Walter Sisulu University

PROFESSORIAL INAUGURAL LECTURE

22 MAY 2012
15H00
MTHATHA HEALTH RESOURCE CENTRE

Topic: Cardiovascular disease and metabolic syndrome in health transition and evidence-based medicine: a perspective from Africa.

Professor B Longo-Mbenza
Professor of Cardiology and
Research Professor
Faculty of Health Sciences
Walter Sisulu University
Eastern Cape
South Africa

Auditorium, Mthatha Health Resource Centre, Mthatha, Eastern Cape

A developmental university...
• technological • scientific • innovative • responsive

WSU
Walter Sisulu University

WALTER SISULU UNIVERSITY
PROFESSOR OF CARDIOLOGY AND
RESEARCH PROFESSOR
FACULTY OF HEALTH SCIENCES

TOPIC
CARDIOVASCULAR DISEASE AND METABOLIC SYNDROME IN HEALTH TRANSITION AND EVIDENCE-BASED MEDICINE: A PERSPECTIVE FROM AFRICA

BY
B LONGO-MBENZA

DATE: 22 MAY 2012

VENUE: MTHATHA HEALTH RESOURCE CENTRE AUDITORIUM

The Administrator,
Deputy Vice Chancellor, Academic and Research,
Deputy Vice Chancellor, Planning, Quality Assurance and Development,
Registrar,
- Executive Deans of Faculties,
Director: Research Development,
Director: Postgraduate Studies,
Heads of departments,
Members of the University Community and Colleagues,
- Distinguished Guests,
Ladies and Gentlemen,
Friends,
Students and Comrades,

I am pleased to stand here before you this afternoon to deliver this Professorial Inaugural Lecture on behalf of the faculty of Health Sciences. The word "Physician" is not mentioned in the book of wisdom, the Bible. Neither the word Physician nor the word Doctor "doctus" "IATROS", is used in the Bible. This process will NOT be perceptible to the five senses, nor will it be understood by those who are not spiritual. It will not be perceptible nor understood by doctors, scientists, philosophers, scholars and religious leaders.

In the Holy Bible, Jeremiah 8:22 says: "Is there no balm in Gilead? Is there no physician there? Why then is there no healing of my people? In a rather humorous vein, one may ask: Is there no medicine in poor Eastern Cape Province of South Africa? Is there no physician there? Why then poor health among the sons and daughters of this province?

Daniel 1:4 talks about "young men in whom was no blemish, but handsome, and skilful in all wisdom and gifted in knowledge and understanding science and such as had ability in them to stand in the king's palace and whom they might teach the learning and the language of the Chaldeans."

I do stand to present strategic mandate, Vision, Mission and Core values of WSU through the present Professorial Inaugural Lecture. In this lecture, I attempt to converse in a broad sense about physiology, pathophysiology, mathematics, physics, molecular biology, cardiology, epidemiology and ecology.

I became an orphan at age 3 when my father died. He was an assistant physician during the Belgian colonial era in Congo.

I was born in Congo, which was under the Belgian colonization till 1960.
I take this time to thank the WSU management for appointing me as Research Champion Professor in order to reinvigorate research since 2009.

INTRODUCTION

I embrace all the scientific, clinical and public health publications that address "Cardiovascular Disease (CVD) and Metabolic Syndrome in Health Transition and Evidence-based Medicine: a perspective from Africa".

The CVD pandemic worldwide presents a true challenge today with a high health burden that is only expected to rise. I address the causes and prevention of CVD, as well as CVD rehabilitation and physiology. As a member of the American Heart Association and European Society of cardiology, I practice under the level of evidence and the strength of recommendation of particular treatment options, as outlined in the tables below.

Classes of recommendations

Class I	Evidence and/ or general agreement that a given treatment is beneficial
Class II	Conflicting evidence and / or a divergence of opinion about the efficacy of the treatment
Class III	Evidence or general agreement that the given treatment is not useful

Level of evidence

Level of evidence A	Data derived from multiple randomized clinical trials or Meta-analyses
Level of evidence B	Data derived from a single randomized clinical trials or large non-randomized studies
Level of evidence C	Consensus of opinion of the expert and/of small studies, retrospective studies registries

Organization of the Inaugural Lecture

This lecture consists of the definitions, the aims, the Data sources, the data selection, the Data extraction and synthesis and the conclusion.

DEFINITIONS

CVD includes diseases such as atherosclerotic, particularly coronary heart disease (heart attack, heart failure, angina), stroke (brain attack, cerebrovascular accident when the blood flow to the brain is interrupted by blood clot), and peripheral artery.

The life-long impact of heart and other chronic diseases and their traditional risk factors.

PREVENTION WHOLE POPULATION	PREVENTION WHOLE POPULATION	DIAGNOSIS, COST-EFFECTIVE MANAGEMENT	
Before birth • Genes • In the womb **Environment** • Poverty • Cultural Factors • Political Factors • Urbanization • Climate change	**Unhealthy lifestyle** • Unhealthy diet • Tobacco use • Excessive alcohol • Physical inactivity • Stress	**Modifiable risk factors** • Obesity • Hypertension • Tobacco addition • Diabetes • High blood cholesterol legs other fats	**Morbidity And Mortality** Blood vessels Heart Brain Kidneys Lungs Eyes

The new risk factors and biomarkers of CDV are now emerging through structural and biochemical parameters:

- Low birth weight < 2500g;
- Inflammation: fibrinogen
- Infection: HIV, Helicobacter pylori;
- Metabolic syndrome/ Insulin resistance;
- Uric acid;
- renal disease;
- stress
- oxidative stress/ antioxidant.

The metabolic syndrome is characterized by a clustering of CVD, metabolic, inflammatory and haemostatic risk factors.

The scope of the problem: past, present and future.

Africa is caught in the middle of disruptive epidemiologic, demographic, economic and nutritional transitions (Health transition). In contrast to the decrease in industrialized countries of non communicable diseases (NCD) such as CVD, there is a surprising rise in the morbidity and the mortality of CVD in Africa.

Our knowledge of the disease burden components of Africa populations was fragmentary in the past. Historically, the communicable diseases (CD with infectious and perinatal issues) have been emphasized. But, as some populations have undergone socio-economic, political changes (independences, end of apartheid), vital statistics have described a change in the pattern of disease. The picture is of a decline in infectious diseases and an increase in CVD with hypertension, diabetes mellitus and renal failure.

OBJECTIVE

I focus today on the emergence of CVD, and the metabolic syndrome as the paradigmatic example. Consequently, the lecture aims at providing the prevalence, the incidence, the risk factors, the predictors, the paradoxes, and the prevention of CVD.

DATA SOURCES

The criticism of Literature review was obtained through PubMed, PMC, Schoolar google, Honours, MSc, PhD, Proceedings and monographs search.

Published articles, Posters and abstracts from national, regional and international conferences were reviewed. The abstracts and complete articles relevant to the Professorial Inaugural Lecture were selected, read and analyzed to extract information.

The previous WSU Inaugural Lecture series helped me to consolidate my multidisciplinary and interdisciplinary Pedagogy with Problem-based Learning.

METHODS

Despite the official new approach touting improvement in health indicators, current trends jeopardize population health, and hospital and population based surveys in Africa were related to methods with previous moderate and recent high levels of Evidence-based Medicine.

Inclusion criteria ensured that results maximized relevance, validity, and reliability while producing comparable in developed nations, emerging economic nations and African countries.

The World Health Organization (WHO) STEPwise approach to chronic disease risk factors surveillance helped me to use structured and standardized methods with comparable valid data worldwide for prevention of basis, objectives of surveillance, risk factor definition, major behavioural factors and rationale for inclusion of core risk factor.

The following diagrams illustrate the general concept of the Stepwise approach.

Risk factors surveillance.

Stroke surveillance.

Global school-based student health survey.

RESULTS

Extensive findings are presented below.

The metabolic syndrome in Africa

Contrary to the past, metabolic syndrome is no longer rare in Africa. The prevalence is increasing, and it tends to increase with age.

HIV/AIDS

Prevalence of metabolic syndrome by NCEP criteria was 26.6%. 15.7% and 21.9% for HIV on ART, ART naïve HIV and HIV negative individuals, respectively.

Exposure to ART in HIV-patients and metabolic syndrome are the independent risk factor of Insulin resistance.

Incidence of diabetes mellitus among HIV-patients

CVD and HIV/AIDS

Risk factors among school children

Risk factors in workplace

CVD, metabolic syndrome and helicobacter pylori

Metabolic syndrome, pulse pressure, hypertension, nutrition, oxidative stress and diabetic ocular diseases

Seasons, climate and CVD

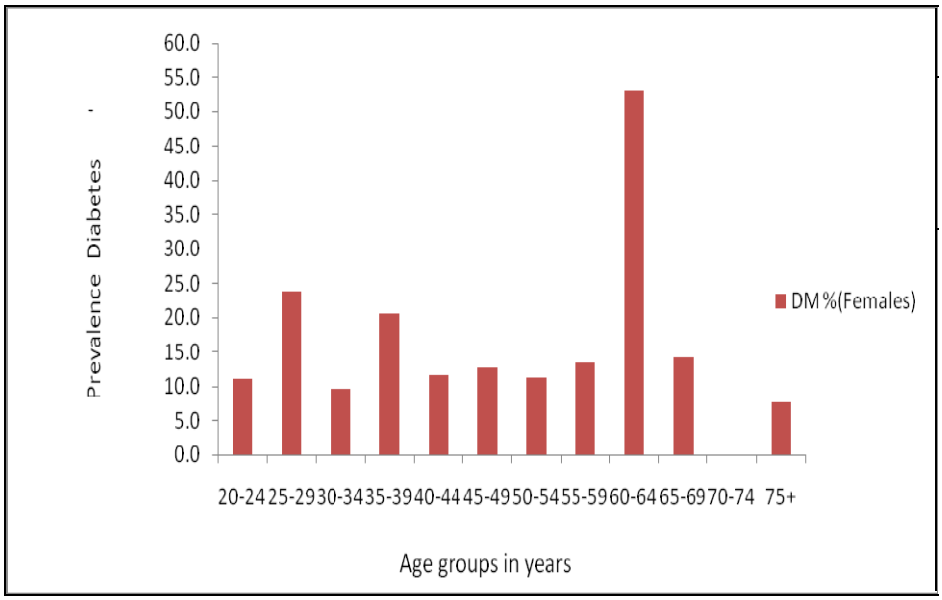
Hypertension: genetic pattern for Africans

