*\#}$	4.93
Vitamin B_6 (mg)	1.16*	0.43	0.90$^{\#}$	0.38	1.22	0.45	1.39	0.59	1.53$^{*\#}$	0.59
Vitamin B_{12} (µg)	4.56	3.2	7.8	7.1	6.02	5.00	4.90	3.45	6.74	5.5
Folic acid (µg)	205.7	103.2	160.5	40.6	178.1	74.06	241.2	98.78	182.28	63.26
Vitamin C (mg)	44.78*	38.0	24.0$^{\#}$	16.04	31.24$^{\&}$	19.18	77.83	63.92	82.15$^{*\#\&}$	79.16

$^{*,\#,\&}$ **means with the same symbol differ significantly (ANOVA for means and standard deviations, Tukey tests for honest significance, p<0,05)**

Table 5.9 (continued)

Age 25-34.9 n=151	Stratum									
Nutrient	1		2		3		4		5	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Vitamin A (RE)	498.2	439.7	849.5	639.4	1091.8	1332.1	1020.6	822.0	1173.2	968.7
Vitamin E (mg)	9.67	5.11	11.01	6.0	10.1	6.6	11.8	8.2	10.8	7.40
Thiamine (mg)	1.16	0.53	1.19	0.37	1.20	32.4	1.19	0.41	1.22	0.45
Riboflavin (mg)	1.27*	0.55	1.73	0.79	1.55	0.83	1.54	0.57	1.90*	0.96
Niacin (mg)	12.80	6.49	11.67	5.57	15.88	6.21	16.07	5.4	16.90	6.23
Vitamin B_6 (mg)	0.94*	0.55	0.92#	0.33	1.23	0.57	1.31	0.55	1.50*#	0.63
Vitamin B_{12} (µg)	8.38	3.63	8.67	4.17	8.44	3.80	9.44	3.51	11.06	4.92
Folic acid (µg)	186.0	69.11	233.5	105.0	194.4	80.0	234.1	89.2	239.2	104.1
Vitamin C (mg)	39.9	43.8	23.1	10.2	37.0	37.8	74.0	87.3	101.35	90.6

*,# means with the same symbol differ significantly (ANOVA for means and standard deviations, Tukey tests for honest significance, p<0,05)

Age 35-44.9 n=108	Stratum									
Nutrient	1		2		3		4		5	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Vitamin A (RE)	656.5	395.8	498.1	368.2	785.1	368.2	1031.0	1013.7	1384.2	952.7
Vitamin E (mg)	8.29	4.46	9.16	5.09	8.97	5.45	9.95	5.16	10.84	5.39
Thiamine (mg)	1.11	0.48	0.92	0.41	1.01	0.34	1.01	0.33	1.11	0.37
Riboflavin (mg)	1.24	0.57	1.25	0.87	1.53	0.69	1.62	0.91	1.78	0.61
Niacin (mg)	11.98	5.08	8.49	4.88	13.62	5.01	13.92	4.7	15.48	4.95
Vitamin B_6 (mg)	0.93*	0.46	0.74#	0.32	1.08	0.56	1.20	0.39	1.40*#	0.58
Vitamin B_{12} (µg)	3.91	3.03	3.47	3.49	5.83	6.55	6.97	8.13	10.00	7.6
Folic acid (µg)	194.0*	78.9	164.4#	62.1	163.6&	63.7	227.6	94.2	252.1*#&	97.3
Vitamin C (mg)	35.4*	24.0	17.0#	7.79	25.4&	14.3	52.8	55.3	78.6*#&	45.8

*,#,& means with the same symbol differ significantly (ANOVA for means and standard deviations, Tukey tests for honest significance, p<0,05)

Table 5.9 (continued)

Age 45-54.9 n=103	Stratum									
Nutrient	1		2		3		4		5	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Vitamin A (RE)	894.5	765.9	517.8	363.9	1446.5	1227.0	823.5	632.5	1739.6	1440.8
Vitamin E (mg)	10.67	6.18	5.49	4.84	10.91	7.38	8.54	4.41	9.41	5.60
Thiamine (mg)	1.21	0.39	0.81	0.44	1.1	0.43	1.01	0.34	1.16	0.50
Riboflavin (mg)	1.46	0.67	1.03	0.58	1.82	1.03	1.34	0.49	1.75	0.75
Niacin (mg)	14.55	5.27	6.93	3.12	14.8	5.71	12.91	5.10	15.52	6.33
Vitamin B_6 (mg)	1.03	0.43	0.61	0.31	1.36	0.79	0.98	0.29	1.50	0.60
Vitamin B_{12} (µg)	5.60	7.00	3.41	2.86	11.80	12.82	5.71	5.90	9.29	8.29
Folic acid (µg)	224.8	85.9	144.3	75.4	205.6	77.8	210.6	57.2	255.6	93.3
Vitamin C (mg)	42.41	39.4	18.6	17.5	62.5	62.16	45.03	37.0	88.7	69.9

Age 55-64.9 n=63	Stratum									
Nutrient	1		2		3		4		5	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Vitamin A (RE)	977.5	877.6	624.0	205.2	589.6	300.0	748.4	515.2	1699.3	1050
Vitamin E (mg)	8.95	4.4	6.5	1.9	8.6	3.9	8.08	3.3	15.2	7.1
Thiamine (mg)	1.15	0.58	0.92	0.35	1.28	0.5	0.9	0.40	1.44	0.12
Riboflavin (mg)	1.40	0.60	1.28	0.55	1.44	0.47	1.22	0.53	2.11	0.41
Niacin (mg)	12.4	5.4	7.3	2.4	14.4	6.7	10.83	3.8	18.53	1.42
Vitamin B_6 (mg)	0.98	0.39	0.74	0.23	0.89	0.36	0.87	0.36	1.52	0.51
Vitamin B_{12} (µg)	5.78	6.48	3.38	1.50	4.31	3.15	4.48	4.8	13.0	8.7
Folic acid (µg)	228.8	88.0	154.2	56.7	182.5	75.6	164.1	50.1	318.7	60.0
Vitamin C (mg)	63.0	88.1	20.3	8.9	27.6	18.8	26.4	24.2	53.7	28.8

Table 5.10 Mean and standard deviation of reported vitamin intakes for men

Age 15-24.9 n=110	Stratum									
Nutrient	1		2		3		4		5	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Vitamin A (RE)	587.0	331.0	400.2*	307.8	638.5	480.0	955.4*	677.4	882.0	685.4
Vitamin E (mg)	11.3	3.6	9.2	5.2	12.3	7.9	14.3	7.9	13.0	8.4
Thiamine (mg)	1.24	0.42	0.92*	0.40	1.14	0.50	1.36	0.50	1.39*	0.45
Riboflavin (mg)	1.28*#	0.53	1.21&	0.69	1.58	0.30	1.98*&	0.55	1.86#	0.82
Niacin (mg)	15.3	6.2	10.2*#	4.9	12.0	5.01	20.3*	3.34	18.1#	6.1
Vitamin B_6 (mg)	1.18*	0.33	0.92#&	0.35	1.34	0.39	1.61#	0.67	1.44*&	0.33
Vitamin B_{12} (µg)	4.39	2.49	3.10*	4.79	5.39	3.68	7.82*	5.67	6.05	5.56
Folic acid (µg)	242.4	91.5	158.5	57.0	240.3	97.4	276.1	106.0	243.1	90.3
Vitamin C (mg)	38.3*	28.0	22.2#	17.8	31.1&	25.9	59.6	50.1	77.2*#&	58.9

*,#,& means with the same symbol differ significantly (ANOVA for means and standard deviations, Tukey tests for honest significance, p<0,05)

Age 25-34.9 n=70	Stratum									
Nutrient	1		2		3		4		5	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Vitamin A (RE)	768.5	808.3	386.1*	358.3	527.7	344.1	856.8	622.3	1031*	612.4
Vitamin E (mg)	10.7	4.1	7.1*	3.7	12.2	6.4	12.0*	6.4	12.0	5.02
Thiamine (mg)	1.06	0.50	1.09	0.47	1.24	0.49	1.26	0.37	1.16	0.30
Riboflavin (mg)	1.33	0.60	1.41	0.74	1.74	0.59	1.63	0.45	1.52	0.34
Niacin (mg)	14.5	8.4	11.1*#	6.63	19.1	7.01	19.0*	5.2	19.9#	4.6
Vitamin B_6 (mg)	0.95	0.51	0.81	0.33	1.44	0.57	1.34	0.60	0.43	
Vitamin B_{12} (µg)	4.26	3.41	3.37	2.45	5.4	3.8	6.45	4.9	7.54	4.65
Folic acid (µg)	192.7	95.4	169.5	91.9	231.8	94.9	231.3	97.7	229.8	65.4
Vitamin C (mg)	27.2*	19.9	17.8#	10.5	22.7&	15.5	30.4@	14.8	77.5*#&@	60.9

*,#,&,@ means with the same symbol differ significantly (ANOVA for means and standard deviations, Tukey tests for honest significance, p<0,05)

Table 5.10 (continued)

Age 35-44.9 n=58	Stratum									
Nutrient	1		2		3		4		5	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Vitamin A (RE)	744.5	728.2	539.4	506.5	1813	1944	416.4	242.0	1416	1031
Vitamin E (mg)	11.4	6.8	6.9	3.8	11.6	5.9	12.8	5.12	10.9	5.08
Thiamine (mg)	1.45	0.39	1.14	0.36	1.03	0.21	1.22	0.33	1.17	0.40
Riboflavin (mg)	1.67	0.67	1.23	0.42	1.87	0.80	1.59	0.37	1.80	0.70
Niacin (mg)	18.61*	7.0	9.92#	4.26	18.4	7.43	20.43#	5.39	16.67	5.26
Vitamin B_6 (mg)	1.24	0.63	0.63*#	0.26	1.19	0.51	1.43*	0.62	1.36#	0.49
Vitamin B_{12} (µg)	4.77	4.11	3.91	4.83	13.80	19.02	3.9	2.1	8.58	8.69
Folic acid (µg)	240.7	102.6	177.1	74.0	247.8	130.5	230.5	59.6	227.1	88.2
Vitamin C (mg)	65.5	56.3	15.1	9.2	33.0	18.9	41.5	35.0	58.2	51.5

*,# means with the same symbol differ significantly (ANOVA for means and standard deviations, Tukey tests for honest significance, p<0,05)

Age 45-54.9 n=61	Stratum									
Nutrient	1		2		3		4		5	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Vitamin A (RE)	889.3	598.5	541.7*	395.0	828.4	632.0	629.8	390.3	1379*	1188
Vitamin E (mg)	10.52	4.54	7.51	4.53	6.44	3.22	11.89	7.77	13.7	6.46
Thiamine (mg)	1.17	0.48	1.18	0.48	1.01	0.35	1.27	0.39	1.17	0.36
Riboflavin (mg)	1.73	1.02	1.56	0.56	1.51	0.69	1.63	0.50	1.72	0.69
Niacin (mg)	16.31	7.78	10.23*	5.07	14.4	5.62	16.9*	4.82	17.50	7.35
Vitamin B_6 (mg)	1.16	0.52	0.75	0.30	0.96	0.36	0.99	0.38	1.20	0.19
Vitamin B_{12} (µg)	5.67	4.45	4.72	3.36	6.54	5.59	4.59	3.32	9.20	7.01
Folic acid (µg)	255.1	99.1	195.6	93.3	195.1	75.9	200.6	85.0	231.0	78.5
Vitamin C (mg)	83.7	153.9	19.8	10.7	15.9	7.9	26.9	24.8	48.7	18.5

*: Strata for which mean dietary intakes differ significantly

Table 5.10 (continued)

Age 55-64.9 n=39	Stratum									
Nutrient	**1**		**2**		**3**		**4**		**5**	
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
Vitamin A (RE)	700.4	748.2	519.6	245.8	527.8	265.7	952.6	1072.3	1032	191.6
Vitamin E (mg)	9.39	3.71	5.68	2.69	10.06	7.21	9.60	6.37	9.60	5.52
Thiamine (mg)	1.22	0.64	1.04	0.42	1.17	0.60	1.26	0.62	1.21	0.43
Riboflavin (mg)	1.22	0.78	1.37	0.50	1.49	0.79	1.80	0.61	1.96	1.26
Niacin (mg)	14.9	8.4	10.3	4018	15.73	6.87	18.8	7.32	16.05	3.46
Vitamin B_6 (mg)	1.04	0.65	0.71	0.25	1.03	0.56	1.20	0.58	1.22	0.19
Vitamin B_{12} (µg)	4.85	5.28	3.18	1.70	3.75	1.97	8.72	9.15	6.95	5.02
Folic acid (µg)	224.3	116.6	169.9	70.5	213.2	108.7	229.1	97.1	262.5	57.3
Vitamin C (mg)	27.8	34.4	11.6	6.4	15.0	9.5	13.5	8.4	55.2	17.2

Table 5.11 Mean and standard deviation of reported mineral intakes for women for the different age groups.

Age 15-24.9 n=108	Stratum									
Nutrient	**1**		**2**		**3**		**4**		**5**	
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
Calcium (mg)	450.4	178.6	604.5	348.6	405.1	247.3	452.2	226.5	440.6	192.3
Iron (mg)	9.8	2.6	8.4	6.6	7.8	2.7	10.3	4.0	9.8	3.3
Magnesium (mg)	332.0	67.3	234.7	77.2	271.1	61.9	305.8	122.2	266.2	88.5
Potassium (mg)	2168	583.4	1844	600.3	1962	491.7	2370	898.6	2189	737.2
Sodium (mg)	1490	748.0	1101	703.8	1437	575.6	1789	836	1841	706.4
Zinc (mg)	9.5	2.3	7.9	3.3	7.8	2.4	10.2	4.0	10.2	3.8

Age 25-34.9 n=151	Stratum									
Nutrient	**1**		**2**		**3**		**4**		**5**	
	Mean	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
Calcium (mg)	437.6	219.1	645.5	302.6	375.7	196.0	444.8	233.7	524.0	300.4
Iron (mg)	8.4	3.6	8.7	4.2	8.4	3.8	9.4	3.5	11.1	4.9
Magnesium (mg)	310.6	134.6	322.3	106.8	303.8	122.9	318.0	107.3	318.1	107.3
Potassium (mg)	2032	780.1	2298	577.6	2104	791	2368	753	2451	931
Sodium (mg)	1257	773.7	1094	447.5	1465	866	1561	683	1653	664.8
Zinc (mg)	8.1*	2.8	7.9	2.7	8.3	3.3	9.7	2.9	10.6*	3.5

*** means with the same symbol differ significantly (ANOVA for means and standard deviations, Tukey tests for honest significance, p<0,05)**

Table 5.11 (continued)

Age 35-44.9 n=127	Stratum									
Nutrient	1		2		3		4		5	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Calcium (mg)	397.1	289.8	572.7	333.4	431.8	208.0	417.9	151.0	522.5	256.7
Iron (mg)	8.3*	3.2	5.9	3.1	8.0	3.6	8.3	2.8	10.1*	3.5
Magnesium (mg)	357	124.1	250.4	110.2	296.1	136.0	288.6	86.7	288.0	91.9
Potassium (mg)	2008	820.5	1892	729.0	2035	580.8	2147.3	521.6	2417	725.5
Sodium (mg)	1098*	511.2	1043.6	546.1	983.2	557.5	1497.1	848.5	1564*	699.0
Zinc (mg)	7.8	2.7	6.7	3.6	7.9*	2.6	8.9	3.1	10.9*	3.1

* means with the same symbol differ significantly (ANOVA for means and standard deviations, Tukey tests for honest significance, p<0,05)

Age 45-54.9 n=103	Stratum									
Nutrient	1		2		3		4		5	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Calcium (mg)	525.1	256.6	485.2	363.9	393.7	223.8	395.3	209.9	603.2	238.2
Iron (mg)	9.7	3.5	4.7*	2.6	10.0	4.9	7.7#	3.01	11.1*#	4.1
Magnesium (mg)	351.2	109.3	217.2	125.8	309.9	140.5	286.46	103.1	304.7	127.3
Potassium (mg)	2317	671.4	1639*	753.4	2249	832.6	2029.8	557.9	2580.7*	919.7
Sodium (mg)	1350	582.0	702.4*	624.4	1291.6	728.0	997.9#	437.8	171*#	958
Zinc (mg)	9.9	3.4	5.3	2.7	8.6*	4.0	7.9	2.5	10.8*	3.8

*,# means with the same symbol differ significantly (ANOVA for means and standard deviations, Tukey tests for honest significance, p<0,05)

Age 55-64.9 n=63	Stratum									
Nutrient	1		2		3		4		5	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Calcium (mg)	435.5	229.2	751.2	366.7	437.9	228.8	436.1	331.3	567.0	132.5
Iron (mg)	9.5	6.2	4.9	1.1	8.3	3.2	7.5	3.3	12.8	2.2
Magnesium (mg)	305.4	133.5	232.7	76.3	333.9	128.5	263.6	105.6	375.7	25.6
Potassium (mg)	2166	737.9		717.0	2057	2034	1948	702	3062	442.7
Sodium (mg)	1062*	477.1	895.3	463.7	1305	666.1	881.3#	421.6	2105.7*#	490.2
Zinc (mg)	8.8	3.8	6.4	1.5	8.6	2.7	6.8	2.4	13.3	2.4

*,# means with the same symbol differ significantly (ANOVA for means and standard deviations, Tukey tests for honest significance, p<0,05)

Table 5.12 **Mean and standard deviation of reported mineral intakes for men for the different age groups**

Age 15-24.9 n=110	Stratum									
Nutrient	1		2		3		4		5	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Calcium (mg)	533.8	314.4	585.0	342.2	550.0	343.1	664.2	282.0	575.4	276.3
Iron (mg)	10.1	3.4	6.6[#]	3.1	9.2	4.1	11.6[*]	4.7	11.0[#]	4.1
Magnesium (mg)	353.1	153.6	250.0[*]	109.9	313.1	132.5	395.0[*]	148.2	355.1	116.9
Potassium (mg)	2342	773.1	1906	772.4	2005	1104	2842	1041	2632	223
Sodium (mg)	1845[*]	801	1058[*#&]	552	1395	733.5	2144[&]	881	1885[#]	790
Zinc (mg)	10.6	5.0	7.5[*]	2.9	9.8	4.7	12.0[*]	4.2	11.7	4.3

*,#,& means with the same symbol differ significantly (ANOVA for means and standard deviations, Tukey tests for honest significance, p<0,05)

Age 25-34.9 n=70	Stratum									
Nutrient	1		2		3		4		5	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Calcium (mg)	497.5	302.7	604.1	425.4	622.6	237.3	438.3	171.1	420.3	143
Iron (mg)	9.29	5.6	6.71	3.2	10.9	2.4	9.6	3.1	9.8	2.9
Magnesium (mg)	328.4	170.3	316.7	149.1	395.6	116.3	351.0	104.7	336.5	97.2
Potassium (mg)	2173	890.0	1960	851.1	2655.4	568.7	2339	534.7	2489	600.4
Sodium (mg)	1241	739.5	984.6[*]	507.3	1796	694.8	1548	653.8	1819[*]	851.1
Zinc (mg)	9.0	4.3	8.0	2.9	11.9	5.5	10.6	3.9	11.2	3.0

* means with the same symbol differ significantly (ANOVA for means and standard deviations, Tukey tests for honest significance, p<0,05)

Age 35-44.9 n=58	Stratum									
Nutrient	1		2		3		4		5	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Calcium (mg)	512.6	285.3	527.1	299.3	384.5	184.7	502.2	246.7	494.1	222.1
Iron (mg)	10.5	3.9	6.1[*]	2.0	10.0	3.6	9.7	3.7	10.3[*]	3.5
Magnesium (mg)	439.5	177.6	310.2	69.0	358.0	225.0	376.3	83.7	308.0	98.8
Potassium (mg)	2668	961.0	1989	428.4	2255	767.1	2548	674.1	2337	731.2
Sodium (mg)	1467	762	743	541	1074	541.0	1619	754	1593.7	892.4
Zinc (mg)	10.4	3.6	6.8	1.8	10.2	3.7	11.5	5.7	10.6	4.2

* means with the same symbol differ significantly (ANOVA for means and standard deviations, Tukey tests for honest significance, p<0,05)

Table 5.12 (continued)

Age 45-54.9 n=39	Stratum									
Nutrient	1		2		3		4		5	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Calcium (mg)	616.4	467.5	705.5	275.4	376.1	220.2	477.0	215.2	372.4	131.1
Iron (mg)	9.7	3.4	6.8	3.4	6.7	3.2	8.0	2.4	9.7	2.8
Magnesium (mg)	345.9	128.4	337.6	156.2	318.3	131.3	393.8	116.8	303.0	99.5
Potassium (mg)	2438	860.8	2248	591.9	1950	601.2	2243	631.8	2254.7	581.6
Sodium (mg)	1331	582.8	832.6	428.2	983.8	355.2	1171	464.9	1058	445.6
Zinc (mg)	9.8	4.4	7.8	3.4	7.3	2.6	8.6	2.2	10.7	4.1

Age 55-64.9 n=39	Stratum									
Nutrient	1		2		3		4		5	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Calcium (mg)	345.1	194.8	612.6	362	426.7	125.2	423.1	244.2	412.5	89.8
Iron (mg)	9.5	5.1	6.3	2.1	7.8	4.4	7.8	4.4	8.0	3.8
Magnesium (mg)	338.0	168.7	317.4	125.1	355.8	192.7	370.3	133.5	319.0	127.3
Potassium (mg)	2147	962.0	2006	743.2	2152	1011	2257	850.7	2279	433.5
Sodium (mg)	1219	820.8	908.8	451.5	1175	888.0	1238	769.2	1385	461
Zinc (mg)	9.5	5.0	6.7	2.1	7.7	3.7	9.7	4.2	10.3	2.8

5.5.2 Vitamin A intakes

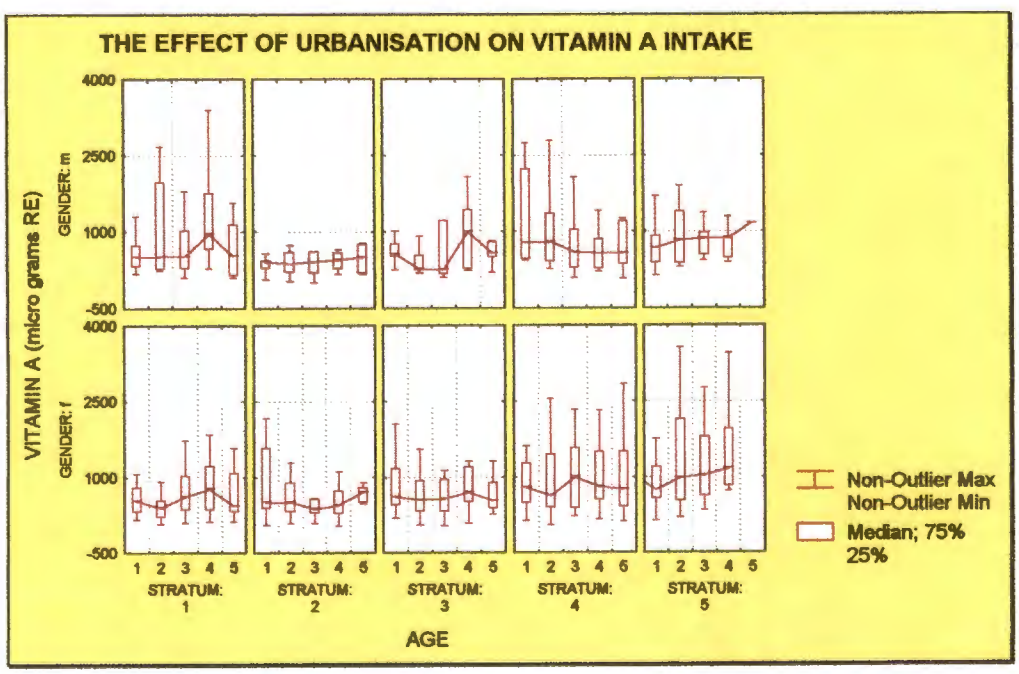


Figure 5.8 **Median vitamin A intake of men and women of the different age groups of strata 1 - 5.**

Vitamin A intake is depicted in Figure 5.14. Here it can be seen that there were not marked differences. In both genders the RDA was achieved (between 800-1200). Thus it can be said that their intakes were adequate.

