Coping, stress hormones and cardiovascular function in urbanised Africans

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For it is by grace you have been saved, through faith – and this is not from yourselves, it is a gift from God. *Ephebians 2:8*

OPSOMMING

AFRIKAANSE TITEL: Coping, streshormone en kardiovaskulêre funksie in verstedelikte Afrikane

Motivering: Verstedeliking is tans 'n meer universele verskynsel as ooit tevore. Die negatiewe uitwerking van verstedeliking op gesondheid, onder andere kardiovaskulêre disfunksie, hoë voorkomssyfers van hipertensie en endokriene strespatrone, kom meer prominent voor by swart Afrikane as hul blanke eweknieë. Die verband tussen die onderliggende fisiologiese en psigologiese meganismes van hiérdie verskynsel is egter steeds onduidelik.

Doelstelling: Die hoof doelstelling van hiérdie studie is om die verband tussen coping-style, streshormone en kardiovaskulêre funksie by landelike en stedelike Afrikane na te vors en om sodoende te bepaal of daar 'n verband tussen 'n spesifieke coping-styl, streshormoonvlakke en kardiovaskulere disfunksie is.

Metodologie: Hierdie studie is 'n onderdeel van die "**T**ransition and **H**ealth during **U**rbanisation in **S**outh **A**frica" (THUSA) – studie, 'n deursneebeeld van 'n vergelykende epidiomologiese studie wat tussen 1996-1998 afgehandel is. Landdrosdistrikte (37) in Noordwes is geïdentifiseer en ewekansig geselekteer om alle vlakke van verstedeliking te verteenwoordig. Die aanvanklike groep proefpersone het bestaan uit 821 oënskynlik gesonde manlike en vroulike Afrikane tussen die ouderdomme van 16 en 70 jaar. Na die verwydering van onvolledige stelle gegewens het hierdie studiegroep bestaan uit 353 manlike en vroulike Afrikane. Antropometriese metings is gedoen deur geregistreerde antropometriste en vraelyste is voltooi met behulp van opgeleide veldwerkers en gekwalifiseerde sielkundiges. Proefpersone is verdeel in 'n aktiewe coping (AC) groep en een wat passief cope (PC), soos afgelei van hulle reaksies op die S-COPE-vraelys, as gevalideerde en betroubare Setswana vertaling van die oorspronklike COPE-vraelys. Die proefpersone is daaarna verdeel volgens hulle vlak van verstedeliking, d.w.s. in landelike en verstedelikte groepe. Die landelike groep het bestaan uit proefpersone van stamgebiede en plaaswonings, met beperkte toegang tot lopende water en krag. Die verstedelikte groep het bestaan uit blou-boordjie werkers wat aan die buite-verstedelikte rand van die groter metropolitaanse gebiede woon, met toegang tot lopende water en krag. Rustende bloeddrukwaardes is gemeet aan die hand van die FINApres-metode. Proefpersone is verdeel in normotensiewe en hipertensiewe

groepe in ooreenstemming met bloeddrukwaardes van die FINApres-metode en die Riva-Rocci/Korotkoff-metode. Vastende, rustende serum-streshormoonwaardes van kortisol, prolaktien en testosteroonvlakke is gemeet en gekorreleer met die kardiovaskulêre waardes, afhanklik en onafhanklik van copingstyle. Betekenisvolle verskille tussen die veranderlikes is bepaal met variansie analises onafhanklik van ouderdom, liggaamsmassa-indeks (BMI) en leefstylfaktore soos fisiese aktiwiteit, alkoholverbruik en rookgewoontes. Ingeligte toestemming is verkry van alle deelnemers, met die toestemming van die ouers van minderjarige adolessente. Die Etiek Komitee van die Potchefstroomse Universiteit (PU vir CHO) het die studie goedgekeur. Die leser word verwys na die opsomming aan die begin van die manuskrip in Hoofstuk 3 vir 'n beskrywing van die proefpersone, navorsingsontwerp en analitiese metodes wat in hierdie verhandeling gebruik is.

Resultate: Resultate van die THUSA-studie toon aan dat die verstedelikte proefpersoongroep jonger, fisies meer aktief is, meer alkohol verbruik en hoër hipertensie-voorkomssyfers getoon het as hulle landelike eweknieë. Verstedelikte mans het 'n kardiovaskulêre patroon van hoër vaskulêre aktiwiteit, (totale perifere weerstandwaardes (TPR), laer kardiale omset (CO) en meegewendheidswaardes (Cw) maar hoër hartfrekwensie as landelike persone getoon. Die endokriene profiel van verstedelikte mans het hoër prolaktien en laer testosteroonwaardes getoon, gekoppel aan kleiner kortisol:prolaktien en groter kortisol:testosteroonverhoudings. Verstedelikte vrouens het dieselde tendens getoon met 'n patroon van hoër vaskulêre aktiwiteit (hoër diastoliese bloeddruk (DBP) en TPR waardes, met laer CO, Cw) en prolaktienwaardes vergeleke met landelike vrouens. As copingstyle in berekening gebring word was dit die AC verstedelikte mans vergeleke met landelike eweknieë, wat ook 'n patroon soortgelyk aan dié van alle verstedelikte mans getoon het, betreffende leefstylfaktore, bloeddruk en hipertensie-voorkomssyfers. Bykomend het hulle hoër hartfrekwensie- en laer Cw waardes, gekoppel aan groter prolaktien- en kleiner testosteroonwaardes asook 'n groter kortisol:prolaktien-verhouding getoon. Die verstedelikte AC en PC vrouens het beide meer alkoholverbruik en hoër vaskulêre aktiwiteit getoon maar slegs die AC vrouens het hoër hipertensie-voorkomssyfers vergeleke met landelike eweknieë getoon. Net die PC verstedelikte vrouens het hoër hoër prolaktienwaardes getoon en fisiese aktiwteit gerapporteer vergeleke met hul landelike eweknieë.

Gevolgtrekking: Verstedeliking onder Afrikane kan geassosieer word met 'n swakker kardiovaskulêre patroon gekoppel aan 'n verergerde stres-ervarende

patroon, vergeleke met landelike proefpersone. Hiérdie patroon is meer beklemtoon in AC stedelike mans wat moontlike chroniese stres-ervarings toon, met 'n groter Kortisol:prolaktien-verhouding.

SUMMARY

TITLE: COPING, STRESS HORMONES AND CARDIOVASCULAR FUNCTION IN URBANISED AFRICANS

Motivation: Urbanisation is now a more universal phenomenon than ever before. The negative effects of urbanisation on health, i.e. cardiovascular dysfunction, hypertension prevalence rates and endocrine patterns of stress are more prominently found in black Africans compared to white counterparts. The association between the underlying physiological and psychological mechanisms of this phenomenon, is however, still unclear.

Objectives: The main objective of this study is to investigate the association between coping styles, stress hormones and cardiovascular function in rural and urban Africans.

Methodology: This study is part of the "**T**ransition and **H**ealth during **U**rbanisation in **S**outh **A**frica" (THUSA) study, a cross-sectional comparative epidemiological study, which ran from 1996-1998. Magistrate districts (37) in the North-West province was identified and randomly selected to be representative of all levels of urbanisation. The initial subject group consisted of 821 apparently healthy African men and women between the ages of 16 and 70 years. After removal of incomplete datasets, this study group consisted of 353 African men and women. Anthropometric measurements were taken with assistance from biokinetisists and questionnaires were completed with the help of trained field workers and qualified psychologists. Subjects were divided into an active coping (AC) group or passive coping (PC) group, dependent on their responses on the S-COPE questionnaire, which is a validated and reliable Setswana translated version of the original COPE questionnaire. The subjects were subsequently divided according to their level of urbanisation i.e rural or urban groups. The rural group consisted of subjects from tribal areas and farmland dwellings who had limited access to water and electricity. The urban group consisted of blue-collar workers living on the peri-urban fringe of greater metropolitan areas, who had access to water and electricity. Resting blood pressure values were measured using the FINApres method. Subjects were divided into normotensive and hypertensive groups in accordance with blood pressure values from the FINApres method and Riva-Rocci/Korotkoff method. Fasting, resting serum stress hormone values of cortisol, prolactin and testosterone levels were measured

and correlated with the cardiovascular values, dependent and independent of coping styles. Significant differences between the variables were determined with variance-analyses independent of age, body mass index (BMI) and lifestyle factors i.e. physical activity, alcohol consumption and smoking habits. Informed consent was obtained from all the participants, with consent from parents of under-aged adolescents. The Ethics Committee of the Potchefstroom University for Christian Higer Education (PU for CHE) approved the study. The reader is referred to the abstract at the beginning of the manuscript in Chapter 3 for a description of the subjects, study design and analytical methods used in this paper.

Results: Results from the THUSA study indicated that the urbanised subject group were younger and physically more ative but with higher levels of alcohol consumption and hipertension prevalence rates than their rural counterparts. Urbanised men also revealed a cardiovascular pattern of higher vascular activity - total peripheral resistance (TPR), lower cardiac output (CO) and compliance (CO) but with higher heart rate compared to rural subjects. The endocrine profile of urbanised men revealed higher prolactin and lower testosterone values, coupled to smaller cortisol:prolactin and larger cortisol:testosterone values. Urbanised women revealed a similar pattern of higher vascular activity (higher diastolic blood pressure values (DBP) and TPR values, with lower CO and Cw) and prolactin values when compared to rural women. When coping styles were added into the equation, the AC urbanised men revealed a pattern similar to all urbanised men in regards to lifestyle factors, blood pressure values and hipertension prevalence rates . Additionally, they revealed higher heart rates and lower Cw values coupled to larger prolactin and smaller testosterone values as well as a larger cortisol:prolactin relationship. Urbanised AC and PC women had higher alcohol consumption values and higher vascular activity, with only AC women revealing higher hypertension prevalence rates compare to their rural counterparts. Only PC urbanised women revealed higher prolactin values and physical activity levels compared to their rural counterparts.

Conclusion: Urbanisation in Africans can be associated with a poorer cardiovascular and a greater stress experiencing pattern compared to rural subjects. This pattern is more accentuated in AC urbanised men, who showed signs of chronic stress and a larger cortisol:prolactin ratio.

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LIST OF ABBREVIATIONS

♂	-	Men
♀	-	Women
µg/dl	-	Microgram per decilitre
AC	-	Active coping
α	-	Alpha
ANOVA	-	Analysis of variance
ANCOVA	-	Analysis of co-variance, adjusted for a variable
β	-	Beta
C:P	-	cortisol:prolactin ratio
C:T	-	cortisol:testosterone ratio
CI	-	Confidence intervals 95%
CO	-	Cardiac Output
Cw	-	Arterial compliance/Windkessel effect
DBP	-	Diastolic blood pressure
e.g.	-	For example
F (df)	-	Degrees of freedom
HDL	-	High-density lipoprotein cholesterol
HPA	-	Hypothalamic-pituitary-adrenal
HT	-	Hypertension
i.e.	-	That is

LDL	-	Low-density lipoprotein cholesterol
LH	-	Luteinising hormone
mmol/l	-	Milli molar per litre
mmHg	-	Millimetre mercury
N	-	Number of subjects
NS	-	Not significant
PC	-	Passive coping
P:T	-	prolactin:testosterone ratio
RAS	-	Renin-angiotensin system
SAM	-	Sympatho-adrenal-medullary system (SAM)
S-COPE	-	Adapted Setswana-translated COPE questionnaire
SE	-	Standard error
SNS	-	Sympathetic nervous system
THUSA	-	Transition in Health during Urbanisation in South Africa
TPR	-	Total peripheral resistance
WHO	-	World Health Organisation

CHAPTER ONE

PREFACE AND OUTLINE OF THE STUDY

1.1 PREFACE

This dissertation consists of one manuscript in Chapter 3, which will be submitted for publication in a peer-reviewed journal*. Chapter 2 gives a literature overview of all the applicable variables under discussion in this study. The different coping styles, stress hormones and cardiovascular function are discussed in detail. Interactions between coping, stress hormones and cardiovascular dysfunction are also discussed. A graphic representation of the above-mentioned interactions is given at the end of Chapter 2. The relevant references are provided at the end of Chapter 2 and Chapter 4 as instructed by the mandatory style enforced by the North-West University, Potchefstroom Campus, Potchefstroom, South Africa. Once the article is submitted to the relevant journal for publication, the references will be in accordance to the prescribed style of the journal itself.

* Manuscript (Chapter 3): Journal for submission – International Journal of Psychophysiology.

1.2 OUTLINE OF STUDY

The outline of the study is as follows:

Chapter 1: Preface and outline of the study

Chapter 2: Introduction, literature overview, questions arising from the literature, motivation, aims and hypotheses

Chapter 3: **Manuscript** – Coping, stress hormones and cardiovascular function in urbanized Africans

Chapter 4: Summary, discussion and findings, bias, power of study, conclusion and recommendations

1.3 AUTHORS' CONTRIBUTIONS

The contribution of each of the researchers involved in this study is given in the following table:

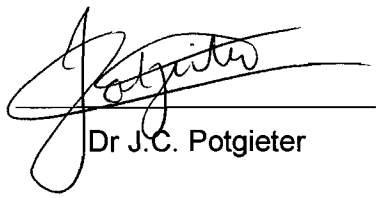
Table 1.1: Authors' contribution list

Ms Wilna Oosthuyzen (B.Sc) (Physiologist)	Responsible for literature searches, statistical analyses, design and planning of manuscript, interpretation of results and writing of manuscript.
Dr L. Malan (Ph.D) (Physiologist)	Supervisor. Supervised the writing of the manuscript, collection of data, initial planning and design of manuscript.
Dr J.C. Potgieter (Ph.D) (Psychologist)	Co-supervisor. Supervised the writing of the manuscript.
Prof. N.T. Malan (D.Sc) (Physiologist)	Co-supervisor. Supervised the writing of the manuscript and collection of data.