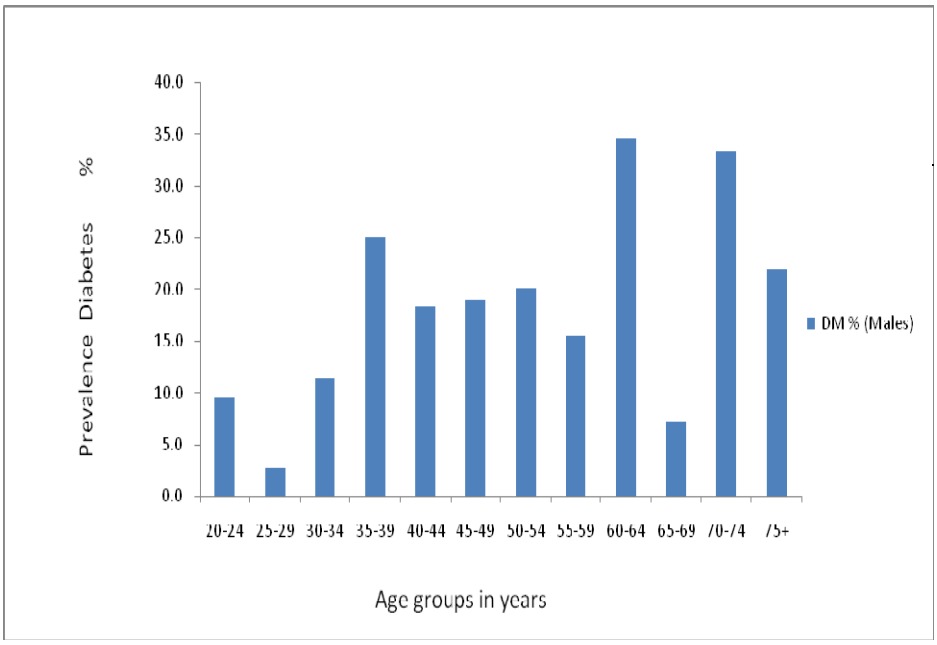
Epidemic of diabetes mellitus

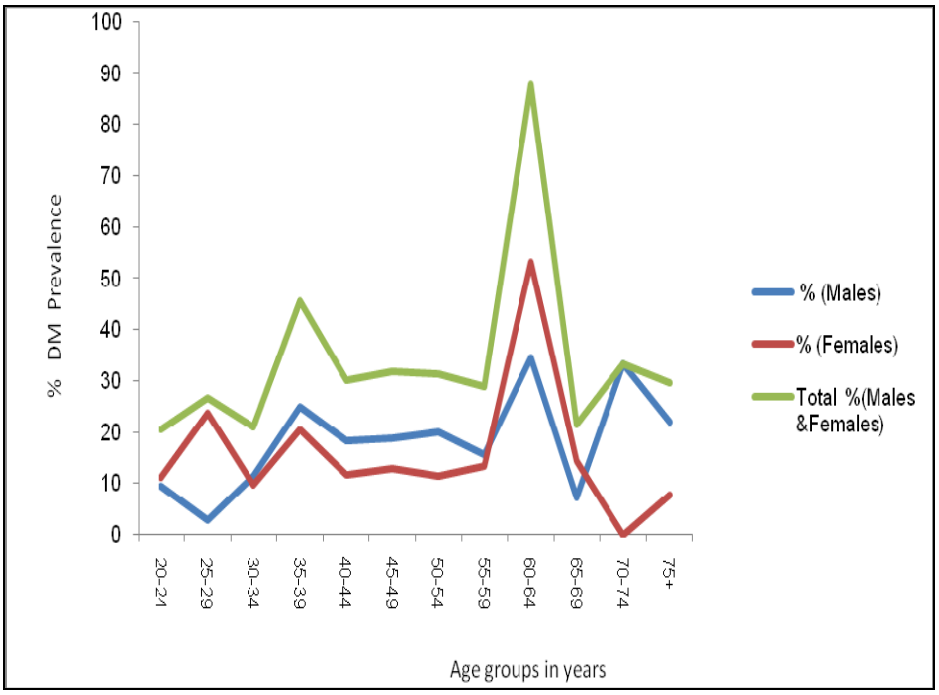
Vegetables intake and pre-eclampsia

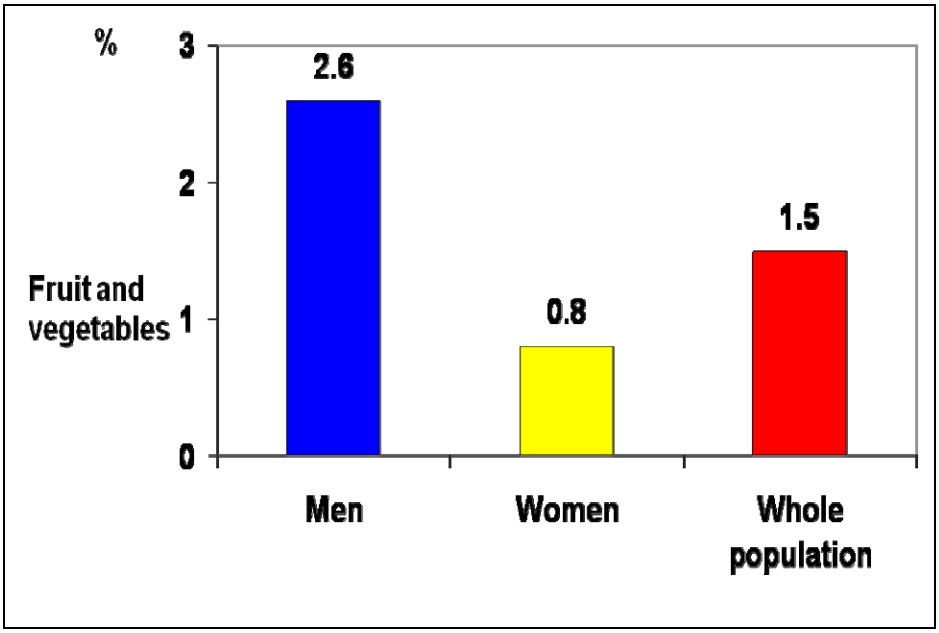
Lipids, Atherosclerosis and Sickle Cell Disease


PROVINCE ORIENTALE
EQUATEUR
NORD-KIVU
SUD-KIVU
KASAI-ORIENTAL
MANIEMA
KASAI-OCCIDENTAL
BANDUNDU
KINSHASA
BAS-CONGO
KATANGA

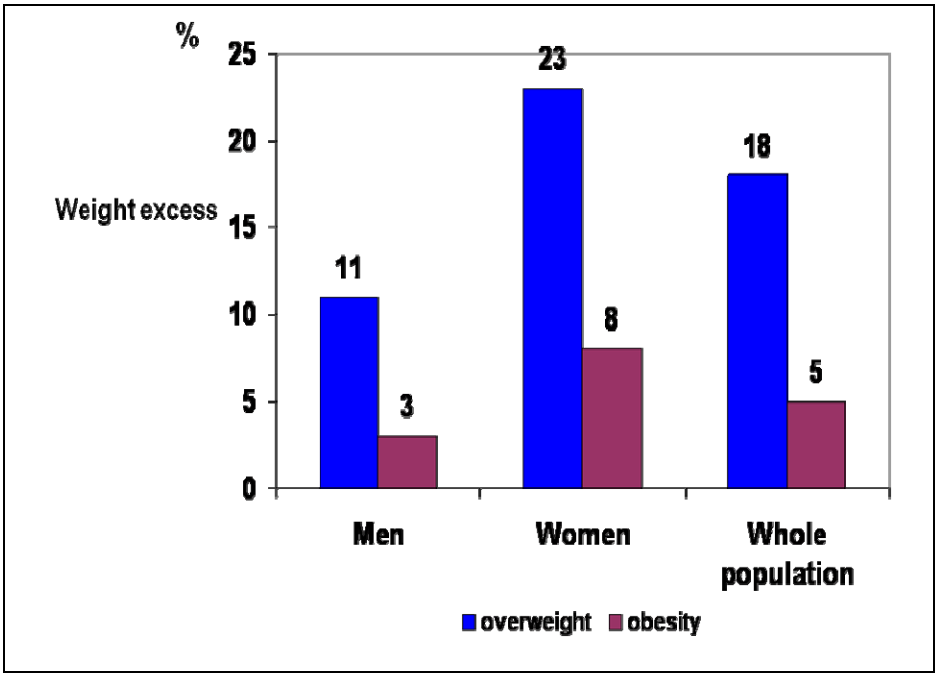

Prevalence Diabetes %
60.0
55.0
50.0
45.0
40.0
35.0
30.0
25.0
20.0
15.0
10.0
5.0
0.0
20-24 25-29 30-34 35-39 40-44 45-49 50-54 55-59 60-64 65-69 70-74 75+
DM %(Females)
Age groups in years


Prevalence Diabetes %
40.0
35.0
30.0
25.0
20.0
15.0
10.0
5.0
0.0
20-24 25-29 30-34 35-39 40-44 45-49 50-54 55-59 60-64 65-69 70-74 75+
DM % (Males)
Age groups in years

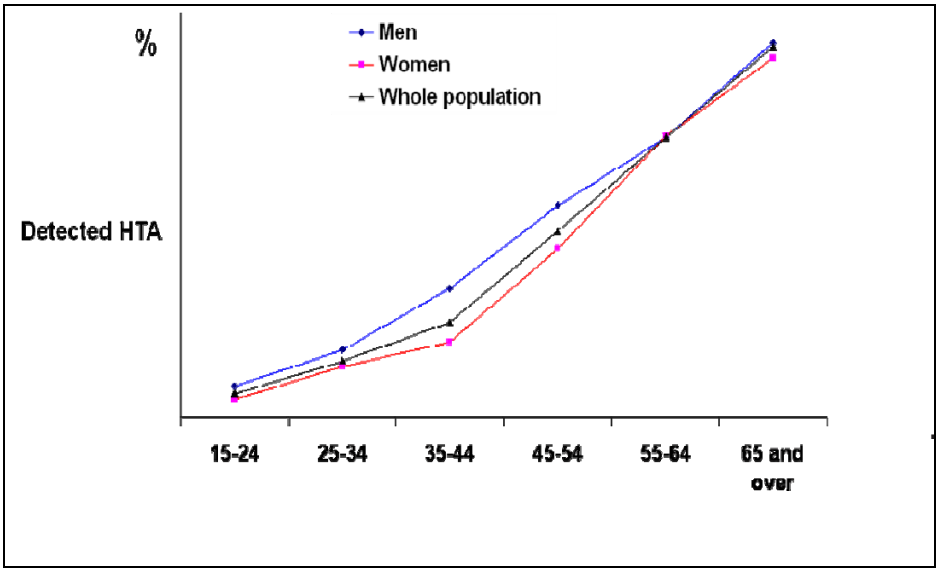




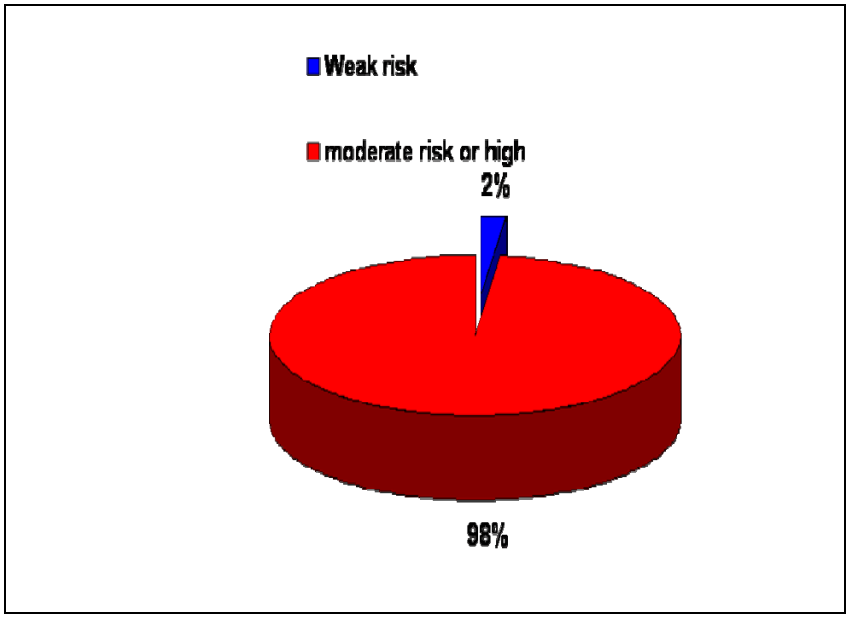
Fruit and vegetables consumption in the population and by sex.



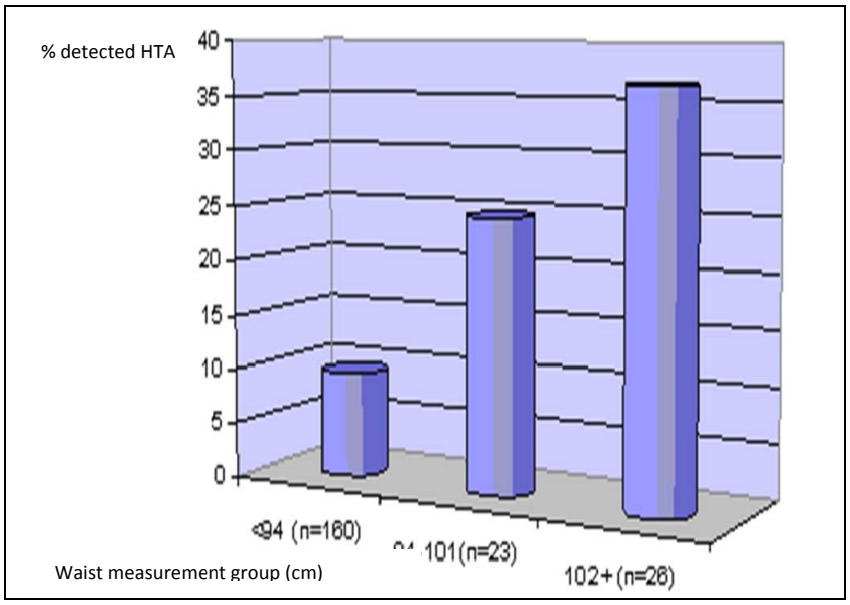
Overweight and obesity in the whole population and by sex.