5.5.3 Vitamin E intakes

Table 5.9 shows that mean vitamin E intakes ranged from 5.49 to 15.2 mg per day for the women and 5.68 to 14.3 mg per day for men. The farm workers had the lowest intake. It appears as if urbanisation did not markedly affect vitamin E intakes, other than slightly higher intakes in the two urban strata. The mean intakes for men and women across strata is shown in Table 5.13 (summarised from Tables 5.9 and 5.10).

Table 5.13 Mean vitamin E intake (mg) per stratum.

Stratum	Men aged 15-64.9	Women aged 15-64.9
1	10.66±0.81	9.44±0.89
2	7.28±1.27	7.88±2.21
3	10.51±2.47	9.95±1.14
4	12.12±1.71	9.87±1.58
5	11.84±1.64	11.42±2.2

* Means calculated from means of different age groups as shown in Tables 5.9 and 5.10

5.5.4 Vitamin C (ascorbic acid) intakes

The mean and standard deviations of intakes of vitamin C for men and women of the different age groups in each stratum, are shown in Tables 5.9 to 5.10. In Table 5.14, mean intakes for men and women of all ages (15-64.9 years) for each stratum are summarised.

Table 5.14 Mean vitamin C intakes (mg/day) per stratum *

Stratum	Men aged 15-64.9	Women aged 15-64.9
1	48.5±25.0	45.1±10.6
2	17.3±4.2	26.1±7.6
3	23.5±8.3	36.7±15.1
4	34.4±17.3	55.2±21.2
5	63.4±13.2	80.9±17.5

* Means calculated from means of different age groups as shown in Tables 5.9 and 5.10.

Tables 5.9, 5.10 and 5.14 show very low mean intakes of vitamin C with large standard deviations, indicating that within groups there were large variations in vitamin C intake. The RDA for adult men and women is 60 mg/day (Garrow & James, 1993). Clearly, many subjects did not have adequate vitamin C intakes. The lowest intakes were observed in the farm workers (stratum 3) and the highest in stratum 5, the only stratum with adequate mean intakes.

5.5.5 Folate intakes

Table 5.15 shows the mean folate intakes of men and women calculated from mean and standard deviation values reported in Tables 5.9 and 5.10.

Table 5.15 Mean folate intakes (μg/day) per stratum *

Stratum	Men aged 15-64.9	Women aged 15-64.9
1	231±24.1	208±18.8
2	174±13.8	151±12.4
3	226±21.5	185±16.0
4	234±26.9	216±30.9
5	239±14.8	250±48.8

* Means calculated from means of different age groups as shown in Tables 5.9 and 5.10.

The RDA for folate for adult men and women is 200 and 150μg/day respectively (Food and Nutrition Board, 1989; Garrow & James, 1993). From the above table it seems as if mean folate intakes were generally adequate except for the farm workers who had low intakes. The highest intakes were seen in stratum 5.

5.5.6 Vitamin D intakes

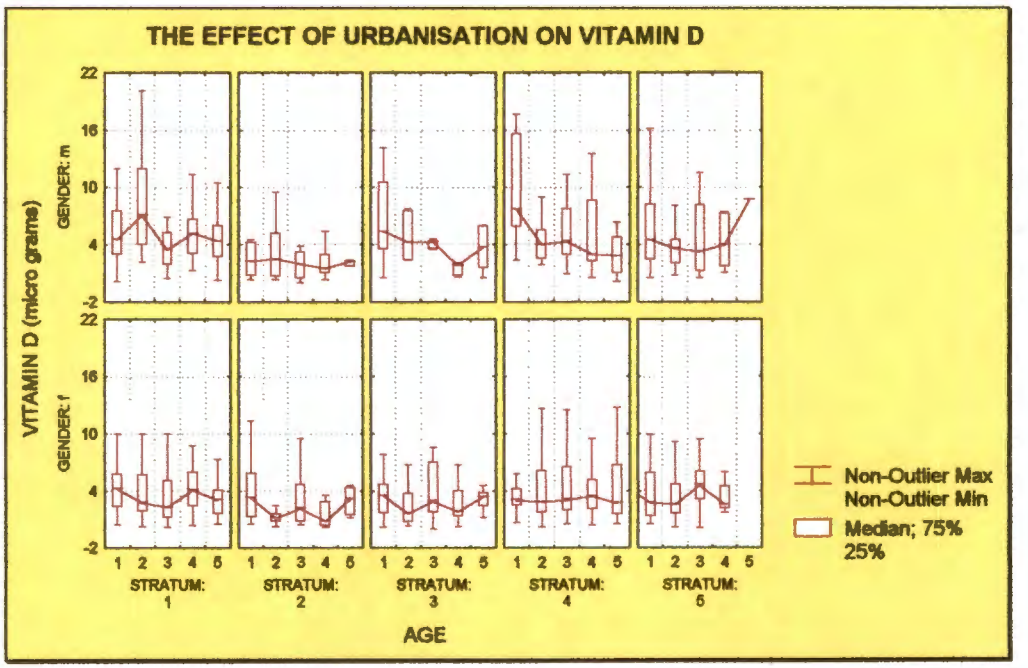


Figure 5.9 **Median of vitamin D intake of men and women of the different age groups of strata 1 - 5.**

The effect of urbanisation on reported vitamin D intakes is illustrated in Figure 5.9. The figure shows no definite pattern of differences between groups. It seems that urbanisation did not influence vitamin D intake to any marked extent. The RDA for vitamin D is 5µg/day. Figure 5.9 shows that in many groups, intakes seem to be low. However, vitamin D values are not available for all South African foods and judgement of adequacy of intake is therefore not possible.

5.5.7 Calcium intakes

Mean and standard deviations of calcium intakes are shown for women in Table 5.11 and men in Table 5.12. From these tables, means were calculated, combining the age groups to show the effect of stratum (Table 5.16).

Table 5.16 Mean calcium intakes (mg/day) per stratum *

Stratum	Men aged 15-64.9	Women aged 15-64.9
1	501±98	449±47
2	607±65	612±98
3	472±109	409±26
4	501±96	429±23
5	455±80.3	532±61

* Means calculated from means of different age groups as shown in Tables 5.11 and 5.12.

The RDA for adult men and women is 800 mg (Food and Nutrition Board, 1989), while the lower reference nutrient intake (Garrow & James, 1993) is 400 mg.

The changes in serum calcium in each age group across all 5 strata are illustrated in Figure 5.10. Values were mostly in the normal range of 2.20-2.60 mmol/L (DAX, 1996). In men it looks as if there is a slight but progressive decrease in serum calcium with urbanisation. Despite the higher reported intakes of men and women from the farms, their serum calcium concentrations were mostly lower than those of subjects in strata 1 and 3. Although the mean values in all strata were less than the RDA, all groups had a mean value of > 400 mg. In contrast to other nutrient intakes, the farm workers had the highest mean intake, possibly because of the availability of fresh milk on farms.

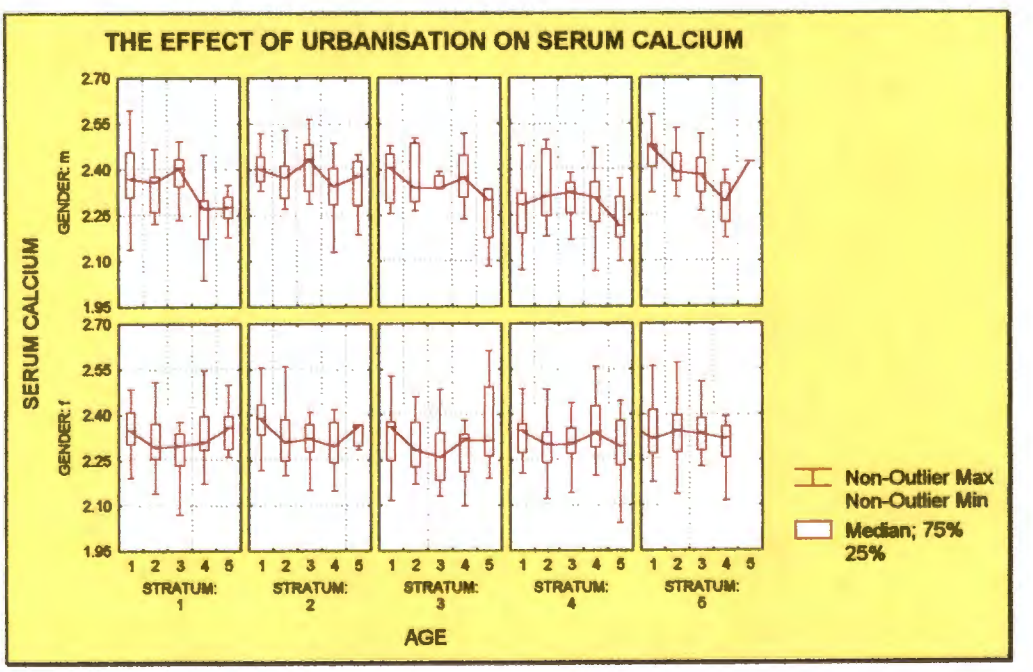


Figure 5.10 Median serum calcium of men and women of the different age groups of strata 1 - 5.

5.5.8 Iron intakes

Mean iron intakes, calculated from the means showed in Tables 5.11 and 5.12 are summarised in Table 5.17.

Table 5.17 Mean iron intakes (mg/day) per stratum*

Stratum	Men aged 15-64.9	Women aged 15-64.9
1	9.82±0.48	9.14±0.73
2	6.50±0.29	6.52±1.91
3	8.92±1.68	8.50±0.87
4	9.34±1.54	8.64±1.19
5	9.76±1.11	11.0±1.17

* Means calculated from means of different age groups as shown in Tables 5.11 and 5.12.

The RDA for iron for adult women is 15 mg/day and for men 10 mg/day (Food and Nutrition Board, 1989). The lower reference nutrient intake is 4.7 mg/day for men and 8 mg/day for women (Garrow & James, 1993). From Tables 5.11 and 5.12 it seems that none of the female groups had a mean intake near 15 mg/day, while 8 of the male groups had a mean intake ≥ 10 mg/day. Table 5.17 shows that the farm workers in stratum 3 had the lowest intakes. The men met the lower reference nutrient intake but the women had a mean value of 6.52 mg/day, substantially lower than the recommended minimum of 8 mg/day. The highest intakes were seen in strata 1 and 5, possibly because rural people in stratum 1 often had their own meat supply, while those in stratum 5 could afford a more varied diet.

5.6 Meat, chicken and egg intakes (gram per day)

Table 5.18 Mean intakes of meat and meat dishes (g/day) of men and women of the different strata*

TOTAL MEAT INTAKE	Men aged 15-64.9	Women aged 15-64.9
	Mean	**Mean**
Stratum 1	54.12	32.02
Stratum 2	26.56	31.81
Stratum 3	64.8	57.36
Stratum 4	75.98	64.85
Stratum 5	100.77	94.68

* Means calculated from means of different age groups as shown in Table 9.1, addendum 9

Table 5.19 Mean intakes of chicken and chicken dishes (g/day) of men and women of the different strata*

TOTAL CHICKEN INTAKE	Men aged 15-64.9	Women aged 15-64.9
	Mean	Mean
Stratum 1	43.6	21.6
Stratum 2	16.2	18.87
Stratum 3	24.49	28.06
Stratum 4	33.02	30.2
Stratum 5	29.15	28.18

* Means calculated from means of different age groups as shown in Table 9.1, addendum 9

Tables 5.18 and 5.19 and 5.20 summarise the total mean intakes of meat, chicken and eggs respectively by subjects in the 5 strata. The contributions of the different types of meat and preparation methods of meat and chicken for the different strata are given in Addendum 9, Table 9.1. The mean values are illustrated in Figures 5.12 and 5.13.

From these tables and figures it is clear that the farm workers had the lowest intake of both red meat and chicken. The total amount of 42.7 g/day for men and 50.7 g/day for women is only half a portion of meat or chicken per day, indicating that meat or chicken was not eaten every day.

It is also clear that in both men and women the intake of red meat increased substantially from stratum 1 to 3, 4 and 5. Men from stratum 5 had almost double the intake than those from stratum 1 and women almost three times higher intakes.

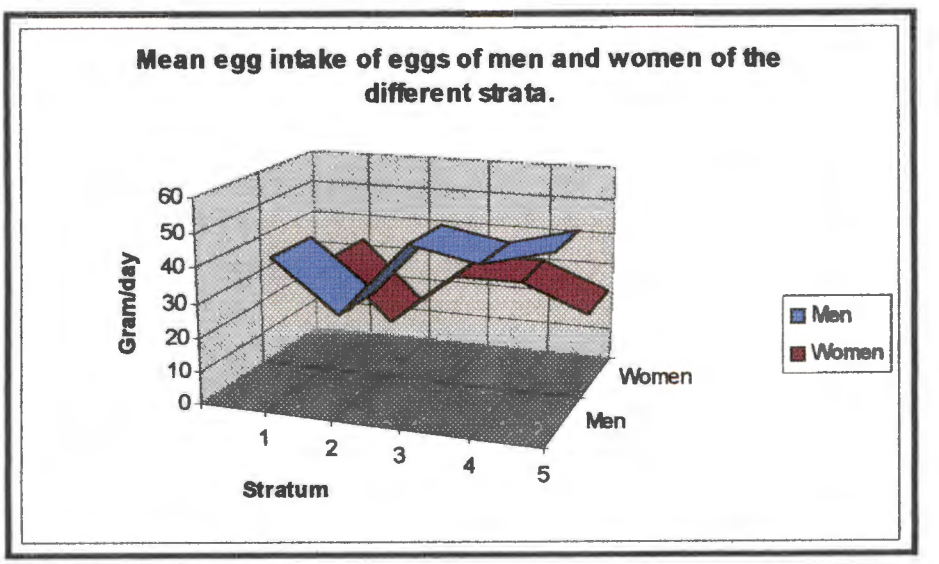


Figure 5.11 Mean meat intake by subjects in the 5 strata

The egg intake of this population were relatively low and still remained low as portrayed in the above mentioned Figure 5.11

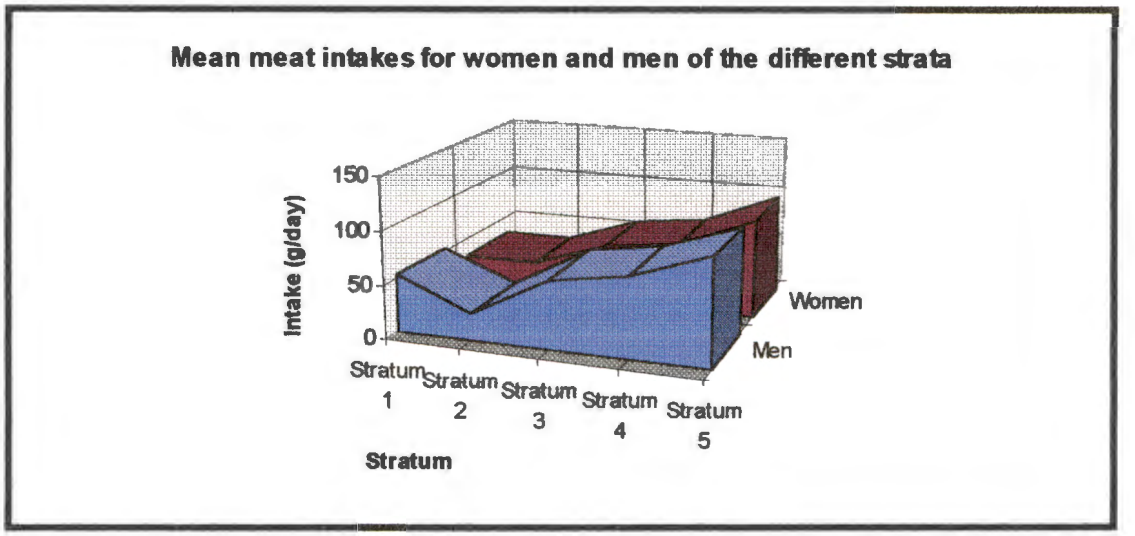


Figure 5.12 Mean meat intake by subjects in the 5 strata.

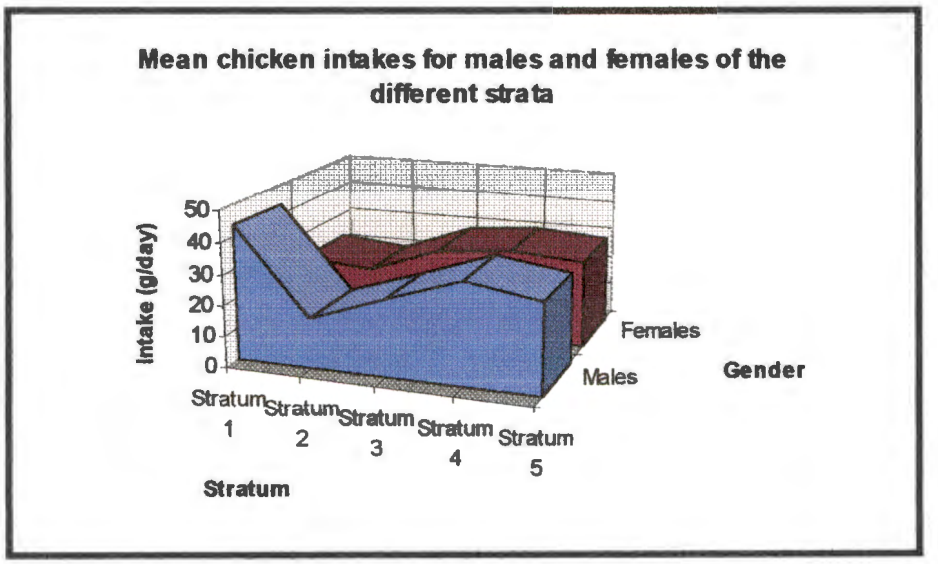


Figure 5.13 Mean chicken intakes by subjects in the 5 strata.

Chicken intakes were approximately a third of red meat intakes and did not increase across strata. Women generally had slightly lower intakes than men.

Table 9.1 (Addendum 9) shows that in all strata the cooking method mostly used for red meat (mostly chuck) was braising with fat while that for chicken was roasting with skin.

5.7 VEGETABLE AND FRUIT INTAKES

5.7.1 Vegetable intake

Mean and standard deviations of intakes of all the different types of vegetables by men and women of all 5 strata are shown in Table 10.1 (Addendum 10) and are summarised in Table 5.20.

Table 5.20 Mean total intakes of vegetables (g/day) of men and women in the different strata*

Stratum	Men aged 15-64.9	Women aged 15-64.9
1	113.9	100.2
2	67.9	87.3
3	91.0	105.4
4	101.9	115.1
5	114.5	128.9

* Calculated from the means of individual vegetables reported in Table 10.1.