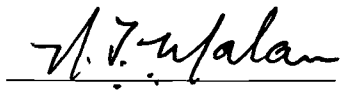
The following is a statement from the co-authors confirming their individual roles in this study and giving their permission that the manuscript may form part of the dissertation.

I declare that I have approved the above-mentioned manuscript, that my role in the study, as indicated above, is representative of my actual contribution and that I hereby give my consent that it may be published as part of the M.Sc. dissertation of Wilna Oosthuyzen.

Dr L. Malan



Dr J.C. Potgieter



Prof. N.T. Malan

CHAPTER TWO

INTRODUCTION AND LITERATURE OVERVIEW

2.1 INTRODUCTION

Urbanisation is now more wide spread than ever before. It is estimated that by 2015, more than half of Africa's population will be living in urbanised areas (Tarver, 1994).

The effects of urbanisation on the human body are quite substantial. In a study conducted on black Africans, hereafter only referred to as Africans, Vorster *et al.* (2000) found urbanisation to be associated with the more negative spectrum of health effects, i.e. a higher HIV infection prevalence, increases in obesity and other risk factors for non-communicable diseases. It was further indicated that urbanisation of Africans may be associated with psychosocial symptoms, such as increased alcohol consumption and smoking (Vorster *et al.*, 2000). In line with this, a study conducted on Kenyan Africans revealed that urbanisation was associated with increases in blood pressure (Cappucio, 1997). This finding was substantiated by Seedat (2000) who indicated that urbanisation in sub-Saharan Africans was associated with increases in blood pressure and higher hypertension prevalence rates.

On a psychosocial level, the process of urbanisation may lead to social disruption, which in turn may lead to increased stress levels (Rahman, 1997; Van Rooyen *et al.*, 2002). The stress theory, as described by Dressler (2004) posits that there are certain social environmental stressors or demands to which an individual needs to adjust. Emphasis is placed on the social and psychological resources which a person has at his/her disposal to cope with these demands (Dressler, 2004). Cassel proposed a hypothesis termed "host resistance", which states that every person is subjected to stressors and that illness is not dependent on these stressors, but rather on the person's innate ability to resist or cope with these stressors (Dressler, 2004). During urbanisation, Africans tend to move away from their collectivistic nature towards a more individualistic culture (Chang, 1996) and may therefore lose some of their ability to cope.

This ability to cope may greatly influence the negative effect of stress on the body as Malan and co-workers (2006) indicated that in Africans, the influence that stress has on the body is greatly dependent on the individual's ability to cope with the situation. This is further substantiated by Herd (1991) who indicated that the environment and previous experiences influence the behavioural reactions towards a stressor. In this behavioural reaction towards a stressor, three biological systems are of importance: neural, neuro-endocrine and neuroendocrine-immune reactions (McEwen, 2004).

Neuro-endocrine activity, the so-called fight-or-flight reaction with activation of the peripheral sympathetic nervous system and the concomitant release of nor-epinephrine and epinephrine from the adrenal medulla, promote the necessary adaptation in the face of a stressor (Gerra *et al.*, 2001). The allostatic stress model posits that the body is able to maintain stability through change (McEwen, 2004). Prolonged exposure to stress may, however, lead to wear and tear of these controlling systems and the systems that first protected, now damages (McEwen, 2004). The two physiological systems that are especially of importance are the sympathetic adrenal medullary (SAM) and the hypothalamic pituitary adrenocortical (HPA) system. Helhammer *et al.* (2006) revealed that the chronic stimulation of the SAM may contribute to the development of cardiovascular disease, while the chronic stimulation of the HPA-axis may be associated with the metabolic changes that are related to an increased risk of cardiovascular disease. Gerra and co-workers (2001) further hypothesized that these two systems may be two different physiological coping systems activated by different thresholds and features of stress. The SAM is non-specific, activated by all types of stress, while the HPA-axis is more specific and activated by aggression and defence reactions (Gerra *et al.*, 2001). Possible habituation of these systems in chronic stressful situations has been elucidated extensively (Gerra *et al.*, 2001; Ostrander *et al.*, 2006).

Christensen and Jensen (1995) indicated that dysfunctional coping styles seem to be the culprit concerning the harmful effects of stress in psychosocial stress, such as are caused by urbanisation. It was indicated that a specific response to stress and the coping style employed may be more harmful to health than a hyperactive HPA-axis and concomitant hypersecretion of cortisol (Christensen & Jensen, 1995). This observation was substantiated by Malan *et al.* (2006) who revealed the association between specific coping styles and different patterns of cardiovascular reactivity in urbanised Africans. Their findings showed that urbanised men and women revealed increased peripheral vascular α-adrenergic responses and greater hypertension prevalence rates when compared to rural men and women. Furthermore, these effects on peripheral vascular responses were found to be more prominent in the active coping (AC) subjects (Malan *et al.*, 2006).

Urbanisation, as a psychosocial stressor can therefore elicit a certain stress experiencing pattern through the activation of neuro-endocrine systems. This stress pattern may, however, be greatly influenced by the individual's inherent ability to cope with the situation. Although the association between psychosocial stress,

coping styles and cardiovascular effects in Africans have been indicated (Malan *et al.*, 2006), few studies have been done to indicate the association between coping styles, the stress pattern and cardiovascular function in Africans. The stress pattern can be interpreted by respectively viewing the stress hormone levels of cortisol, testosterone and prolactin.

Therefore, the general aim of this study is to determine whether the stress hormone levels and cardiovascular function of urbanised Africans who employ a specific coping style differ in any way from their rural counterparts. In the following literature overview the specific associations between coping styles, stress hormone levels and cardiovascular function will be discussed in detail.

2.2 COPING, STRESS HORMONES AND CARDIOVASCULAR FUNCTION IN URBANISED AFRICANS

2.2.1 Coping and coping styles

Lazarus and Folkman (1984) conceptualised coping as a continuous process that could be divided into the following three phases: primary appraisal, the process of determining whether a situation poses a threat to the self; secondary appraisal, the process of determining if there is anything that can be done about the stressful event as well as thinking of possible responses towards this perceived threat; and coping, the process of executing the chosen response (Lazarus & Folkman, 1984). A person's choice of coping response (during the secondary appraisal phase) can influence the outcome of the coping process, which might in turn alter the perception of the situation as not as threatening and therefore, not as stressful (Lazarus & Folkman, 1984). The different coping responses will subsequently be discussed.

2.2.1.1 Problem-focused and emotion-focused coping

Problem-focused coping is a direct approach in the management of stressors, during which the individual strives to solve the problem by getting to the root of the problem itself. Emotion-focused coping, on the other hand, is aimed at reducing or managing the emotions or distress associated with a situation (Carver, Scheier & Weintraub, 1986).

In stressful situations, both types of coping are usually employed. Problem-focused coping predominates when a person feels that something constructive can be done to change the situation. On the other hand, emotion-focused coping predominates

when the person experiences the situation as being out of control and that it should simply be endured (Carver *et al.*, 1989). Rueda and Perez-Garcia (2006) indicated that the continuous use of emotion-focused coping strategies can, in the long-term, effectively decrease both psychological and physiological well-being. Different situations require different coping styles, however. Magaya, Asner-Self and Schreiber (2005) indicated that Zimbabwean adolescents employed emotion-focused coping more often than problem-focused coping, and found that this type of coping style was not seen as a maladaptive form of functioning, as it effectively reduced the stress experience on the short term (Magaya *et al.*, 2005; Matheson & Cole, 2004). It is only when emotion-focused coping and downplaying of the importance of a problem undermine the motivation to adopt a more strategic response to manage the stressor that it becomes maladaptive (Matheson & Cole, 2004).

2.2.1.2 Active and passive coping

Henry, Stephens and Ely (1986) proposed a model in which the stress reaction is divided into a defence or active coping response and a defeat or passive coping response. Active coping involves either behavioural or psychological responses aimed at changing the nature of the stressor. Activities like planning; restraint and suppression of competing activities form an important part of the active coping process (Carver *et al.*, 1989) and show a strong semblance to the problem-focused approach that was discussed in die previous section. On the other hand, defeat or passive coping responses are avoidant by nature and are thought to be a psychological risk factor or marker for more adverse responses (Holohan & Moos, 1987). This passive form of coping, as termed by Lazarus and Folkman (1984), is closely associated with emotion-focused coping (Carver *et al.,* 1989) that was discussed in the previous section. For the purpose of this study the digitome classification of coping is used, referring to active coping (AC) and passive coping (PC) when discussing coping styles.

2.2.2 The stress response

Selye's theory of the General Adaptation Syndrome divides the stress response into three separate stages (Scollan-Koliopoulos, 2005). The first stage is the 'alarm reaction', the classic fight-or-flight reaction in which the sympathetic nervous system (SNS) is immediately activated. Thereafter, the 'stage of resistance' takes place, during which the HPA-axis is chronically activated. Finally, the 'stage of exhaustion' takes place during which serious damage to organs and bodily systems occurs

(Scollan-Koliopoulos, 2005). The time-span between each of these stages may differ from individual to individual, dependent on the severity and duration of a specific situation, the person's own subjective appraisal of the situation, as well as individual biological differences (Scollan-Koliopoulos, 2005).

Stimulation of the SNS during the alarm reaction leads to the release of epinephrine (E) and norepinephrine (NE) from the adrenal medulla. E has a stimulatory effect on the β-receptors and thereby increases heart rate, stroke volume and contractility (Opie, 2004). Furthermore, E and the associated stimulation of the β-adrenergic receptors are associated with an active or 'defence' coping style (Anderson, 1989; Henry *et al.*, 1986; Scollan-Koliopoulos, 2005).

A study by Ma and Morilak (2005) indicated that the medial amygdala (MeA) plays a central role in the stress response, through the release of NE. The release of NE from the adrenal medulla leads to a cascade of physiological events. Firstly, NE activates α1-receptors which facilitate the stimulation of the HPA-axis with the concomitant release of corticotrophin releasing hormone (CRH) (Ma & Morilak, 2005). Secondly, Opie (2004) elucidated the two-fold action of NE on the cardiovascular system. When NE binds to β-adrenergic receptors, it causes an increase in heart rate and contractility, with the binding of NE to α-adrenergic receptors leading to an increase in peripheral vascular resistance (Opie, 2004). Studies have further shown that NE outflow is associated with the adoption of a more passive or 'defeat' coping style in handling a situation (Scollan-Koliopoulos, 2005).

The stress response is mediated by two components of the hypothalamus and the brainstem: the CRH-receptors and the locus coeruleus-nor-epinephrine/ sympathetic system (Rosmond, 2005). The stage of resistance could imply increased activity of these systems with concomitant abnormal levels of cortisol and the accompanied effects on bodily systems (Rosmond, 2005). Chronic stress can, however, decrease the responsiveness of the HPA-axis and desensitize it, thereby leading to initial high levels of cortisol which stabilizes thereafter (Lundberg, 2005; Ostrander *et al.*, 2006). In severe and chronic stressful situations the HPA-axis might even become hypo-responsive, which in turn can lead to hypocortisolism (Kristenson *et al.*, 2004). Huisman and co-workers (2002) discovered this phenomenon in urbanised Africans where cortisol levels were found to be lower in the face of continued stressful situations. This response could imitate the phase of exhaustion, as termed by Selye (Scollan-Koliopoulos, 2005). However, the stress response is clearly a complex system which is, even now, not fully understood.

2.2.3 Stress hormones

As discussed above, the HPA-axis is the controlling centre for endocrine activity in stressful situations (Black & Garbutt, 2002; Kruk *et al.*, 1998; Lundberg, 2005). The stress hormones investigated for the purpose of this study are cortisol, testosterone and prolactin. Increases in cortisol levels are indicative of stress, as cortisol levels could increase with the expectation of an oncoming stressful situation (Guyton & Hall, 2006; Henry, 1992). Cortisol levels are furthermore also good indicators of hypo- or hyperactivity of the HPA-axis and could thereby also indicate the nature of the stressor exposed to (Gaab *et al.*, 2006; Ostrander *et al.*, 2006). Testosterone levels on the other hand are a strong indication of an expectation of success (Henry, 1992) and can, therefore, provide valuable information regarding the expectancy and mindset of the individual within a specific situation. Prolactin is indicated as a stress hormone, as prolactin levels could be associated with a feeling of control (Henry, 1992) and this, in turn, can also be indicative of the mindset of the individual.

These stress hormones can, therefore, clearly indicate the activity and responses of the HPA-axis within the stress experience as well as the psychological responses involved. Each of these hormones will subsequently be discussed in detail to investigate each hormone's characteristics and function.