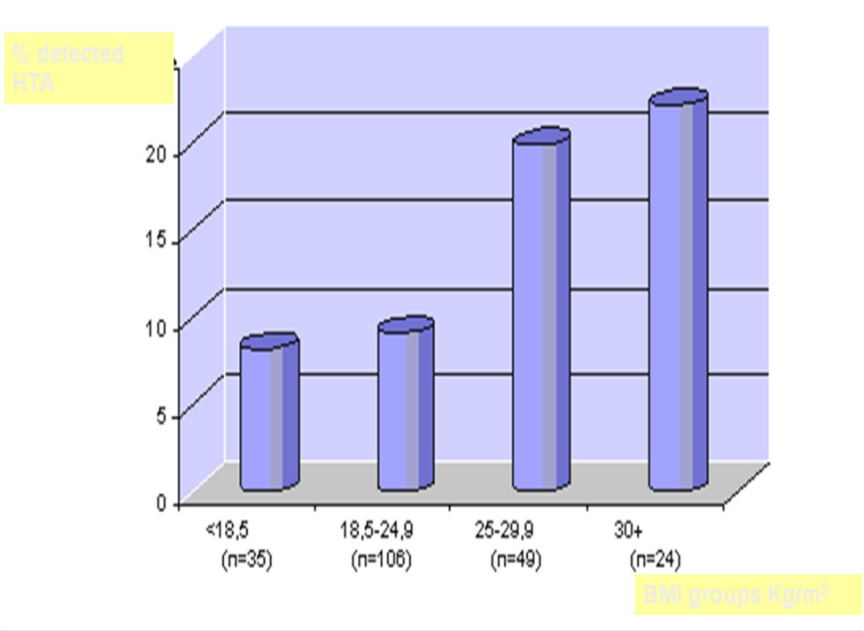


Increase of detected HTA rate with age in the whole population, men and women.



Weak risk and high risk in the population.





Positive relation between detected HTA rate and BMI groups.

Effects of physical training on obesity, arterial hypertension, brachial arterial stiffness / sub-clinical atherosclerosis, dysglycemia and heart rate in sedentary employees and urban employees from Kinshasa DR Congo

Kikontwe Louise*, Longo-Mbenza B**,

*Physiotherapist, Master student, University of Kinshasa, Faculty of medicine, Kinshasa, DRC
* Research Champion Professor, Walter Sisulu University, Faculty of Health Sciences, South Africa

Background

Physical inactivity is well established as another of the major risk factors for atherosclerosis. However, longitudinal epidemiologic studies have consistently shown that regular physical activity prevents atherosclerosis cardiovascular diseases (CVD). There is no data about urban employees of National Company of Electricity seated for more than 14 hours per day in their offices at the headquarters of Kinshasa, DRC. The study sought to determine whether regular physical training reduces higher CVD risk related to obesity, arterial hypertension, dysglycemia, and sub-clinical atherosclerosis to increase cardiorespiratory fitness.

Methods

A short-term follow-up and interventional study was conducted from 6 June 6th to October 19th 2005 within the occupational medicine service. 21 Sedentary employees , and treated for unknown moderate CVD risk (obesity, sub-clinical atherosclerosis, arterial hypertension, hypercholesterolemia dysglycemia) were prescribed a 3-moth, period non pharmacological treatment including appropriate diet and regular physical training with an increase in intensity of exercise and improvement of cardiorespiratory fitness (pre-, during and post-exercise heart rate). Base line values of these CVD risks factors were compared with those post-physical training.

Results

At the baseline these 21 males were defined by a mean age of 55+- 10 years, 14% smokers, 57% of excessive alcohol intake, 100% of physical inactivity, 95% of hypertension, 47% of sub-clinical atherosclerosis, 66.7% of dysglycemia, 42.4% of hypercholesterolemia, 33.3% of overweight, 38.1% of total obesity, and 66.7% of abnormal obesity. There was an indifferent effect of regular physical training on dysglycemia, hypercholesterolemia, and obesity. However, there was a significant (P<0.005), values of mean blood pressure and pulse pressure (-61%), but a significant improvement of cardiorespiratory fitness.

Conclusion

Despite its limited effect on obesity, hypercholesterolemia, and dysglcemia, regular physical training demonstrated a significant control of hypertension and sub-clinical atherosclerosis in these sedentary employees. Increased exercise intensity. Appropriate diet and pharmacological treatment are recommended.

Tableau 1. Composantes de la pression artérielle avant et après entraînement cardiovasculaire

Variables d'intérêt	Avant entraînement	Après entraînement	P
PAS (mmHg)	145,9 ±12,2	131,1± 8,6	<0,00001
PAD (mmHg)	93,4 ± 11,4	82,9 ± 9,4	<0,00001
Pression pulsée (mmHg)	53,6 ± 9,9	48,2 ±8,4	<0,00001

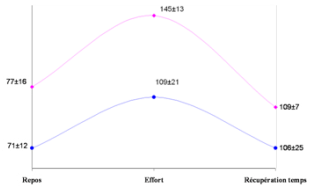


Tableau 2. Fonction respiratoire avant et après entraînement cardiovasculaire

Variables d'intérêt	Avant entraînement	Après entraînement	P
CVF (Litre/sec.)	2,04 ± 0,67	2,45 ± 0,59	NS
VEMS (Litre/sec.)	1,781 ± 0,744	2,241 ± 0,506	0,06
TIFF (%)	86,3 ± 23,2	92 ± 6,6	< 0,05
DEP (Litre/sec.)	5,8 ± 1,9	6,7 ± 1,4	< 0,05

Figure 1. Evolution de FC avant ( ) et après ( ) entraînement cardiovasculaire au repos, à l'effort et à la récupération.

Tableau 4. Dérivées de la fréquence cardiaque avant et après l'entraînement

Variables d'intérêt	Avant entraînement	Après entraînement	p
Fréquence cardiaque Maximale de réserve	85,7 ± 8,7bpm	64,8±11,7bpm	<0,05
Réserve de la Fréquence cardiaque	62,4±24,5 bpm	38,2 ±22,5 bpm	<0,00001
Fréquence cardiaque Cible			
▪ Limite inférieure	131,9 ±7,8 bpm	97,8 ±13,9 bpm	<0,00001
▪ Limite supérieure	145,7 ± 6,5 bpm	97,9 ± 13,9 bpm	<0,00001

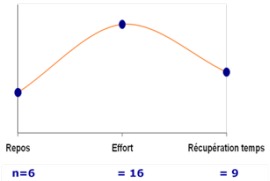
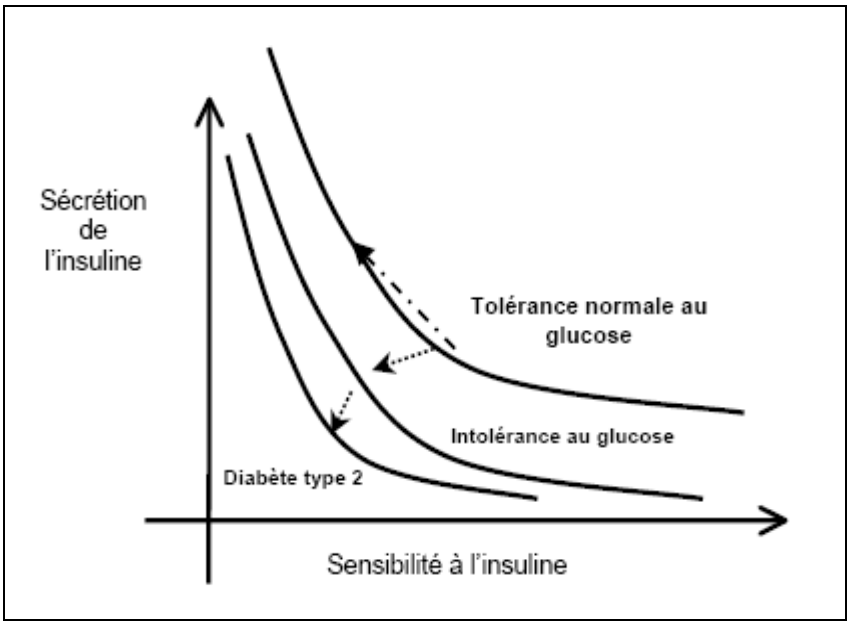
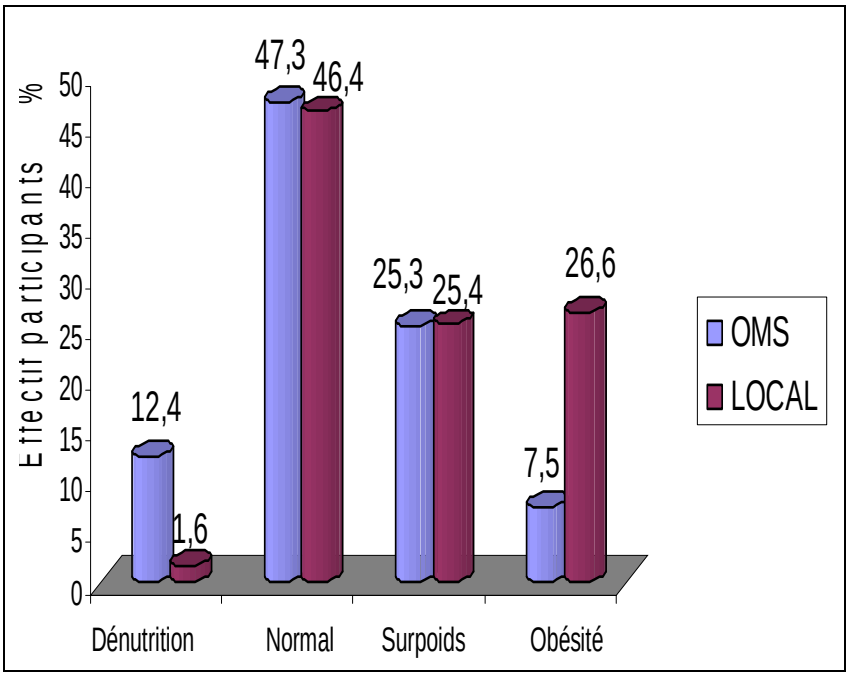
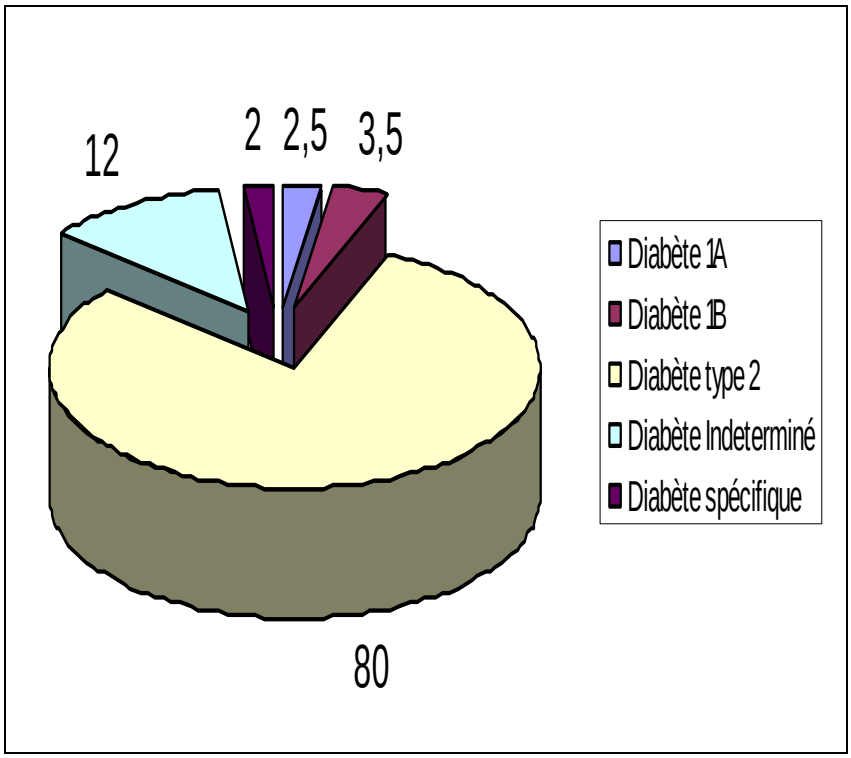
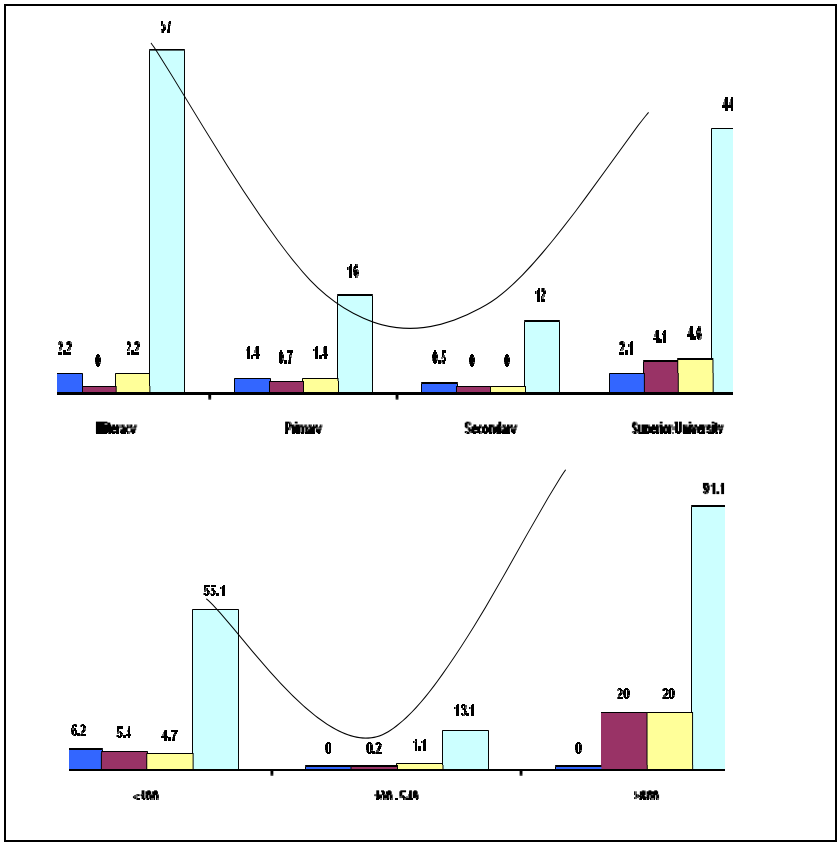