Table 5.20 shows that the mean intake of vegetables in these subjects were very low. If 100g cooked vegetables is regarded as a portion (Langenhoven *et al.* 1986), only one portion of vegetables per day was eaten. The smallest intakes were seen in the men and women of stratum 3 and the largest intake of 129 g/day in the women of stratum 5. Men from stratum 1 and stratum 5 had larger intakes than those of the other strata. Table 10.1 (Addendum 10) shows that potato, onions, tomatoes, dried beans, cabbage, pumpkin and beetroot were the most popular vegetables.

5.7.2 Fruit intakes

Mean intakes of individual fruits are shown in Table 11.1 (Addendum 11) while total intakes per strata for men and women are summarised in Table 5.21. From this table it can be seen that fruit intakes were also low. If 100 g is regarded as a portion (Langenhoven *et al.* 1986), the farm workers had less than half a portion daily. Men and women from strata 1, 3 and 4 ate between one and one and a half portion daily while those from strata 5 just more than two portions daily.

Table 5.21 Mean total intakes of fruit (g/day) of men and women of the different strata*

Stratum	Men aged 15-64.9	Women aged 15-64.9
1	116.4	100.6
2	39.9	32.2
3	99.8	132.6
4	128.1	170.1
5	207.1	239.5

* Calculated from means of individual fruit shown in Table 11.1 (Addendum 11)

Table 11.1 indicates that in all strata apples, oranges and bananas were the fruits mostly eaten, with grapes, pears and fresh peaches following.

5.8 OTHER LIFESTYLE RISK FACTORS

5.8.1 Body mass index (BMI)

The mean and standard deviations of the BMI in kg/m^2 of the men and women of different age groups for all the strata are given in Table 5.22. The mean BMI for strata, combining the age groups, are also given.

Table 5.22 Mean BMI in kg/m^2 of men and women.

BMI: Men

Age group	n	Stratum 1	n	Stratum 2	n	Stratum 3	n	Stratum 4	n	Stratum 5
15-24.9	27	19.1±2.40	13	19.9±1.52	11	21.0±1.55	9	19.5±1.38	35	20.3±2.95
25-34.9	11	22.9±6.51	14	20.9±2.34	8	21.0±3.95	19	21.6±2.43	18	23.9±3.42
35-44.9	18	20.6±2.64	12	25.2±13.07	5	19.1±2.38	15	21.7±3.65	13	25.1±1.91
45-54.9	14	24.8±6.05	21	21.2±3.93	8	19.1±2.68	18	23.2±5.43	6	25.7±3.18
55-65	15	22.0±3.70	9	19.2±1.51	5	20.3±2.69	11	22.0±7.30	1	25.7±-
Total 15-64.9	**85**	**21.9±2.2**	**69**	**21.3±2.3**	**37**	**20.1±1.0**	**72**	**21.6±1.3**	**73**	**24.1±2.3**

Table 5.22
BMI: Women

Age group	n	Stratum 1	n	Stratum 2	n	Stratum 3	n	Stratum 4	n	Stratum 5
15-24.9	29	22.5±4.04	13	21.4±4.65	15	25.3±5.19	13	24.1±4.11	34	23.2±4.62
25-34.9	31	25.2±5.51	13	23.0±4.54	19	29.3±8.18	48	27.1±6.44	27	28.1±6.03
35-44.9	30	28.6±6.32	14	28.5±7.73	22	28.3±6.70	27	30.3±7.52	29	29.9±5.75
45-54.9	33	26.1±7.76	14	27.0±4.16	11	27.5±8.17	22	30.7±9.92	11	29.9±4.43
55-65	16	29.4±6.41	6	23.4±4.03	10	30.9±5.93	14	26.3±6.88	-	-
Total 15-64.9	**139**	**26.4±2.8**	**60**	**24.7±3.0**	**77**	**28.3±2.1**	**124**	**27.7±2.8**	**101**	**27.8±3.2**

From Table 5.22 it can be seen that the mean BMI's of men were substantially lower than those of the women. In all the strata, mean BMI of women increased with age. In men, this was only noticeable in stratum 5. In men, the mean BMI of strata 1 to 4 were similar. Men of stratum 5 had a mean BMI of 24.1 kg/m^2 compared to the 20.6 - 21.6 kg/m^2 of men in the other strata. In women, the two rural strata (1 and 2) showed mean BMI's of 26.4 and 24.7 kg/m^2, compared to the mean values of 28.3, 27.7 and 27.8 kg/m^2 of the three urban strata. Therefore, it seems that in addition to an effect of age in women, there was a slight increase in BMI with urbanisation in women across all strata and of men in stratum 5.

5.8.2 Adult height

The mean and standard deviations of the height of the different groups of subjects in each stratum is given in Table 5.23. Because it is possible that subjects from the 15-24.9 year old age group has not reached adult height, in the calculation of the mean heights per stratum of men and women, only the subjects aged 25 and older were included.

Table 5.23 shows that the mean heights for men and women of stratum 5 were higher than those of the other strata. The men and women from the farms had the lowest height.

Table 5.23 Mean heights (cm) of men and women.

Men

Age group	n	Stratum 1	n	Stratum 2	n	Stratum 3	n	Stratum 4	n	Stratum 5
15-24.9	27	1.68±0.06	13	1.71±0.04	11	1.68±0.05	9	1.73±0.07	35	1.71±0.07
25-34.9	11	1.72±0.06	14	1.68±0.07	8	1.69±0.05	19	1.68±0.06	18	1.71±0.07
35-44.9	18	1.69±0.07	12	1.66±0.05	5	1.68±0.04	15	1.67±0.08	13	1.71±0.05
45-54.9	14	1.68±0.06	21	1.68±0.06	8	1.68±0.04	18	1.67±0.07	6	1.72±0.07
55-65	15	1.68±0.06	9	1.66±0.08	5	1.66±0.05	11	1.66±0.06	1	1.75± -
Total mean *	**58**	**1.69±0.02**	**56**	**1.67±0.01**	**26**	**1.68±0.01**	**63**	**1.67±0.01**	**38**	**1.71±0.01**

Women

Age group	n	Stratum 1	n	Stratum 2	n	Stratum 3	n	Stratum 4	n	Stratum 5
15-24.9	29	1.58±0.05	13	1.56±0.06	15	1.58±0.06	13	1.59±0.07	34	1.60±0.07
25-34.9	31	1.57±0.07	13	1.56±0.04	19	1.57±0.07	48	1.58±0.07	27	1.61±0.06
35-44.9	30	1.58±0.04	14	1.56±0.07	22	1.56±0.06	27	1.57±0.06	29	1.59±0.06
45-54.9	33	1.58±0.06	14	1.57±0.06	11	1.57±0.06	22	1.56±0.06	11	1.58±0.03
55-65	16	1.59±0.05	6	1.50±0.05	10	1.55±0.07	14	1.54±0.04	-	-
Total mean *	**110**	**1.58±0.01**	**47**	**1.55±0.03**	**62**	**1.57±0.01**	**111**	**1.56±0.02**	**67**	**1.59±0.02**

* Without the subjects aged 15-24.9 years, and excluding the one man in stratum 5.

In the younger subjects (15-24.9 year old group), an increase in height across strata is observed. This is especially noticeable in the women, with the exception of women from stratum 2, who were shorter than those from the other groups.

5.8.3 Smoking habits

Table 5.24 The frequency of smokers and snuff takers in the different age groups of men and women in all strata

		AGE GROUPS					
Stratum	**Gender**	**15-24.9**	**25-34.9**	**35-44.9**	**45-54.9**	**55-64.9**	**TOTAL MEAN**
1	M	7.4	63.6	89.5	60.0	50.0	48.9
1	F	17.2	25.8	40.0	45.5	50.0	34.5
2	M	53.8	73.3	76.9	85.7	77.8	74.6
2	F	38.5	61.5	57.1	64.3	16.7	51.7
3	M	72.7	62.5	100.0	75.0	80.0	75.7
3	F	33.3	26.3	66.7	72.7	50.0	49.4
4	M	77.8	47.3	60.0	55.6	72.7	59.7
4	F	7.7	27.1	22.2	50.0	66.6	32.8
5	M	25.7	38.9	69.2	16.7	-	36.1
5	F	2.9	3.7	3.4	18.2	-	5.0

* Percentage of total subjects in group

The percentage of subjects who smoked are given for men and women of each age group for all strata in Table 5.24. The mean total for each stratum is also shown. In both men and women, more subjects in strata 2 and 3 smoked. Less subjects smoked in stratum 4 than in 2 and 3, and stratum 5 had lower percentages of smokers than stratum 1. More men than women smoked. It should be noted that "smoking" data included women who snuffed tobacco, which accounted for the majority of the women "smokers". The high percentage of young smokers (age 15-24.9) in strata 2, 3 and 4 (men) is particularly noticeable. The mean "packet years" of smoking, calculated as

the product of the number of years the subject has been smoking and the number (or fraction) of packets of 20 cigarettes, are shown in Table 5.25. The gram tobacco snuffed or used in pipes or home-made cigarettes are also shown in the table.

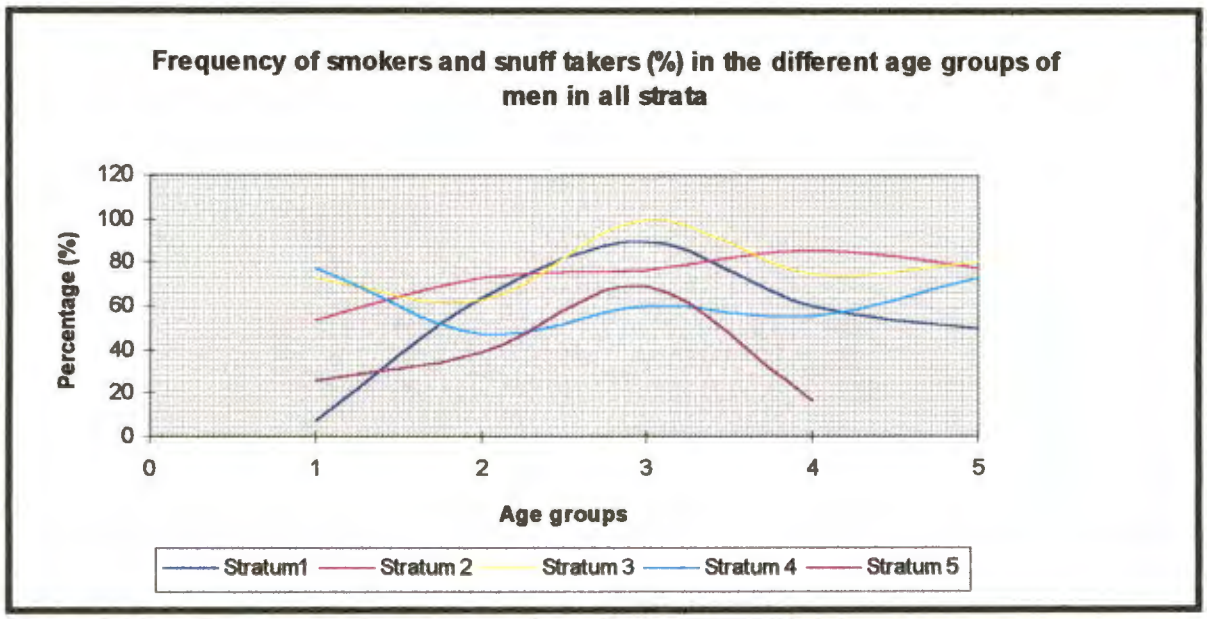


Figure 5.14 Frequency of smokers and snuff takers.

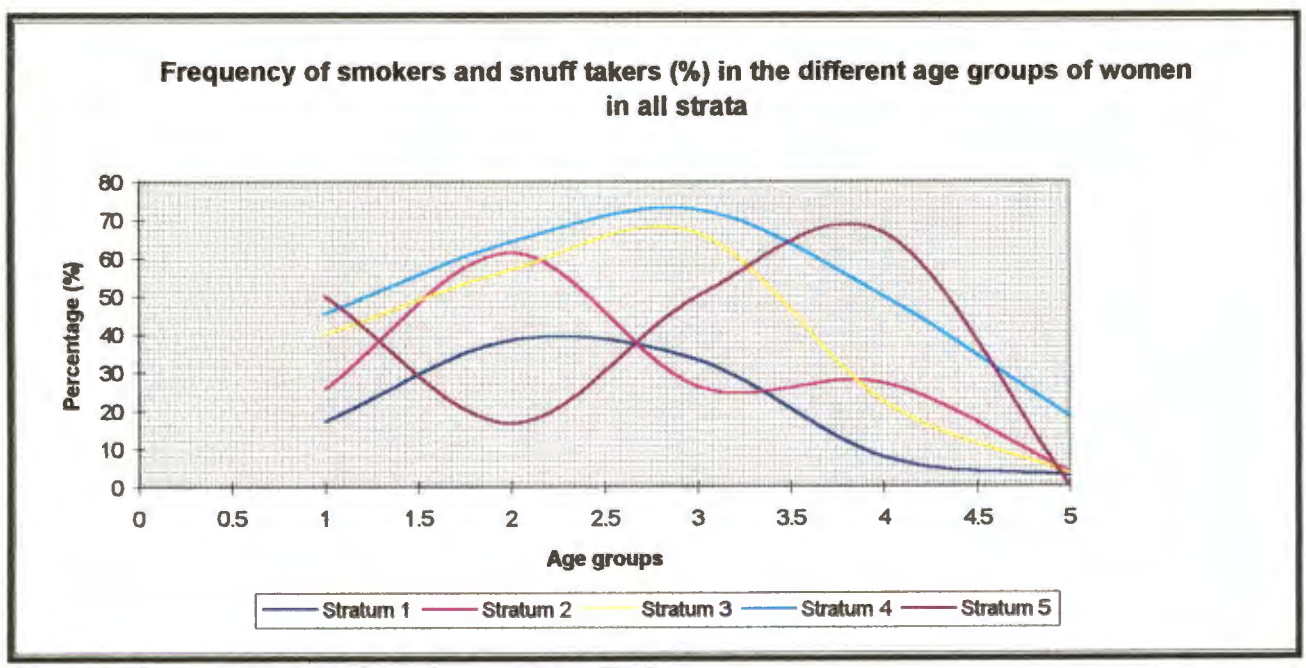


Figure 5.15 Frequency of smokers and snuff takers.

Table 5.25 **Pack years of smokers (and snuff takers) in the different age groups of women and men in all strata.**

Women	Stratum 1		Stratum 2		Stratum 3		Stratum 4		Stratum 5	
Age group										
15-24.9	-	-	-	-	-	-	-	-	-	-
25-34.9	-	-	0.046	0.166	0.028	0.119	0.467	2.626	-	-
35-44.9	0.369	1.989	-	-	1.054	2.890	0.185	0.962	-	-
45-54.9	-	-	-	-	0.020	0.063	2.157	7.834	-	-
55-64.9	-	-	-	-	0.625	1.768	1.097	2.426	-	-

Men	Stratum 1		Stratum 2		Stratum 3		Stratum 4		Stratum 5	
Age group										
15-24.9	0.006	0.029	0.258	0.871	1.468	2.232	1.827	1.880	0.144	0.380
25-34.9	0.201	0.585	0.470	1.554	2.676	5.098	2.895	4.828	1.268	2.350
35-44.9	1.032	3.652	2.182	5.693	6.720	9.888	3.243	5.964	2.320	3.268
45-54.9	1.746	4.992	2.571	9.739	0.375	1.061	8.413	29.48	0.833	2.041
55-64.9	2.200	8.521	-	-	15.60	19.68	7.89	15.30	-	-

Table 5.26 **Daily gram tobacco used by smokers and snuff takers in the different age groups of women and men in all strata.**

Women	Stratum 1		Stratum 2		Stratum 3		Stratum 4		Stratum 5	
Age group										
15-24.9	2.078	5.310	6.838	12.25	9.75	27.83	0.145	0.521	0.441	2.572
25-34.9	6.19	20.37	6.479	9.62	1.05	2.54	2.951	7.204	0.278	1.443
35-44.9	4.967	12.08	8.821	15.82	9.617	14.72	2.19	7.712	0.517	2.795
45-54.9	7.606	12.43	25.79	39.02	3.86	10.57	2.964	6.858	0.205	0.630
55-64.9	11.08	16.42	-	-	7.750	11.69	4.033	9.441	-	-

Men	Stratum 1		Stratum 2		Stratum 3		Stratum 4		Stratum 5	
Age group										
15-24.9	1.037	5.389	15.39	29.82	9.091	30.15	-	-	-	-
25-34.9	18.55	24.73	16.64	21.10	12.50	35.36	0.497	2.168	-	-
35-44.9	25.92	29.87	36.49	35.33	25.00	43.30	-	-	-	-
45-54.9	12.78	20.37	30.94	29.66	15.24	12.66	12.20	19.42	-	-
55-64.9	19.97	36.60	32.44	38.92	2.500	5.590	12.50	26.81	-	-

5.8.4 Activity levels

Table 5.27 **The frequency (%)* of subjects who had low, medium or high activity levels#**

Stratum	Gender	#	AGE GROUPS 15-24.9	25-34.9	35-44.9	45-54.9	55-64.9	TOTAL MEAN
1	M	L	3.7	0.0	22.2	13.3	43.8	16.6±17.5
		M	48.2	90.0	72.2	66.6	50.0	65.4±17.2
		H	48.2	10.0	5.6	20.0	6.3	18.0±17.8
1	W	L	34.5	50.0	20.0	16.2	43.8	32.9±14.7
		M	58.6	50.0	76.7	74.2	56.3	63.2±11.7
		H	6.9	0.0	33.3	9.7	0.0	10.0±13.7
2	M	L	15.4	20.0	7.7	14.3	66.7	24.8±23.8
		M	76.0	73.3	61.5	76.2	22.2	61.8±23.0
		H	7.7	6.7	30.8	9.5	11.1	13.2±10.0
2	W	L	0.0	15.4	28.6	28.6	33.3	21.1±13.5
		M	100.0	69.2	64.3	64.3	66.7	72.9±15.3
		H	0.0	15.4	7.1	7.1	0.0	5.9±6.4

3	M	L	27.3	37.5	20.0	50.0	40.0	35.0±11.6
		M	72.7	50.0	80.0	50.0	60.0	62.5±13.5
		H	0.0	12.5	0.0	0.0	0.0	2.5±5.6
3	W	L	40.0	76.5	65.2	63.6	60.0	61.1±13.3
		M	60.0	23.5	30.4	27.3	30.0	34.2±14.7
		H	0.0	0.0	4.3	9.1	10.0	4.7±4.8
4	M	L	11.1	16.7	20.0	23.5	55.0	25.3±17.2
		M	66.7	50.0	66.7	70.6	45.0	59.8±11.5
		H	22.2	33.3	13.3	5.9	0.0	14.9±13.2
4	W	L	38.5	60.9	46.2	27.3	53.3	45.1±13.1
		M	23.1	32.6	50.0	63.6	26.7	39.2±17.1
		H	38.5	2.2	3.8	9.1	20.0	14.7±15.0
5	M	L	5.7	5.5	7.7	16.7	-	8.9±5.3
		M	60.0	94.9	92.3	50.0	-	74.2±22.5
		H	34.3	0.0	0.0	33.3	-	16.9±19.5
5	W	L	18.2	25.9	27.6	18.2	-	22.5±5.0
		M	57.6	66.7	69.0	63.6	-	64.2±4.9
		H	24.2	7.4	3.4	18.2	-	13.3±9.6

* Percentage of total

\# Low : Subjects sat most of the times

Medium: Own housework; walk medium distances; stand and walk during working hours

High : As in medium plus regular participation in sport or exercise activities.

The percentages of subjects who reported low, medium or high activity levels, are shown in Table 5.27. Activity levels were categorised after questions on doing own housework, type of job, distances walked each day and participation in sport or other exercise were answered. Low, medium or high categories are therefore only a rough indication, but because the same standards for judgement were used in all subjects, comparisons across strata are possible. The table indicates that strata 1, 4 and 5 had the highest percentages of subjects with high activity. In stratum 1 it was mainly on long distance walked daily while in strata 4 and 5 more subjects participated in sport (mainly soccer or running). Stratum 3 had the highest percentages of subjects with low activity. The proportion of subjects who reported medium activity were the largest in urban men (stratum 5) and women living on farms (stratum 2).

5.9 MAIN FINDINGS OF THIS STUDY

In Table 5.28 the effects of urbanisation on the diet and lifestyle related risk factors found in this study are summarised.