2.2.3.1 Cortisol

Cortisol is a glucocorticoid hormone secreted by the adrenal cortex, whose secretion is closely regulated by adrenocorticotrophic hormone (ACTH). Greenspan and Strewler (1997) indicated that the circadian rhythm of ACTH secretion, responsiveness of the HPA-axis to stress and the feedback inhibition of cortisol through ACTH secretion, are all under neuroendocrine control. A study revealed that while the HPA-axis is not abnormally active during rest, it does, however, become hyperactive and it stimulates the release of cortisol with stimulation (Björnthorp, 1997)

Cortisol enhances vascular reactivity in response to stimuli such as NE (Widmaier, Raff & Strang., 2006) and thus it has a permissive effect on the secretion of NE from the adrenal medulla. Ma and Morilak (2005) further indicated the effect that NE has on cortisol secretion through stimulating CRH secretion, as discussed above. The effect that elevated cortisol levels have on the cardiovascular system can thereby be closely linked with the effects of NE on the cardiovascular system. Whitworth and co-

workers (1995) substantiated this statement by elucidating the effects of cortisol on the cardiovascular system especially. They found that elevated levels of cortisol may be associated with hypertension through several mechanisms, including sodium retention, haemodynamic changes, hyperinsulemia, vascular responsiveness and increased SNS activity.

Elevated cortisol levels can, therefore, be seen as a stress hormone marker associated with various damaging effects on the cardiovascular system. Malan *et al.* (1996) elucidated the effect of cortisol further by attributing the adverse effects that stress, such as is caused by urbanisation, has on health, to the increase in glucocorticoid hormones in chronic and acute stress situations. Huisman and co-workers (2002) could, however, not substantiate this association in another subgroup of Africans, as cortisol levels were not associated with increases in blood pressure of vascular responsiveness. A possible explanation for this is given by Gaab and co-workers (2006) who found that the chronic release of cortisol due to stress may have deleterious effects on health, through a deregulated HPA-axis. Therefore, the available literature on the mechanisms of cortisol and especially HPA-axis activity in the face of a stressful situation, such as urbanisation, is still contradictory and extremely complex.

2.2.3.2 Testosterone

Testosterone is part of the 19-carbon class of steroids, known as androgens. In men, it is secreted by the Leydig cells within the testes in response to stimulation by circulating luteinizing hormone (LH) (Greenspan & Strewler, 1997). The secretion of testosterone starts with the activation of the hypothalamus, which leads to the release of gonadotrophin-releasing hormone (GnRH). GnRH then circulates to the anterior pituitary where it stimulates the secretion of LH. LH is absorbed by the Leydig cells where it eventually leads to the secretion of androgens, and mainly testosterone (Greenspan & Strewler, 1997). In women, small amounts of androgens are secreted by the ovaries while larger amounts of androgens are secreted by the adrenal cortex under HPA-axis control (Widmaier *et al.*, 2004). The hypothalamus is, therefore, directly involved in the secretion of testosterone. Elman (2001) found a negative correlation between testosterone and epinephrine levels with activation of the adrenomedullary hormonal system, leading to decreases in testosterone levels. Henry (1992) substantiated this finding by indicating that increased testosterone levels can be associated with a feeling of control over the situation. Therefore, in

stressful situations testosterone levels may decrease, yet with a feeling of control it will increase again.

The role testosterone might play in the development of hypertension was elucidated by Reckelhoff (2001) who indicated that testosterone is known to increase blood pressure values in both men and women. Huisman and co-workers (2002) found contradictory evidence in African women, where they indicated that testosterone correlated with compliance with low levels of testosterone, associated with increased vascular responsiveness.

It thus follows that, the effect testosterone has on cardiovascular and stress responses in men and women are still contradictory and complex concerning Africans.

2.2.3.3 Prolactin

Prolactin is a 198-amino-acid polypeptide that is secreted by the anterior pituitary (Greenspan & Strewler, 1997). One of its functions in women is to inhibit gonadotropin secretion; hyperprolactinemia has been shown to lead to hypogonadism (Greenspan & Strewler, 1997). This finding was further substantiated by a study which revealed that high prolactin levels might indeed suppress testosterone levels in urbanised African men (Huisman *et al.*, 2002). This interaction might, in turn, be associated with increases in vascular responsiveness in Africans (Huisman *et al.*, 2002).

The available research of prolactin and psychological responses is currently scarce and contradictory. An important function of prolactin within the stress response is its actions as a cortisol antagonist, which has a modulatory effect on the anti-inflammatory action of cortisol (Gala, 1990) and therefore, these actions indicate the severity of the stress experience.

Additionally, Henry (1992) indicated that prolactin levels will increase as the perception of control over a situation decreases. Prolactin secretion might be stimulated by neurotransmitters such as dopamine, serotonin and NE. Prolactin levels may therefore be indicative of the activity of the central dopaminergic system (Hardan *et al.*, 1999). It can therefore, be deduced that the HPA-axis also plays a role in the secretion of prolactin in stressful situations. Dostal and his team (2003), however, found that prolactin levels will increase initially, but not continually, in the face of a prolonged stressful situation.

The interpretation of prolactin levels is complex, but of importance as it may be indicative of a feeling of control or depression in the long run. Its effect on testosterone is also of importance to better understand the concomitant cardiovascular effects these hormones may have.

2.2.4 Stress hormone ratios

In Africans, Malan and co-workers (1996) found the relationship between endocrine activity and the emotional experience of stress to be very complex. Henry *et al.* (1986) indicated the association between a larger cortisol:testosterone ratio and a depressed state of mind. Malan *et al.* (1996) found this to be true in rural Africans, as they were subjectively experiencing the most stress in the experimental setup. The urbanised Africans showed a smaller resting cortisol:testosterone ratio, which in turn could be indicative of a lesser stress experience (Malan *et al.*, 1996).

A larger cortisol:prolactin ratio (C:P) is suggestive of control over the situation, as Henry *et al.* (1986) indicated that a smaller C:P indicate a feeling of loss of control. In contrast to this, a larger C:P might also indicate habituation of the HPA-axis with the concomitant decrease in cortisol secretion, as experienced during prolonged exposure to a stressful situation (Ostrander *et al.*, 2006). A further explanation for this phenomenon is given by Greenspan and Strewler (1997) who indicated that cortisol is a prolactin antangonist and thereby effectively decreases prolactin levels.

The relationship between the prolactin:testosterone ratio (P:T) and the stress experienced is currently not known. From the available literature, it is, however, possible to deduce that a greater P:T may indicate a feeling of loss of control and depression as high levels of prolactin together with low levels of testosterone are associated with a depressed state of mind (Henry, 1992). This deduction is in accordance with results from Malan and co-workers' (1996) research which indicated that high prolactin and low testosterone levels are indicative of a loss of control and the experience of more stress in Africans.

2.2.5 Cardiovascular variables

Blood pressure = Cardiac output (CO) x Total peripheral resistance (TPR) (Opie, 2004). From the equation the importance of TPR in the control of blood pressure is quite apparent. The following three mechanisms are responsible for regulating TPR within the cardiovascular system: Autonomic control – vasoconstriction vs. vasodilatation, vasoconstrictive hormones and endothelial control (Opie, 2004).

Neurotransmitters such as NE are known to be vasoconstrictive due to local α-adrenergic stimulation, with adenosine and nitrous oxide (NO) having a vasodilatory action on the arterioles (Opie, 2004). When TPR increases, diastolic blood pressure (DBP) is more susceptible to increases than systolic blood pressure (SBP) (Opie, 2004) as TPR is a good indication of the resistance against which the heart must pump (Guyton & Hall, 2006).

Cardiac output (CO), on the other hand, is a good indicator of the working of the heart itself (Guyton & Hall, 2006; Opie, 2004). It is defined as the amount of blood pumped into the aorta by the heart each minute (Guyton & Hall, 2006) and it is mainly regulated by two factors, namely TPR and heart rate (HR) (Guyton & Hall, 2006).

Compliance can be defined as the change in volume of the artery per unit pressure (Guyton & Hall, 2006) and is can therefore described as the ability of the artery to maintain volume flow despite changes in pressure. The windkessel effect (Cw), as described by Middlemost (1999), is the ability of the arteries to store part of the stroke volume (SV) during systole and drain it during diastole and thereby maintaining volume flow and ensuring smooth blood flow. Compliance and TPR are seen as the two main determinants of afterload on the heart (Middlemost, 1999). Furthermore, Cw can be seen as a risk factor for cardiovascular events, as Cw is decreased in the early phases of hypertension (Middelmost, 1999).

The cardiovascular system is therefore a closely interlinked system with small changes in one area leading to big effects in another, as can be seen in the interaction between TPR, CO, HR and Cw. This system is also very sensitive for other influences, increased SNS activity has been shown to lead to increased vascular muscle tone, as can be reflected in an increased TPR (Guyton & Hall, 2006) and concomitant decreased compliance and is thereby a good indicator of future cardiovascular dysfunction (Resnick *et al.*, 2000).

2.2.6 Cardiovascular risk factors

For the purpose of this study, several risk factors that may have adverse effects on the cardiovascular system were identified within the African group. These risk factors cannot be ignored when interpreting the results and it is therefore of importance to understand the effects each of these factors better.

2.2.6.1 Gender as a risk factor in the development of cardiovascular dysfunction

The WHO recognises gender as one of the non-changeable risk factors in the development of cardiovascular disease (WHO, 2003). It is now well known that men are more at risk concerning the development of cardiovascular dysfunction than their female counterparts. Opie and Seedat (2005) found that hypertension prevalence rates were higher in African men than those found in women. Another study substantiated this finding by indicating that men have a slower return to a normotensive state after a stressful event than women, which can be attributed to the vasodilatory effect of estrogen (Scollan-Koliopoulos, 2005). This is probably due to the negative influence androgens may have on the cardiovascular system, coupled to the protective function of estrogens against cardiovascular disease (ANON, 2002). Reckelhoff (2001) supported this finding showing through her study that the higher prevalence of hypertension found in men, could be attributed to the role of testosterone and the stimulatory effect it may have on the renin-angiotensin system (RAS).

2.2.6.2 Race as a risk factor in the development of cardiovascular dysfunction

Fray and Douglas (1993) indicated that 25% of the reported 60 million hypertensive patients in North America today are African-American. They further stated that cardiovascular disease found in African-Americans are more severe, diagnosed at an earlier age and more fatal at a younger age, when compared to their Caucasian counterparts (Fray & Douglas, 1993). A possible explanation for this phenomenon is that African-Americans show a greater total peripheral resistance (TPR), probably due to hypertrophy of the vasculature or an increased reactivity. Additionally, it has been indicated that African-Americans (Anderson, 1989; Ergul, 2000) and Africans (Van Rooyen *et al.*, 2000) show greater sympathetic and cardiovascular reactivity (Anderson, 1989) towards a stressor with concomitant increases in vascular resistance and higher prevalence rates of hypertension.

Mufunda and Sparks (1992) attempted to understand this greater prevalence of cardiovascular disease better by investigating salt sensitivity in Africans. They, however, concluded that while Africans showed an increased salt sensitivity, this increase is not sufficient to be the cause of hypertension on its own. Supporting this profile, Africans with a greater inherent salt sensitivity and low renin-angiotensin hypertension (Opie, 2004) were found to show higher plasma renin activity during

acute stress, which may contribute to the higher hypertension prevalence rates found amongst urbanised Africans (Malan *et al.*, 2006; Malan *et al.*, 1996).

The need has, however, arisen to view race not as a risk factor, but rather as a risk marker in the development of cardiovascular disease (Anon, 2002; Appel, Harrell & Deng, 2005). Appel and co-workers (2005) indicated that other factors such as body mass index (BMI) and educational levels are more reliable predictors of cardiovascular risk. Other studies supported this by showing that the high prevalence of cardiovascular disease found among African-Americans is mediated by other risk factors and not purely race (Kristenson *et al.*, 2004; Thomas, Eberly, Smith, Neaton & Stamler, 2005). It is therefore necessary to proceed with caution when viewing race as a risk factor, since it may be necessary to refer to contextual model as conceptualised by Anderson and McNeilly (1992) which states that race should not be viewed as a proxy for genetic differences, but rather as a proxy for the effect of exposure to chronic social and sociocultural factors.

2.2.6.3 Urbanisation as a risk factor in the development of cardiovascular dysfunction

Seedat (2000) calls the spread of cardiovascular disease through developing countries, a "second wave epidemic". Opie and Seedat (2005) substantiated this by indicating that the high prevalence of urbanisation among Africans, accounts in part for the high occurrence of hypertension, especially in African men. Urbanisation is further associated with an increase in both systolic and diastolic blood pressure in Africans (Schutte *et al.*, 2004). Schutte and co-workers (2004) concluded that the urbanisation of African men is associated with an increase in blood pressure through a peripheral mechanism. Another study elaborated that all urbanised individuals are more at risk for the development of hypertension through the effects on the vascular system (Van Rooyen *et al.*, 2002). Malan *et al.* (2006) supported these findings by indicating that urbanised Africans showed higher hypertension prevalence rates and vascular responsiveness compared to rural groups. Additionally, Knardahl (2000) indicated that environmental and predispositional factors are of importance in the development of cardiovascular disease. This statement is supported by the work of Mufunda and Sparks (1992) who revealed that although Africans show an increase in salt sensitivity, it is rather psychosocial stress caused by urbanisation coupled with Westernised living habits such as increased alcohol consumption, more obesity, increased consumption of carbohydrates and altered dietary electrolytes, that play a role in the development of hypertension in urbanised Africans.