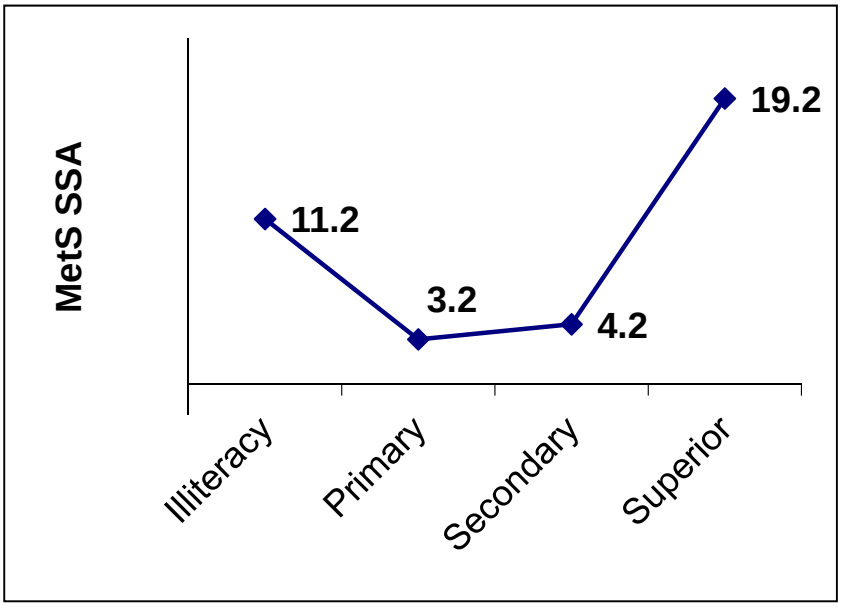
Figure 2. Economie d'énergie cardiaque ≥ 15 % sous l'effet de l'entraînement, au repos, à l'effort et à la récupération.

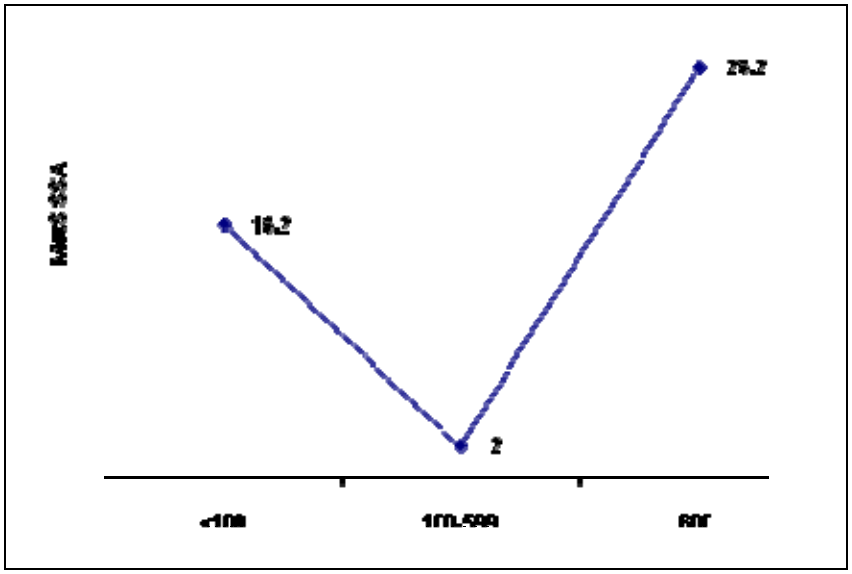

Sécrétion
de
l'insuline
Tolérance normale au
glucose
Intolérance au glucose
Diabète type 2
Sensibilité à l'insuline

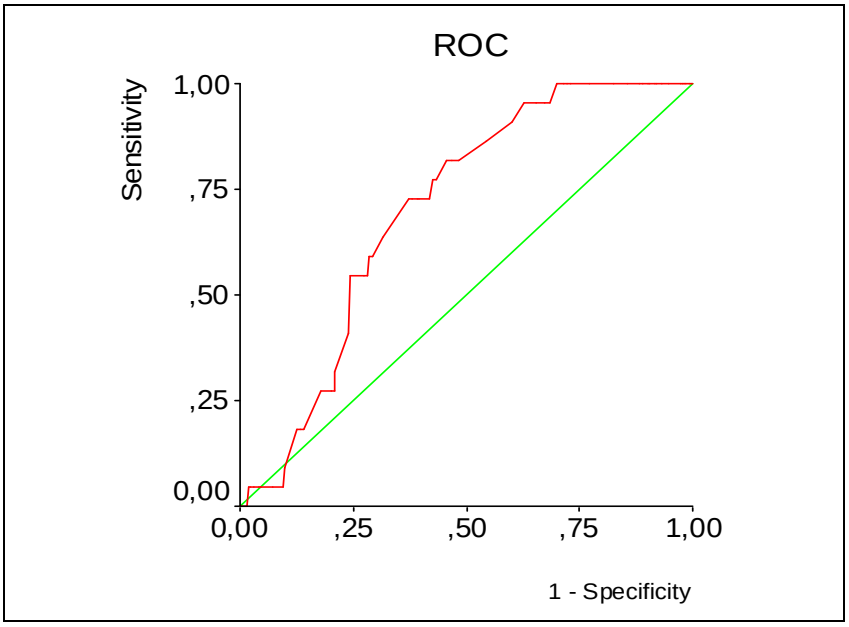

Effectif participants %
50
45
40
35
30
25
20
15
10
5
0
12,4
1,6
47,3
46,4
25,3
25,4
7,5
26,6
OMS
LOCAL
Dénutrition
Normal
Surpoids
Obésité

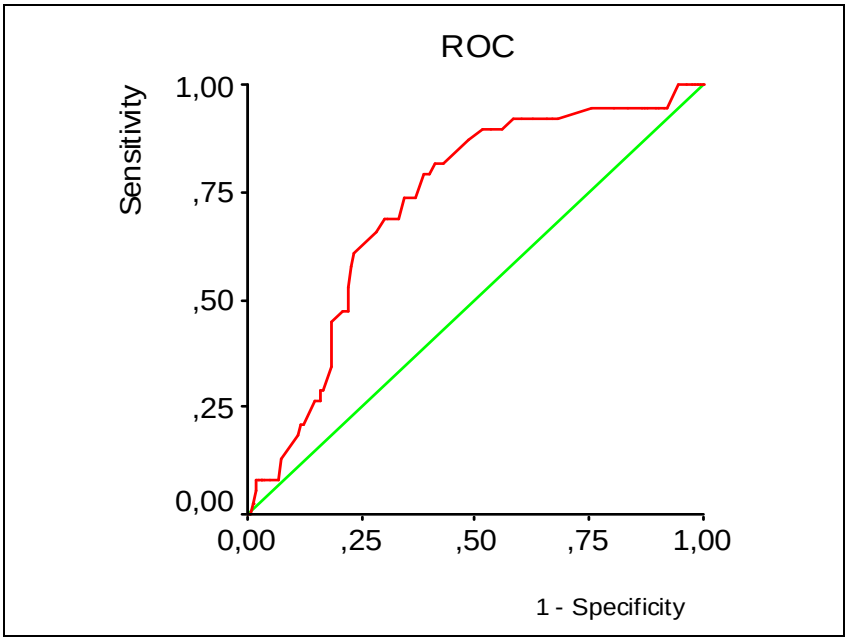

12
2
2,5
3,5
80
Diabète 1A
Diabète 1B
Diabète type 2
Diabète Indeterminé
Diabète spécifique


57
16
12
44
2.2
0
2.2
1.4
0.7
1.4
0.5
0
0
2.1
4.1
4.4
Literacy
Primary
Secondary
Superior-University
55.1
13.1
91.1
6.2
5.4
4.7
0
0.2
1.1
0
20
20


