LIFESTYLE, BODY FAT DISTRIBUTION AND INSULIN-RELATED CORONARY HEART DISEASE RISK FACTORS IN HYPERTENSIVE FEMALES

by

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Submitted in partial fulfilment of the requirements for the degree

D. Phil. (Human Movement Science)

in the

Faculty of Humanities

University of Pretoria

PRETORIA

September 2000

ACKNOWLEDGEMENTS

The author wants to thank the following persons who made this study possible:

- God, my heavenly Father, who gave me inner strength
- My promotor, Prof. J.M.. Loots, for his guidance and sempiternal patience
- My wife Sonia, for her moral support and endless help on the computor
- Mrs. Rita Olwagen, for her assistance with the statistical analysis
- Mr. Ian Cook and Dr. Sanette Brits, for their support and encouragement
- Dr. Mariaan Alberts and Dr. Tessa van der Merwe, for their advice on the blood tests
- Dr. Willie Kamffer and Dr. Henk Stander, for the sonars and scans
- Mrs. Carol-Lynn Nel, for her editing of the script
- Jani and Imé, my two daughters, who had to sacrifice so much

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% BF	Percentage body fat
5-HT_1	5-Hydroxytryptamine-1
A/C	Albumin Creatinine ratio
AA	Arachidonic Acid
AAR	Alliance for aging research
ACE inhibitors	Angiotensin Converting Enzyme inhibitors
Ach	Acetylcholine
ACTH	Corticotropin
ADP	Adenosine Diphosphate
ALLHAT	Anti-hypertensive and Lipid-lowering to Prevent Heart Attack Trial
ANG II	Angiotensin II
ANP	Atrial Natriuretic Peptide
Apo B	Apoprotein B-100
ARIC study	Atherosclerosis Risk in Communities study
AT I	Angiotensin I
ATG	Angiotensinogen
AV3V	Anteroventral Third Ventricle
AVP	Arginine Vasopressin
BF	Black females
BK	Bradykinin
BMI	Body Mass Index
BNF	British Nutrition Foundation
BW	Body weight
C	Cholesterol
CAD	Coronary artery disease
CAMP	Cyclic Adenosine Monophosphate
CE	Cholesterol ester
CETP	Cholesterol ester transfer protein
CHAOS	Cambridge Heart Anti-oxidant Study
CGMP	Cyclin Guanosin Monophosphate
CHD	Coronary Heart Disease
CHEC study	Community Hypertension Evaluation Clinic study
CM	Chylomicrons
CORIS study	Coronary Risk Factor Study.
CRF	Corticotropin-releasing hormone
CT	Computed tomography
Db	Body density
DBP	Diastolic Blood Pressure
DGA	Dihomo-gammalinolenic acid
DHA	Docosahexaenoic Acid
DHEA	Dehydroepiandrosterone

ECE	Endothelin Converting Enzyme
ECF	Extracellular Fluid
EDHF	Endothelium-derived Hyperpolarizing Factor
EDNO	Endothelium-derived nitric oxide
EDRF	Endothelium-derived Relaxing Factor
EE	Energy Expenditure
EFA	Essential Fatty Acid
EOLS	Endogenous Ouabain-like Substances
EPA	Eicosapentaenoic Acid
Factor VIIc	Coagulent activity of Factor VII
FAI	Functional Aerobic Impairment
FFA	Free Fatty Acid
FSH	Follicle stimulating hormone
FT	Fast Twitch fibres
GH	Growth hormone
GHRH	Growth hormone releasing hormone
GLA	Gamma Linolenic Acid
Glucose-6-P	Glucose 6 Phosphate
HDL cholesterol	High-density lipoprotein cholesterol
HL	Hepatic lipase
HOMA	Homeostasis Model Assessment
HOT study	Hypertension Optimal Treatment study
HPA axis	Hypothalamo-pituitary-adrenal axis
HT	Height
IBM	Ideal Body Mass
IDDM	Insulin Dependent Diabetes Mellitus
IDL	Intermediate Density Lipoprotein
IGF I	Insulinlike Growth Factor I
IMT	Intimal-medial thickness
ISH	Isolated Systolic Hypertension
ISH	International Society for Hypertension
J-G	Juxtaglomerular
JNC	Joint National Committee on the Detection Evaluation and Treatment of High Blood Pressure
L-arg	L-arginine
LA	Linoleic Acid
LCAT	Lecithin-cholesterol acetyl transferase
LDL cholesterol	Low density lipoprotein cholesterol
LH	Luteinizing hormone
Lp (a)	Lipoprotein a
LPL	Lipoprotein Lipase
LRC	Lipid Research Clinics
LVH	Left ventricular hypertrophy