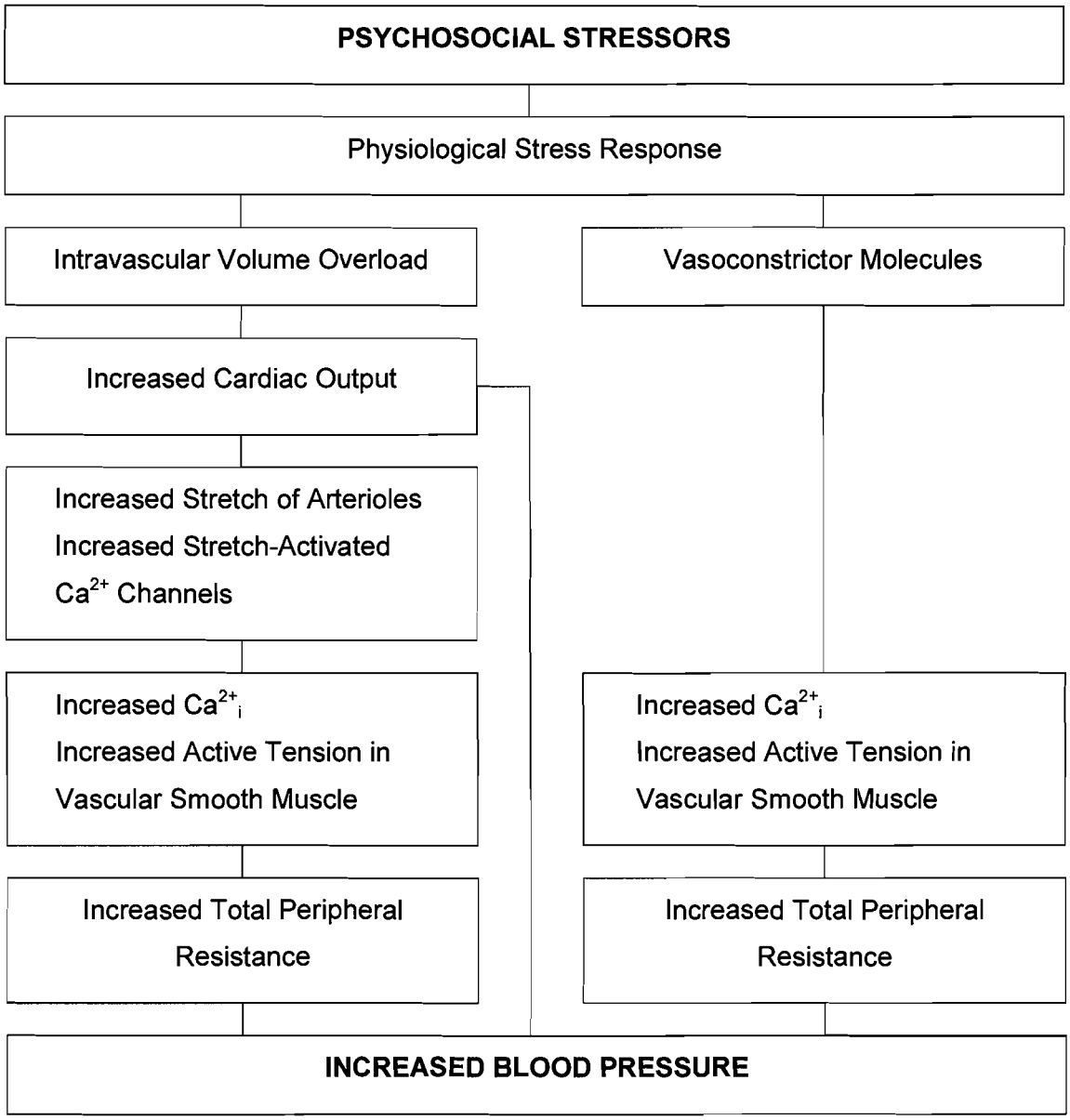


Figure 2.1: Pathogenesis of sustained hypertension in blacks (Fray & Douglas, 1993)

Figure 2.1 provide a graphic representation of the pathogenesis of hypertension in Africans. Fray and Douglas (1993) additionally indicated that neural influences might be the profound influence in the physiological stress response leading to vascular reactivity, increased CO and TPR through an α-adrenergic response. This model enforces the term "psychosocial stressor induced-hypertension" as termed by Fray and Douglas (1993) to be a more accurate description in the development of hypertension in Africans, than essential hypertension.

2.2.7 Interaction between stress hormones and cardiovascular function

A study by Nyklicek (2005) has indicated that some hypertensive subjects not only have a hyperactive cardiovascular system, but also an overactive HPA-axis and immune reactivity towards stress. Furthermore, a study conducted on cynomolgus monkeys by Rosmond (2005) showed that the continuous activation of the HPA-axis can lead to a series of physiological events that may result in myocardial ischemia, ventricular fibrillation, plaque rupture and coronary thrombosis. The effect of cortisol has been shown to increase heart rate and blood pressure causing constriction of the coronaries and accumulating lipids in the intima, all of which can be the precursor of the above cardiovascular dysfunction (Rosmond, 2005). Cortisol seems to have an indirect, rather than a direct effect on the cardiovascular system through the effects it may have on fat distribution and insulin resistance (Björntorp, 1997; Rosmond, 2005). Additionally, cortisol may further exercise its effect on cardiovascular function through the permissive effect it has on NE secretion (Widmaier *et al.*, 2004) as was discussed earlier.

Research on the specific effect of testosterone on the cardiovascular system is currently contradictory. Some studies found the testosterone levels of Africans to be negatively associated with hypertension (Huisman *et al.*, 2002) while Reckelhoff (2001) indicated a positive association with the development of hypertension. In his attempt to elucidate the effect of testosterone on cardiovascular function, Khalil (2005) has shown that testosterone and other sex hormones stimulate endothelial cell growth and inhibit smooth muscle cell proliferation. Relaxation of the endothelium also occurs with stimulation of the sex hormone receptors (Khalil, 2005), thereby leading to vaso-relaxation. On the other hand, increases in androgens, such as testosterone, is known to increase blood pressure (Reckelhoff, 2001) through the hypothesised mechanism of sodium reabsorption and its effect on the renin-angiotensin system (Reckelhoff, 2001). Additionally, renin activity (Malan *et al.*, 1996) and salt sensitivity are both seen as risk markers for the development of hypertension in Africans (Ergul, 2000, Opie, 2004). Huisman *et al.* (2002) indicated that lower testosterone and elevated prolactin levels are associated with increased diastolic blood pressure (DBP) and total peripheral resistance (TPR) reactivity in urbanised African women. This might also be an explanation of the higher hypertension prevalence found in this specific subject group compared to the rural African women (Huisman *et al.*, 2002).

From the literature, cortisol is known to increase the risk of cardiovascular incidents. However, information regarding the specific effect testosterone has on the cardiovascular system, is currently contradictory, and the available research on the effect of prolactin is insufficient.

2.2.9 Interaction between coping styles and cardiovascular function

The contextual model as discussed above, considers the role of psychosocial factors in the development of hypertension in African-Americans. Dressler, according to Fray and Douglas (1993), showed that the risk of hypertension in African-Americans may be caused by a high status lifestyle, low class rank and little perceived social support.

The specific interaction between coping styles and cardiovascular function is however, still complex and contradictory. Numerous studies have indeed indicated that African-Americans who actively cope with life demands through increased effort, determination and with the perception of little chance of success ("John Henryism"), run a higher risk for the development of cardiovascular disease (Fray & Douglas, 1993) and hypertension prevalence rates (Fernander *et al.*, 2004). Fray and Douglas (1993) enforced this finding by revealing that the final manifestation of active coping (AC) is increased cytosolic calcium with its concomitant cardiovascular effects. Finally, it was indicated that active coping together with its associated SNS induced vascular hyperreactivity and hypertension are associated with salt retention in African-Americans. This deranged salt metabolism may be due to altered sodium transport in the kidneys and may be the pathogenic manifestations of chronic exposure to psychosocial stressors (Fray & Douglas, 1993).

The increased cardiovascular risk associated with active coping styles was supported by Malan and co-workers' (2006) study, which indicated that AC urbanised subjects showed higher blood pressure values, especially regarding vascular values, when compared to AC rural subjects.

Contrary to these findings, Henry *et al.* (1986) proposed in his model that AC is characterised by an increase in β-adrenergic activity and cardiovascular reactivity, with an increase in cardiac output (CO) and a decrease in total peripheral resistance (TPR). Passive coping (PC), on the other hand, is characterised by an increase in α-adrenergic activity and cardiovascular reactivity with a decrease in CO and an increase in TPR. Additionally, the vascular hyperactivity model states that "beta-

activity" causes increases in heart rate, stroke volume and CO, with "alpha-activity" causing increased TPR (Fray & Douglas, 1993).

It thus appears that the available research on the association between coping styles and cardiovascular function is still unclear, especially with regard to Africans.

2.2.9 Interaction between coping styles and stress hormones

It has been shown that perceived control and the adoption of an active style of coping lead to decreased physiological reactivity and concomitant decreased cardiovascular and endocrine reactivity (Matheson & Cole, 2005). According to Matheson and Cole (2005), this is true especially for cortisol, due to a decrease in glucocorticoid and sympathetic nervous system activity.

Other studies have indicated that the perception of control over a situation is the cause of different endocrine activation patterns (Henry, 1992; Salvador, 2005).orde Henry's (1992) animal model has revealed that with an expectation of success and the concurrent feeling of control, testosterone levels rise. As the animal gradually loses control, the coping strategies employed become more passive and the prolactin levels rise. During stressful situations, the initial cortisol levels are high, but with prolonged exposure to stress, the cortisol levels return to normal (Henry, 1992). According to Huisman and co-workers (2002) urbanised African females indicated an endocrine pattern of lower cortisol levels coupled with low testosterone and high prolactin levels, which might indicate a perception of unavoidable/passive coping and ongoing stress, which may lead to a desensitizing of the HPA-axis with lower cortisol levels. In a study regarding the influence of acculturation on endocrine reactivity in Africans, Malan *et al.* (1996) showed that the endocrine reactivity of rural Africans differed significantly from urban Africans and Caucasians, and that these patterns were altered during the urbanisation process. Additionally, urbanised African men showed the highest cortisol levels, low testosterone and high prolactin levels when compared to rural groups, which was indicative of the experience of stress (Malan *et al.*, 1996).

Henry *et al.* (1986) also indicated that a greater cortisol to testosterone ratio (C:T) is indicative of a tendency to a depressed state of mind, while a low cortisol to prolactin ratio (C:P) is more suggestive of the perception of the situation not being as stressful.

From existing literature it, therefore, seems that there is a clear interaction between stress experience and the secretion of stress hormones. However, the nature of this

interaction, as well as the possible role that specific coping styles might play in mediating the relationship between stress and endocrine activity in Africans, still warrants further research attention.

2.2.7 Questions arising from the literature

From the literature the following questions arise:

- Is there any association between coping styles i.e. AC or PC, stress hormone levels and the underlying stress hormone ratios in urban and rural Africans?
- Is there any association between coping styles, stress hormones and cardiovascular function? In other words, is a specific endocrine and cardiovascular pattern inherent to a specific coping style in urban and rural Africans?

2.2.8 Main aim, motivation and hypotheses

The main aim of this study was to examine the association between coping styles, stress hormones and cardiovascular function in rural and urban Africans.

The hypotheses were as follows:

- Urbanised subjects will show increased hypertension prevalence rates and a stress experiencing pattern with increased cortisol and prolactin and decreased testosterone levels. Urbanised subjects will show larger cortisol:prolactin, prolactin:testosterone and cortisol:testosterone ratios.
- PC urban subjects compared to AC urbanised subjects will show increased hypertension prevalence rates and an increased stress experiencing pattern with increased cortisol and prolactin and decreased testosterone levels with larger cortisol:prolactin, prolactin:testosterone and cortisol:testosterone ratios.
- PC urbanised subjects compared to PC rural subjects will show an increased cardiovascular risk, coupled with an increased stress experiencing pattern with increased cortisol and prolactin and decreased testosterone values together with larger cortisol:prolactin, prolactin:testosterone and cortisol:testosterone ratios.

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CHAPTER THREE

MANUSCRIPT

Instruction for authors: International Journal of Psychophysiology

- An abstract of not more 250 words should follow the title and should provide a concise description of the purpose of the report or article.
- The abstract should include a maximum of 8 keywords which reflect the index.
- Articles should be divided into sections headed by a caption (Introduction, Materials and Methods, Results, Discussion, Acknowledgments, References).
- Citation of literature references in the text should be given at the appropriate place by the author's name followed by year in parentheses. Should there be more than two authors, the first author's name should be followed by *et al.*
- All references in the text should be listed at the end of the paper on a separate page (double space according to the Harvard system).
- Tables of numerical data should be typed (double spaced) on a separate page, numbered in sequence in Arabic numerals, provided with a heading and referred to in the text as Table 1, Table 2.

CHAPTER 3

COPING, STRESS HORMONES AND CARDIOVASCULAR FUNCTION IN URBANISED AFRICANS

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ABSTRACT

The aim of this study is to investigate the role of coping styles in urbanized Africans on stress hormone and cardiovascular values. This cross-sectional comparative epidemiological study included 353 African men and women. Responses on the S-COPE-questionnaire divided the subjects into active (AC) and passive coping (PC) groups. Resting blood pressure variables were measured with the Riva-Rocci/Korotkoff and the FINApres method. Fasting, resting serum values of cortisol, prolactin and testosterone were determined using radio-immuno assays. ANCOVA's adjusting for age, BMI and lifestyle factors revealed that urbanized groups were younger, physically more active, consumed more alcohol with higher vascular activity (peripheral resistance (TPR), diastolic blood pressure (DBP), lower cardiac output (CO) and compliance (Cw), higher hypertension prevalence rates and prolactin values than rural subjects. Urbanized men showed smaller testosterone values, cortisol:prolactin and larger cortisol:testosterone ratios. Adding coping styles AC urbanized subjects indicated higer vascular activity, hypertension prevalence and a pattern similar to urbanized subjects regarding lifestyle factors, vascular activity and hypertension prevalence rates, prolactin and testosterone levels (men only). These factors were emphasized in AC men with higher heart rates and larger cortisol:prolactin ratio. AC and PC urbanized women showed increased alcohol consumption, only AC women showed higher hypertension prevalence rates compared to rural subjects. PC urbanized women revealed higher TPR and prolactin values, despite being more active than PC rural women. Urbanization in Africans can be associated with a poorer cardiovascular and stress endocrine pattern. This pattern is more accentuated in AC urbanized men, who indicated possible chronic stress experiencing.