Table 5.28 Effect of urbanisation on colorectal cancer risk factors

Risk factor	Changes observed in mean values across strata and interpretation of data reported in Chapter 5
FOOD INTAKES Red meat	The highest intakes (101 and 95 g red meat/day) observed in men and women of stratum 5. Men had almost double the intake of those in stratum 1 and women three times higher. Farm workers had very low intakes - only about half a portion per day.
Chicken	Was about a third of red meat intake and did not increase across strata.
Vegetables	Intakes across all strata were low - only one portion per day. Rural men (S1) and urban women (S5) had the highest intake (113 and 121g/day).
Fruit	Intakes generally low. Farm workers ate only half a portion per day. Men and women from strata 1, 3 and 4 had approximately one and a half portion daily and those in stratum 5 about 2 portions.
NUTRIENT INTAKES Energy	No consistent changes across strata observed. Intakes seem adequate, except in some farm workers.
Total protein	Intakes were adequate. In most age groups there was a progressive increase in men and women across strata except that groups in stratum1 often had higher intakes than some in 2 and 3.
Plant/animal protein ratios	Clear indications that in men and women the intake of animal protein increased across strata and that the ratio of plant protein decreased with urbanisation.
Total fat	A progressive increase in fat intake across strata was observed. The farm workers generally had the lowest intakes. Men and women of stratum 5 took in more than 30% of their energy as fat.
P/S ratio of the diet	The P/S ratio gradually decreased across strata (urban strata took in more saturated fat) but the diet of the farm workers also had a low P/S ratio.
Total carbohydrate (CHO)	A decrease in the % of total energy provided by carbohydrates was observed. All age and gender groups in stratum 5 still had intakes > 50% of total energy. The farm workers had the highest intakes.
Dietary fibre (g/MJ)	Relatively low values in all ages and strata observed. Farm workers had the lowest intakes. Intakes in other strata very similar. No change with urbanisation.
Calculated starch	Starch intakes decreased progressively across strata. In men there was a ±62 g/day difference between strata 1 and 5, and in women ±45 g/day.
Alcohol	Men reported higher intakes than women, confirmed with serum GGT values. Alcohol intake probably increase with urbanisation in young men and in women across strata up to 55 years of age. The other men already had high intakes.
Vitamin D	No consistent changes across strata observed. Intakes seem adequate, except in some farm workers.
Vitamin E	The farm workers had the lowest intakes and the urban subjects from strata 4 and 5 the highest. Little difference between strata observed.
Vitamin C	Only subjects in stratum 5 had adequate mean intakes. All other strata, especially 1, 2 and 3 showed exceptionally low intakes.
Folate	Mean intakes seemed generally adequate except in farm workers. Highest mean intake seen in stratum 5.
Calcium	Mean intakes were < RDA but > LRI. The farm workers had the highest intake. No discernible pattern across strata.
Iron	Farm workers had very low intakes. The highest intakes were seen in strata 1 and 5. Mean intakes of women were < RDA.
OTHER Body mass index (BMI)	A clear influence of age in women observed (but not in men, except for stratum 5). BMI increased across strata in women.
Height	Urban groups had greater height than rural groups. Women from stratum 2 (farm workers) were the shortest.
Smoking	More subjects smoked in strata 2 and 3 than in 1. Stratum 5 had the lowest % of smokers.
Activity	No clear effect of urbanisation on activity levels observed. Strata 1, 4 and 5 had the most subjects with high activity levels and strata 3 with low levels.

CHAPTER 6

DISCUSSION

6.1 INTRODUCTION

The main objective of this study was to examine the effect of urbanisation and acculturation of black South Africans living in the Northwest Province, on their risk factors (mainly diet and lifestyle) for colorectal cancer. The motivation underlying this study is the low prevalence and incidence of colorectal cancer in blacks compared to the other ethnic groups in South Africa. The reasons for these differences are not known and the objective of this discussion is to evaluate changes of risk factors in the African population in order to develop a hypothesis that could explain these low rates, despite marked increases in other chronic diseases of lifestyle (Fourie & Steyn, 1995).

The design of the study is a cross-sectional survey and not (at present) a long-term follow-up of subjects. Comparisons of the presence and level of risk factors between subjects living in rural and urban communities were done. Observed differences in subjects stratified for gender and age group and level of urbanisation (in 5 strata from rural to urban) are seen as a possible effect of urbanisation. "Changes in risk factors" are therefore equated to differences between strata for the same age and gender group.

In addition to body mass index, smoking habit and activity levels, the colorectal cancer risk factors examined in this study were the dietary factors. Accurate and reliable measurement of dietary intakes was therefore necessary. A team, under leadership of Ms. Una MacIntyre, took responsibility for measurement of dietary patterns, habits and nutrient intakes (MacIntyre, 1998). The quantitative food frequency questionnaire used, was designed after a pilot study among Africans in the Northwest Province. This questionnaire was validated extensively (MacIntyre, 1998) and administered by Setswana-speaking fieldworkers, specially trained in interviewing techniques. Therefore, the dietary and nutrient intake data reported in this

thesis, is regarded as being as accurate as possible. Mean intakes of individual nutrients and percentages of subjects not reaching recommended amounts (Food and Nutrition Board, 1989) were compared with intakes reported for other groups of rural and urban Africans (reviewed by Vorster *et al.* 1997) and seemed realistic.

6.2 CHARACTERISTICS OF THE POPULATION

The African population of the Northwest Province is mainly rural and of the Tswana tribe. Other cultural groups are also present in this Province such as Zulu's, Shangaans and Xhosa's. Looking at each stratum, one can summarise the characteristics of the population in the different strata as follows:

Stratum 1: Rural	The subjects in this stratum lived in villages. Some had their own homes and land (plots) with livestock such as goats, oxen and chicken. Not many had vegetable gardens or crops because provision of electricity and adequate water is not that available.
Stratum 2: Farm workers	These subjects were very poor and mostly illiterate. They were also the group which were the most undernourished. Their living conditions were bad as a result of their poverty. Families lived in brick houses provided by the white farmers on the land. Water and electricity were available but not everywhere. Only a few had vegetable gardens.
Stratum 3: Squatters	This population is the population in transition. They lived in shacks (makuku's) and informal settlements at the fringes of major cities. Their houses were built from corrugated iron and other material. Unemployment is a major problem under these subjects and as a result the crime and violence rates are high. Water and sanitation as well as electricity supply is a problem. Many squatters have to walk several kilometres to get water.
Stratum 4 + 5: Urban	These subjects, S4 (middle class) and S5 (upper class) were from the established areas near or adjacent to big cities and towns or formerly white residential areas. Most of these subjects were professional people or employees that lived in brick houses owned by themselves. Water and electricity is not a problem.

6.3 RISK FACTORS FOR COLORECTAL CANCER

6.3.1 Evidence from the literature

In Table 6.1 the dietary and lifestyle factors thought to be related to the risk of colorectal cancer are summarised (WCRF & AICR, 1997).

Table 6.1 Dietary and lifestyle risk factors of colorectal cancer*

Strength of evidence	Decreased risk	No relationship	Increased risk
Convincing	Physical activity (colon) Vegetables Aspirin; NSAIDS#		Smoking tobacco
Probable			Red meat, alcohol
Possible	NSP/fibre Starch Carotenoids	Calcium Selenium Fish	High body mass index (BMI) Greater adult height Frequent eating Sugar (sucrose) Total fat Saturated/animal fat Processed meat Eggs Heavily cooked meat Iron Total energy intake (NJP)▲ MUFAT (NJP) Cholesterol (NJP) Protein (NJP)
Insufficient	RS• Vitamin C Vitamin D Vitamin E Folate Methionine Cereals Coffee PUFA (NJP)†	Poultry (NJP) Dairy products (NJP)	

* Adapted from WCRF & AICR (1997)
\# NSAIDS : Non-steroid anti-inflammatory drugs
• RS : Resistant starch
▲ NJP : No judgement possible
MUFAT : Mono-unsaturated fat
† PUFA : Polyunsaturated fatty acids

From Table 6.1 it can be seen that the major lifestyle factors for which there is convincing evidence that they protect against colorectal cancer are physical activity and vegetable intake. The only promotive lifestyle factor that has this type of evidence is smoking tobacco. The **probable** promotive factors are the intake of red meat and alcohol. The **possible** protective factors are dietary fibre (NSP), starch and the carotenoids. Among the **possible** promotive factors are high BMI, greater adult height, frequent eating, intakes of sugar, total fat, saturated fat, processed and heavily cooked meat and eggs.

Table 6.2 Summary of changes in risk factors found in this study

		RURAL (S1 + S2)		S3		URBAN (S4 + S5)	
Type of factor	Risk factor*	Men	Women	Men	Women	Men	Women
Protective factors (Convincing)	Physical activity						
	Vegetable intake	L	L	L	L	L↑	L↑
Protective factors (Possible)	Dietary fibre (NSP)	L	L	L	L	L ↔	L ↔
	Starch	H	H	H	H	H ↓	H ↓
	Carotenoids						
Protective factors (Insufficient evidence)	Vitamin C	L	L	L	L	N↑	N↑
	Vitamin D			↔	↔	↔	↔
	Vitamin E	L-A	L-A	A	A	A↑	A↑
	Folate	A	A	A	A	↑	↑
Protective factors (no judgement possible)	PUFA	L-A	L-A	↔	↔	↓	↓
	Pulses						
	Fruit	L	L	L	L	L↑	L↑
Promotive factors (Convincing)	Smoking tobacco			↑	↑	↓	↓
Promotive factors (probable)	Red meat	L	L	L	L	L ↑	L ↑
	Alcohol	H	L	H ↔	L ↔	H ↔	L ↔
Promotive factors (possible)	High BMI	N	N	N	H ↑	N	H ↑
	Greater height	L	L	L ↔	L ↔	L	L ↔
	Sugar (sucrose)	L	L	L ↔	L ↔	L ↔	L ↔
	Total fat	L	L	L	L	H ↑	H ↑
	SATFAT	L-N	L-N	L	L	H ↑	H ↑
	Eggs						
	Heavily cooked meat						
Promotive factors (insufficient evidence)	Iron	L	L	L	L	A ↑	L ↑
	Total energy intake	A ↔	A ↔	A ↔	A ↔	A ↔	A ↔
Possible no relationship	Calcium	L-A	L-A	L-A	L-A	L-A	L-A
	Selenium						
	Fish						
	Poultry (NJP)	L	L	L	L	L	L
	Dairy products (NJP)						

NJP : No judgement possible
* : Adapted from WCRF & AICR, (1997)
↑ : Increase from S_{1+2}
↓ : Decrease from S_{1+2}
↔ : no change from S_{1+2}

L : Low
H : High
A : Adequate
N : Normal

Table 6.2 summarises the changes in those risk factors measured in this study. The findings of the risk factors measured in this thesis will now be discussed, against the background of the evidence that these factors are related to colorectal cancer risk (WCRF & AICR, 1997).

6.3.2 The protective factors

6.3..2.1 Physical activity

A large number of cohort and case-control studies produced evidence that physical activity, especially when lifelong, decreases the risk of colon cancer in both sexes. The evidence that physical activity influences rectal cancer is more limited and according to the panel of the WCRF and AICR (1997) report, no judgement on its role is possible. The panel pointed out that neither dietary intakes nor body size confound the association between physical activity and colon cancer risk. These factors, however, may interact in a complex fashion so that the highest risk (especially in men) is seen for those with the lowest physical activity, highest energy intake and highest body mass (Slatterly *et al.* 1997).

The high physical activity levels of rural Africans, walking long distances to collect wood and water, to shops, schools or to their jobs, have been documented (Walker, 1994). The proportion of subjects who had high physical activity levels did not differ from the rural to urban strata in this study, although there were indications that the type of activities differed. The increases observed in BMI of women from the rural to urban strata and of men in stratum 5, despite no reported increases in energy intakes, suggest that physical activity could have declined with urbanisation. It is therefore difficult to judge whether physical activity changed with urbanisation in these subjects. It was clear, however, that the men and women from the squatter areas were the least active. It is also difficult to judge on basis of the present data if desirable activity levels were achieved. Again, using mean BMI as an indicator, it seems as if the men balanced energy expenditure with intake, but not the women. With the small differences in reported physical activity as well as energy intakes and BMI across strata, it seems unlikely that changes in physical activity levels in this population contribute to differences in colorectal cancer risk.

There are a number of possible mechanisms by which physical activity may protect against colon cancer. One hypothesis is that it stimulates colon

peristalsis and therefore decrease the time that possible carcinogens reside in the colon (Simon, 1984; WCRF & AICR, 1997). Another is that it has favourable effects on the immune system (Simon, 1984). In addition, high physical activity and lower body mass, are associated with a metabolic profile of low insulin, good glucose tolerance, low serum lipids and possibly lower levels of other growth factors that is less favourable to cancer growth (McKeown-Eyssen, 1994).

The above emphasise that in evaluating physical activity as a risk factor in this population, bowel habits (constipation and defecation frequency) and their metabolic profiles should also be taken into account.

6.3.2.2 Vegetable consumption

A large variety of cohort and case-control studies have examined vegetable and fruit consumption in relation to colon cancer, rectal cancer, colorectal cancer and colorectal adenomatous polyps (summarised in the WCRF & AICR, 1997). Many of these studies measured relative risks and odds ratios in vegetarians and seven-day Adventists compared tot populations with lower vegetable intakes. The panel of the WCRF and AICR (1997) report is of the opinion that the evidence is convincing that diets rich in vegetables protect against cancers of the colon and rectum. The suggested mechanisms by which high intakes of vegetables may protect against colorectal cancer are based on beneficial effects of the fibre and non-digestible fructo-oligosaccharides on fermentation and bacterial growth (Cummings, 1997). Also vegetables contain many substances such as the vitamin antioxidants, phenols, flavonoids, isothiocynates and indoles which have anticarcinogenic properties (WCRF & AICR, 1997).

Vegetable consumption in these Africans were low. Mean daily intakes across strata varied from 67.9g (men in stratum 2) to 128.9g (women in stratum 5). The World Cancer Research Fund (WCRF & AICR, 1997) recommends that between 400 and 800g or five or more portions a day of a variety of vegetables and fruit should be eaten to protect against a number of cancers. If daily fruit intakes are added to vegetable intake, values varied from 108g (men in stratum 2) to 369g (women in stratum 5). Although an

increase in vegetable and fruit intake from rural to urban strata was observed the highest intake in stratum 5 is still lower than the minimum recommended amount of 400g/day.

The traditional African diet (Vorster *et al.* 1994; Walker, 1994) was characterised by a high vegetable and fruit intake supplemented with legumes, grains, seeds and nuts. The present study suggests that rural and urban Africans in the Northwest are no longer following this diet. There are several possible reasons for this, with poverty probably the underlying, common factor. Climatic conditions may also contribute. The Northwest is a comparatively dry region of South Africa and subject to heavy frosts and bitter colds in the high lying areas in winter. The result is that the season for growing vegetables and fruit is relative short in most areas and there's a lack of easily accessible water for irrigation in most rural areas. Therefore, many people are dependent on bought vegetables and fruit and cannot afford sufficient amounts. However, the Northwest is known as an excellent maize producing area. Maize is the staple of the diet of Africans in this area, and although intakes are lower in the urban than in the rural areas (MacIntyre, 1998) it still is the major contributor to energy and carbohydrate in the urban African diet.

Carotenoids, another possible protective factor were not measured in this study because the South African Food Tables (Kruger & Langenhoven, 1991) do not give values for these substances. However, because fruit and vegetables are the major dietary sources of carotenoids, it can be accepted that intakes would be low.

From the above it seems that the low vegetable intake of this population must contribute to an increased risk of colorectal cancer in both rural and urban subjects, but that the observed increased intake with urbanisation may lessen this risk.

6.3.3 The possible protective factors

6.3.3.1 Dietary fibre (NSP) consumption

The traditional African diet was high in fibre. Intakes of up to 40g/day of total dietary fibre have been reported in the past (reviewed by Vorster *et al.* 1997; Walker, 1994). The present study showed low fibre intakes in all groups. None of the groups reached the nutrient goal of >30g/day published for Europe, nor the limits for population average intakes (lower:27 and upper 40g/day) published by the World Health Organisation (James, 1993).

The reasons for the low fibre intakes are the low intakes of fruit and vegetables and the fact that refined "white" maize meal is preferred. The data indicate that the traditional high fibre intake is not present any more in Africans - not even in the rural areas. However, the data further indicate that with urbanisation and possibly better economic circumstances a more varied diet, containing more fruit and vegetables are eaten with small increases in fibre intake. Therefore, it seems as if the low fibre intakes of this population may increase their risk for colorectal cancer, and that the increase in fibre intake with urbanisation may decrease risk.

6.3.3.2 Starch intakes

The panel of the WCRF and AICR (1997) judged that diets high in starch possibly decrease the risk of colorectal cancer, that any protective effect is more likely if starchy diets are relatively unrefined and that diets high in resistant starch may reduce risk. However, the evidence for the latter is still insufficient. The mechanisms through which starch and its resistant fractions protect against colorectal cancer, is hypothesised to be because of its fermentation in the large bowel (Cummings & Bingham, 1987). It should be noted that diets high in starch, and especially unrefined starchy products, will mostly also be high in dietary fibre and low in fat. The effects of starch could therefore also be mediated through a replacement of fat in the diet.

The mean percentage of energy provided by carbohydrate in the diets of the men and women of the different strata in this study varied from 54-66%. Calculated starch varied from 193 to 286g/day. The major dietary source of

carbohydrates was maize porridge. Clearly, the subjects ate a high carbohydrate diet. It is further suspected that because of the custom to cook a large pot of maize porridge and eat it reheated or cold, a substantial portion of the starch will be in a resistant form, with a lower glycaemic index (Venter *et al.* 1990).

There were indications that total carbohydrate and especially starch intakes are declining with urbanisation. It therefore seems as if the protective effect of the high starch intake by these subjects may decrease in the future as people start eating a more varied diet and less maize porridge.

6.3.4 Factors that may be protective: insufficient evidence

6.3.4.1 Vitamin C

According to the WCRF & AICR (1997) vitamin C intake may reduce the risk of colorectal cancer but insufficient evidence still exists. Vitamin C intake in these Africans varied from 11.6 to 101.4 mg/day. Fruits and vegetables are the most important sources of vitamin C. Looking at this population's fruit and vegetable intake, one can see that the recommendations of at least five portions per day were not met. Mean vitamin C intakes of the farm workers and squatters were less than the recommended daily allowance of 60mg/day. The intake may be low due to the cost and availability of fruit and vegetables. Mean values found in this study were also lower than the 104 and 74mg/day reported for urban African men and women and the 61 and 71mg/day for rural men and women (Vorster *et al.* 1997).

6.3.4.2 Vitamin D

The evidence that vitamin D may reduce the risk of colorectal cancer was suggested by the panel of the WCRF & AICR (1997) although present evidence is still insufficient. The data presented in this study show no definite pattern. For this population the vitamin D sources are mainly exposure to sunlight. The RDA for vitamin D was achieved in both sexes of all the age groups.

6.3.4.3 Vitamin E

Data suggests that vitamin E might reduce the risk of colorectal cancer (WCRF & AICR, 1997) but more evidence is needed. Main sources of vitamin E are fats and oils as well as other sources such as fish, fruits and vegetables. Vitamin E intakes were adequate.

6.3.4.4 Folate

Higher intakes of folate may decrease the risk of colorectal cancer although the evidence is still insufficient (WCRF & AICR, 1997; Glynn *et al.* 1996). The population of this study had folate intakes between 144.3 to 262.5 μg/day. Green leafy vegetables, nuts, grains and liver are the main sources of folate (Halsted, 1993). Because vegetable intakes were low in this population, especially in the rural areas, folate intakes were not always what is recommended for adults (RDA = 200μg/day). Increased folate intake may lessen the risk of developing cancer of the colon and rectum or precancerous colonic polyps. Diminished folate status appears to promote carcinogenesis (Mason & Levesque, 1996).

6.3.5 The promotive factors

6.3.5.1 Smoking

Although colorectal cancer is not one of the major tobacco related cancers, a possible association with tobacco use and colorectal cancer has been suggested (D'Avanzo *et al.* 1995). Smoking is the single most important cause of cancer (Austoker, 1995). Smoking habits in stratum 4 and 5 were not as high as in 2 and 3. More men than women smoked. Smoking is most common in the age groups 25-34.9 years. The percentage of smokers in all age groups have declined with bigger decreases in the older age groups and those in stratum 4 and 5. In a cohort study by Nyren *et* al. (1996) no indication of an excess risk of colon cancer in males, who were long-term heavy smokers, were found. Smoking is undoubtedly associated with the

problems of poverty and unemployment such as the situation in stratum 3 of this population. Men in stratum 3 also had the highest alcohol consumption

6.3.6 The probable promotive factors

6.3.6.1 Red meat

Red meat as well as processed meat most likely increases the risk of developing colorectal cancer by the effect of fat on bile acid production, and the formation of carcinogens when meat is cooked or processed (WCRF & AICR, 1997). The mean meat intakes of this population progressively increased with urbanisation. Mean intakes varied from 16.2 to 100.7 g/day in both sexes of all the age groups. However, 100g/day is still regarded as a low intake (less than a medium portion/day). Clearly, the low intakes of meat must play a protective role. It is possible that the increase in intakes has not reached levels where it would confer a risk although the literature indicates that there are increases in risk with "higher" intakes of meat, and that much lower amounts of meat intake were reported in some of the study groups (WCRF & AICR, 1997).

6.3.6.2 Alcohol intake

Excessive consumption of alcoholic beverages might be associated with an increased risk for cancer (Tomatis, 1990; Goldbohm *et al.* 1994). Associations have been observed between the frequency of cancer of the rectum and beer consumption (Goldbohm *et al.* 1994; Meyer & White, 1993; Newcomb *et al.* 1993). No association have been shown with cancer of the colon, except in the study by Newcomb *et al.* (1993) where liquor and colon cancer were related (Newcomb *et al.* 1993). Epidemiological studies have suggested that high alcohol intake and low folate intake might be jointly associated with colorectal tumours via DNA metabolism (Boutron-Ruault *et al.* 1996).

In this study men in the age group 25-44.9 years from the farms and informal settlements consumed the most alcohol. Reported intakes of men from strata

4 and 5 were lower than those from strata 2 and 3, indicating beneficial effects of urbanisation probably related to education.

6.3.7 The possible promotive factors

6.3.7.1 BMI

The BMI of men were lower than that of the women in this study. Mean BMI increased in women with age and were generally higher in urban than in rural groups across all age ranges. In men this was only noticeable in stratum 5. According to the panel of the WCRF & AICR (1997) there is evidence that obesity increases the risk of colorectal cancer in men, while data for women have been more inconsistent.