MetS SSA
11.2
3.2
4.2
19.2
Illiteracy
Primary
Secondary
Superior


19.2
16.2
2


ROC
Sensitivity
1,00
,75
,50
,25
0,00
0,00
,25
,50
,75
1,00
1 - Specificity


ROC
Sensitivity
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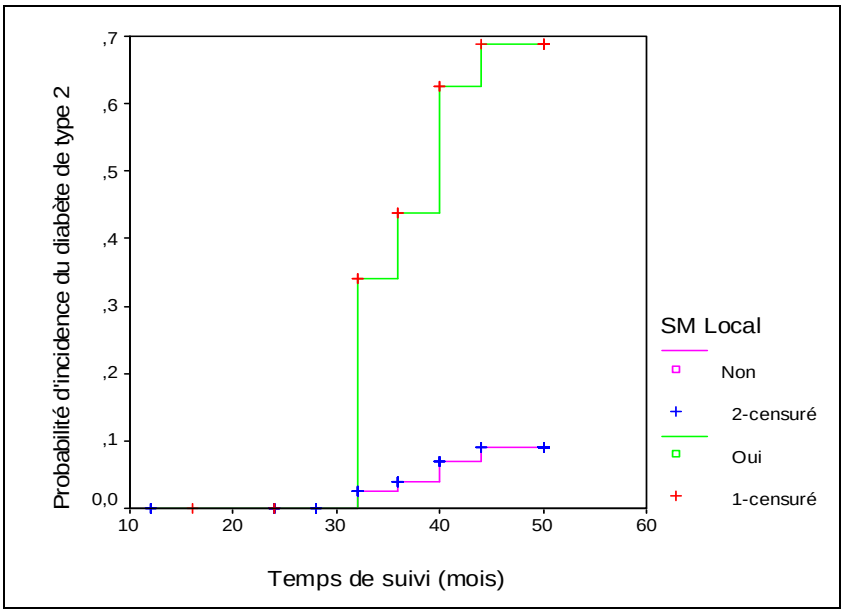
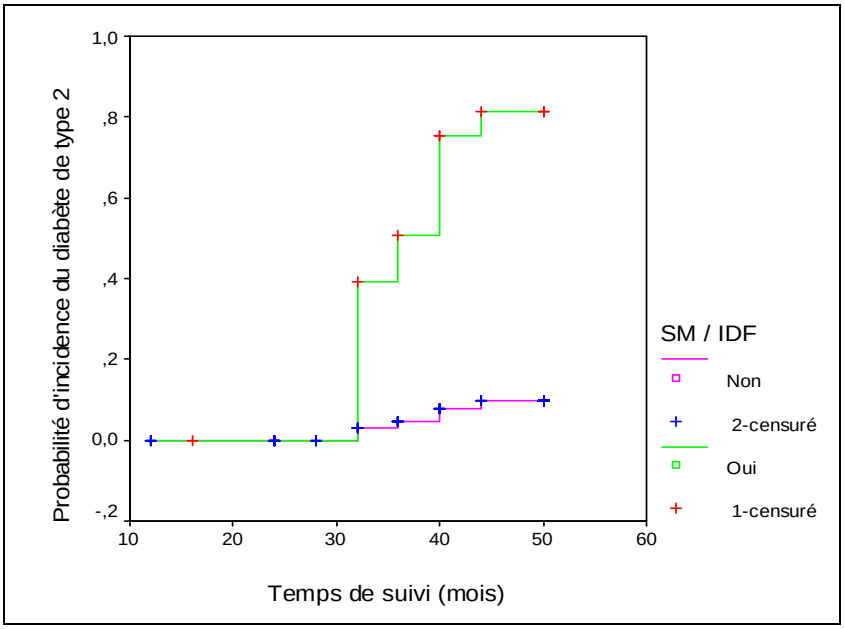
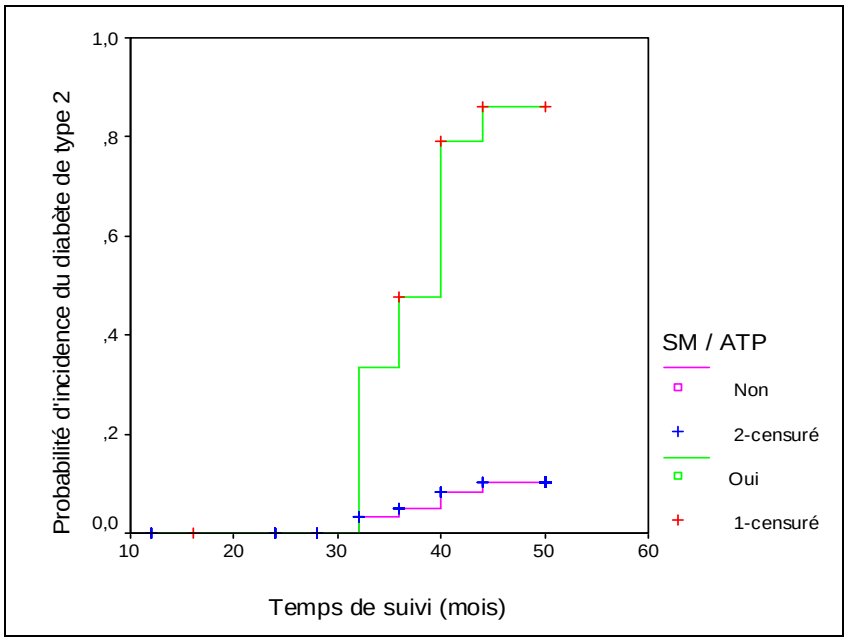
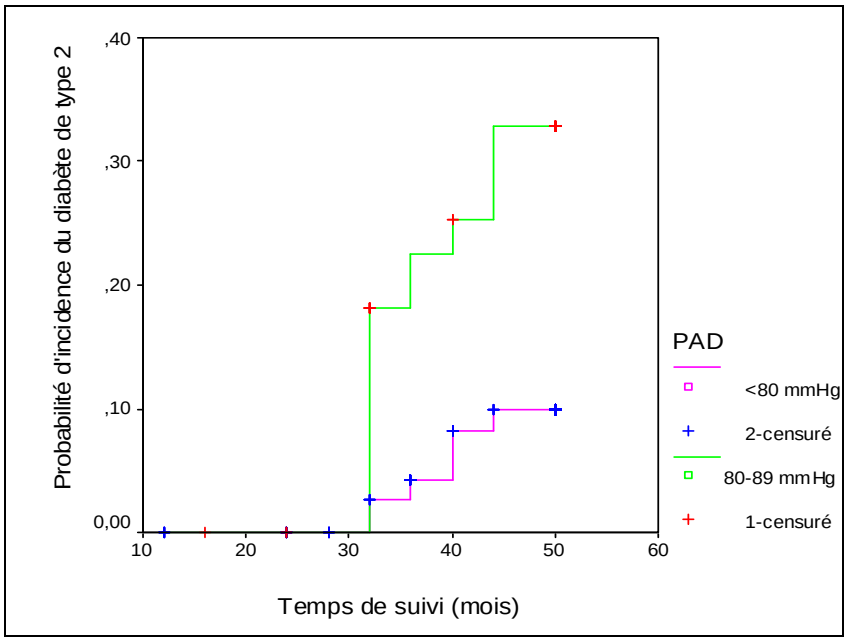
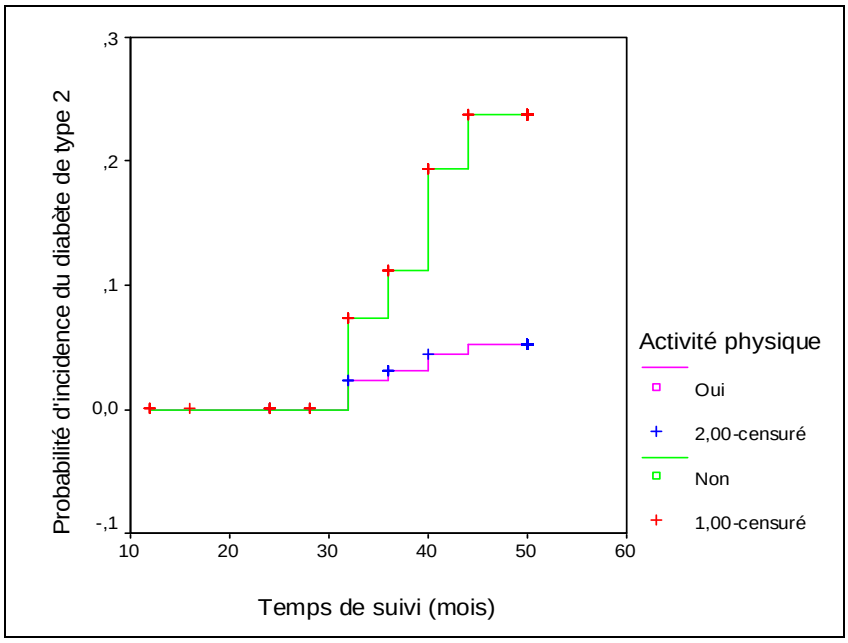
0,00
0,00
,25
,50
,75
1,00
1 - Specificity

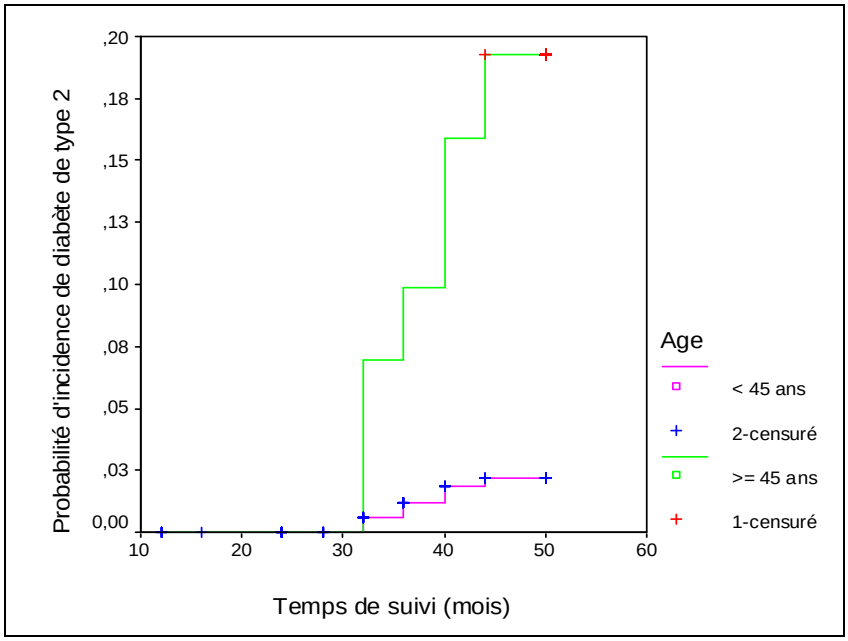

Probabilité d'incidence du diabète de type 2
,7
,6
,5
,4
,3
,2
,1
0,0
10
20
30
40
50
60
Temps de suivi (mois)
SM Local
Non
2-censuré
Oui
1-censuré

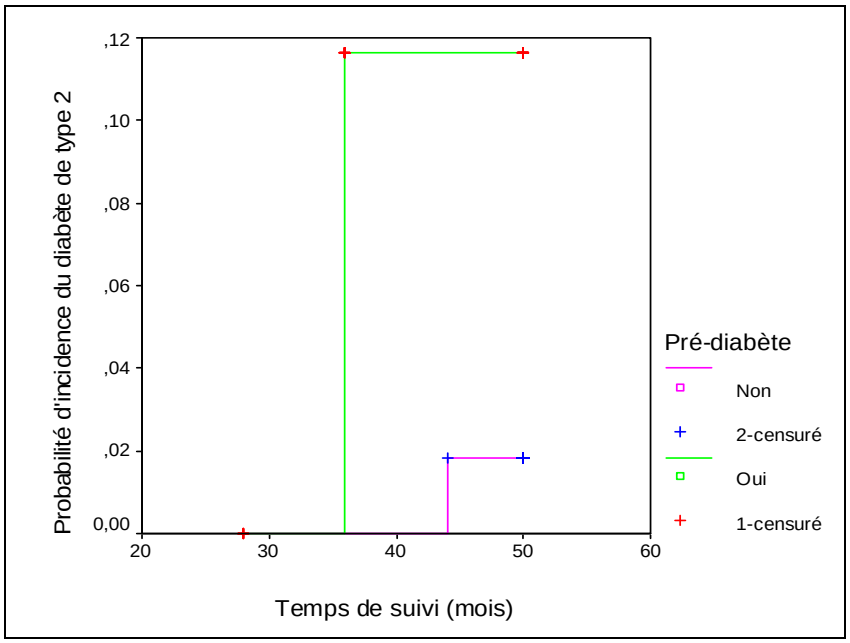

Probabilité d'incidence du diabète de type 2
1,0
,8
,6
,4
,2
0,0
-,2
10
20
30
40
50
60
Temps de suivi (mois)
SM / IDF
Non
2-censuré
Oui
1-censuré