Word count: 250

Keywords: urbanization, Africans, coping styles, stress hormones, cardiovascular variables

INTRODUCTION

Urbanization is an even more universal phenomenon now than ever before. It is estimated that by 2015, more than half of Africa's population will be living in urbanized areas (Tarver, 1994).

The effects of urbanization on the human body are quite substantial. In a study conducted on black Africans, hereafter only referred to as Africans, Vorster *et al.* (2000) found urbanization to be associated with the more negative spectrum of health effects i.e. a higher HIV infection prevalence, increases in obesity and other risk factors for non-communicable diseases. This finding was substantiated by Seedat (2000) who indicated that urbanization in sub-Saharan Africans was associated with increases in blood pressure (Cappucio, 1997) and higher hypertension prevalence rates.

The process of urbanization may lead to social disruption which in turn may lead to increased stress levels (Rahman *et al.*, 1997; Van Rooyen *et al.*, 2002). The stress theory, as described by Dressler and co-workers (2004) posits that there are certain social environmental stressors or demands to which an individual needs to adjust. Emphasis is placed on the social and psychological resources which a person has at his/her disposal to cope with these demands (Dressler *et al.*, 2004).

Malan and co-workers (2006) indicated that in Africans, the influence that stress has on the body is greatly dependent on the individual's ability to cope with the situation. This is further substantiated by Herd (1991) who indicated that the environment and previous experiences influence the behavioural reactions towards a stressor. In this behavioural reaction towards a stressor, three biological systems are of importance: neural, neuro-endocrine and neuroendocrine-immune reactions (McEwen, 2004). Neuro-endocrine activity, the so-called fight-or-flight reaction with activation of the peripheral sympathetic nervous system and the concomitant release of nor-epinephrine and epinephrine from the adrenal medulla, promote the necessary adaptation in the face of a stressor (Gerra *et al.*, 2001). The allostatic stress model posits that the body is able to maintain stability through change (McEwen, 2004). Prolonged exposure to stress may, however, lead to wear and tear of these controlling systems and the systems that first protected, now damages (McEwen, 2004). The two physiological systems that are especially of importance here, are the sympathetic adrenal medullary (SAM) and the hypothalamic pituitary adrenocortical (HPA) system. Helhammer *et al.* (2006) revealed that the chronic stimulation of the SAM may contribute to the development of cardiovascular disease, while the chronic stimulation of the HPA-axis may be associated with the metabolic changes that are related to an increased risk for cardiovascular disease.

Gerra and co-workers (2001) further hypothesized that these two systems may be two different coping systems activated by different thresholds and features of stress. The SAM is non-specific, activated by all types of stress, while the HPA-axis is more specific and activated by aggression/control and defence/active coping (AC) reactions (Gerra *et al.*, 2001; Henry *et al.,* 1986). Possible habituation of these systems in chronic stressful situations has been elucidated extensively (Gerra *et al.*, 2001; Ostrander *et al.*, 2006).

Christensen and Jensen (1995) indicated that dysfunctional coping styles seem to be the culprit concerning the harmful effects of stress in psychosocial stress/urbanization. It was indicated that an inadequate response to stress and the coping style may be more harmful to health than a hyperactive HPA-axis and concomitant hypersecretion of cortisol (Christensen and Jensen, 1995). This observation was supported by Malan *et al.* (2006) who revealed the association between a specific coping style and cardiovascular reactivity in urbanized Africans. Their findings showed that urbanized men and women revealed increased peripheral vascular α-adrenergic responses and greater hypertension prevalence rates when compared to rural men and women. Furthermore, these effects on peripheral vascular responses were found to be more prominent in the active coping (AC) subjects (Malan *et al.*, 2006).

Urbanization, as a psychosocial stressor can therefore elicit a certain stress experiencing pattern through the activation of neuro-endocrine systems. This stress pattern may, however, be greatly influenced by the individual's inherent ability to cope with the situation. Although the association between psychosocial stress, such as urbanization, coping styles and cardiovascular effects in Africans have been indicated (Malan *et al.*, 2006), few studies have been done to indicate the association between coping styles, the stress pattern and cardiovascular function in Africans. The stress pattern can be interpreted by respectively viewing the stress hormone levels of cortisol, testosterone and prolactin.

The hypotheses put forward are therefore, firstly, that urbanized subjects will show increased hypertension prevalence rates and a stress experiencing pattern with increased cortisol and prolactin and decreased testosterone values coupled with larger cortisol:prolactin (C:P), prolactin:testosterone (P:T) and cortisol:testosterone (C:T) ratios. Secondly, PC urbanized subjects compared to AC urbanized subjects will show increased hypertension prevalence rates and stress pattern with increased cortisol and prolactin and decreased testosterone values coupled with larger C:P, P:T and C:T ratios. And lastly, PC urbanized subjects compared to PC rural subjects will show an increased cardiovascular risk with hypertension prevalence rates, coupled with a stress experiencing pattern with increased

cortisol and prolactin and decreased testosterone values together with larger C:P, P:T and C:T ratios.

METHODS

Study design

This study is nested in the THUSA study (Transition and Health during Urbanization in South Africa), a cross-sectional comparative epidemiological project, which extended over a period of three years from 1996-1998. Magistrate districts or study sites (37) in the North-West Province were randomly selected representing urbanization status. In the weeks preceding the collection of data the sites were visited to obtain consent from tribal chiefs, government officials and the Department of Health to work in the area and to notify the local community of the visit of the research team. Fieldworkers were recruited and trained with regard to the recruitment of subjects and to supply information about the study to the subjects in their own language. The design and methodology of the THUSA study was published by Vorster *et al.* (2000).

Subjects

The subject sample included 821 recruited subjects, complying with the inclusion criteria of apparently healthy Setswana-speaking men and women, aged 16 – 70. The final sample size, after incomplete data sets were removed, was 353 urbanized and rural African men and women. Exclusion criteria were: pregnancy, lactation, casual visitors to the study site, body temperature above 37.5°C, inebriation, malnutrition, a history of/or current psychotherapy, acute or chronic medication (for infectious diseases including tuberculosis, hypertension, epilepsy and diabetes mellitus), incomplete data set and subjects scoring high on the utilization of active as well as passive coping styles.

Subjects were divided into two groups: (a) active coping (AC) and (b) passive coping (PC) men and women. This was done according to their responses to an adapted, translated and validated Setswana COPE Questionnaire (S-COPE) (Stapelberg, 1999), based on the original Carver COPE questionnaire (Carver *et al.*, 1989). These two groups (AC and PC) were then subdivided into rural-urbanized groups. The rural group included subjects living in tribal areas and in farmland dwellings with limited access to running water and electricity. The urbanized group included blue collar subjects living in the peri-urban fringe area of the greater metropolitan area and in established townships with full access to running water and electricity.

The Ethics committee of the PU for CHE approved the study. The study protocol conforms with the ethical guidelines of the Declaration of Helsinki (The World Medical Association Declaration of Helsinki, 2004). Informed consent was obtained from the subjects and the parents of under-aged adolescents.

Experimental procedure

Data collection was performed between 07:00 and 13:00 and each subject was involved for approximately two hours. Anticipation stress was minimized by first introducing the subjects to the experimental setup. After the introduction, subjects completed the demographic and psychological questionnaires individually in a structured interview format in their home language with the aid of trained African fieldworkers under the supervision of psychologists. Physical acitivity, alcohol consumption and smoking habits were also determined. Anthropometric measurements were taken by trained biokineticists with each subject standing upright with the head in the Frankfort plane. An anthropometrist measured height and weight of subjects in their underwear with calibrated instruments (Invicta Stadiometer, IP 1465, U.K.; Precision Health Scale, A & D Company, Japan; Holtain unstretchable metal tape). Each subject's weight and height were measured in their underwear. Subsequently, the subject was connected to the Finapres apparatus and blood pressure was recorded continuously whilst in a sitting position. After a resting period of at least 10 minutes, continuous blood pressure values of one minute were recorded. Blood pressure was regarded as resting when the systolic pressure did not change by more than 10 mmHg during the last minute of this period; otherwise the resting period was extended by a maximum of two minutes.

Measuring instruments and apparatus

Psychological questionnaires

The COPE Questionnaire (Carver *et al.*, 1989) is a well-known and established self reporting questionnaire with 53 items. Each item requires the subject to respond to statements regarding the utilization of specific coping strategies on a 4-point Likert scale, which varies from 1 (I usually don't) to 4 (I usually do). Higher scores indicate more frequent use of a particular coping strategy. The COPE was adapted for culture sensitivity and translated into Setswana. Stapelberg (1999) extracted an emic factor pattern from the original COPE through exploratory factor analysis (principal factors - maximum likelihood method of factor extraction with varimax rotation), indicating three clear and reliable subscales with

loadings >0.30 and eigenvalues of 6.53, 2.52 and 1.07, forming the S-COPE (Setswana-COPE). These subscales are (1) Active outreach-to-others, (2) Surrender and resignation and (3) Overt expression of distress. Cronbach alpha-reliability values varied from 0.85 to 0.70 for sub-scales in the S-COPE (Stapelberg, 1999). Ten items of the original COPE did not load significantly on one of the above subscales and were omitted to form the 43-item S-COPE. The S-COPE has construct validity, but convergent and discriminant validity have not been determined (Stapelberg, 1999; Wissing & Van Eeden, 2002).

The first subscale was taken to indicate active coping (an approach strategy with strong emphasis on engagement in active coping, actively seeking social support, commitment to tasks and controllability) (Cronbach alpha-reliability, 0.85). The second and third factors were combined for the purposes of this study to form a measurement of passive coping (an avoidance strategy with strong emphasis on appraisals of uncontrollability and expression of distress) (Cronbach alpha-reliability, 0.75). The active coping subscale included items such as "I talk to someone who could do something helpful about the problem", "I take direct action to deal with the problem" and "I try to find comfort in my religion". The passive coping subscale included items such as "I reduce the amount of effort I am putting into solving the problem", "I just give up trying to reach my goal", and "I become upset and am very aware of my feelings".

Participants in this study were grouped into active and passive coping groups according to the scores obtained on above subscales, by using median splits. Subjects presenting low and high scores on both subscales were excluded (men, N = 25; women, N = 33) and, therefore, only independent clear responders for active or passive coping were retained.

Blood pressure

Resting blood pressure was recorded by the method of Peñáz (Silke & McAuley, 1998). The FINger-Arterial-PRESsure device is non-invasive and monitors finger arterial blood pressure continuously (Silke & McAuley, 1998). Although the Finapres is not ideal for obtaining absolute values (Imholz et al., 1998), the resting values for systolic (SBP) and diastolic blood pressure (DBP) were also substantiated by means of the Riva-Rocci/Korotkoff method and subjects who had resting systolic ≥ 140mmHg and/or diastolic ≥ 90mmHg values were classified as hypertensive according to the WHO cut-off points (WHO, 2003). The Finapres device is not ideal for obtaining absolute values but it is validated and suitable for determining relative changes in comparative studies (Imholz *et al.*, 1998). Data were stored on magnetic tape by means of a Kyowa RTP-50A four-channel data recorder and digitized for further analysis by means of the Fast Modelflo software program. The Fast Modelflo three

element model integrates the subject's gender, body mass and stature and determines an age dependent aortic flow curve from the surface area beneath the pressure/volume curve and can, therefore, additionally calculate cardiac output (CO), total peripheral resistance (TPR) and arterial "Windkessel" compliance (Cw) of the arterial system (Wesseling *et al.*, 1993). When comparing Modelflo software-determined SV with invasive intra-arterial thermo-dilution-determined SV, the Modelflo software determined aortic flow from non-invasively determined finger pressure during orthostasis correctly (Harms *et al.*, 1999). This technique is thus an alternative to the invasive intra-arterial measurements, without the risks and ethical questions inherent to invasive measurements (Wesseling *et al.*, 1993).

Biochemical analyses

Fasting, resting blood samples (using a winged infusion set – 21G, with a heparin block – 0.5ml in 9ml normal saline) were obtained from the medial cubital vein or vena cephalica in the non-dominant arm by a registered nurse. All the biochemical analyses were executed in the same laboratory using standardized methods as follow: cortisol (CA-1529, CA-1549; Clinical Assays, GammaCoat) and testosterone (CA-1558; Clinical Assays, GammaCoat) were measured using 125I RIA kits of INCSTAR Corp. Minnesota USA. Prolactin (Catalog nr. 40-2165; Radio-isotopic Assay) were measured using the kit from the Nichols Institute Diagnostics, California USA. The intra-assay and inter-assay coefficients of variation for cortisol were respectively 7.7% and 9.8% and did not differ between men and women; for testosterone the intra-assay coefficients were 8.59% for men and 6.19% for women. The inter-assay coefficients were 13.64% and 6.86% respectively for men and women. Prolactin had an intra-assay coefficient of 3.5% and inter-assay coefficient of 6.9%, irrespective of gender.