6.3.7.2 Adult height

The expert panel of WCRF & AICR (1997) summarised the evidence that greater stature is associated with an increased risk of colon cancer. They quote studies from the USA which showed that women taller than 168cm and men ≥ 185.4cm were at increased risk. Although there were indications that heights increased in both men and women with urbanisation, maximum mean heights were substantially lower (approximately 10cm) than the values mentioned above.

6.3.7.3 Sugar (sucrose) intakes

There is evidence that high sucrose intake is a possible risk factor of developing colorectal cancer (WCRF & AICR, 1997). Fructose, mostly found in fruits, is not related to the risk of colorectal cancer because of the potentially protective constituents in fruit (WCRF & AICR, 1997). Sugar (sucrose) intake in the subjects of this study were low and showed no constant increase with urbanisation. This, therefore, pose no direct risk of developing colorectal cancer.

6.3.7.4 Total fat intake

There is a definite increase in total fat and saturated fat intake during urbanisation. The farm workers had the lowest intakes. Total fat intake has

been indicated to be a very important risk factor for the development of colorectal cancer (WCRF & AICR, 1997). Looking at the data presented in this study it seems as if the population's risk of developing colorectal cancer with urbanisation will increase because of an increased total fat intake.

6.3.7.5 Saturated/animal fat

The majority of studies describe a positive association between intake of animal fats, especially saturated fats, and colon cancer incidence (Miller *et al.* 1994; WCRF & AICR, 1997). There is a marked increase in total fat intake with urbanisation, except the subjects from stratum 2 (farm workers). Overall, the urban groups are at higher risk considering their fat intake, especially animal (saturated fat) intake.

6.3.7.6 Eggs

In a study by Steinmetz & Potter (1993) a positive correlation between colon cancer risk and egg consumption was found in females. This suggests a role for cholesterol in the aetiology of colon cancer. The data reported in this study shows that egg intakes relative low and very little changes were observed with urbanisation.

6.3.8 Factors that could be promotive: insufficient evidence

6.3.8.1 Iron

Micronutrients, especially folate, calcium and iron affect the risk of colorectal cancer (Tseng *et al.* 1996). The subjects in this study had relatively low iron intakes (see Table 5.17, p.30). One would, therefore, assume they are not in a high risk group when considering the WCRF & AICR (1997) assumption that high iron intakes increase risk of colorectal cancer, although the evidence is insufficient.

6.4 BALANCE IN RISK FACTORS

In Table 6 the contributors to colorectal cancer risk in this population is summarised and observed effects of urbanisation indicated.

Table 6. Colorectal risk factor situation in adult Africans of the Northwest Province.

RISK FACTOR	PRESENT RISK* (Rural Population)	CHANGES WITH URBANISATION **
Physical activity	NJP	↔
Vegetable	↑	↓
Smoking habits	↑	↓
Red meat	↓	↑ but still ↓
Alcohol	↑ (m)	NJP ↑
Dietary fibre	↑	↓
Starch	↓	↑ but still ↓
BMI	(w)↑ (m) ↓	↑
Adult height	↓	↑ but still ↓
Sugar	↓	↔ ∴ ↓
Total fat	↓	↑
Saturated fat	↓	↑
Eggs	↓	↔

* : Based on evaluation of level of factor in all strata
** : Comparison of urban with rural strata
↑ : Increased risk
↓ : Decreased risk
↔ : No change
NJP : No judgement possible
w : women
m : men

Of the 13 accepted, probable or possible risk factors for colorectal cancer considered, 8 in men and 7 in women were related to low risk, while 4 in men and 5 in women were related to high risk. On the available information it was not possible to judge the effects or contribution of physical activity to risk.

Of the factors associated with low risk, six: red meat, starch and fat (total and saturated) intake, BMI in women, as well as adult height changed with urbanisation in a direction to increase risk. However, of these six, three (red meat and starch intake and adult height, although changed were still under cut-point levels where they present increased risk.

Of the five factors associated with an increased risk of colorectal cancer (low vegetable intake, smoking habit, high alcohol consumption by men, low

dietary fibre intakes, and high BMI in women) three actually improved with urbanisation, possibly conferring a lower risk. These were increases in vegetable and fire intake and a decrease in smoking habits of urban men, especially those in stratum 5.

Because the contribution of a particular risk factor to total risk of colorectal cancer is not known, it is not possible to judge whether these changes will result in a lower overall risk. However, it is clear that with urbanisation, detrimental effects in some risk factors are not large enough to reach levels where they operate as risk factors, while in some instances, beneficial changes were observed in factors associated with high risk, possibly resulting in a lowering of risk.

CHAPTER 7

CONCLUSIONS AND RECOMMENDATIONS

The results of this study indicated that both protective and promotive risk factors for colorectal cancer were present in rural African men and women. The protective factors were high intakes of starch, low intakes of total and saturated fat, low consumption of meat, eggs and sugar, low BMI in men, and relatively low adult height. The promotive factors were the smoking habit (especially in men), high alcohol consumption by men and low intakes of vegetables and dietary fibre. No judgement on the role of physical activity was possible.

During urbanisation (comparing the levels of the risk factors in urban men and women to those in the rural areas) increases and decreases of both protective and promotive factors were observed. It is concluded that the urbanisation process at this stage (point in time) has resulted in positive and negative changes in colorectal cancer risk resulting, in balance, in no change in total risk. This may explain the low rates of colorectal cancer in urban Africans, despite increases in other chronic diseases of lifestyle.

7.2 NEW HYPOTHESIS

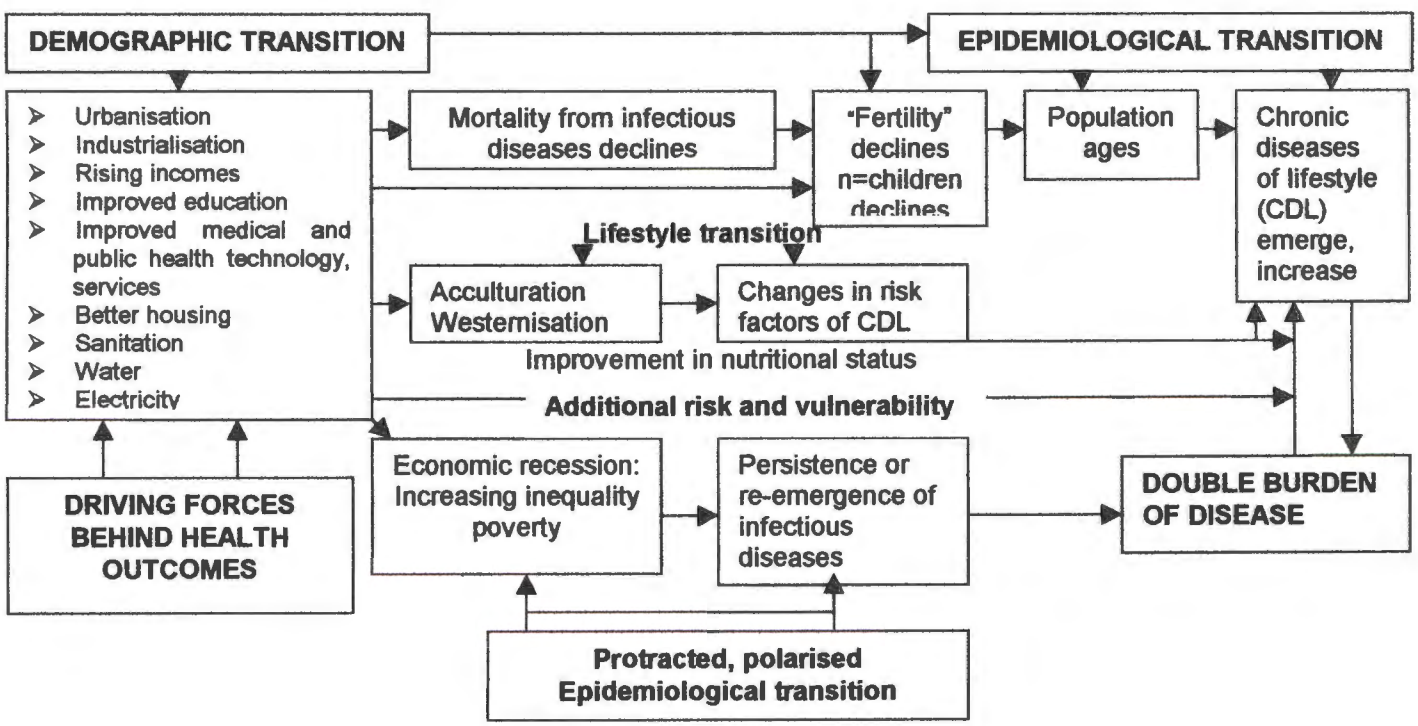
Figure 7.1 illustrates the driving forces behind the changes in health and nutritional status of people living in developing countries. The *demographic transition* with accompanied changes in many circumstances will result in an *epidemiological transition*, characterised by an older population and emergence of chronic diseases of lifestyle, also because of the *lifestyle and nutrition transition*. It is hypothesised that the emergence of the different chronic diseases of lifestyle will differ, depending on the balance of effects of the lifestyle and nutrition transition on the various risk factors of these diseases. It is further hypothesised that changes in lifestyle and nutrition during early urbanisation of the African population of the Northwest Province as seen at present, will result in positive *and* negative changes in risk factors

for colorectal cancer, with the result that on balance, total risk of this cancer is not affected, which may explain why this disease has not emerged in urban Africans.

From the above, it is clear that with longer exposure to western influences and greater dietary changes, it is possible that risk factors of colorectal cancer will change to such an extent that the disease will emerge in black South Africans in time.

It is of course possible that Africans may have a genetic protection against colorectal cancer. However, the experience in the USA which clearly indicates an increase in colorectal cancer, with comparable incidence rates in blacks and whites (Tomatis, 1990) argues against a genetic protection.

THE HEALTH TRANSITION IN DEVELOPING COUNTRIES



(Adapted from Bourne, 1996)

7.3 RECOMMENDATIONS REGARDING DIET AND CANCER PREVENTION

These recommendations were compiled by the expert panel of the World Cancer Research Fund and American Institute for Cancer Research (1997). The recommendations are based on an the requirements of and adult consuming 2000 Cal/day (8400 kJ/day).

Recommendation	Description
Plant based diets ➢ Vegetables/fruit ➢ Pulses and starch foods	Contains essential nutrients. Low in fat and calories. Filling, thus reduce less healthy food eaten. ⅔ of food on plate should be plant based Add beans to soups and reduce meat, or poultry Eat fruits in stead of creamy, sugary puddings For snacks: raisins, dried fruit or carrot, celery sticks
Weight control	Balanced calorie intake and level of physical activity Weight gain in adulthood < 5kg Eat low-calorie, nutrient rich foods Use low fat ingredients, cooking methods Eat wholegrain bread – fill up. Exercise
Exercise	Take 1 hours brisk walk/day and vigorously 1 hour/week Will stop you from becoming over weight or obese Incorporate physical activity into everyday life Climb stairs in stead of lifts
Vegetables/fruit	400-800g or 5+ portions a day Leaf green vegetables, carrots, tomatoes, citrus fruits partially protective Variety is the key Fresh fruit juice in morning Mid morning, afternoon and 2 portions in main meal
Starch	600-800g or 7+ portions per day Cereals, pulses, roots, tubers Minimally processed foods Limit refined sugar Cereal + wholegrain toast for breakfast Baked potato for lunch, pasta dish for supper Grapes, bananas, peaches and strawberries
Alcohol	Not recommended. If consumed less than 2 drinks per day for men, and 1 for women. Although evidence exists that small amounts of alcohol protect men, and probably women for CHD not the case for cancer.
Meat	Limit intake of red meat to less than 80g/day. Choose fish, poultry, wild animals in stead of red meat Major source of animal fat (saturated)
Iron	Meat good source (animal) Vitamin C helps absorbs iron from vegetables When eating more vegetables and fruit less meat.
Fat	Limit fatty foods from animal origin Choose vegetable oils Saturated fat found in animal products (meat and dairy) Unsaturated fat – vegetables or plant oil 30% of total calorie intake Use less cooking oil and fat, bake, grill or boil food Lower fat dairy products Remove skin and visible fat
Food preparation	Don't eat food that has been charred Avoid burning of meat juices Cooking in direct flames causes production of carcinogenic compounds Remove charred bits before serving
Tobacco	Don't smoke (or snuff!)

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ADDENDUM 1

Potchefstroomse Universiteit
vir Christelike Hoër Onderwys

The THUSA project:

information for participating clinic sisters

Dear Sister

Thank you for being willing to help us in organising this very important project. We are sure that the project will contribute to improved health of all the people of the North West Province.

The aim of the project is to get enough information regarding level of urbanisation, eating patterns, life-style and certain health indicators, to plan appropriate health and nutrition intervention strategies.

For the baseline survey, which will be done from February 1996 to March 1997, we need 2500 subjects who must be representative of the African population of the North West. These subjects will be drawn in matched groups from randomly selected traditional rural areas, from farms, from squatter camps, from urban towns and cities and also from professional people. Therefore, 500 subjects from 5 different levels of urbanisation. To control for seasonal influences, we will examine only a quarter of each group (± 128 subjects) during a specific visit. A visit will be from a Monday morning 08:00 to Friday afternoon 12:00. During each visit, we would like to get subjects from 4 different areas or points (around a clinic for example). Therefore, 32 subjects from four different places in your area. In total, the 128 subjects measured during our visit to your area, must include:

- 64 men, aged 15-65
- 64 women, aged 15-65

We would appreciate it if the ages are evenly distributed, as follows:

12 or 13 men or women in each 10 year span: 15-25; 25-35; 35-45; 45-55; 55-65.

What do we need from you?

1 To help us to set up the survey in your area. We will visit you for final arrangements at least two weeks before the field visit (survey) date. You must please help to identify the places where we can get subjects to participate.

2 We would need 2 or 3 rooms either in a clinic, hospital, school or other building to work in. If possible, also ± 5 tables and 10 chairs.

- A nurse or another leader in the community to help with the motivation and recruitment of healthy subjects. In other words, to explain the project to the subjects in their mother-tongue and to get signed informed consent to take a blood sample. To ask subjects to bring their ID with them.

- A nurse (one or more) to help with the taking of the blood samples. We will show the nurse exactly how the sample should be taken. We will centrifuge the sample and do all other measurements ourselves.

- Please note, we **will not test for HIV or AIDS.** Sterilised disposable needles, syringes and gloves will be used.

- Additional subjects for every diabetic and people suffering from diseases will have to be recruited.

- If possible, a nurse to assist with the blood pressure measurements.

- We have motivated for funds to pay an honorarium to all helpers. If these funds realise, we plan to pay R10 per hour per helper.

We will visit your area on the following dates:

..... for preliminary talks and arrangements of main visit

..... for actual survey

- While in your area, we will be staying at .....

- Please call (0148) 299 2481 or 299 2469 for further information.

Kind regards

PROF HH VORSTER

ADDENDUM 2

Potchefstroomse Universiteit
vir Christelike Hoër Onderwys

The THUSA project:

information for participating subjects

- **What is the THUSA project?**

THUSA stands for Transition, Health and Urbanisation of South Africans. In the project the effects of a change in eating patterns and life-style on health during the movement from rural to urban areas will be monitored. The main aim is to plan appropriate interventions that will ensure good health for all. During the baseline survey from February 1996 to March 1997, health indicators and eating patterns of Africans living in remote rural areas, on farms, in squatter camps and established towns and cities in the North West Province will be measured.

- **Why is the project so important?**

In all developing countries, the transition from rural to urban life-styles has been characterised by an increase in the diseases of overnutrition such as obesity, diabetes, high blood pressure, stroke, heart disease and certain forms of cancer. We know that it is possible to alleviate **hunger** and **undernutrition** and at the same time to **prevent** overnutrition, if people are helped to make the right food choices.

In this project we want to analyse the situation in the North West so that our Department of Health and Developmental Social Welfare will know which strategies to follow to ensure optimal nutrition and health for all. Therefore, by participating in this project, you will help all the people of the North West.

- **What will be measured in the project?**

  - Eating and drinking habits - food and nutrient intakes
  - Socio-economic background
  - Medical history
  - Activity levels
  - Smoking habits
  - Weight, height, waist, hip and arm measurements
  - Clinical signs of malnutrition (appearance of hair, skin, eyes, thyroid)
  - Blood sample: markers of nutritional status and disease. Please note, **NO** HIV or AIDS testing
  - Urine sample: excretion of minerals
  - Blood pressures
  - Diabetes

- **Who may participate?**

  Healthy Africans living in the North West Province who are between 15 and 65 years of age. People will be asked to participate and may refuse. Therefore, only **volunteers** will be asked to sign the informed consent form to participate.

- **What are the benefits for you?**

  Many health and nutritional status indicators of yourself will be measured. You will receive feedback during which a member of the investigation team will explain your health risk to you. You will receive dietary advice and will be referred to your clinic or doctor if necessary.

- **What do we expect from you?**

  - Please bring your ID; we need to know your birth date.
  - We will appreciate it if you will report fasting on the day of your participation. It means that for 10 - 12 hours before your blood sample is taken, you must not eat or drink anything but pure water.
  - You will be asked to sign a form giving consent to participate in the project.
  - We will ask you a number of questions regarding your health, age, income, family, smoking and drinking habits, etc.
  - Then you will receive a **number** for the project.
  - You will be weighed and measured.
  - We will take your blood pressure to determine stroke risk.
  - You will be asked to give us a urine sample
  - We will then take a blood sample with a thin, sterilised butterfly needle and a number of small sterile syringes.
  - Your temperature will be taken (orally).
  - Your finger will be pricked twice to measure blood sugar. You will also be asked to drink a sugar cool drink.
  - You will be questioned in detail about your eating habits.
  - Every third person will be asked to participate in a blood pressure project.
  - You will receive journals to read while you wait!
  - If you have any questions about the project, please do not hesitate to ask any one of the field workers.

Thank you for your participation!

ESTé VORSTER

ADDENDUM 3

THUSA PROJECT : PU FOR CHE
RECRUITMENT AND INFORMED CONSENT FORM

Title of the project: Nutritional and health status of Africans in transition

Name: ..... No.....

Address: .....

Tel no:.....

Age: .....

Are you pregnant? .....

Are you lactating? .....

Do you suffer from diabetes? hypertension? Other disease?.....

When did you have your last meal?

.....

or anything but water to drink? .....

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 volunteer

Witnesses

....

Signed at ..... on .....

For subjects under the age of 21, signed consent of a parent or legal guardian is necessary.

I, ... (full names) the parent/legal guardian of the person named above, hereby consent that he/she may participate in the THUSA project.

Signature ..... Date .....

Relationship .....

THUSA PROJECT : PU FOR CHE
RECRUITMENT AND INFORMED CONSENT FORM

Title of the project: Nutritional and health status of Africans in transition

Name: ..... No.....

Address: .....

Tel no:.....

Age: .....

Are you pregnant? .....

Are you lactating? .....

Do you suffer from diabetes? hypertension? Other disease?.....

When did you have your last meal?

.....

or anything but water to drink? .....

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 volunteer

Witnesses

....

Signed at ..... on .....

For subjects under the age of 21, signed consent of a parent or legal guardian is necessary.

I, ... (full names) the parent/legal guardian of the person named above, hereby consent that he/she may participate in the THUSA project.

Signature ..... Date .....

Relationship .....

ADDENDUM 4

Potchefstroomse Universiteit vir Christelike Hoër Onderwys

THE THUSA PROJECT

Subject name:_____ No:_____ S:_____

		CHECK / CONTROL
STATION 1	RECRUITMENT DEMOGRAPHIC QUESTIONNAIRE	
STATION 2	CLINICAL EXAMINATION Blood presusre	
	Oral temperature	
	Thyroid	
	Hair	
	Skin	
	Eyes	
	Blood sample YES NO	
STATION 3	ANTHROPOMETRY	
STATION 4	DIETARY QUESTIONNAIRE	
STATION 5	URINE SAMPLE	
STATION 6	GLUCOSE TOLERANCE	t_0 = t_{120} =
STATION 7	STRESS TESTS	
STATION 8	FOOD SECURITY QUESTIONNAIRE	
STATION 9	PHYSICAL ACTIVITY QUESTIONNAIRE	
STATION 10	ATTITUDE QUESTIONNAIRE (WOMEN ONLY)	
STATION 1	BACK TO CHECK / CONTROL	_____ SIGNATURE
LUNCH		
STATION 11	PSYCHOLOGICAL TESTS	

ADDENDUM 5

Potchefstroomse Universiteit
vir Christelike Hoër Onderwys

Subject number				
Date	D	M	Y	
Place				
Interviewer				

Home address _____

_____

_____

_____

Sex		
	Male	1
	Female	2

Age				
Date of birth	D	M	Y	

First Language		
	Tswana	1
	Afr	2
	Eng	3
	Xhosa	4
	Zulu	5
	Other	6

Second Language		
	Tswana	1
	Afr	2
	Eng	3
	Xhosa	4
	Zulu	5
	Other	6

What is your marital status?		
	Never married	1
	Married	2
	Divorced	3
	Widowed	4

Do you suffer from:			
	High blood	Yes	1
		No	2
	Diabetes	Yes	1
		No	2
	CHD	Yes	1
		No	2
	Stroke	Yes	1
		No	2

Does anyone in your family suffer from:			
	High blood	Yes	1
		No	2
	Diabetes	Yes	1
		No	2
	CHD	Yes	1
		No	2
	Stroke	Yes	1
		No	2

Do you take medicine regularly?		
	Yes	1
	No	2
If yes - what do you take?		