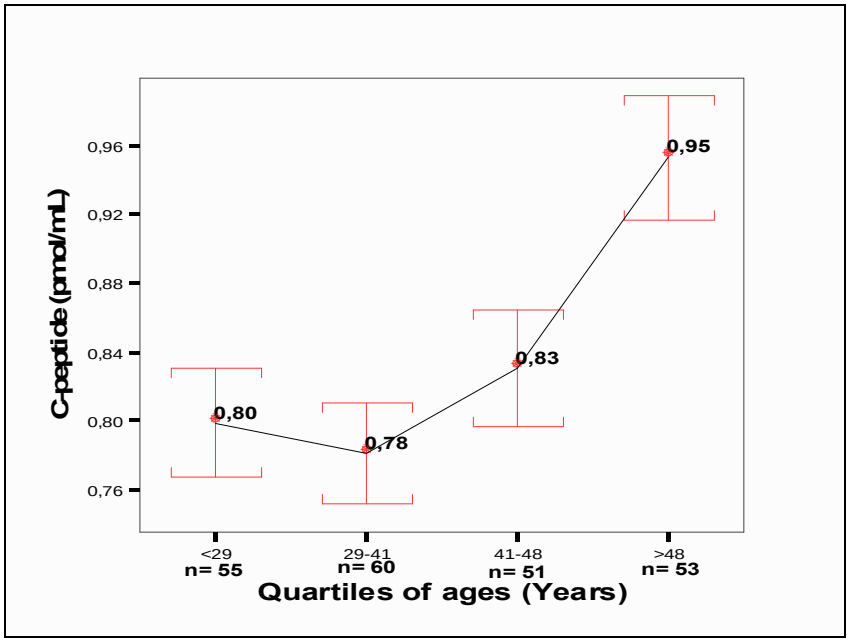

Probabilité d'incidence du diabète de type 2
1,0
,8
,6
,4
,2
0,0
10
20
30
40
50
60
Temps de suivi (mois)
SM / ATP
Non
2-censuré
Oui
1-censuré

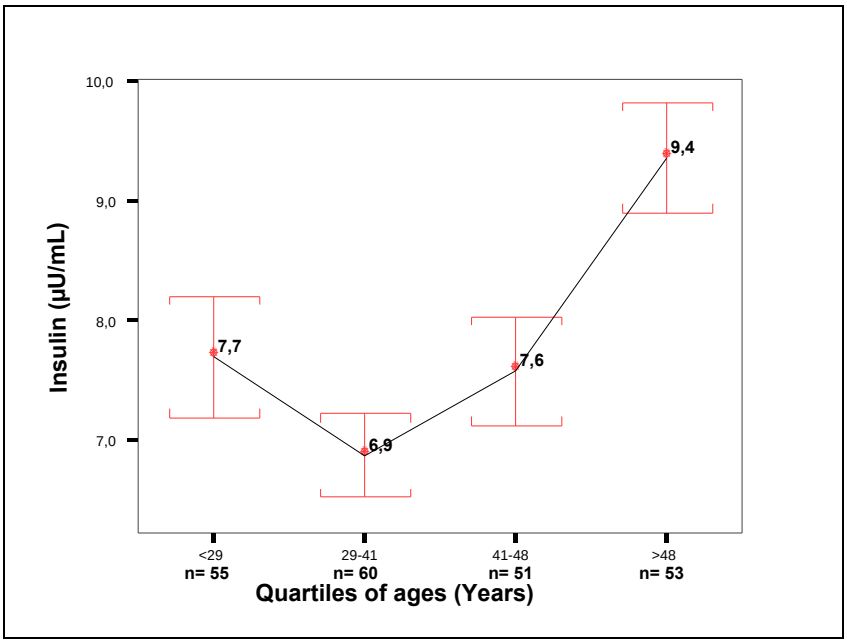

Probabilité d'incidence du diabète de type 2
,40
,30
,20
,10
0,00
10
20
30
40
50
60
Temps de suivi (mois)
PAD
<80 mmHg
2-censuré
80-89 mmHg
1-censuré

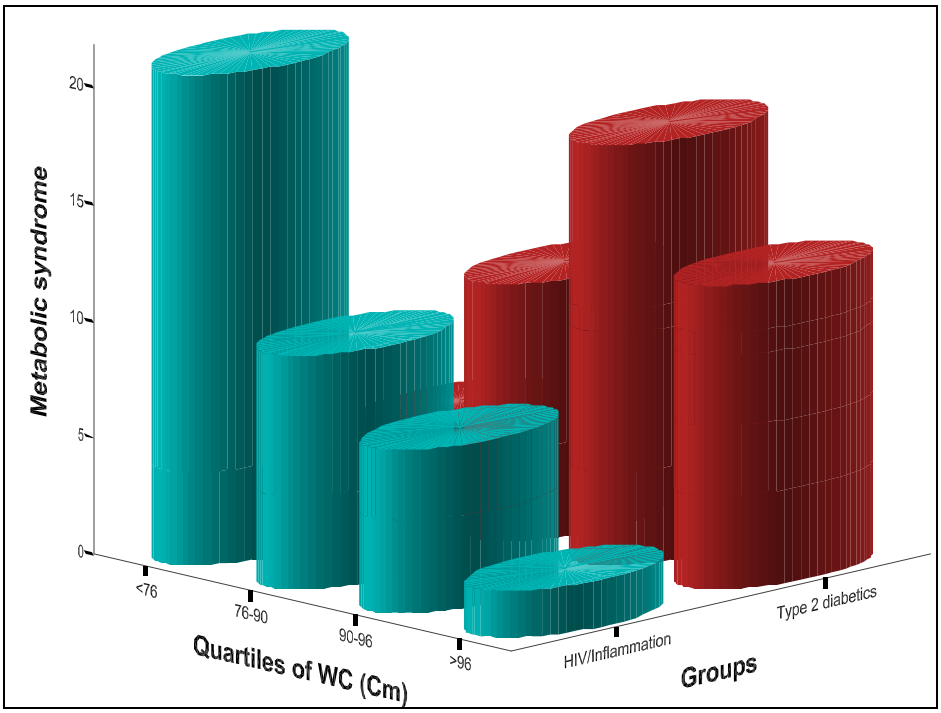

Probabilité d'incidence du diabète de type 2
,3
,2
,1
0,0
-,1
10
20
30
40
50
60
Temps de suivi (mois)
Activité physique
Oui
2,00-censuré
Non
1,00-censuré

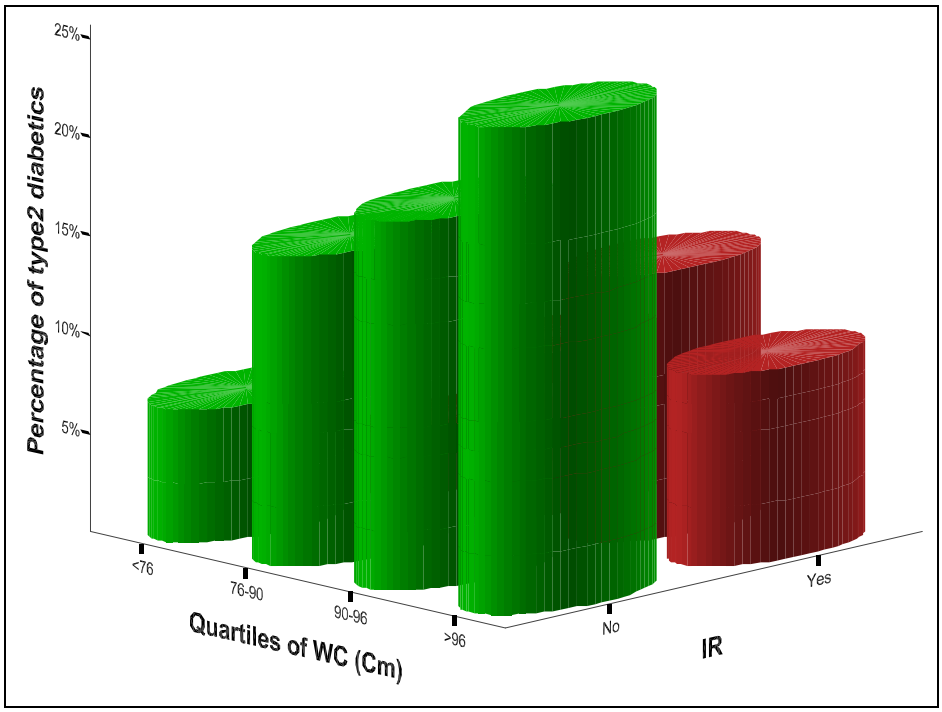

Probabilité d'incidence de diabète de type 2
,20
,18
,15
,13
,10
,08
,05
,03
0,00
10
20
30
40
50
60
Temps de suivi (mois)
Age
< 45 ans
2-censuré
>= 45 ans
1-censuré

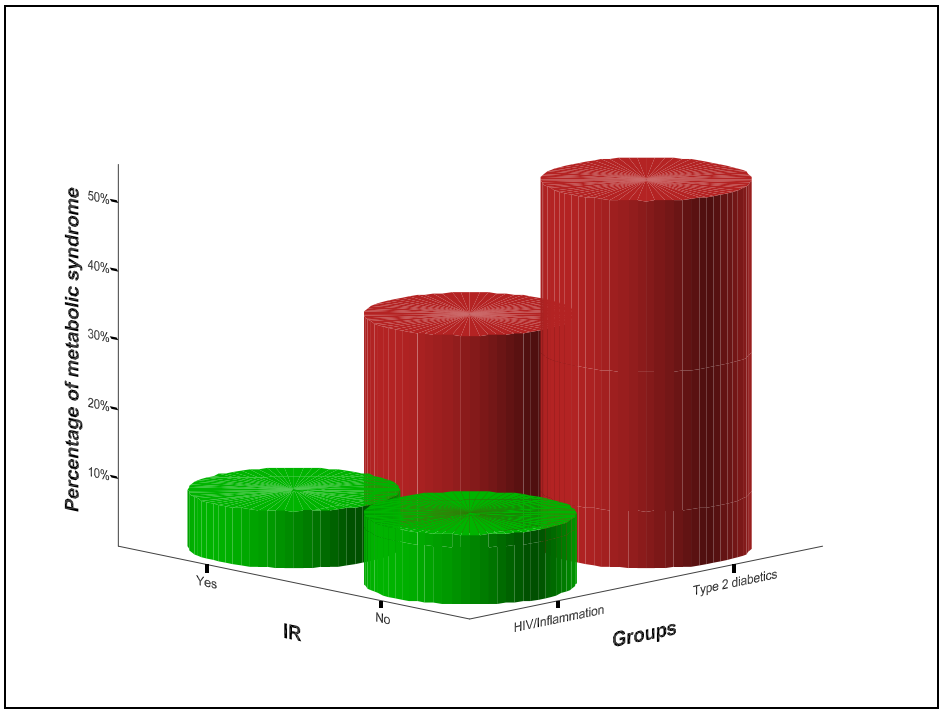

Probabilité d'incidence du diabète de type 2
,12
,10
,08
,06
,04
,02
0,00
20
30
40
50
60
Temps de suivi (mois)
Pré-diabète
Non
2-censuré
Oui
1-censuré

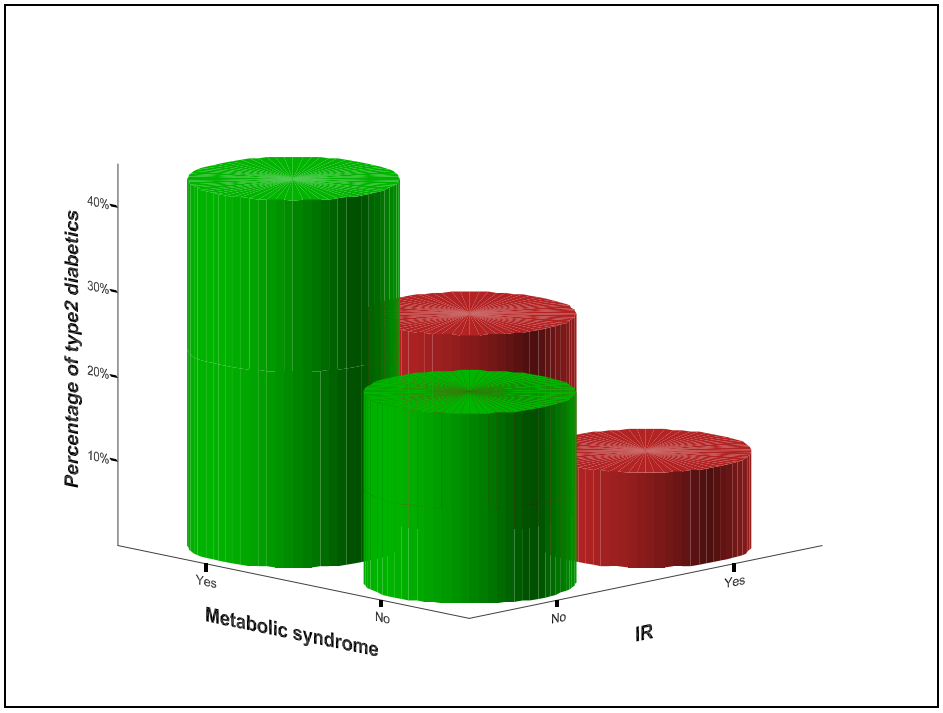

C-peptide (pmol/mL)
0,96
0,92
0,88
0,84
0,80
0,76
0,80
0,78
0,83
0,95
<29
n= 55
29-41
n= 60
41-48
n= 51
>48
n= 53
Quartiles of ages (Years)