MARS	Monitored Atherosclerosis Regression Study
MBP	Membrane Binding Protein
MRC	Medical Research Council
n-3	omega 3 poly-unsaturated fatty acid
n-6	omega 6 poly-unsaturated fatty acid
NEFA Non	esterified fatty acids
NHANES I	National Health and Nutrition Examination Survey I
NIDDM	Non-insulin dependent diabetes mellitus
NO	Nitric Oxide
PAI I	Plasminogen activator inhibitor I
PG	Prostaglandin
PGI_3	Prostacyclin
PHYLLIS	Plaque Hypertension Lipid Lowering Italian Study
PL	Phospholipids
POMS	Profile of Mood States
PRA	Plasma Renin Activity
PUFA	Poly-unsaturated Fatty acid
PV	Plasma Volume
RDA	Recommended dietary allowance
RIA	Radioimmunoassay
RR	Relative risk
SAG	Sagittal height
SANSS	South African Nutritional Status Survey
SBP	Systolic Blood Pressure
Sf	Svedberg unit
SHEP	Systolic Hypertension in the Elderly Program
SKF	Skinfold
SNS	Sympathetic Nervous System
SPSS	Statistical Package for Social Sciences
ST	Slow Twitch fibers
t-PA	Tissue plasminogen activator
TEE	Total Energy Expenditure
TG	Triglyceride
TGF	Transforming Growth Factor
TGRLP	Triglyceride Rich Lipoprotein
Thr	Thrombin
TMD	Total Mood Disturbances
UAER	Urinary albumin excretion rate
UBO	Upper Body Obesity
UK	United Kingdom
USA	United States of America
VIGHOR	Vanderbijlpark Information project re: Gesondheid/Health Obesity Risk Factors

VLDL	Very Low Density Lipoprotein
WF	White females
WHO	World Health Organization
WHR	Waist-to-hip ratio

SINOPSIS

TITLE: Lifestyle, body fat distribution and insulin-related coronary heart disease risk factors in hypertensive females

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There seems to be a dearth of information on insulin related coronary heart disease risk factors in South-Africa. The main objective of the present study therefore, was to determine if the lifestyles and body fat distributions of black and white female hypertensives increase their susceptability to insulin resistance and CHD. Such information may be used to optimize anti-hypertensive treatment of females and to lower their overall CHD risk.

A total of 60 females (30 black and 30 white) with uncomplicated essential hypertension were recruited in the Pietersburg area. Their physical activities and health were assessed with a questionnaire developed by Paffenbarger *et al.* (1993). A quantitative food frequency questionnaire was used to collect information on their habitual diets. The aerobic capacity of the subjects was measured indirectly with a 1 mile walking test. Overall obesity levels, percentage body fat and fat distribution were measured with standard anthropometric measurements such as body mass index (BMI), sum of skinfolds, waist-to-hip ratios (WHR) and sagittal heights. Abdominal fat was measured with computed tomography (CT) scans and ultrasound (sonars). The following tests were done on overnight fasting blood samples: full lipograms, insulin, glucose, uric acid, fibrinogen, plasminogen and leptin. Albumin and creatine levels were measured in random urine samples. Insulin resistance was calculated from fasting glucose and insulin concentrations according to the computer-solved homeostasis model of Matthews *et al.* (1985):Insulin

resistance = insulin/ ($22.5e^{linglucose}$).

Although black and white subjects in the present study were equally obese with similar WHRs, CT scans revealed that white subjects carried more intra-abdominal fat and black subjects more subcutaneous abdominal fat. This may explain why twice as many white subjects (17 vs 8) had large insulin related CHD risk clusters. Obesity thus seems to be more benign in the majority of black females, but those with a propensity for visceral accumulation, may be particularly at risk of CHD. They not only matched their white counterparts, as far as the sizes of their intra-abdominal fat areas and the number of their CHD risk factors are concerned, but they were all diabetic as well. The different fat distributions of black and white subjects could not be explained by differences in their diets, smoking, drinking and stress levels, but it should be noted that aerobic capacity showed strong inverse correlations with visceral fat areas in both groups. Although these correlations may be epiphenomena of genetic factors, it can be envisaged that an increase in aerobic activity would not only decrease the visceral fat areas of most subjects, but it would also improve their insulin sensitivity and CHD metabolic risk profiles. These prophylactic effects of aerobic exercise would be enhanced even more by a decrease in sugar, refined carbohydrates and saturated fat intake and an increased intake of fish and whole foods containing complex carbohydrates. For some females even assiduous attention to diet, body mass control and exercise may not be enough to lower their CHD risk signficantly and they should seriously consider supplementation with vitamin E, C and B complex.

Key words: hypertension, diet, physical acitivity, stress, insulin resistance syndrome, coronary heart disease, obesity, computed tomography, ultrasound, body fat distrution, visceral fat, type 2 diabetes.