Do you snuff?		
	Yes	1
	No	2
Do you smoke?		
	Yes	1
	No	2
If no - have you smoked regularly before?		
	Yes	1
	No	2
If yes - what do you smoke?		
	Cigarettes	1
	Tobacco/pipe	2
	Other	3
If other - describe		
How much do you smoke? per day		
per week		
For how long have you been smoking (years)		
Calculate pack years		

What is your highest qualification?	None	1
	< St.6	2
	St. 6-8	3
	St. 6-8 + trade	4
	St. 9-10	5
	St. 9-10 + trade	6
	St. 9-10 + academic	7

What is your occupation?	

Do you have a job at the moment?	Yes	1
	No	2

If yes - what kind of job?	

On which days of the week do you work?	Irregular (piece work)	1
	Part time (1-4 days)	2
	Full time (5-6 days)	3

How much money do you earn? Is it between...	R0-100	
	R101-500	
	R501-1000	
	R1000-2000	
	R2000-3000	
	R3000+	

What is the source of this income?	

Do you receive any additional pensions?	Yes	1
	No	2

How much pension do you receive per month? _____

Interviewer - *Re-evaluate final income category*	R0-100	1
	R101-500	2
	R501-1000	3
	R1000-2000	4
	R2000-3000	5
	R3000+	6

Who else contributes money to your household? How much?

Yes	1
No	2

_____

_____

_____

_____

Who else contributes other resources eg. food, sharing work/chors to your household? - specify!

_____
_____
_____
_____

Yes	1
No	2

Does any member of your household have the right to use any property as his/her own?	Yes	1
	No	2

What type of property?	

How do you use it?	

Please name the members of your household

Member	Age	Education	Present job

What type of house do you live in?	Traditional	1
	Mokuku	2
	Brick house	3
	Other	4
Specify other		

Do you share a toilet with other households?	Yes	1
	No	2

What type of toilet do you have?	Communal	1
	None	2
	Bucket system	3
	Outside longdrop	4
	Outside chemical	5
	Outside water flush	6
	Inside water flush	7

Where do you get your drinking water from?	Fountain, river	1
	Communal tap	2
	Tap on premises	3
	Tap in house	4
	Other	5
If other specify		

Do you have access to electricity inside your house?	Yes	1
	No	2

What type of stove do you have?	None	1
	Coal/wood	2
	Gas or parrafin	3
	Electric	4

What type of fridge do you have?	None	1
	Parraffin	2
	Gas	3
	Electric	4

How long have you been living here? (years)	

Where did you live before coming here?	Rural area	1
	Farm	2
	Squatter camp	3
	Township	4

ADDENDUM 6

STUDIENOMMER _____ DEEL I

INSTRUKSIE: Skryf die proefpersoon se antwoord in die blokke langs die vraag OF omkring die nommer langs die proefpersoon se antwoord.

VOORBEELD:

1. Hoeveel maaltye het jy gister geeet? Gister=Maan1 Dins2 Woens3 Don4 Vry5 Sat6 Son7				
2.1.1 Omtrent hoe laat het jy jou eerste maaltyd geëet?				
2.1.2 Waar het jy die maaltyd geëet? Huis Werk Ander spesifiseer_____ Nie van toepassing				

Beantwoord asseblief die volgende vrae:

1. Hoeveel maaltye het jy gister geëet? Gister=Maan1 Dins2 Woens3 Don4 Vry5 Sat6 Son7				
2.1.1 Omtrent hoe laat het jy jou eerste maaltyd geëet?				
2.1.2 Waar het jy die maaltyd geëet? Huis Werk Ander spesifiseer_____ Nie van toepassing				1 2 3 4
2.2.1 Omtrent hoe laat het jy jou tweede maaltyd geëet?				
2.2.2 Waar het jy die maaltyd geëet? Huis Werk Ander spesifiseer_____ Nie van toepassing				1 2 3 4
2.3.1 Omtrent hoe laat het jy jou derde maaltyd geëet?				
2.3.2 Waar het jy die maaltyd geëet? Huis Werk Ander spesifiseer_____ Nie van toepassing				1 2 3 4
2.4.1 Omtrent hoe laat het jy jou vierde maaltyd geëet?				

2.3.2 Waar het jy die maaltyd geëet? Huis Werk Ander spesifiseer_____ Nie van toepassing					1 2 3 4
2.5 Eet jy hierdie hoeveelheid maaltye op meeste weeksdae?	JA 1		NEE 2		
INDIEN NEE: 2.5.1 Hoeveel maaltye eet jy gewoonlik per weeksdag?					
2.6 Eet jy jou maaltye op meeste weeksdae op omtrent dieselfde tye as bo?	JA 1		NEE 2		
INDIEN NEE:					
2.6.1 Omtrent hoe laat eet jy jou eerste maaltyd gewoonlik?					
2.6.2 Waar eet jy die maaltyd gewoonlik? Huis Werk Ander spesifiseer_____ Nie van toepassing					1 2 3 4
2.6.3 Omtrent hoe laat eet jy gewoonlik jou tweede maaltyd?					
2.6.4 Waar eet jy gewoonlik die maaltyd? Huis Werk Ander spesifiseer_____ Nie van toepassing					1 2 3 4
2.6.5 Omtrent hoe laat eet jy jou derde maaltyd?					
2.6.6 Waar eet jy die maaltyd gewoonlik? Huis Werk Ander spesifiseer_____ Nie van toepassing					1 2 3 4
2.6.7 Omtrent hoe laat eet jy gewoonlik jou ander maaltye?					
2.6.8 Waar eet jy gewoonlik die maaltye? Huis Werk Ander spesifiseer_____ Nie van toepassing					1 2 3 4

3. Het jy gister enigiets geëet of gedrink voor, tussen of na maaltye ?			J 1	N 2
3.1 **INDIEN JA:** Wat het jy geëet of gedrink en waar en wanneer het jy dit geëet of gedrink?				
Tipe voedsel:	Wanneer? (vroeg oggend , oggend, middag, aand)	Waar?		

INSTRUKSIE: Indien proefpersoon JA op vraag 3 geantwoord het, vra 3.2, indien NEE, VRA 3.3		
3.2 Eet of drink jy enigiets voor, tussen of na maaltye op meeste dae?	JA 1	NEE 2
3.3 Alhoewel jy niks tussen maaltye gister geeet het, eet of drink jy gewoonlik iets tussen maaltye?	JA 1	NEE 2
3.3.1 Indien JA, wat eet of drink jy gewoonlik tussen maaltye, en waar en wanneer eet jy dit?		

Tipe voedsel	Waar?	Wanneer?				

4. Hoe word kos gewoonlik in jou gesin bedien?

Moeder bedien almal op individuele borde	1
Elke bedien hom/haarself om individuele borde	2
Vader bedien almal op individuele borde	3
Volwassenes bedien vir hulself, en skep vir kinders in	4
Almal eet uit 'n gemeenskaplike skottel	5
Volwassenes en kinders eet uit aparte gemeenskaplike skottels	6
Volwassesnes eet uit borde, kinders eet uit 'n gemeenskaplike bord	7

Ander Beskryf bv woon alleen of in 'n koshuis	8

5. Hoe dikwels eet jy by restaurante, 'steak houses', Wimpy, kafees, 'fast-food' winkels, wegneem plekke, padkafees?	
Daagliks	1
Weekliks	2
Maandeliks	3
Selde	4
Nooit	5

5.1 **INDIEN DAAGLIKS, WEEKLIKS OF MAANDELIKS,** Waar eet jy en wat eet jy gewoonlik?					
Plek (Naam en beskrywing)	VOEDSEL GEËET				

STUDIENOMMER_____ DEEL II

INSTRUKSIE: Omkring die proefpersoon se antwoord. Vul die hoeveelheid en hoeveel keer geëet in gegewe kolomme in.

Ek gaan jou nou uitvra oor die tipe en die hoeveelheid voedsel wat jy eet. Sê asseblief of jy die voedsel eet, hoeveel jy eet en hoe gereeld jy dit eet. Ons gaan begin met mieliemeel pap.

Vir kantoor gebruik

VOEDSEL	BESKRYWING	HOEVEEL-HEID	HOEVEEL KEER GEEET Per dag	Per week	Per maand	Selde	Nooit	KODE	HVHD/DAG
Eet jy mieliemeel pap? JA 1 NEE 2 Indien JA, watter tipe het jy op die oomblik by die huis? Naam van produk_____1 Weet nie_____2 Maal self_____3 Indien die naam van die produk gegee is: Gebruik jy gewoonlik die produk? JA 1 NEE 2 WEET NIE 3 Waar kry jy jou mieliemeel vandaan? (Mag meer as een antwoord gee) Winkel 1 Werkgewer 2 Oes en maal self 3 Ander - spesifeer 4 Weet nie 5									
Mieliemeel pap	Styf (pap)							e4225 4250	
Mieliemeel pap	Slap							e4225 4250	
Gooi jy melk oor jou slappap? JA 1 NEE 2 INDIEN JA, watter tipe melk (volroom vars, suur, vetvry, 2%, melk mengsel)_____ INSTRUKSIE: Wys die proefpersone die voorbeelde									
INDIEN JA, hoeveel melk?									
Gooi jy suiker oor jou pap? JA 1 NEE 2									
Indien JA, hoeveel suiker?								9012	
'Ting'									
Mabella: tipe: Grof Fyn Rys	Styf							4082	
Gooi jy melk oor jou mabellapap? JA 1 NEE 2 INDIEN JA, watter tipe melk (volroom vars, suur, vetvry, 2%, melk mengsel)_____ INSTRUKSIE: Wys die proefpersone die voorbeelde									
INDIEN JA, hoeveel melk?									
Gooi jy suiker oor jou mabellapap? JA 1 NEE 2									
Indien JA, hoeveel suiker?								9012	
Hawermout								4032	
Gooi jy melk oor jou hawermoutpap? JA 1 NEE 2 INDIEN JA, watter tipe melk (volroom vars, suur, vetvry, 2%, melk mengsel)_____ INSTRUKSIE: Wys die proefpersone die voorbeelde									
INDIEN JA, hoeveel melk?									
Gooi jy suiker oor jou hawerpap? JA 1 NEE 2									

VOEDSEL	BESKRYWING	HOEVEEL-HEID	HOEVEEL KEER GEEET Per dag	Per week	Per maand	Selde Nooit	KODE	HVHD/DAG
Indien JA, hoeveel suiker?							9012	
Ontbyt graankos	Handelsnaam van graankosse tans in die huis: Weet nie__							
Gooi jy melk oor jou ontbytgraan? JA 1 · NEE 2 INDIEN JA, watter tipe melk (volroom vars, suur,vetvry, 2%, melk mengsel)_____ INSTRUKSIE: Wys die proefpersone die voorbeelde								
INDIEN JA, hoeveel melk?								
Gooi jy suiker oor jou ontbytgraan? JA 1 NEE 2								
Indien JA, hoeveel suiker?							9012	
Stampmielies	Gekoop Self gemaal Met vet Sonder vet						4077 4073	
Stampmielies met droë boontjies							A014	
Is die hoeveelheid stampmielies en bone dieselfde as op die foto? JA 1 NEE 2 Indien NEE, gebruik jy meer bone as op die foto of minder? MEER 1 MINDER 2								
Stampmielies met grondboontjies							A013	
Is die hoeveelheid stampmielies en grondboontjies dieselfde as op die foto? JA 1 NEE 2 Indien NEE, gebruik jy meer grondboontjies as op die foto of minder? MEER 1 MINDER 2								
Rys	Wit Bruin Mielierys						4040 4134 4043	
Pastas	Macaroni Spaghetti Ander:						4062	
Jy is baie behulpsaam. Kan ek jou nou uitvra oor vleis? HOENDER, ROOIVLEIS EN VIS Hoeveel keer in die week eet jy rooivleis, hoender , vis of soya produkte? _____ Eet jy hoender? JA 1 NEE 2 INDIEN JA, Waar kry jy jou hoender vandaan? (Mag meer as een antwoord gee) Winkel/supermark/spaza Werkgewer Slag eie Geskenke Ander spesifiseer Eet nie hoender nie								1 2 3 4 5 6
Hoender:	Hoe maak jy hoender gaar?							
	Gekook, niks bygevoeg						1521	
	Gebraai: in deeg/krummels							
	sonder deeg/krummels						1520	
	Gerooster							

VOEDSEL	BESKRYWING	HOEVEEL-HEID	HOEVEEL KEER GEEET Per dag	Per week	Per maand	Selde	Nooit	KODE	HVHD/DAG
	Bredie Watter groente sit jy gewoonlik by?							A002	
Eet jy die hoender se vel?			Altyd 1	Soms 2	Nooit 3				
Hoenderbeen-bredie								A003	
Hoenderpote	Met vel Sonder vel							A004 1609	
Hoender afval	Hoe maak jy dit gaar?							1610	
Waar kry jy jou rooivleis vandaan? (Mag meer as een antwoord)									
								Winkel/supermark/'spaza'	1
								Werkgewer	2
								Slag eie	3
								Geskenke	4
								Ander spesifiseer:	5
								Eet nie rooivleis	6
Rooivleis	Hoe hou jy van vleis? Met vet Sonder vet								
Beesvleis	Hoe maak jy beesvleis gaar?								
	Gebraai met been								
	Gebraai sonder been								
	Gestowe met been							A001	
	Gestowe sonder been							A001	
	Gerooster met been								
	Gerooster sonder been								
	Gemaal							1571	
Skaapvleis	Hoe maak jy skaapvleis gaar?								
	Gebraai met been								
	Gebraai sonder been								
	Gestowe met been							1511	
	Gestowe sonder been							1511	
	Gerooster met been								
	Gerooster sonder been								
	Gemaal							1571	
Varkvleis	Hoe maak jy varkvleis gaar?								
	Gebraai met been								
	Gebraai sonder been								
	Gestowe met been								
	Gestowe sonder been								

VOEDSEL	BESKRYWING	HOEVEEL-HEID	HOEVEEL KEER GEEET Per dag	Per week	Per maand	Selde	Nooit	KODE	HVHD/DAG
	Gerooster met been								
	Gerooster sonder been								
	Gemaal							1598	
Afval	Derms: gekook, niks bygevoeg								
	Gestowe met groente								
	Maag							1546	
	Hart							1565	
	Longe								
	Lewer							1515	
	Niertjies							1518	
Watter groente word gewoonlik by die bredies bygevoeg?									
Wors	Gebraai Gerooster							1526	
Spekvleis								1501	
Koue vleis	Polonie							1514	
	Ham							1564	
	Weenseworsies							1531	
	Ander - spesifiseer								
Vleispastei	Tuisgemaak Gekoop							1548	
Blikkies vleis	'Bully beef' Ander Wys voorbeelde							1535	
Hamburger	Tuisgemaak Gekoop							A015	
Droë bone/ertjies/ lensies	Hoe maak jy dit gaar?								
Sojaprodukte bv Toppers	Handelsname van sojaprodukte wat gebruik word: Weet nie___ Wys voorbeelde							3527	
Sardyne in tamatie/chilli sous	Heel							2503	
	Fyngemaak met gebraaide uie							A005	
Gebraaide vis	Met deeg/krummels Sonder deeg/krummels							2509 2523	
Ander ingemaakte vis	Tuna Gepekelde vis Ander:							2547	

VOEDSEL	BESKRYWING	HOEVEEL-HEID	HOEVEEL KEER GEEET Per dag	Per week	Per maand	Selde Nooit	KODE	HVHD/DAG
Viskoekies	Tuisgemaak Bevrore (gevries) Gekoop						2531	
Eiers	Gekook/ geposjeer Roereier Gebraai						1001 1003	

ONS KOM NOU BY GROENTE EN VRUGTE

Waar kry jy jou groente vandaan? (Mag meer as een antwoord)

Eie groente tuin	1
Werkgewer/ werkgewer se plaas	2
Eie plaas	3
Winkel/Supermark/ groentewinkel	4
Smous	5
Veld (bv morogo)	6
Geskenke	7
Ander spesifiseer	8

Hoeveel keer in die week eet jy groente? _____

VOEDSEL	BESKRYWING	Hoeveel-heid	HOEVEEL KEER GEEET Per dag	Per week	Per maand	Selde Nooit	KODE	HVD/DAG
Kool	Hoe maak jy kool gaar?							
	Gekook, niks bygevceg						8066	
	Gekook met aartappel, ui en vet						A006	
	Gebraai, niks bygevoeg						A007	
	Gekook dan gebraai met aartappel en uie						A006	
	ander: Weet nie:							
Spinasie/ morogo/ ander groen blaargroente	Hoe maak jy spinasie gaar?							
	Gekook, niks bygevoeg						8071	
	Gekook, vet bygevoeg							
	Gekook met uie, tamatie en vet						A011	
	Gekook met uie,tamatie en aartappel							
	Gekook met grondboontjies							
	Ander: Weet nie:							

VOEDSEL	BESKRYWING	Hoeveelheid	HOEVEEL KEER GEEET Per dag	Per week	Per maand	Selde Nooit	KODE	HVD/DAG
Tamatie en uie sous	Tuisgemaak met vet -sonder vet Ingemaak {Is dit die hoeveelheid pap wat jy eet? Hoeveel meer of minder? }						A012 A016 8221	
Pampoen	Hoe kook jy pampoen?							
	Gaargemaak in vet en suiker						A010	
	Gekook, bietjie suiker en vet						A009	
	Ander_____ Weet nie_____							
Wortels	Hoe kook jy wortels?							
	Gekook met suiker en vet						8129	
	- met aartappel en uie						A008	
	Rou, slaai						8015	
	Ander_____ Weet nie_____							
Mielies/ suikermielies	Hoe eet jy mielies? Aan stronk met vet - sonder vet						8033	
	Van die stronk af met vet sonder vet						8261	
Beetslaai	Tuisgemaak Gekoop						8005	
Aartappels	Hoe maak jy aartappels gaar?							
	Gekook/gebak met skil						8046	
	- sonder skil						8045	
	Fyngedruk							
	Gebraai						8189	
	Skyfies						8048	
	Slaai Ander spesifiseer: Weet nie						8226	
Patats	Hoe maak jy patats gaar?							
	Gekook/gebak met skil sonder skil						8057 8214	
	Fyngedruk met vet en suiker							
	Ander spesifiseer: Weet nie___							

VOEDSEL	BESKRYWING	Hoeveel-heid	HOEVEEL KEER GEEET Per dag	Per week	Per maand	Selde Nooit	KODE	HVD/DAG
Slaai groente	Rou tamatie,						8059	
	Blaarslaai						8031	
	Komkommer						8025	
Ander groente	Spesifiseer:							

VRUGTE

Hou jy van vrugte? JA 1 NEE 2

Hoeveel keer in 'n week of in 'n maand eet jy vrugte? _____

Waar kry jy jou vrugte vandaan?

Eie vrugte bome	1
Werkgewer/werkgewer se plaas	2
Eie plaas	3
Winkel/supermark/groentewinkel	4
Smous	5
Veld	6
Geskenke	7
Ander spesifiseer	8

VOEDSEL	BESKRYWING	Hoeveel-heid	HOEVEEL KEER GEËET Per dag	Per week	Per maand	Selde Nooit	KODE	HVD/DAG
Appels/Pere	Vars Geblikte pere						7001 7054	
Piesangs							7009	
Lemoene / naartjies							7031	
Druiwe							7020	
Perskes	Vars Geblikte perskes						7036 7038	
Appelkose	Vars Geblikte perskes						7003 7004	
Mango's	Vars						7026	
Koejawels	Vars Geblikte koejawels						7021 7023	
Indien proefpersoon geblikte vrugte eet: Eet jy vla saam met die geblikte vrugte? ALTYD 1 SOMS 2 NOOIT 3								
Vla	Tuisgemaak Langlewe (Ultramel)						0004	
Wildevrugte/ bessies	Stamvrugte Noem-noem Klappers Maroelas Nastergals Ander -spesifiseer						7070	

VOEDSEL	BESKRYWING	Hoeveelheid	HOEVEEL KEER GEËET Per dag	Per week	Per Selde maand	Nooit	KODE	HVD/DAG
Gedroogte vrugte	Tipes:							
Ander vrugte:								
BROOD EN BROODSMEERE								
Brood Broodrolle	Wit						4001	
	Bruin						4002	
	Volgraan						4003	
Smeer jy enigiets op die brood? Altyd 1 Soms 2 Nooit 3 Indien ALTYD of SOMS, wat smeer jy op?								
Margarien	Wat is die handelsnaam van die margarien wat jy tans gebruik? _____ Weet nie __ Wys voorbeelde							
Botter	Wat is die handelsnaam van die botter wat jy tans gebruik? _____ Tuisgemaak Weet nie ___						6502	
Grondboontjie botter							6509	
Konfyt/stroop/ heuning							9008	
Marmite/Fray Bentos ens							9501	
Vis/vleis smeer							1512	
Kaas	Tipe:							
Atjar							A017	
Polonie							1514	
Ander smere:	Spesifiseer:							
Kluitjie							4001	
Vetkoek							4057	
Provita, kraak-beskuitjies								
VETTE: Watter vette gebruik jy en waarmee saam gebruik jy dit?								