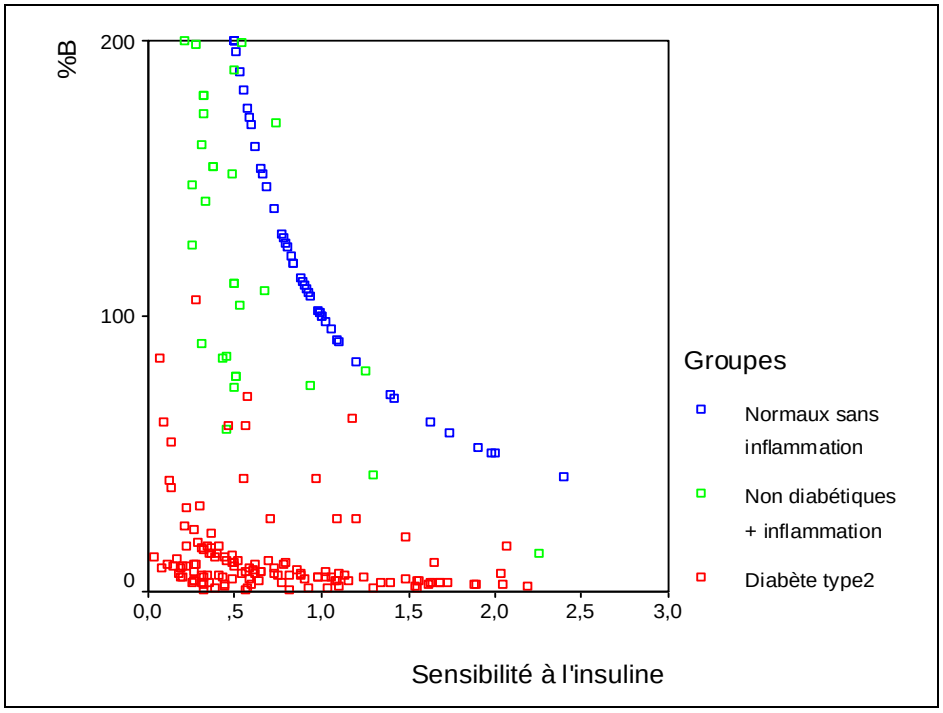

10,0
9,0
8,0
7,0
Insulin (µU/mL)
7,7
6,9
7,6
9,4
<29
n= 55
29-41
n= 60
41-48
n= 51
>48
n= 53
Quartiles of ages (Years)

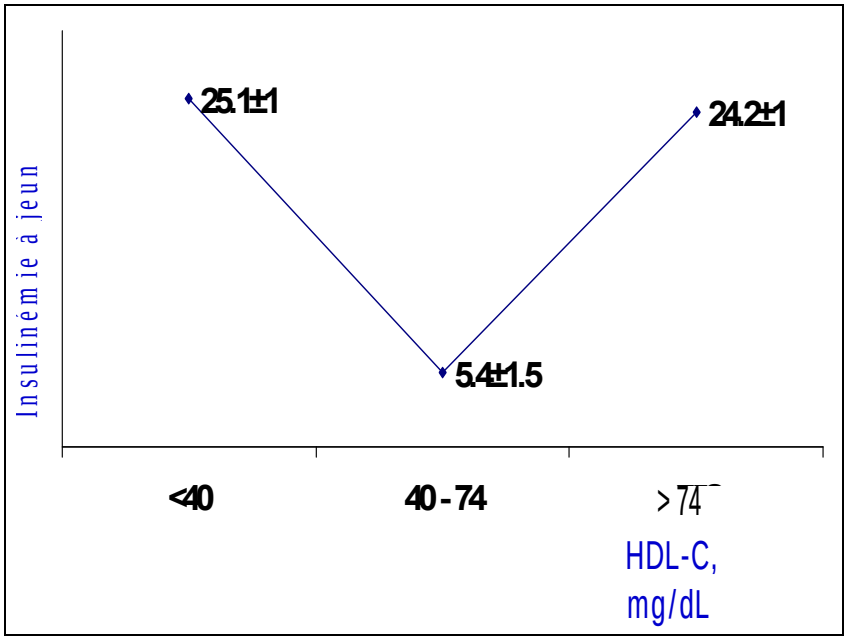

20
15
10
5
0
Metabolic syndrome
<76
76-90
90-96
>96
Quartiles of WC (Cm)
HIV/Inflammation
Type 2 diabetics
Groups

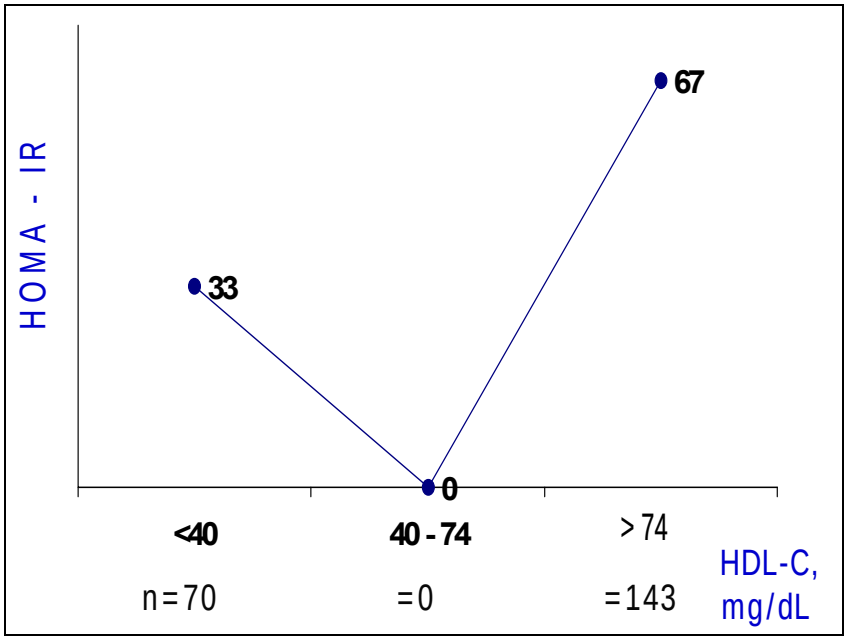

Percentage of type2 diabetics
25%
20%
15%
10%
5%
<76
76-90
90-96
>96
Quartiles of WC (Cm)
No
Yes
IR


Percentage of metabolic syndrome
50%
40%
30%
20%
10%
Yes
No
IR
HIV/Inflammation
Type 2 diabetics
Groups


Percentage of type2 diabetics
40%
30%
20%
10%
Yes
No
No
Yes
Metabolic syndrome
IR


%B
200
100
0
0,0
,5
1,0
1,5
2,0
2,5
3,0
Sensibilité à l'insuline
Groupes
Normaux sans inflammation
Non diabétiques + inflammation
Diabète type2


Insulinémie à jeun
25.1±1
5.4±1.5
24.2±1
<40
40 - 74
> 74
HDL-C, mg/dL


HOMA - IR
33
0
67
<40
40 - 74
> 74
n = 70
= 0
= 143
HDL-C, mg/dL

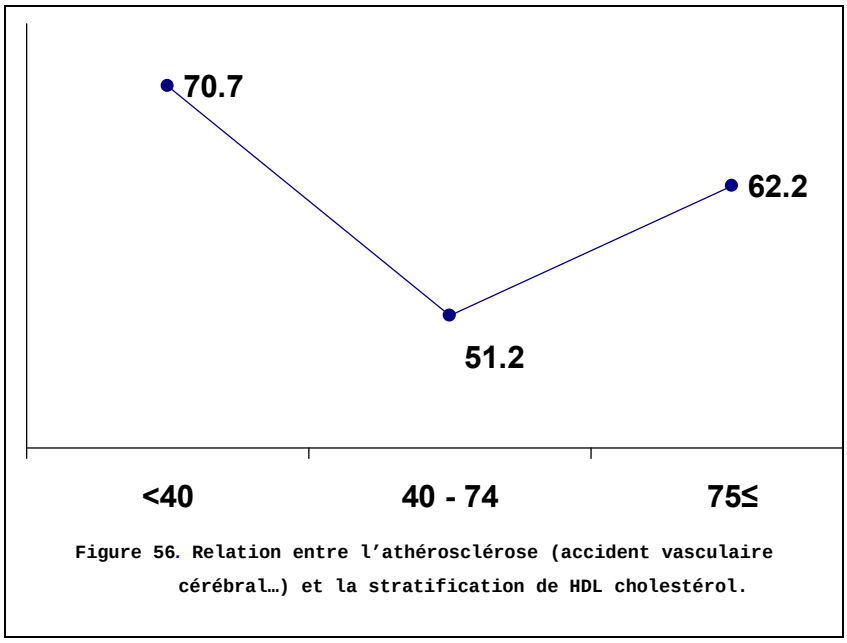
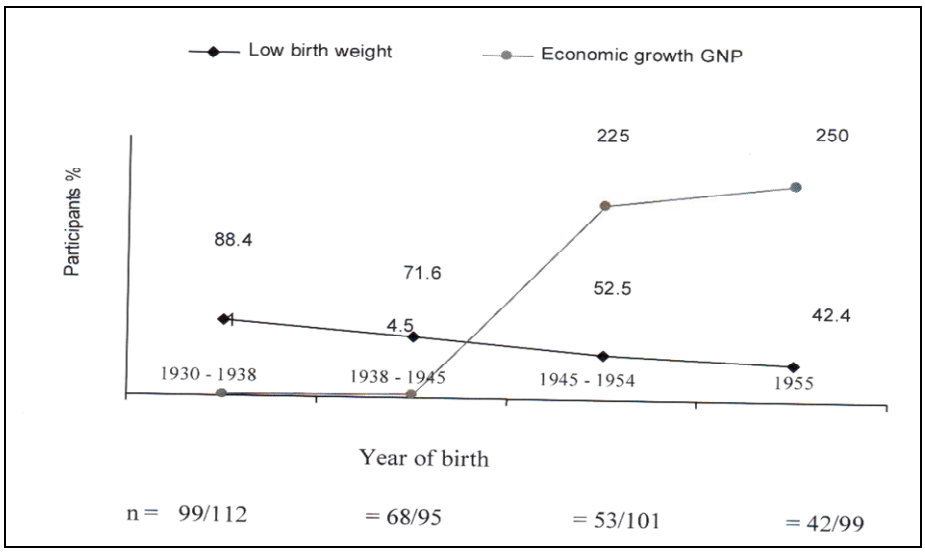


Figure 56. Relation entre l'athérosclérose (accident vasculaire cérébral…) et la stratification de HDL cholestérol.



Distribution of participants with low birth weight by the year of birth (P for trend= <0.0001) and economic growth (GNP) in % for Congo between 1930 and 1955.Current characteristics of 407 participants categorized by level of their birth weight.