SAMEVATTING

TITEL: Lewenstyl, verspreiding van liggaamsvet en insulienverwante risikofaktore vir koronêre hartsiekte in hipertensiewe vroue

NAAM VAN KANDIDAAT: L.A.S. du Plessis

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GRAAD: D. Phil.

Dit wil voorkom asof daar 'n gebrek aan inligting aangaande insulienverwante risikofaktore vir koronêre hartsiekte in Suid-Afrika is. Die hoofdoel van die huidige studie was dan juis om te bepaal of die lewenstyl en liggaamsvetverspreiding van swart - en wit hipertensiewe vroue hulle vatbaarheid vir insulienweerstand en koronêre hartsiekte verhoog. Hierdie inligting kan gebruik word om anti-hipertensiewe behandeling te optimaliseer en om algehele koronêre risiko te verlaag.

Sestig vroue (30 swart en 30 wit) met ongekompliseerde essensiële hipertensie is in die Pietersburgdistrik gewerf. Hulle fisieke aktiwiteit en gesondheid is geëvalueer deur 'n vraelys wat deur Paffenbarger *et al.* (1993) ontwikkel is. 'n Kwantitatiewe voedselfrekwensievraelys is gebruik om inligting in te samel oor die kos wat hulle normaalweg eet. Die aërobiese kapasiteit van die proefpersone is op indirekte wyse gemeet met 'n 1-myl staptoets. Algehele obesiteitsvlakke, persentasie liggaamsvet en vetverspreiding is gemeet met standaard antropometriese metings soos liggaamsmassa-indeks, som van velvoudiktes en middel-tot-heupomtrek-ratio's. Abdominale vet is gemeet met gerekenariseerde aksiale tomografiese skandering en ultraklank (sonar). Die volgende bloedtoetse is gedoen op oornagse vastende bloedmonsters: vol lipogramme, insulien, glukose, uriensuur, fibrinogeen, plasminogeen en leptien. Albumien- en kreatinienvlakke is gemeet in lukraakgekose urienmonsters. Insulienweerstand is bepaal vanaf vastende glukose- en insulienkonsentrasies volgens die homeostaserekenaarmodel van Matthews

et al. (1985): Insulienweerstand = insulien/ ($22.5e^{linglukose}$).

Alhoewel swart - en wit proefpersone in hierdie studie ewe obees was met soortgelyke middel-tot-heupratio's, het skandering gewys dat wit vroue meer intra-abdominale, en swart vroue meer onderhuidse abdominale vet dra. Dit mag dalk verklaar hoekom tweemaal soveel wit - as swart proefpersone (17 vs 8) hoë insulienverwante koronêre risikogroeperinge gehad het. Obesiteit blyk dus meer onskadelik te wees in die meerderheid swart vroue, maar dié met 'n geneigdheid tot akkumulering van viserale vet, mag 'n besonderse hoë risiko hê vir koronêre hartsiekte. Hulle was nie net op gelyke voet met hulle wit eweknieë in terme van die grootte van hulle viserale vetoppervlaktes en hoeveelheid koronêre risikofaktore nie, maar hulle was boonop almal diabeties. Die verskil in vetverspreiding van swart - en wit vroue kon nie verklaar word deur verskille in hulle diëte, rook- en drinkgewoontes en stresvlakke nie, maar daar moet op gelet word dat aërobiese kapasiteit sterk negatiewe korrelasies toon met viserale vetoppervlakte in beide groepe. Alhoewel hierdie korrelasies slegs epifenomena van genetiese faktore kon wees, sou mens kon verwag dat 'n verhoging in aërobiese kapasiteit nie net sou gepaardgaan met 'n krimping in viserale vetoppervlaktes in meeste proefpersone nie, maar ook met verhoogde insuliensensitiwiteit en 'n beter koronêre risikoprofiel. Die profilaktiese effek van aërobiese oefening kan verder verhoog word deur 'n afname in suiker, verfynde koolhidraat en versadigte vetinname, gepaardgaande met 'n toename in vis, asook kos wat bestaan uit gekompliseerde koolhidrate. Vir sommige vroue mag volharding met die regte dieet, massabeheer en oefening selfs nie genoeg wees om hulle koronêre risiko beduidend te verlaag nie, en hulle behoort dit ernstig te oorweeg om aanvullende vitamien E, C en B kompleks in te neem.

Sleutelterme: hipertensie, dieet, fisieke aktiwiteit, stres, insulienweerstandsindroom, koronêre hartsiekte, obesiteit, gerekenariseerde aksiale tomografiese skandering, ultraklank, liggaamsvetverspreiding, viserale vet, tipe 2- diabetes.