VOEDSEL	BESKRYWING	Hoeveelheid	HOEVEEL KEER GEËET Per dag	Per week	Per maand	Selde Nooit	KODE	HVD/DAG
Margarien	Waar gebruik op brood met groente Hoeveelheid lepels ___per nommer gesinslede_____ ander-spesifiseer							
Botter	Waar gebruik: op brood met groente ander-spesifiseer Hoeveelheid lepels ___per nommer gesinslede_____							
Holsum /plantvette	Waar gebruik: Hoeveelheid lepels ___per nommer gesinslede____						6508	
Olie	Waar gebruik: Hoeveelheid lepels ___per nommer gesinslede____						6510	
Uitgebraaide vet	Waar gebruik: Hoeveelheid lepels ___per nommer gesinslede____							
Gemengde vet (makhuru)	Waar gebruik: Hoeveelheid lepels ___per nommer gesinslede____							
Niervet	Waar gebruik: Hoeveelheid lepels ___per nommer gesinslede____						6520	
Mayonnaise/ slaaisous	Hoeveelheid lepels ___per nommer gesinslede____						6573	
Room	Vars/Langlewe/ geblik 'Orley whip'	-					6503	
DRANKE:								
Tee							9514	
Suiker/koppie tee							9012	
Melk/koppie tee	Watter tipe melk gebruik jy in jou tee?							
	Vars/langlewe volroom						0006	
	Vars/langlewe 2%							
	Vars/langlewe vetvry						0072	
	Volroommelkpoeier naam_____						0009	

VOEDSEL	BESKRYWING	Hoeveelheid	HOEVEEL KEER GEËET Per dag	Per week	Per maand	Selde Nooit	KODE	HVD/DAG
	Afgeroomde melkpoeier naam_____						0008	
	Melkmengsel naam_____						0068	
	Verromer naam_____						0039	
	Gekondenseerde melk						0002	
	Ingedampte melk						0003	
	Geen							
Koffie							1513	
Suiker/koppie koffie							9012	
Melk/koppie koffie	Watter tipe melk gebruik jy in jou koffie? Vars/langlewe volroom						0006	
	Vars/langlewe 2%							
	Vars/langlewe vetvry						0072	
	Volroommelkpoeier naam_____						0009	
	Afgeroomde melkpoeier naam_____						0008	
	Melkmengsel naam_____						0068	
	Verromer naam_____						0039	
	Gekondenseerde melk						0002	
	Ingedamptemelk						0003	
	Geen							
Melk as sulks	Vars/langlewe volroom						0006	
	Vars/langlewe 2%							
	Vars/langlewe vetvry						0072	
	Suurmelk(maas)						0006	
	Volroommelkpoeier naam_____						0009	
	Afgeroomde melkpoeier naam_____						0008	
	Melkmengsel naam_____						0068	
	Gekondenseerde melk						0002	
	Ingedampte melk						0003	
Melkdranke	Nestle Milo Ander:							
Yoghurt	Drink yoghurt Dik yoghurt						0044 0020	

VOEDSEL	BESKRYWING	Hoeveelheid	HOEVEEL KEER GEËET Per dag	Per week	Per Selde maand	Nooit	KODE	HVD/DAG
Kwas	Sweeto						9013	
	SixO						9013	
	Oros/Lecol-met suiker						9002	
	- nagemaakte versoeter						9013	
	Kool Aid						9002	
	Ander:							
	Wys voorbeelde							
Vrugtesap Spesifiseer tipes:	Vars/ Liquifruit/Ceres							
	Tropica							
	Konsentrate bv Halls							
	Nektars							
	Handelsname:_____ Wys voorbeelde							
Gaskoeldranke bv Coke, Fanta	Versoet Dieet						9001 9013	
Mageu /Motogo								
Tuisgemaak							9516	
Tlokwe							9516	
Bier							9506	
Spirituliee							9510	
Rooiwyn							9508	
Witwyn							9518	
Likeur							9517	
Ander: spesifiseer								
PEUSELHAPPIES EN LEKKERGOED:								
Aartappelskyfies							8049	
Grondboontjies	Rou Gebraaide Gesout Sonder sout						6001 6007	
Kaaskrulle Niknaks ens							4076	
Rosyntjies							7022	
Grondboontjies en rosyntjies								
Sjokolade	Naam							
Suikerlekkers	Sugas, gommetjies						9009	
Lekkers	Toffies, Karamels						9014	

VOEDSEL	BESKRYWING	Hoeveelheid	HOEVEEL KEER GEËET Per dag	Per week	Per maand	Selde Nooit	KODE	HVD/DAG
Beskuitjies	Tipe							
Koeke en terte	Tipe							
Skons							4029	
Beskuit							4160	
Southappies	Vleisrolletjies Samoosas Beskuitjies bv Bacon kips Ander spesifiseer:						1534 4196 4162	
POEDINGS:								
Ingemaakte vrugte	Tipe:							
Jellie							9004	
Vla	Tuisgemaak Langlewe (Ultramel)						0004	
Gebakte poeding							4181	
Kitspoeding							4066	
Roomys Sorbet							6507 6516	
Ander spesifiseer:								
SOUSE / GEURMIDDELS:								
Atjar							A017	
Tamatiesous Worcestersous							9505	
Blatjang							9524	
Piekels							8176	
Pakkies sop							4069	
Ander:								
INSEKTE:								
Sprinkane								
Mopani wurms								
Ander:								
WILDE VOELS OF DIERE (Gejag in landelike gebiede of op plase)								

VOEDSEL	BESKRYWING	Hoeveelheid	HOEVEEL KEER GEËET Per dag	Per week	Per maand	Selde Nooit	KODE	HVD/DAG
ALLERLEI: Noem asseblief enige ander voedsel wat meer as een/twee keer per week geeet word waaroor ons nie gepraat het nie.								

SOUT GEBRUIK :
Die volgende paar vrae is om uit te vind of jy sout gebruik, waar jy dit gebruik en hoeveel gebruik.

Voeg jy sout by kos terwyl dit gaargemaak word?

Altyd 1	Soms 2	Nooit 3	Weet nie 4

Voeg jy sout by kos nadat dit gaargemaak is?

Altyd 1	Soms 2	Nooit 3

Hou jy van souterige kosse soos gesoute grondboontjies, aartappelskyfies ens?

Baie lief daarvoor 1	Hou daarvan 2	Hou nie baie daarvan,eet dit soms 3	Eet dit nooit 4

BÊRE VAN VOEDSEL:

Bêre jy voedsel van een maaltyd om tydens 'n volgende maaltyd te eet?

Altyd 1	Soms 2	Nooit 3

Indien ALTYD of SOMS, watter voedsel bêre jy?
Eet jy hierdie oorskiet kosse koud of maak jy dit warm?

VOEDSEL	Her-verhit	Koud

Gebruik jy enige van die volgende?

	Naam van produk	Hoeveelheid /dag
Vitamiene/vitamiene & minerale		
Tonikums		
Gesondheidskosse		
Liggaamsbou-preparate		
Dieetvesel supplement		
Ander: spesifiseer		

DANKIE VIR U SAMEWERKING EN GEDULD!

TOTSIENS!

ADDENDUM 7

Potchefstroomse Universiteit
vir Christelike Hoër Onderwys

QUESTIONNAIRE 3 **THUSA PROJECT**

NO .....

ALL THE INFORMATION OF THIS QUESTIONNAIRE IS

CONFIDENTIAL

1. Are you using any form of contraceptive No - 1/Yes - 2

2. If yes, state type
   1. IUD
   2. Condoms
   3. Other
   4. Pill
   5. Injection

   If 4 or 5 state product name .....

3. At what age did you start using contraceptives

   Duration of using

4. Has your method of contraceptive changed during this time N-1/Y-2

5. If yes, how and when .....
   .....
   .....
   .....
   .....

6. At what age did you experience your first menstruation
   1. ≤ 12
   2. 13-14
   3. ≥ 17

7. What is the date of the first day of your last menstruation
   day ..... mo .....

8. How many days between menstruation .....

   Phase at present

9. How old were you when your first child were born
   1. ≤ 24
   2. 25-29
   3. ≥ 30

10. Have you experience your menopause No - 1/ Yes - 2

11. If yes, at what age
    1. Pre
    2. < 50
    3. > 50

12. Have you had a historectomy No-1/ Yes - 2

13. If yes, at what age
    1. ≤ 24
    2. 25-29
    3. ≥ 30

14. How many children do you have

15. What are the ages of your youngest and oldest
    Youngest
    Oldest

16. How many of your children were breast fed

17. How long did you breast fed each child

1.	7.
2.	8.
3.	9.
4.	10.
5.	11.
6.	12.

18. Have you had any previous problems with your breasts N-1/Y-2

19. If yes, state what .....
    .....
    .....
    .....

20. Do you have a family history of breast problems No - 1/ Yes -2

ADDENDUM 8

Potchefstroomse Universiteit
vir Christelike Hoër Onderwys

The THUSA project:

feedback to participants

Dear Participant

No. .....

Name:.....

Your tests have shown that

- Everything measured were normal, no signs of disease .....
- You must see your clinic or doctor to check for:

  - goitre .....
  - high blood pressure .....
  - blood sugar (diabetes) .....
  - anaemia .....
  - heart disease .....

- You must try to loose weight.....
- You must include more of the following foods in your diet.....

  .....

  .....

  .....

Thank you for participating in this project.

ADDENDUM 9

Table 9.1: Mean and standard deviations of intakes of meat and chicken of men and women of the different strata

Stratum 1	Men		Women	
	Mean (g/day)	SD	Mean (g/day)	SD
Beef (g):				
Chuck, braised (with fat)	17.63	21.21	14.46	68.21
Boerewors, cooked (beef/pork)	9.90	12.68	6.22	10.49
Chuck braised (fat trimmed)	3.82	10.98	?	?
Polony	3.18	6.88	2.73	6.87
Corned canned (med.fat, 5% cereal	2.78	8.01		
Minced savoury reg (tomato & onion	1.88	5.84	1.46	6.61
Minced savoury lean (tomato & onion	-	-	0.03	0.40
Liver, beef fried	1.54	2.72	1.63	3.91
Rump steak, fried (with fat)	0.82	7.25	0.27	1.99
Beef patty frozen grilled	0.55	3.33	0.18	0.78
Rump steak, fried (fat trimmed)	0.08	0.68	-	-
Meat paste (beef,chick etc.)	0.01	0.11	-	-
Sausage, vienna canned (beef/pork)	-	-	0.85	2.61
Mutton:				
Loin chop grilled (with fat)	1.85	4.85	1.06	2.42
Stew vegetable (fat trimed)	1.42	5.75	2.37	15.70
Shoulder braised (with fat)	1.40	10.29	0.11	0.90
Leg roasted (with fat trimmed)	0.15	0.95	-	-
Chop grilled (fat trimmed)	-	-	0.10	0.65
Minced pan-fried	-	-	0.03	0.32
Samoosa (mutton filling)	-	-	0.01	0.16
Pork:				
Loin chop grilled (with fat)	0.71	2.54	0.51	2.90
Thick rib/breast braised + fat	0.14	1.25	-	-
Salami (Pork/beef) Russians	0.13	1.13	-	-
Chicken:				
Roasted (light & dark + skin)	15.78	22.28	8.93	11.49
Boiled (Light & dark + skin)	4.95	15.94	4.57	9.32
Feet, raw	4.09	8.78	3.77	7.75
Giblets cooked	2.27	5.12	3.20	6.77
Fried batter dipped (Kentucky)	0.31	2.07	0.31	1.99
Giblets curried (Sun oil, onion)	-	-	0.62	7.68
Chicken boiled (light/dark, no skin)	-	-	0.07	0.44
Chicken liver (cooked)	-	-	0.13	1.62
Ofal				
Tripe, brawn, brain, tongue	6.13	10.29	-	-

Table 9.1 (continued)

Stratum 2	Men		Women	
	Mean (g/day)	Amount/ person (g/day)	Mean (g/day)	Amount/ person (g/day)
Beef (g):				
Chuck, braised (with fat)	8.41	14.95	8.98	14.37
Boerewors, cooked (beef/pork)	4.87	8.79	4.27	6.92
Chuck braised (fat trimmed)	2.54	7.25	2.07	6.71
Polony	0.52	1.50	0.88	1.89
Corned canned (med.fat, 5% cereal	2.36	5.36	2.32	4.99
Minced savoury reg (tomato & onion	1.29	5.01	1.03	3.58
Minced savoury lean (tomato & onion	-	-	-	-
Liver, beef fried	-	-	1.49	3.22
Rump steak, fried (with fat)	0.54	3.01	-	-
Beef patty frozen grilled	-	-	-	-
Rump steak, fried (fat trimmed)	-	-	-	-
Meat paste (beef,chick etc.)	-	-	-	-
Sausage, vienna canned (beef/pork)	0.26	1.13	0.95	2.73
Heart cooked (no fat)	0.17	0.73	0.19	0.68
Kidney (cooked)	0.14	1.20	-	-
Mutton:				
Loin chop grilled (with fat)	1.68	3.49	3.00	4.98
Stew vegetable (fat trimed)	0.22	1.04	1.03	5.57
Shoulder braised (with fat)	-	-	0.22	1.69
Shoulder braised (no fat)	0.03	0.24	-	-
Leg roasted (with fat trimmed)	-	-	-	-
Loin chop grilled (fat trimmed)	0.20	1.18	0.08	0.65
Minced pan-fried	0.04	0.36	-	-
Pork:				
Loin chop grilled (with fat)	0.77	2.45	1.19	3.78
Thick rib/breast braised + fat	0.01	0.12	-	-
Salami (Pork/beef) Russians	-	-	0.08	0.65
Bacon, fried (with fat)	-	-	0.07	0.52
Chicken:				
Roasted (light & dark + skin)	5.19	8.63	9.78	14.68
Boiled (Light & dark + skin)	7.65	15.99	4.29	11.92
Feet, raw	3.07	7.01	2.36	3.92
Giblets cooked	-	-	2.44	6.94
Fried batter dipped (Kentucky)	0.29	1.77	-	-
Chicken boiled (light/dark, no skin)	-	-		
Ofal				
Cooked (Tripe, brawn, brain, tongue)	2.51	4.30	2.76	4.13
Tripe (cooked in milk)	-	-	0.20	1.56

Table 9.1 (continued)

Stratum 3	Men		Women	
	Mean (g/day)	Amount/ person (g/day)	Mean (g/day)	Amount/ person (g/day)
Beef (g):				
Chuck, braised (with fat)	17.15	28.85	10.50	13.69
Boerewors, cooked (beef/pork)	10.37	12.43	9.83	10.23
Chuck braised (fat trimmed)	2.32	5.72	1.81	4.65
Polony	7.17	14.42	3.79	7.27
Corned canned (med.fat, 5% cereal	2.05	4.88	0.95	2.23
Minced savoury reg (tomato & onion	2.37	6.48	2.25	5.55
Minced savoury lean (tomato & onion	1.32	7.97	-	-
Liver, beef fried	2.71	6.32	4.09	8.32
Rump steak, fried (with fat)	1.34	3.95	1.58	5.14
Beef patty frozen grilled	0.85	4.56	1.71	5.46
Rump steak, fried (fat trimmed)	-	-	0.63	5.59
Meat paste (beef,chick etc.)	0.05	0.31	0.04	0.25
Sausage, vienna canned (beef/pork)	1.29	3.33	1.91	4.43
Minced pan fried (reg)	0.20	0.87	0.38	1.88
Heart cooked (no fat)	-	-	0.04	0.25
Tongue ox simmered	-	-	0.03	0.22
Mutton:				
Loin chop grilled (with fat)	5.02	12.28	6.44	10.21
Stew vegetable (fat trimed)	0.83	3.73	0.06	0.40
Shoulder braised (with fat)	0.17	1.09	0.54	4.81
Leg roasted (with fat trimmed)	0.32	2.03	-	-
Chop grilled (fat trimmed)	0.66	3.50	0.11	1.01
Minced pan-fried	-	-	0.10	0.70
Samoosa (mutton filling)	-	-	0.08	0.67
Stew vegetable (fat trimmed)	-	-	0.06	0.40
Pork:				
Loin chop grilled (with fat)	1.93	6.73	1.24	5.66
Thick rib/breast braised + fat	-	-	0.54	4.81
Salami (Pork/beef) Russians	-	-	0.19	1.04
Bacon fried with fat	0.17	1.09	0.05	0.22
loin, braised (lean)	-	-	0.16	1.45
Chicken:				
Roasted (light & dark + skin)	12.02	13.66	13.94	18.07
Boiled (light & dark + skin)	3.00	8.08	4.84	12.97
Feet, raw	2.29	5.28	4.45	10.40
Giblets cooked	5.51	13.70	2.08	7.58
Fried batter dipped (Kentucky)	0.59	3.02	2.29	7.44
Chicken boiled (light/dark, no skin)	0.10	0.62	0.30	1.17
Boiled dark meat with skin	0.98	6.25	-	-
Chicken roasted dark meat with skin	-	-	0.16	1.45
Ofal				
Tripe, brawn, brain, tongue	6.51	13.01	8.15	12.00
Tripe cooked in milk	-	-	0.10	0.89

Table 9.1 (continued)

Stratum 4	Men		Women	
	Mean (g/day)	Amount/ person (g/day)	Mean (g/day)	Amount/ person (g/day)
Beef (g):				
Chuck, braised (with fat)	18.49	31.85	13.06	19.39
Boerewors, cooked (beef/pork)	13.27	14.99	10.11	13.17
Chuck braised (fat trimmed)	4.72	12.39	4.75	10.02
Polony	6.59	9.57	5.32	11.84
Corned canned (med.fat, 5% cereal	2.59	6.60	2.68	7.35
Minced savoury reg (tomato & onion	2.96	10.80	4.60	11.77
Minced savoury lean (tomato & onion	0.80	4.77	0.06	0.76
Liver, beef fried	2.27	4.28	2.58	4.64
Rump steak, fried (with fat)	2.34	11.10	0.42	2.11
Beef patty frozen grilled	3.23	9.73	1.40	6.62
Rump steak, fried (fat trimmed)	0.06	0.47	0.94	8.23
Meat paste (beef,chick etc.)	0.11	0.60	-	-
Sausage, vienna canned (beef/pork)	2.24	9.10	2.23	6.32
Heart cooked (no fat)	0.35	1.28	0.06	0.39
Minced, pan fried (reg)	0.20	1.44	0.41	2.51
Kidney cooked	0.01	0.12	0.02	0.19
Tripe cooked in milk	-	-	0.15	1.16
Bolo/topside, braised lean	-	-	0.45	5.37
Mutton:				
Loin chop grilled (with fat)	5.54	12.53	3.60	10.50
Stew vegetable (fat trimed)	0.13	0.75	1.26	4.70
Shoulder braised (with fat)	0.59	3.24	0.40	2.08
Leg roasted (with fat trimmed)	-	-	-	-
Chop grilled (fat trimmed)	0.46	2.45	0.64	3.69
Minced pan-fried	-	-	0.39	2.47
Samoosa (mutton filling)	-	-	0.06	0.76
Pork:				
Loin chop grilled (with fat)	0.38	1.35	0.69	3.02
Thick rib/breast braised + fat	-	-	0.03	0.34
Salami (Pork/beef) Russians	0.27	2.03	0.09	0.82
Ham cooked/ canned (reg, 11% fat)	-	-	0.04	0.35
Bacon fried (fat)	-	-	0.13	1.43
Chicken:				
Roasted (light & dark + skin)	18.13	19.07	14.55	19.72
Boiled (Light & dark + skin)	6.55	17.05	5.24	13.44
Feet, raw	2.66	11.21	3.70	7.16
Giblets cooked	4.90	14.22	5.27	12.42
Fried batter dipped (Kentucky)	0.65	3.52	0.82	3.31
Chicken boiled (light/dark, no skin)	0.13	0.74	0.46	3.04
Roasted dark meat + skin)	-	-	0.08	1.01
Boiled (light & dark no skin)	-	-	0.04	0.50
Pie, comm. (flaky past. hm)	-	-	0.04	0.42
Ofal Tripe, brawn, brain, tongue cooked	8.38	10.90	8.28	11.53

Table 9.1 (continued)

Stratum 5	Men		Women	
	Mean (g/day)	Amount/ person (g/day)	Mean (g/day)	Amount/ person (g/day)
Beef (g):				
Chuck, braised (with fat)	20.15	25.11	19.93	23.77
Boerewors, cooked (beef/pork)	15.42	19.70	11.77	27.06
Chuck braised (fat trimmed)	5.86	15.70	7.30	14.47
Polony	7.97	12.13	5.91	9.29
Corned canned (med.fat, 5% cereal	2.06	5.56	3.14	9.72
Minced savoury reg (tomato & onion	5.71	12.00	9.52	17.48
Minced savoury lean (tomato & onion	-	-	0.22	1.28
Liver, beef fried	2.75	4.82	3.94	5.65
Rump steak, fried (with fat)	2.73	10.79	1.33	5.76
Beef patty frozen grilled	5.28	9.64	4.74	9.91
Rump steak, fried (fat trimmed)	0.22	1.91	0.33	2.82
Meat paste (beef,chick etc.)	0.20	1.47	0.11	0.41
Sausage, vienna canned (beef/pork)	7.04	16.70	6.24	12.73
Minced pan fried (reg)	-	-	0.02	0.18
Heart cooked (no fat)	0.04	0.34	-	-
Kidney cooked	0.09	0.56	-	-
Pie, steak & kidney (comm)	-	-	0.58	6.35
Tripe cooked in milk	-	-	0.09	1.01
Tongue, ox simmered	-	-	0.05	0.55
Meat pie (flaky pastry, hm)	-	-	0.05	0.55
Rib braised (with fat)	-	-	0.03	0.28
Mutton:				
Loin chop grilled (with fat)	6.37	12.32	6.81	11.35
Stew vegetable (fat trimed)	2.27	7.77	3.16	9.13
Shoulder braised (with fat)	2.16	7.60	0.74	3.43
Chop grilled (fat trimmed)	0.14	1.13	0.71	5.75
Minced pan-fried	1.52	5.10	0.58	3.47
Samoosa (mutton filling)	-	-	0.05	0.55
Pork:				
Loin chop grilled (with fat)	4.35	10.78	0.98	2.47
Thick rib/breast braised + fat	0.04	0.34	-	-
Salami (Pork/beef) Russians	0.22	1.91	-	-
Bacon fried with fat	0.61	2.19	0.27	0.95
Ham, cooked/canned (reg. 11% fat)	0.11	0.45	0.14	1.23
Chicken:				
Roasted (light & dark + skin)	18.04	17.79	16.99	16.78
Boiled (light & dark + skin)	3.44	9.65	4.50	10.72
Feet, raw	3.81	12.36	1.58	5.63
Giblets cooked	2.08	5.06	3.23	6.66
Fried batter dipped (Kentucky)	1.75	5.89	1.75	6.35
Chicken boiled (light/dark, no skin)	0.03	0.23	0.03	0.22
Liver coooked	-	-	0.05	0.55
Ofal				
Tripe, brawn, brain, tongue	7.46	12.27	5.94	7.86

ADDENDUM 10

Table 10.1 Mean and standard deviation of vegetable intake of men and women of the different strata.