Statistical analyses

All of the processed data were transferred to Microsoft Excel XP and further analysed by means of the software computer package STATISTICA, version 7 (Statsoft, 2004). The reliability of the S-COPE Questionnaire was determined by using the Cronbach alpha (α) reliability coefficient. A single 2 X 2 X 2 analysis of covariance (ANCOVA), adjusted for age, body mass index (BMI) and lifestyle factors (physical activity, smoking habits and alcohol consumption), on the main effects (coping style X gender X urbanization level) for significant differences between cardiovascular variables and endocrine/stress hormones was done. Significant interaction on the main effects led to subsequent 2 X 2 ANCOVAs and one-way ANCOVAs using Post-Hoc Tuckey HSD tests, adjusting for age, BMI and lifestyle factors.

Partial correlations, adjusting for age, BMI and lifestyle factors were thereafter computed to determine any significant underlying associations between cardiovascular and endocrine variables in each gender group.

Forward stepwise logistical regression analyses were performed with hypertension prevalence (HT) as the dependent variable and the coping style, urbanization level, cardiovascular and endocrine variables as the independent variables. Hypertension prevalence rates were determined by dividing subjects into groups (0/1) according to the WHO (2003) cut-off points for hypertension (SBP ≥ 140mmHg and/or DBP ≥ 90mmHg). All results were regarded as statistically significant when $p \leq 0.05$ and as highly significant when $p \leq 0.01$.

RESULTS

An initial 2 X 2 X 2 ANCOVA was performed to determine any interaction between the main effects, i.e. coping style, gender and urbanization level and each of the different variables. Significant interaction on the main effects showed for heart rate ($F(1,4.4329) = 787.35$; $p=0.04$) and testosterone ($F(1,3.596) = 0.2470$; $p=0.06$).

In Table 1, one way ANCOVAs show the characteristics of rural and urbanized subjects. Urbanized men showed higher vascular values with SBP ($F(1,223) = 310.8$; $p = 0.002$), heart rate ($F(1,223) = 186.06$; $p = 0.036$), DBP ($F(1,223) = 170,06$, $p = 0.000$), TPR ($F(1, 223) = 0.091$; $p = 0.006$), lower CO ($F(1,223) = 1.13$; $p = 0.036$) and compliance ($F(1,223) = 0.954$; $p = 0.001$) values., They showed higher hypertension prevalence rates ($p = 0.002$) despite their group being younger ($F(1,263) = 161.62$; $p = 0.011$), physically more active ($F(1,263) = 2.046$; $p = 0.000$) and consuming more alcohol ($F(1,263) = 0.543$; $p = 0.005$) compared to their rural counterparts Additionally, their endocrine pattern revealed higher prolactin ($F(1,187) = 0.05$; $p = 0.000$), lower testosterone ($F(1, 170) = 0.25$; $p = 0.002$) values coupled to a smaller C:P ($F(1,170) = 0.25$; $p = 0.001$) and a larger C:T ratio ($F(1,154) = 0.88$; $p = 0.044$).

In the urbanized women, the same trend regarding the cardiovascular pattern found in the urbanized men, was reflected in their higher DBP ($F(1,272) = 143.93$; $p = 0.000$), TPR ($F(1, 272) = 0.04$; $p = 0.000$), lower CO ($F(1, 272) = 0.74$; $p = 0.010$) and compliance ($F(1, 272) = 0.04$; $p = 0.000$) and higher hypertension prevalence rates ($p = 0.02$) when compared to rural women. The urbanized female group was younger ($F(1, 325) = 167.79$; $p = 0.000$), physically more active ($F(1, 325) = 1.112$; $p = 0.000$) but consumed more alcohol ($F(1, 325) =$

0.431; p = 0.000. When compared to rural women, urbanized women only revealed higher prolactin values (F(1, 246) = 0.08; p = 0.002).

Table 1: Comparing descriptive statistics, blood pressure and hypertension prevalence rates values (mean ± 95% CI) of rural versus urbanized subjects independent of age, BMI and life-style factors

RURAL	MEN (N = 88)			WOMEN (N = 142)		
* Age (years)	37.41	(34.1;40.74)		37.20	(34.9;39.493)	
* BMI (kg/m^2)	20.23	(19.64;20.80)		25.60	(24.55;26.62)	
* Alcohol (g/d)	2.72	(2.51;2.93)		2.63	(2.5;2.76)	
* Smoke (%)	1.50	(1.39;1.61)		1.91	(1.86;1.96)	
* PAI	3.34	(3.01;3.67)		2.74	(2.62;2.85)	
SBP (mmHg)	111.82	(108.63;11)5.01)		111.51	(108.79;114.23)	
DBP (mmHg)	71.19	(68.5; 73.88)		67.90	(66.00;69.81)	
HT	5			5.08		
URBANIZED	MEN (N = 142)		p values	WOMEN (N = 137)		p values
* Age (years)	32.96	(30.57;35.35)	**0.010**	30.85	(28.53;33.17)	**< 0.001**
* BMI (kg/m^2)	20.41	(19.9;20.92)	0.87	25.23	(24.18;26.27)	0.75
* Alcohol (g/d)	3.03	(2.93;3.12)	**0.004**	2.93	(2.84;3.02)	**< 0.001**
* Smoke (%)	1.41	(1.33;1.49)	0.26	1.91	(1.86;1.95)	0.66
* PAI	4.31	(4.01;4.60)	**0.001**	3.18	(2.95;3.41)	**< 0.001**
SBP (mmHg)	118.98	(115.7;122.27)	**0.002**	114.27	(111.02;111.52)	0.18
DBP (mmHg)	80.07	(77.88;82.25)	**0.001**	73.26	(71.13;75.39)	**< 0.001**
HT	22.33		**0.002**	13.40		**0.002**

5 % CI, 95 % confidence interval, N, number of subjects; BMI, body mass index (kg/m^2); Alcohol consumption (grams per day); Smoke (%), percentage of subjects smoking; PAI, physical activity index; SBP (mmHg), systolic blood pressure (millimeter mercury); DBP, diastolic blood pressure; HT, percentage hypertension prevalence rates where SBP ≥ 140mmHg and/or DBP, ≥ 90 mmHg (WHO, 2003);*, not adjusted. Values highlighted differed significantly, P ≤ 0.05. All other values are insignificant.

Table 2: Comparing cardiovascular and endocrine parameters (mean ± 95% CI) between rural-urbanized groups independent of age, BMI and lifestyle factors

RURAL	MEN (N = 46)			WOMEN (N = 108)		
HR (b/m)	71.73	(68;75)		81.29	(78;84)	
CO (l/min)	3.71	(3.3;4.1)		3.12	(2.9;3.3)	
TPR (mmHg s/ml)	1.67	(1.4;1.9)		1.82	(1.7;2.0)	
Cw (ml/mmHg)	1.74	(1.6;1.8)		1.21	(1.17;1.25)	
Prolactin (ng/ml)	0.84	(0.7;0.9)		0.82	(0.08;0.9)	
Cortisol (µg/dl)	1.19	(1.15;1.2)		1.05	(1.0;1.1)	
Testosterone (ng/ml)	0.71	(0.7;0.8)		0.76	(0.7;0.8)	
C:P	1.56	(1.4;1.7)		1.31	(1.0;1.64)	
C:T	1.83	(1.6;2.0)		2.22	(1.1;3.4)	
P:T	1.28	(1.1;1.4)		1.41	(0.9;1.9)	
URBANIZED	MEN (N = 102)		p values	WOMEN (N = 97)		pvalues
HR (b/m)	75.98	(73;79)	**0.036**	84.67	(82;87)	0.12
CO (l/min)	3.19	(3.0;3.4)	**0.036**	2.93	(2.7;3.1)	**0.010**
TPR (mmHg s/ml)	2.18	(2.0;2.4)	**0.006**	2.15	(2.0;2.3)	**< 0.001**
Cw (ml/mmHg)	1.56	(1.5;1.6)	**0.001**	1.11	(1.1;1.2)	**0.000**
Prolactin (ng/ml)	0.91	(0.9;1.0)	**< 0.001**	0.92	(0.9;1.0)	**0.002**
Cortisol (µg/dl)	1.19	(1.1;1.2)	0.78	1.04	(1.0;1.1)	0.36
Testosterone (ng/ml)	0.61	(0.55;0.64)	**0.002**	0.79	(0.7;0.8)	0.43
C:P	1.39	(1.3;1.5)	**0.001**	1.37	(1.2;1.5)	0.59
C:T	2.11	(1.9;2.3)	**0.044**	1.37	(1.1;1.7)	0.451
P:T	1.54	(1.3;1.8)	0.102	1.26	(1.1;1.5)	0.448

95 % CI, 95 % confidence interval, N, number of subjects; AC, active coping; PC, passive coping; HR, heart rate (beats/min); CO, cardiac output (l/min); TPR, total peripheral resistance (mmHg s/ml); Cw, Windkessel compliance (ml/mmHg); C:P, cortisol:prolactin; C:T, cortisol:testosterone; P:T, prolactin:testosterone. Values highlighted differed significantly, $p \leq 0.05$.

Subjects were subsequently compared according to their urbanization level, i.e. rural or urbanized and coping style i.e. AC or PC. No significant differences could, however, be found for any of the variables.

Thereafter, AC urbanized subjects were compared to AC rural subjects to determine any significant differences between the urbanization levels. The same procedure was performed for the PC subjects.

In Tables 3 and 4 the characteristics of AC urbanized subjects were compared to AC rural subjects. The same trend existed although more accentuated in AC subjects as been found in urbanized subjects regarding vascular activity, lifestyle factors (i.e. alcohol consumption and physical activity). AC urbanized men revealed that they consumed more alcohol ($F(1, 261) = 0.545$; $p = 0.037$) whilst reporting higher physical activity ($F(1, 261) = 2.053$; $p = 0.001$). Furthermore, AC urbanized men showed higher vascular blood pressure values with higher SBP ($F(1,221) = 313.08$; $p = 0.034$), DBP ($F(1,221) = 170,37$; $p = 0.001$), slower heart rate ($F(1, 221) = 182.14$; $p = 0.004$), lower compliance ($F(1,221) = 0.09$; $p = 0.002$) and higher hypertension prevalence rates ($p = 0.005$) in comparison with AC rural men.. On endocrine level, AC urbanized men showed higher prolactin ($F(1, 185) = 0.05$; $p = 0.016$) and lower testosterone ($F(1,168) = 0.04$; $p = 0.037$) values, coupled with a larger C:P ratio ($F(1, 168) = 0.256$; $p = 0.037$) compared to their rural counterparts.

AC urbanized women consumed more alcohol ($F(1, 323) = 0.431$; $p = 0.003$) and revealed higher vascular blood pressure values (SBP: ($F(1,196) = 295.35$; $p = 0.040$); DBP, lower compliance ($F(1, 196) = 0.04$; $p = 0.026$) and hypertension prevalence rates ($p = 0.03$) when compared to AC rural women.

When PC urbanized men were compared to PC rural men, PC urbanized men revealed higher DBP values ($F(1, 221) = 170.37$; $p = 0.004$) as well as greater physical activity ($F(1, 261) = 2.054$; $p = 0.008$). The PC urbanized women group was not only younger ($F(1, 323) = 167.31$; $p = 0.001$) and physically more active ($F(1, 323) = 1.108$; $p = 0.002$), but also consumed more alcohol ($F(1, 323) = 0.431$; $p = 0.001$) with higher TPR values ($1, 196) = 0.58$; $p = 0.029$) and higher prolactin values ($F(1, 196) = 0.08$; $p = 0.029$) than their rural counterparts.

Partial correlation coefficients and regression analyses were calculated, yet no significant correlations could be found.

Table 3: Comparing descriptive statistics and blood pressure values (mean ± 95% CI) between rural-urbanized subjects in active and passive coping groups independent of age, BMI and life-style factors

RURAL											
	AC						PC				
	MEN (N = 28)			WOMEN (N = 53)			MEN (18)			WOMEN (55)	
* Age (years)	38.75	(32.73;44.76)		34.6	(31.78;37.41)		37.55	(29.25;45.85)		37.145	(33.57;40.72)
* BMI (kg/m^2)	21.01	(19.84;22.17)		26.35	(24.58;28.08)		19.25	(17.96;20.55)		25.02	(23.32;26.73)
* Alcohol (g/d)	2.68	(2.33;3.03)		2.87	(2.63;3.09)		3.00	(2.52;3.48)		2.45	(2.28;2.62)
* Smoke (%)	1.5	(1.3;1.70)		1.96	(1.91;2.01)		1.55	(1.30;1.81)		1.945	(1.88;2.01)
* PAI	3.26	(2.67;3.83)		2.98	(2.78;3.18)		3.57	(2.69;4.45)		2.64	(2.46;2.83)
SBP (mmHg)	107.77	(102;113)		106.85	(103;111)		110.38	(104;117)		113.85	(108;118)
DBP (mmHg)	65.59	(61;70)		65.93	(63;69)		71.21	(66;77)		69.15	(66;72)
HT (%)	5.56			0.00			4.17			10.17	

URBANIZED												
	AC						PC					
	MEN (N = 47)			WOMEN (N = 46)		p-values	MEN (55)			WOMEN (51)		
* Age (years)	28.23	(33.75;42.7)	0.38	34.28	(29.77;38.78)	0.13	30.52	(27.06;33.99)	0.39	31.37	(27.22;35.52)	**0.001**
* BMI (kg/m^2)	19.98	(19.19;20.7)	0.84	26.42	(24.36;28.50)	0.99	21.15	(20.3;22.00)	0.94	24.92	(23.14;26.72)	0.985
* Alcohol (g/d)	3.00	(2.8;3.19)	**0.037**	2.97	(2.8;3.14)	**0.003**	3.00	(2.85;3.14)	0.62	2.84	(2.71;2.970	**0.001**
* Smoke (%)	1.38	(1.2;1.5)	0.99	1.91	(1.8;1.99)	0.99	1.36	(1.23;1.49)	0.52	1.82	(1.72;1.93)	0.981
* PAI	4.14	(3.56;4.7)	**0.001**	3.42	(2.96;3.88)	0.17	4.62	(4.13;5.11)	**0.008**	3.06	(2.66;3.46)	**0.002**
SBP (mmHg)	121.15	(115;127)	**0.034**	116.05	(111;121)	**0.04**	116.04	(111;121)	0.35	115.02	(109;121)	0.985
DBP (mmHg)	82.52	(78;87)	**0.001**	72.28	(69;75)	**0.046**	78.11	(75;82)	**0.044**	74.39	(70;79)	0.118
HT (%)	29.79		**0.005**	8.7		**0.03**	19.07		0.13	17.65		0.195

95 % CI, 95 % confidence interval, AC, active coping; PC, passive coping; N, number of subjects; BMI, body mass index (kg/m^2); Alcohol consumption (gram per day); Smoke (%), percentage of subjects smoking; PAI, physical activity index; SBP (mmHg), systolic blood pressure (millimeter mercury); DBP, diastolic blood pressure; HT, percentage hypertension prevalence rates where SBP ≥ 140mmHg and/or DBP, ≥ 90 mmHg (WHO, 2003);*, not adjusted. Values highlighted differed significantly, P ≤ 0.05. All other values are insignificant.

Table 4: Comparing cardiovascular and endocrine parameters (mean ± 95% CI) between rural-urbanized subjects in active and passive coping groups independent of age, BMI and lifestyle factors

RURAL	AC				PC			
-	MEN (N = 28)		WOMEN (N53)		MEN (N = 18)		WOMEN (N = 55)	
HR (b/m)	77.22	(73.35;81.09)	82.13	(79.45;84.82)	73.90	(71.03;76.78)	85.86	(82.50;89.21)
CO (l/min)	3.13	(2.59;3.38)	2.90	(2.66;3.14)	3.19	(2.95;3.44)	2.77	(2.53;3.00)
TPR (mmHg s/ml)	1.54	(1.26;1.8)	1.76	(1.58;1.95)	1.84	(1.44;2.24)	1.88	(1.67;2.1)
Cw (ml/mmHg)	1.78	(1.69;1.88)	1.25	(1.19;1.30)	1.66	(1.49;1.84)	1.17	(1.12;1.22)
Prolactin (ng/ml)	0.82	(0.7;0.9)	0.85	(0.77;0.92)	0.87	(0.75;0.98)	0.78	(0.7;0.9)
Cortisol (µg/dl)	1.17	(1.1;1.2)	1.06	(1.01;1.11)	1.22	(1.2;1.3)	1.04	(0.98;1.08)
Testosterone (ng/ml)	0.73	(0.66;0.79)	0.82	(0.74:0.91)	0.67	(0.57;0.78)	0.69	(0.6;0.8)
C:P	1.44	(1.27;1.60)	1.41	(1.17;1.65)	1.35	(1.25;1.45)	1.29	(1.04;1.54)
C:T	2.02	(1.82;2.21)	1.63	(1.34;1.91)	2.18	(1.82;2.54)	1.69	(0.43;2.94)
P:T	1.54	(1.36;1.72)	1.38	(1.09;1.67)	1.52	(1.16;1.88)	1.51	(0.74:2.29)

URBANIZED	AC						PC					
	MEN (N = 47)		p values	WOMEN (N = 46)		p values	MEN (N = 55)		p values	WOMEN (N = 51)		p values
HR (b/m)	68.73	(65.32;72.14)	**0.004**	79.83	(76.87;82.80)	0.34	75.34	(70.84;79.83)	0.95	82.77	(79.05;86.48)	0.78
CO (l/min)	3.50	(3.20;3.80)	0.26	3.05	(2.86;3.24)	0.97	3.54	(3.10;3.97)	0.37	3.14	(2.92;3.37)	0.08
TPR (mmHg s/ml)	2.20	(1.98;2.54)	0.15	1.96	(1.79;2.15)	0.55	2.11	(1.79;2.42)	0.24	2.31	(2.05;2.58)	**0.016**
Cw (ml/mmHg)	1.48	(1.39;1.58)	**0.002**	1.14	(1.08;1.19)	**0.026**	1.67	(1.54;1.72)	0.58	1.17	(1.12;1.22)	0.06
Prolactin (ng/ml)	0.90	(0.8;0.97)	**0.016**	0.90	(0.79;1.0)	0.83	0.92	(0.8;0.98)	0.20	0.94	(0.85;1.0)	**0.029**
Cortisol (µg/dl)	1.19	(1.1;1.3)	0.95	1.05	(0.99;1.12)	0.99	1.19	(1.13;1.25)	0.99	1.02	(0.96;1.07)	0.97
Testosterone (ng/ml)	0.62	(0.58;0.66)	**0.018**	0.77	(0.69;0.84)	0.80	0.59	(0.54;0.65)	0.60	0.81	(0.74:0.88)	0.21
C:P	1.73	(1.35;1.78)	**0.037**	1.40	(1.19;1.61)	1.00	1.54	(1.35;1.74)	0.32	1.24	(0.65;1.84)	0.97
C:T	1.71	(1.46;1.96)	0.40	1.48	(1.13:1.83)	0.99	1.92	(1.68;2.16)	0.70	2.84	(0.74:4.94)	0.15
P:T	1.16	(1.00;1.35)	0.99	1.14	(0.95:1.34)	0.91	1.34	(1.09;1.58)	0.08	1.63	(0.72:2.55)	0.50

95 % CI, 95 % confidence interval, N, number of subjects; AC, active coping; PC, passive coping; HR, heart rate (beats/min); CO, cardiac output (l/min); TPR, total peripheral resistance (mmHg s/ml); Cw, Windkessel compliance (ml/mmHg); C:P, cortisol:prolactin; C:T, cortisol:testosterone; P:T, prolactin:testosterone. Values highlighted differed significantly, $p \leq 0.05$.)

DISCUSSION

The aim of this study was to determine whether the stress hormone levels and cardiovascular function of urbanized Africans who employ a specific coping style differ in any way from their rural counterparts. A pattern of a possible greater stress experience was identified in urbanized Africans who revealed increased vascular blood pressure values and higher hypertension prevalence rates accompanied by increased alcohol consumption despite their group being younger and more active than the rural African group. This is in accordance with results from both the Malan and co-workers' (1996) and the Huisman and co-workers' (2002) studies which indicated that urbanized Africans experience greater levels of stress in comparison with their rural counterparts. Additionally, our findings are supported by other studies which revealed that urbanized Africans show greater hypertension prevalence rates as well as vascular responsiveness than rural Africans (Malan *et al.*, 2006; Seedat *et al.*, 2000).

Urbanized men revealed a cardiovascular pattern of increased peripheral vascular activity with increased TPR values and with lower compliance values. Furthermore, urbanized men revealed a greater stress experiencing pattern with larger heart rate, prolactin, smaller testosterone values, coupled with a smaller C:P ratio and a larger C:T ratio. The smaller C:P ratio may, according to Henry *et al.* (1986) be indicative of a feeling of control. This however, is in contradiction to their larger C:T ratio, which in turn indicates a sign of stress and a depressed state of mind (Henry *et al.*, 1986). A possible explanation for these seemingly contradictory results is habituation of the HPA-axis during chronic exposure to stressful situations i.e. as during urbanization and in passive coping (Heim *et al.*, 2000; Ostrander *et al.*, 2006; Lundberg, 2005). As prolactin acts as a cortisol antangonist (Gala, 1990) this could further enforce the idea of a desensitized HPA-axis.

Urbanized women also revealed a cardiovascular pattern of increased vascular or α-adrenergic activity of increased TPR and decreased CO and Cw as posited by Fray and Douglas (1993). Furthermore, urbanized women showed higher prolactin values in comparison with rural women, which tell of a greater stress experience and a decreased feeling of control (Henry *et al.* 1992). These results support the idea that urbanized women experience greater stress compared to rural women.

However, the role that coping styles play in this regard is still unclear. Adding coping styles it was clear that the pattern of more cardiovascular and stress

experiencing risk factors indicated by increased alcohol consumption, higher vascular activity and hypertension prevalence rates was evident in the AC subjects especially the AC urbanized men. Their stress endocrine pattern of larger prolactin and smaller testosterone values coupled with a larger C:P ratio could possibly be indicative of higher levels of stress experiencing. (Dostal *et al.*, 2003; Malan *et al.*, 1996).

When compared to AC rural women, the AC urbanized women revealed increased vascular blood pressure values and lower compliance, as well as increased alcohol consumption. Despite of no changes in their endocrine values it is the AC urbanized women compared to their rural counterparts who revealed the poorest cardiovascular and stress experiencing pattern with increased hypertension prevalence rates This may be indicative that urbanized women experience greater exposure to stressful situations in comparison to rural women, which is in accordance to the findings of Vorster *et al.* (2000). As the PC urbanized women also revealed high vascular activity with additionally larger prolactin values it could be that they experience the situation as stressful.

As the cardiovascular and endocrine pattern, especially in AC urbanized men, indicated a vascular response and greater stress experiencing, it seems reasonable to state that their responses resembled a PC style's physiological response according to Henry's model (1986). Resembling a PC style implies high prolactin values and low testosterone values as indicators of a loss of control over a situation (Henry, 1992). Numerous studies have indicated that the social environment, be it urbanized or rural, is of great importance to the subject's physiological well-being, especially in Africans (Malan *et al.*, 2006; Schutte *et al.*, 2004; Van Rooyen *et al.*, 2000). This study is thus in accordance with the results found in Malan *et al.*'s (2006) study, which indicated that urbanization and especially an AC coping style can be associated with higher blood pressure values, vascular responsiveness and hypertension prevalence rates, especially in men.

The resemblance between an AC subject and a PC subjects' physiological responses as explained above, can be supported by the observable fact that there were no statistical differences to report between AC and PC urbanized subjects or AC and PC rural subjects. A possible explanation for this could be that Africans, within the urbanization process, are moving away from their collectivistic culture towards the more westernized and individualistic culture (Chang, 1996) with its concomitant greater stress experience. This situation may lead to possible

habituation or desensitization of the control systems in the long run, whereby an AC subject will reflect a PC subject's physiological pattern.

In conclusion, it has become evident that urbanization as a psychosocial stressor in Africans leads to a variety of effects on the body. Urbanization in Africans can be associated with poorer cardiovascular and stress experiencing patterns in comparison with rural subjects. This pattern is more accentuated in AC subjects who indicated a greater cardiovascular risk compared to AC rural subjects by revealing increased vascular blood pressure values and increased hypertension prevalence rates together with decreased compliance. This pattern is accentuated in especially AC men, who revealed possible chronic stress experiencing with a larger cortisol:prolactin ratio.

The first hypothesis is partially supported, as urbanized men did indicate increased hypertension prevalence rates and a stress pattern of increased prolactin and decreased testosterone values coupled with a smaller C:P ratio and larger C:T ratio. Urbanized women revealed increased hypertension prevalence rates and increased prolactin values. The second hypothesis is rejected, as there were no significant differences between urbanized and rural AC/PC men and women. And lastly, the third hypothesis is partially supported as only PC urbanized women showed an increased cardiovascular risk and stress experiencing pattern compared to PC rural women with increased TPR values,and increased prolactin values compared to PC rural women. It was, however, AC urbanized men and women who indicated a greater cardiovascular risk compared to AC rural men and women by revealing increased vascular blood pressure values and increased hypertension prevalence rates together with decreased compliance.

LIMITATIONS AND RECOMMENDATIONS

The study was limited by the subjective nature of the coping questionnaire. While it is completely validated (Stapelberg, 1999), the grouping of subjects as purely AC or PC might be an oversimplification of a complex process. It is necessary to investigate the distinction between AC and PC especially within the specific African context, as the possibility exists that both types of coping styles could be both adaptive and maladaptive depending on the situation and cultural context an individual finds him-/herself in.

Since habituation of biological systems is known to occur, the duration of stay in the specific urbanization level is also important to determine, as it could be indicative of the duration of exposure to the stress associated with the process of urbanization.

It is therefore, recommended that studies on the holistic view of individuals be continued to gain a more informed understanding of the intricate balance between the physiological, psychological and social aspects of a person as a human being and to get a step closer to the concept of health, as defined by the WHO (1948).

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CHAPTER 4

GENERAL FINDINGS AND CONCLUSIONS

4.1 INTRODUCTION

A summary of the main findings from the article reported in this dissertation is given. The results will be discussed, interpreted, elucidated and compared to relevant literature reviewed in Chapter 1. Conclusions are subsequently drawn and recommendations are made to researchers investigating coping and stress hormones in urbanized Africans, with the aim of ultimately identifying possible risk markers in the development of cardiovascular dysfunction and concomitant hypertension.