Stratum 1	Men		Women	
	Amount/ person (g/day)	SD	Amount/ person (g/day)	SD
VEGETABLES				
Potato, boiled without skin	17.49	47.47	9.80	9.75
Potato, mashed (WH, HM)	14.35	22.76	9.75	17.81
Onion, cooked	11.56	8.36	9.82	8.76
Tomato, boiled/canned	11.08	9.70	10.89	12.39
Beans, sugar dried cooked	9.56	12.68	7.34	11.00
Cabbage, cooked	9.46	11.66	9.26	12.76
Pumpkin/sqaush, winter ckd (eg.Hubbard)	6.19	7.56	6.12	6.24
Beetroot, salad	4.44	4.83	5.70	5.69
Spinach, cooked	3.90	11.39	2.73	5.91
Sweetpotato, baked + skin (flesh only)	3.49	10.95	2.33	13.13
Beans, white kidney ckd (pot. onion HM)	3.01	10.91	3.25	11.42
Spinach, cooked (pot. onion + HM)	2.74	6.61	4.35	9.94
Potato, roasted in beef fat	2.40	8.01	4.01	13.35
Tomato, raw	2.23	9.20	0.55	2.55
Carrot, cooked	2.23	4.78	1.50	2.71
Salad, potato (Mayo, egg)	1.78	12.97	0.32	1.70
Salad, mixed veg (lett, tom, cucum, HM)	1.26	5.02	1.55	8.17
Sweetpotato, candied (sug. HM)	0.82	4.00	1.33	3.98
Sweetpotato, cooked without skin	0.72	5.07	-	-
Spinach, cooked with HM	0.67	4.64	0.20	2.12
Cole slaw, HM (Mayo)	0.62	3.41	-	-
Beans, green cooked (pot. + onion + sun)	0.50	3.53	-	-
Carrot, candied (sugar, HM)	0.50	2.14	0.70	4.00
Spinach, cooked (pot. onion, sun)	0.47	3.86	1.89	6.61
Potato, boiled + skin (flesh only)	0.37	2.77	2.63	17.79
Potato, mashed (SM + HM)	0.31	2.72	0.33	4.12
Beans, broad dried cooked	0.29	2.60	-	-
Sweetcorn, canned (whole kernel)	0.27	2.38	-	-
Samp: beans	-	-	0.93	11.56
Spinach cooked + butter	-	-	0.24	2.12
Potato, mashed (LFM, HM)	-	-	0.22	2.75
Carrot, raw	1.23	3.97	2.42	5.69

Table 10.1 (continued)

Stratum 2	Men		Women	
	Amount/ person (g/day)	SD	Amount/ person (g/day)	SD
VEGETABLES				
Potato, boiled without skin	5.97	10.02	-	-
Potato, mashed (WH, HM)	9.68	16.86	8.44	13.26
Onion, cooked	7.09	7.79	7.80	8.38
Tomato, boiled/canned	6.07	6.09	8.24	11.32
Beans, sugar dried cooked	6.16	11.90	5.22	8.14
Cabbage, cooked	4.35	5.97	4.83	5.24
Pumpkin/sqaush, winter ckd (eg.Hubbard)	5.23	7.79	8.56	15.14
Beetroot, salad	3.68	4.94	4.56	4.07
Spinach, cooked	2.72	7.93	4.83	23.68
Beans, white kidney ckd (pot. onion HM)	1.29	6.50	1.83	9.60
Spinach, cooked (pot. onion + HM)	5.09	11.86	9.75	16.30
Potato, roasted in beef fat	2.36	6.52	1.54	5.35
Tomato, raw	1.62	5.11	1.03	3.54
Carrot, cooked	1.04	2.27	1.56	3.01
Salad, potato (Mayo, egg)	-	-	0.27	1.51
Salad, mixed veg (lett, tom, cucum, HM)	-	-	-	-
Sweetpotato, candied (sug. HM)	0.29	1.27	0.78	2.39
Sweetpotato, cooked without skin	0.20	0.99	0.78	5.86
Spinach, cooked with HM	-	-	9.75	16.30
Cole slaw, HM (Mayo)	-	-	-	-
Beans, green cooked (pot. + onion + sun)	-	-	-	-
Carrot, candied (sugar, HM)	0.45	2.08	0.75	2.80
Spinach, cooked (pot. onion, sun)	0.61	5.06	4.83	23.68
Potato, boiled + skin (flesh only)	0.25	2.05	0.51	3.91
Potato, mashed (SM + HM)	0.91	6.87	-	-
Beans, broad dried cooked	-	-	-	-
Sweetcorn, canned (whole kernel)	1.41	8.76	-	-
Carrot, raw	0.48	2.27	1.24	4.53
Beans, green cooked, pot. onion, HM	0.71	3.92	0.22	1.35
Spinach, cooked + sun oil	0.25	2.05	-	-

Table 10.1 (continued)

Stratum 3	Men		Women	
	Amount/ person (g/day)	SD	Amount/ person (g/day)	SD
VEGETABLES				
Potato, boiled without skin	8.71	13.08	10.21	27.64
Potato, mashed (WH, HM)	17.10	22.32	20.19	35.54
Onion, cooked	8.29	6.86	8.75	9.44
Tomato, boiled/canned	8.44	10.49	9.31	11.31
Beans, sugar dried cooked	4.56	6.12	5.85	9.68
Cabbage, cooked	4.85	5.29	5.89	5.75
Pumpkin/sqaush, winter ckd (eg.Hubbard)	7.37	11.27	6.91	7.63
Beetroot, salad	7.41	12.70	7.16	6.62
Spinach, cooked	3.37	10.04	1.74	4.70
Sweetpotato, baked + skin (flesh only)	-	-	0.25	2.24
Beans, white kidney ckd (pot. onion HM)	4.73	17.76	3.31	14.71
Spinach, cooked (pot. onion + HM)	1.44	3.87	5.54	9.26
Potato, roasted in beef fat	0.66	2.81	4.91	13.89
Tomato, raw	0.66	2.27	0.89	2.52
Carrot, cooked	1.85	3.31	1.81	3.56
Salad, potato (Mayo, egg)	0.24	1.28	0.91	3.32
Salad, mixed veg (lett, tom, cucum, HM)	1.71	5.67	2.36	6.02
Sweetpotato, candied (sug. HM)	0.61	2.29	1.54	4.91
Sweetpotato, cooked without skin	0.22	1.41	0.56	2.35
Spinach, cooked with HM	1.73	6.37	0.34	1.72
Carrot, candied (sugar, HM)	0.68	2.40	0.66	2.90
Spinach, cooked (pot. onion, sun)	1.02	3.87	0.44	2.19
Beans, broad dried cooked	-	-	0.63	4.13
Sweetcorn, canned (whole kernel)	-	-	0.39	2.39
Cabbage cooked (pot. Onion, HM)	0.73	4.69	-	-
Beans, haricot dried cooked	0.73	2.59	0.64	4.56
Pumpkin/squash, summer candied (HM, sun)	0.39	2.50	-	-
Pumpkin/squash, winter candied	0.32	2.03	-	-
Lettuce, raw	0.29	1.72	-	-
Beans, green cooked (pot. Onion, HM)	0.29	1.87	0.38	1.69
Cucumber, raw	0.27	1.72	-	-
Spinach, cooked (pot, onion, PUM)	0.22	1.41	-	-
Sweetpotato, cooked without skin	0.22	1.41	-	-
Sweetpotato, candied (sug, sun)	-	-	0.36	2.44
Beans, white kidney dried cooked	-	-	0.35	2.91
Carrot, raw	1.88	7.30	3.16	5.84

Table 10.1 (continued)

Stratum 4	Men		Women	
	Amount/ person (g/day)	SD	Amount/ person (g/day)	SD
VEGETABLES				
Potato, boiled without skin	15.10	38.37	9.46	12.04
Potato, mashed (WH, HM)	15.06	20.84	13.72	15.80
Onion, cooked	11.03	9.38	9.63	7.49
Tomato, boiled/canned	13.34	14.39	11.45	11.95
Beans, sugar dried cooked	5.24	8.78	7.42	11.52
Cabbage, cooked	-	-	8.65	13.38
Pumpkin/sqaush, winter ckd (eg.Hubbard)	5.51	5.56	6.47	7.10
Beetroot, salad	9.42	17.18	8.25	7.91
Spinach, cooked	1.24	3.75	1.80	5.29
Sweetpotato, baked + skin (flesh only)	0.32	1.90	1.76	8.18
Beans, white kidney ckd (pot. onion HM)	1.51	12.70	6.18	23.55
Spinach, cooked (pot. onion + HM)	-	-	7.39	12.58
Potato, roasted in beef fat	4.51	19.12	2.69	7.65
Tomato, raw	1.61	5.08	3.15	14.43
Carrot, cooked	2.49	4.49	1.76	2.80
Salad, potato (Mayo, egg)	0.52	2.63	0.94	3.88
Salad, mixed veg (lett, tom, cucum, HM)	1.01	3.39	2.49	8.53
Sweetpotato, candied (sug. HM)	0.42	2.10	1.45	4.88
Sweetpotato, cooked without skin	0.80	2.49	0.29	2.02
Spinach, cooked with HM	0.56	3.43	-	-
Cole slaw, HM (Mayo)	-	-	-	-
Beans, green cooked (pot. + onion + sun)	-	-	-	-
Carrot, candied (sugar, HM)	0.63	2.08	1.09	5.90
Spinach, cooked (pot. onion, sun)	0.31	2.09	0.44	2.95
Potato, boiled + skin (flesh only)	1.77	10.72	1.87	12.65
Potato, mashed (SM + HM)	-	-	-	-
Beans, broad dried cooked	-	-	-	-
Spinach, cooked (pot, onion, HM)	5.44	11.22	-	-
Beans, white kidney ckd (pot, onion, HM)	1.34	5.29	-	-
Beans, haricot dried cooked	0.46	3.92	0.70	8.39
Lettuce raw	0.46	2.41	0.40	2.72
Beans, white kidney ckd (pot, on, PUM)	0.37	2.27	-	-
Beans, green cooked (pot. Onion, HM)	-	-	0.67	7.33
Beans, dried canned tom.sce (baked beans)	-	-	0.51	4.32
Beans, white kidney dried cooked	-	-	0.46	3.14
Beans, green cooked	-	-	0.45	2.31
Samp: Beans 1:1	-	-	0.18	1.26
Carrot, raw	1.41	3.40	3.41	8.93

Table 10.1 (continued)

Stratum 5	Men		Women	
	Amount/ person (g/day)	SD	Amount/ person (g/day)	SD
VEGETABLES				
Potato, boiled without skin	7.65	8.54	6.77	9.14
Potato, mashed (WH, HM)	14.96	14.22	12.81	14.32
Onion, cooked	8.33	6.99	8.46	6.49
Tomato, boiled/canned	11.16	22.32	10.12	9.27
Beans, sugar dried cooked	5.22	6.25	6.37	10.50
Cabbage, cooked	6.54	7.61	8.42	10.26
Pumpkin/sqaush, winter ckd (eg.Hubbard)	5.54	5.14	8.49	10.62
Beetroot, salad	6.72	14.31	8.43	15.08
Spinach, cooked	1.53	4.65	0.97	2.92
Sweetpotato, baked + skin (flesh only)	0.44	3.00	1.03	4.19
Beans, white kidney ckd (pot. onion HM)	2.96	12.22	6.60	26.80
Spinach, cooked (pot. onion + HM)	8.91	12.22	8.30	12.25
Potato, roasted in beef fat	7.08	17.28	5.14	15.29
Tomato, raw	3.11	6.29	2.86	9377
Carrot, cooked	2.35	3.55	1.69	3.32
Salad, potato (Mayo, egg)	0.80	2.29	0.58	2.76
Salad, mixed veg (lett, tom, cucum, HM)	5.85	15.27	6.58	12.62
Sweetpotato, candied (sug. HM)	1.85	4.29	2.86	11.35
Spinach, cooked with HM	0.24	1.50	0.83	3.87
Cole slaw, HM (Mayo)	-	-	0.73	4.59
Carrot, candied (sugar, HM)	0.25	1.40	2.52	8.86
Potato, boiled + skin (flesh only)	0.87	7.76	0.51	4.24
Sweetcorn, canned (whole kernel)	-	-	0.68	3.05
Lettuce, raw	0.78	2.70	0.71	3.80
Tomato, onion, stewed (sug, no short)	0.68	3.97	0.69	5.18
Beans haricot dried cooked	0.61	4.25	-	-
Onion sauteed in HM	0.43	0.96	0.40	0.97
Spinach, cooked (pot, onion, PUM)	0.24	2.14	-	-
Beans white kidney dried cooked	-	-	1.69	12.96
Salad, potato (mayo, egg, cream)	-	-	0.57	1.95
Beans, dried canned Tom sce (baked beans)	-	-	0.52	2.56
Onion, sauteed in HM	-	-	0.40	0.97
Cauliflour, cooked	-	-	0.33	3.59
Cole slaw, commercialo	-	-	0.22	2.39
Vegetales, mix canned/frozen cooked	-	-	0.22	2.39
Beans, green cooked	-	-	0.19	1.77
Carrot, raw	9.35	21.99	8.20	20.02

ADDENDUM 11

Table 11.1 Mean and standard deviation of fruit intake of men and women of the different strata

Stratum 1	Men		Women	
	Amount/ person (g/day)	SD	Amount/ person (g/day)	SD
FRUIT				
Apple, raw + skin	40.47	54.13	31.61	45.51
Orange, raw	40.38	83.85	29.85	49.75
Banana, raw	16.41	30.60	15.10	22.91
Grapes, raw	5.04	17.63	5.58	14.68
Pear, raw	4.28	13.86	2.69	14.69
Peach, raw	3.22	14.07	6.46	17.23
Fruit juice, baby average	1.73	7.38	2.42	10.44
Peach canned in syrup	1.69	4.00	1.63	4.83
Guava, canned/cooked + sugar	1.63	12.91	0.28	1.97
Mango, raw	0.77	5.69	-	-
Guava, raw	0.29	1.91	-	-
Appricot, raw	0.27	1.46	-	-
Appricot, canned in syrup	0.18	0.68	-	-
Orange juice, (Ceres, LF)	-	-	2.44	24.45
Guava juice, sweetened	-	-	1.96	24.25
Peach dried, raw	-	-	0.52	4.28

Stratum 2	Men		Women	
	Amount/ person (g/day)	SD	Amount/ person (g/day)	SD
FRUIT				
Apple, raw + skin	13.35	22.49	10.37	15.96
Orange, raw	7.23	15.44	6.17	13.13
Banana, raw	5.00	7.68	4.59	6.12
Grapes, raw	0.68	3.19	1.07	4.00
Peach, raw	11.65	33.53	7.49	21.45
Fruit juice, baby average	0.75	2.54	0.69	2.47
Peach canned in syrup	1.19	2.73	1.42	2.91
Peach dried, raw	-	-	0.39	2.33

Table 11.1 (continued)

Stratum 3	Men		Women	
	Amount/ person (g/day)	SD	Amount/ person (g/day)	SD
FRUIT				
Apple, raw + skin	34.46	45.30	69.20	94.76
Orange, raw	8.63	18.12	12.54	29.13
Banana, raw	35.98	53.39	28.79	40.08
Grapes, raw	2.66	7.27	4.01	9.63
Pear, raw	7.12	32.41	1.14	5.26
Peach, raw	4.37	13.28	4.51	13.16
Fruit juice, baby average	0.27	1.32	5.60	26.31
Peach canned in syrup	2.46	5.23	3.01	5.37
Guava, canned/cooked + sugar	-	-	0.88	5.05
Appricot, raw	0.41	2.20	-	-
Appricot, canned in syrup	0.46	2.12	0.31	1.07
Orange juice, (Ceres, LF)	2.44	15.62	1.24	7.40
Fruit salad, dried raw	0.29	1.38	0.31	2.09
Pear canned in syrup	0.27	1.72	-	-
Peach, dried raw	-	-	0.76	3.82
Fruit juice, dairy mix	-	-	0.31	2.80

Stratum 4	Men		Women	
	Amount/ person (g/day)	SD	Amount/ person (g/day)	SD
FRUIT				
Apple, raw + skin	53.66	89.38	57.58	84.12
Orange, raw	26.42	51.38	41.20	75.55
Banana, raw	28.15	43.23	24.42	32.26
Grapes, raw	3.87	9.46	8.58	23.99
Pear, raw	2.93	10.66	5.96	20.63
Peach, raw	5.49	16.44	2.83	12.04
Fruit juice, baby average	-	-	12.92	67.49
Peach canned in syrup	4.10	9.38	3.64	9.58
Guava, canned/cooked + sugar	0.35	1.94	-	-
Mango, raw	0.46	3.92	0.44	2.56
Guava, raw	-	-	0.72	4.96
Appricot, raw	0.63	4.14	0.42	3.28
Appricot, canned in syrup	0.25	1.58	0.66	2.80
Orange juice (Ceres, LF)	1.49	11.88	2.91	17.67
Cucumber, raw	0.30	2.37	-	-
Orange juice fresh	-	-	2.26	21.77
Orange juice canned sweetened	-	-	2.11	25.18
Pear juice, Ceres	-	-	2.11	25.18
Pear canned in syrup	-	-	0.70	8.39
Guava, canned/cooked + sugar	-	-	0.32	1.50
Fruit salad, dried raw	-	-	0.32	2.15

Table 11.1 (continued)

Stratum 5	Men		Women	
	Amount/ person (g/day)	SD	Amount/ person (g/day)	SD
FRUIT				
Apple, raw + skin	67.49	68.07	69.63	68.28
Orange, raw	41.77	59.76	42.57	54.62
Banana, raw	31.94	43.76	29.67	34.70
Grapes, raw	7.91	18.49	7.19	12.46
Pear, raw	17.59	44.94	17.42	40.11
Peach, raw	3.65	9.90	4.03	11.46
Fruit juice, baby average	24.75	50.19	31.44	73.17
Peach canned in syrup	3.11	6.63	4.36	6.99
Guava, canned/cooked + sugar	1.25	3.52	1.36	3.89
Mango, raw	0.23	1.11	0.39	2.44
Guava, raw	0.41	2.04	1.04	4.44
Appricot, raw	1.13	3.26	0.96	5.05
Appricot, canned in syrup	0.59	2.23	1.28	5.14
Orange juice, (Ceres, LF)	2.49	14.00	14.71	73.22
Fruit salad, dried raw	2.38	9.18	3.23	16.46
Cucumber, raw	0.18	0.81	0.86	6.39
Orange juice, fresh	-	-	4.91	38.25
Mango juice, Ceres	-	-	3.31	25.54
Prune, dried raw	-	-	0.66	4.93
Carrot, raw (pine, orange, sug)	0.27	2.36	0.48	5.25

ADDENDUM 12

Table 12.1 : Mean and standard deviations of intakes of eggs of men and women of the different strata

	Men		Women	
	Mean (g/day)	SD	Mean (g/day)	SD
Stratum 1	**(n=78)**		**(n=153)**	
Eggs, fried in sun oil	26.46	34.72	23.47	33.72
Egg boiled	8.95	21.36	7.22	17.14
Egg scrambled (WM; HM)	7.47	26.10	2.52	11.60
Total	**42.88**		**33.21**	
Stratum 2	**n=69**		**n=59**	
Eggs, fried in sun oil	18.86	36.50	10.37	15.96
Egg boiled	7.68	22.09	3.97	15.15
Egg scrambled (WM; HM)	1.78	9.56	1.08	5.19
Total	**28.32**		**15.42**	
Stratum 3	**n=41**		**n=80**	
Eggs, fried in sun oil	30.85	47.24	22.39	39.76
Egg boiled	4.71	11.93	2.79	7.88
Egg scrambled (WM; HM)	14.00	39.36	5.93	26.01
Total	**49.56**		**31.11**	
Stratum 4	**n=21**		**n=142**	
Eggs, fried in sun oil	32.44	42.22	21.81	31.80
Egg boiled	4.92	21.70	3.65	11.04
Egg scrambled (WM; HM)	8.85	34.05	5.90	19.98
Total	**46.21**		**31.36**	
Stratum 5	**n=79**		**n=118**	
Eggs, fried in sun oil	38.32	46.24	19.42	27.06
Egg boiled	11.16	22.32		
Egg scrambled (WM; HM)	1.37	9.79	4.13	14.06
Total	**50.85**		**23.55**	