4.2 SUMMARY OF THE MAIN FINDINGS

The significant findings of the article reported in this dissertation were:

4.2.1 Coping, stress hormones and cardiovascular function in urbanised Africans

The main aim of this study was to examine the association between coping styles, stress hormones and cardiovascular function in rural and urbanised Africans. From the results the first hypothesis is partially accepted as urbanised men did indicate increased hypertension prevalence rates and a stress pattern of increased prolactin and decreased testosterone values coupled with a smaller C:P ratio and larger C:T ratio. Furthermore, urbanised men revealed a cardiovascular pattern characterised by increased peripheral vascular responses with increased heart rate and TPR coupled with decreased CO and Cw. Urbanised women, however, only revealed increased hypertension prevalence rates and increased prolactin values coupled with a cardiovascular pattern of increased TPR and decreased CO and Cw. The second hypothesis is rejected as there were no significant differences between urbanised AC and PC men and women. Additionally, there were no significant differences between rural AC and PC men and women. And lastly, the third hypothesis is partially accepted as PC urbanised women did show an increased cardiovascular risk compared to PC rural women with increased TPR values, hypertension prevalence rates and an increased stress experiencing with increased prolactin values compared to PC rural women. It was, however, AC urbanised men and women who indicated greater cardiovascular risk compared to AC rural men and women by revealing increased vascular blood pressure values and increased hypertension prevalence rates together with decreased compliance.

4.3 COMPARISON TO RELEVANT LITERATURE

When the results from this study are compared with existing literature (as presented in Chapter 2), it is evident that certain findings confirmed those found in the literature.

The endocrine pattern of urbanised groups revealed higher prolactin and lower testosterone values. This is in accordance with the results of the Huisman *et al.* (2002) study on Africans, which indicated that this specific pattern could be indicative of a greater stress experience. According to Malan *et al.* (2006) habituation or dissociation of coping styles in urbanised subjects could be responsible for this perceived pattern. This study is, furthermore, in accordance with results from Malan *et al*'s (2006) study which indicated that urbanisation and an AC style in men and women but especially in men is associated with higher blood pressure values, vascular responsiveness and higher hypertension prevalence rates.

Contradictory findings from this study were that AC subjects do not appear to be experiencing greater control over a stressful situation compared to his PC counterpart within the same urbanisation level. No significant differences could be found between coping styles within the same urbanisation level. Contrary to this, Henry *et al.* (1986) indicated that a AC style is associated with control over the situation and associated increases in testosterone levels, while PC is associated with decreases in testosterone. Control is also associated with increased levels of prolactin, as described by Henry's animal model (1992) as prolactin levels increases as the animal loses control over the situation. In this study, it was the AC urbanised subject who revealed the poorest endocrine pattern with increased prolactin and decreased testosterone values. An explanation of these findings is in accordance with the Malan and co-workers study (2006) which indicated that Africans may show dissociation of habituation of physiological systems with chronic exposure to stress (Hellhammer *et al.*, 2005).

Numerous studies have indicated the increased hypertension prevalence rates for urbanised Africans (Huisman *et al.*, 2002; Van Rooyen *et al.*, 2002) and are confirmed by this study. Certain results in this study were, however, not known from the literature. These include the stress hormone ratios and the association it may have with cardiovascular function in Africans. From the literature, it is also not known what influence coping styles might have on these ratios. Findings of this study revealed that urbanisation in Africans is associated with a smaller C:P ratio and a larger C:T ratio. When coping styles are added, it was actually the AC urbanised men

who revealed a larger C:P ratio. This is contradictory to Henry *et al.* (1986) study who associated a larger C:P ratio with an experience of the situation as stressful.

Novel findings from this study that could add value to the available literature are the results that indicated that urbanisation in Africans could be associated with higher vascular blood pressure values, hypertension prevalence rates and peripheral vascular responses coupled with a stress pattern indicated by increased prolactin and decreased testosterone values. In urbanised men, this stress experiencing pattern additionally indicated a larger C:T and smaller C:P ratio. Adding coping styles in an urbanised environment it was actually the AC subjects especially AC men who revealed a higher cardiovascular and endocrine stress experiencing risk .

Discrepancies between the results of this study and the literature might be explained by racial differences, as some of the research in the literature refers to Caucasian or African-American populations. It must further be emphasized that literature regarding the interaction between coping styles, stress hormones and cardiovascular function in Africans, is lacking to a large extent.

4.4 DISCUSSION AND FINDINGS

4.4.1 Chance and confounding

Chance: It is important to reflect on some of the important factors that might have affected the results. There are some methodological issues that could have caused weaknesses in the study, and therefore, might have influenced the different outcomes.

The number of subjects included in this study could be questioned, especially the number of subjects for the AC rural men (n=18). A power analysis using blood pressure values determined that 15 subjects would have been sufficient.

Furthermore, the possibility of chance should be taken into account. Through the use of partial correlations, it was determined that one out of twenty significant correlations might be due to chance.

Confounders: HIV-status, socio-economic status (education and social support) and psychological characteristics could have influenced the results by causing over or under estimation of the associations between cardiovascular function and the various variables studied. By adjusting statistically for age, body mass index and lifestyle

factors (physical activity, smoking and alcohol consumption) these possible confounders could be successfully addressed.

It was necessary to interpret all the statistical results from a physiological perspective, which implies that all statistical significance does not necessarily indicate a physiological significance.

4.4.2 Weaknesses of the study

Weaknesses of this study included:

a) The duration of stay within urbanised areas was not known, which could influence adaptation/habituation of physiological processes.

b) Resting values were taken for each of the stress hormones, which could be better controlled by controlling the time of day of sampling, as hormone levels may vary substantially during the day. Cortisol values are difficult to determine in women, as contraceptive use greatly influences the availability of cortisol within the serum.

c) The subjects were divided into only AC and PC groups according to results from the COPE-questionnaire. A PC style in the traditional, collectivistic African context might not be as negative or dysfunctional as within the Westernized individualistic culture. This may, therefore, be an over-simplification of a very complex phenomenon, since in certain situations a PC approach might be more constructive than an AC approach.

4.4.3 Discussion of main findings

Numerous studies have been conducted to elucidate the specific cardiovascular effects urbanisation, as a psychosocial stressor, has on Africans (Malan, *et al.*, 2006; Malan *et al.*, 1992; Malan, *et al.*, 1996).Opie & Seedat, 2005; Seedat, 2000, 1999; Van Rooyen, *et al.*, 2000). Psychosocial stress, such as that caused by urbanisation is also known to elicit an endocrine pattern, which is greatly altered by the perception of control over the situation or the specific coping style (Matheson & Cole, 2005; Salvador, 2005; Henry, 1986). However, the effect that these coping styles could have on the stress hormone levels and concomitant cardiovascular effects are still unclear.

The main focus of this study was to investigate and elucidate the association between copings styles, stress hormones and cardiovascular function in Africans. Although the findings cannot be generalized to the whole African population, it could serve as a foundation for future studies.

The results from this study are presented schematically in Figure 4.1. It is evident that urbanisation, as a psychosocial stressor, elicits a specific endocrine and cardiovascular pattern with higher blood pressure values, TPR and lower CO and Cw in the urbanised men and women. Furthermore, the urbanised individuals revealed increased prolactin values with the urbanised men showing lower testosterone values compared to their rural counterparts. Additionally, the urbanised men revealed significant differences regarding the stress hormone ratios with smaller C:P and larger C:T than rural men. When adding coping styles, it was actually the urbanised AC men who revealed the poorer cardiovascular and endocrine pattern, with higher blood pressure values as well as higher prolactin and lower testosterone coupled to a larger C:P ratio. It therefore seems that it is indeed the AC urbanised men who reveal a greater stress experience and greater loss of control.

The AC urbanised women revealed a cardiovascular pattern with higher blood pressure values, higher hypertension prevalence and lower Cw than the AC rural women. However, no differences between the endocrine parameters were found. In the comparison between PC urbanised and rural subjects men and women indicated higher vascular activity but only women had a perception of stress experiencing with their higher prolactin values

4.5 CONCLUSION

Urbanisation in Africans could be associated with increased vascular blood pressure values, hypertension prevalence rates and a cardiovascular pattern of increased TPR and decreased Cw and CO. Regarding the stress experiencing pattern, urbanisation in men could be associated with increased prolactin and decreased testosterone values, coupled with smaller C:P and larger C:T ratios. The cardiovascular risk in AC subjects is emphasized in men and women but especially in men with their additional endocrine stress experiencing pattern. with a larger C:P ratio, which could be indicative of a feeling of less control over the situation. PC in urbanised women although associated with increased TPR and increased prolactin values did not show higher hypertension prevalence rates than their rural and urbanised counterparts.

Certain coping styles could therefore be associated with a poorer cardiovascular and stress experiencing pattern in urbanised Africans.

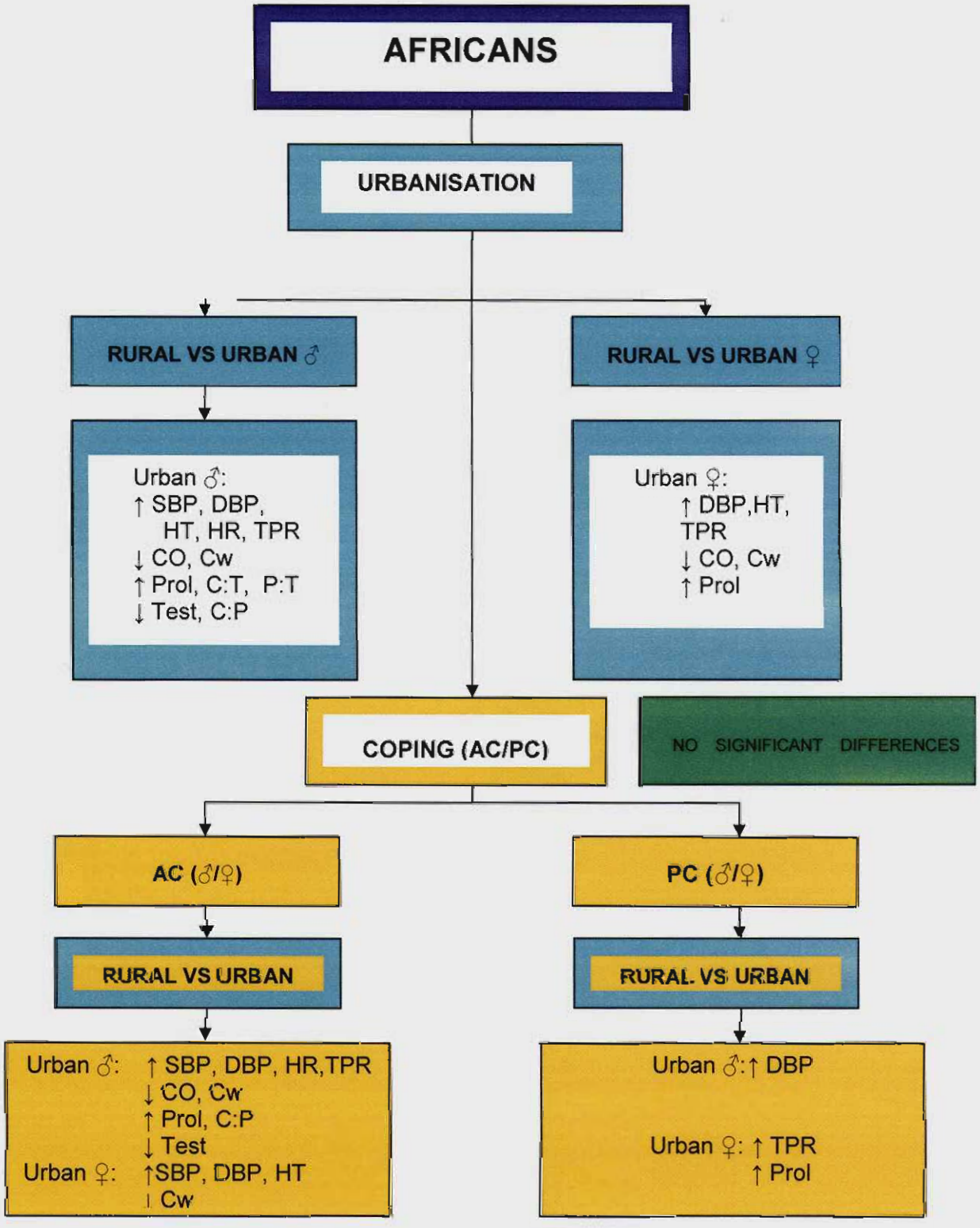

Figure 4.1: Schematic representation of the significant differences between coping styles in Africans and stress hormones and cardiovascular data. Where: ↑, increase; ↓, decrease; AC, active coping; PC, passive coping; ♂, men; ♀, women, SBP, systolic blood pressure, DBP, diastolic blood pressure; HT, hypertension prevalence; HR, heart rate; TPR, total peripheral resistance; CO, cardiac output; Cw, Windkessel compliance; C:P, cortisol:prolactin; C:T, cortisol:testosterone; P:T prolactin:testosterone

4.6 RECOMMENDATIONS

The following recommendations are proposed for future studies:

- Although an AC style compared to a PC style is not associated with an improved cardiovascular and endocrine pattern, one must bear in mind that this subject group might have a different perception of coping. A traditional PC style in their collectivistic culture might not be as destructive as seen by a more westernised society. A closer look at the specific underlying coping strategies employed by Africans is thus necessary to understand the dynamics involved better.

- It could be interesting to focus on emotion-focused and problem-focused coping in future and the concomitant cardiovascular and endocrine effects this classification might hold, in Africans.

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