Current variable	P	With low birthweight (n = 262) Mean ± SD	Without low birthweight (n = 145) Mean ± SD
Age (yrs)	<0.0001	56.9 ± 32.1	46.3 ± 9.8
SBP (mmHg)	<0.0001	170.9 ± 31.9	156.4 ± 34.3
DBP (mmHg)	0.002	98.3 ± 19.3	91.8 ± 22
Pulse pressure (mmHg)	<0.001	72.6 ± 23.2	64.6 ± 21.9
Fasting plasma glucose (mg/dl)	<0.001	104.3 ±49.8	76.7 ± 16.5
Urea Nitrogen (mg/dl)	0.036	31.8 ± 26.2	26.5 ± 19.3
Creatinin (mg/dl)	0.013	1.3 ± 0.8	1.1 ± 0.6
Uric acid (mg/dl)	0.079	5.9 ± 2.6	5.5 ± 2.4
Fifrinogen (mg/dl)	<0.0001	378.8 ± 143.9	318.9 ± 128.3
Total cholesterol (mg/dl)	<0.001	204.2 ± 58.2	185.7 ± 48.6
LViDd (mmm)	0.007	52 ± 9.3	48.9 ± 9.2

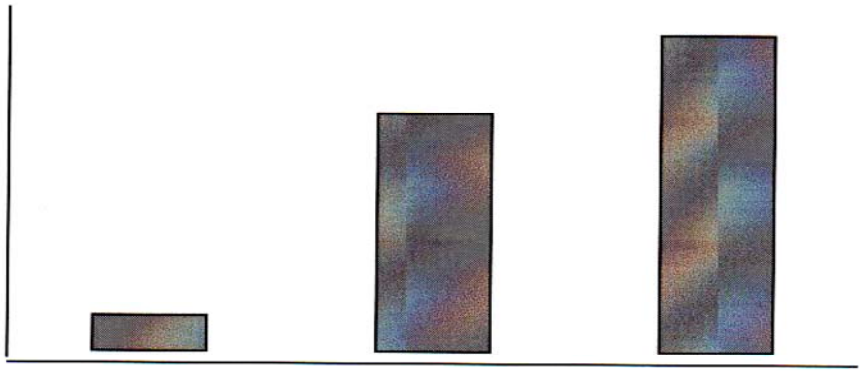
LViDd: End-diastolic LV internal dimension.

Means of continuous variables of 407 participants categorized by the presence of metabolic syndrome (Mets) defined according to WHO criteria.

Variable	With Mets (n = 77) Mean ± SD	Without Mets (n = 330) Mean ± SD	P
Age (years)	60.1 ± 56.8	51.5 ± 11.6	0.012
BMI (Kg/m^2)	33 ± 8.1	27.2 ± 7.4	<0.0001
SBP (mmHg)	174.9 ± 32.3	163.8 ± 33.5	0.016
Pulse pressure (mmHg)	77.3 ± 21.9	68 ± 23	<0.001
Birth weight (g)	2291 ± 413	2751.1 ± 759.1	<0.0001
Fasting plasma glucose (mg/dl)	152.5 ± 66.2	80.9 ± 17.8	<0.0001
Urea Nitrogen (mg/dl)	38.7 ± 30	27.8 ± 22.1	<0.0001
Creatinin (mg/dl)	1.5 ± 0.7	1.2 ± 0.7	0.002
eGFR	72.1 ± 32.6	99.8 ± 50.3	<0.0001
Uric acid (mg/dl)	6.7 ± 2.3	5.5 ± 2.6	<0.0001
Fifrinogen (mg/dl)	434 ± 130.4	339.6 ± 137.9	<0.0001
Total cholesterol (mg/dl)	224.4 ± 69.8	191.4 ± 49.8	<0.0001
LV mass	180 ± 102	150 ± 120	0.042
LViDd (mm)	52.7 ± 9.9	50 ± 9.2	0.022
EDIST (mm)	13.1 ± 5.6	11.6 ± 5.4	0.031

LViDd: end-diastolic left ventricular (LV) internal dimension. EDIST : end-diastolic inter(ventricular septal thickness

CHF	24 (31.2)	62 (18.8)	0.017
Ischemic stroke	30 (39)	70 (21.2)	<0.001
Low birth weight	72 (93.5)	190 (57.6)	<0.001
CKD	30 (39)	69 (20.9)	<0.0001
Hypercholesterolemia	41 (53.2)	102 (30.9)	<0.0001
Hyperuricemia	41 (53.2)	106 (32.1)	<0.001
Elevated fibrinogen	54 (70.1)	124 (37.6)	<0.0001
Elevated Urea nitrogen	45 (58.4)	115 (36.8)	<0.0001



n = 7/310 =31/50 =39/47

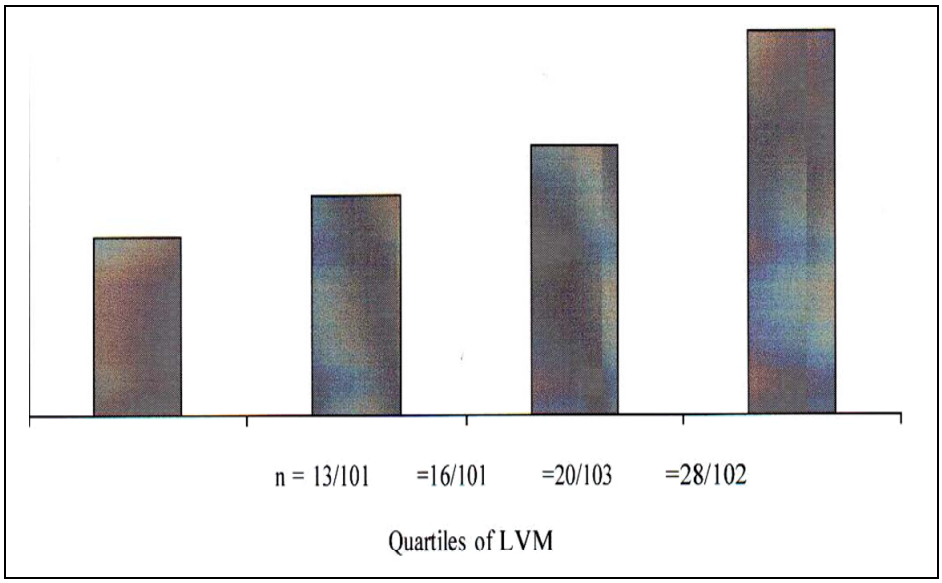
Fasting glucose, mg/dL

Relationship between the prevalence of the metabolic syndrome defined to WHO criteria and the levels of fasting plasma glucose in 407 patients. P for trend<0.0001.

Multivariate regression analysis for factors potentially linked to the presence of the metabolic syndrome defined to WHO criteria among 407 patients.

Independent Individual Variable	Beta coefficient	SE	OR (95% CI)	P
CHD, yes vs. no	0.845	0.373	2.3 (1.1 - 4.8)	0.024
Low birth weight, yes vs. no	2.294	0.482	10 (3.9 - 25.5)	<0.0001
Elevated fibrinogen, yes vs. no	1.247	0.287	3.5 (2 - 6.1)	<0.0001
Constant	-	0.500		<0.0001

CHD: coronary heart disease. SE:

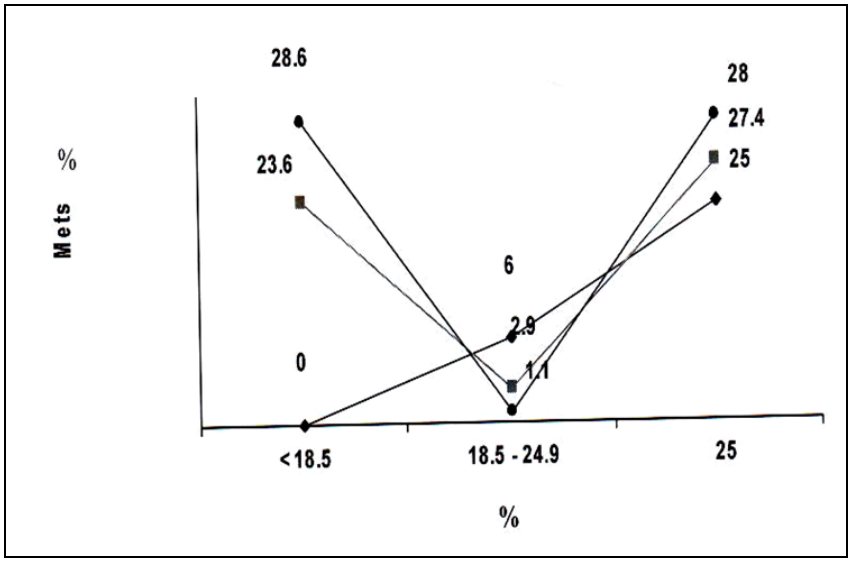


Relationship between prevalence of the metabolic syndrome according to WHO criteria among 407 patients. P for trend=0.007.

Relationship between prevalence of the metabolic syndrome according to WHO criteria among 168 women.

Independent individual variable	Beta coefficient	SE	OR (95% CI)	P
Low birth weight yes vs. no	2.888	1.040	18 (2.3 – 37)	0.005
Elevated urea, yes vs. no	0.968	0.468	2.6 (1.1 – 6.6)	0.038
Elevated fibrinogen, yes vs. no	1.117	0.467	3.1 (1.7 – 9.8)	0.017
Constant	- 4.874	1.059		<0.0001

SE: standard error.



Relationship between the prevalence of metabolic syndrome (Mets) and nutritional status categories in all patients (▬) , men (●) and women (◆).

Means and standard deviation for selected characteristics of all non hypertensive, prehypertensive and normotensive participants.

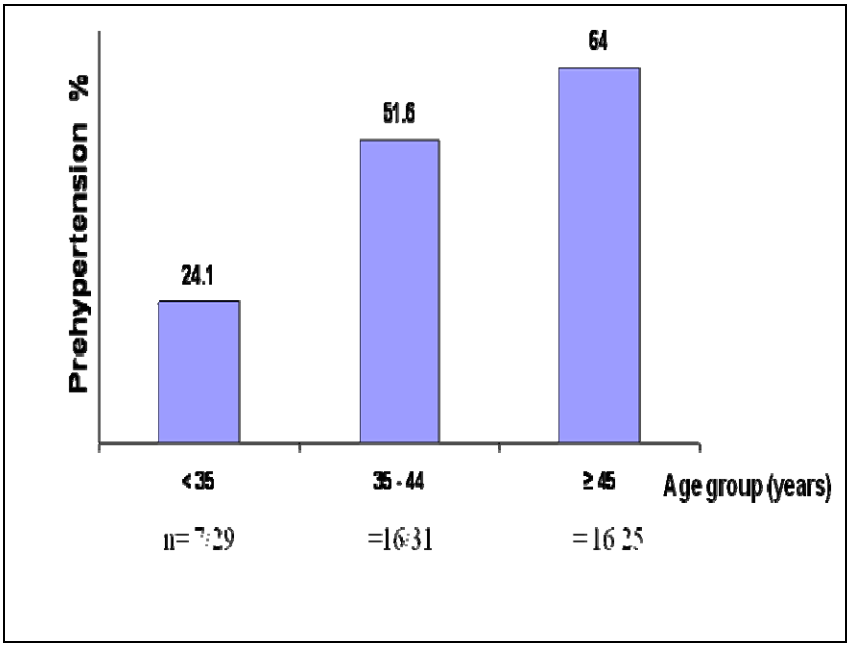
Characteristics	All non hypertensives	Prehypertensive	Normotensive	P
Age (years)	40 ± 9	43.5 ± 8.3	37.4 ± 8.6	0.001
BMI (kg m²)	25.8 ± 4.6	27.3 ± 4.5	24.9 ± 4.5	0.017
WC (cm)	89.4 ± 12.2	92.5 ± 11	87.4 ± 13.1	
Fat mass ( )	29 ± 8	31.5 ± 9.1	26.3 ± 9	0.009
QTc (msec)	0.351 ± 0.002	0.357 ± 0.003	0.343 ± 0.002	0.004
TC (mg dL)	199.5 ± 50.4	197.8 ± 51.8	201.5 ± 47.5	0.756
HDL-C (mg dL)	81.5 ± 33.2	78.5 ± 38.7	85.1 ± 26	0.401
TG (mg dL)	78.6 ± 36.2	80.7 ± 403	74.4 ± 30.7	0.453
LDL-C (mg dL)	101.7 ± 64	102.3 ± 66.6	101.3 ± 60	0.945
Fasting glucose (mg dL)	89.7 ± 26.2	92.2 ± 32.4	86.6 v 18	0.368

Factor loadings for original variables with rotated factors in normotensive employees.

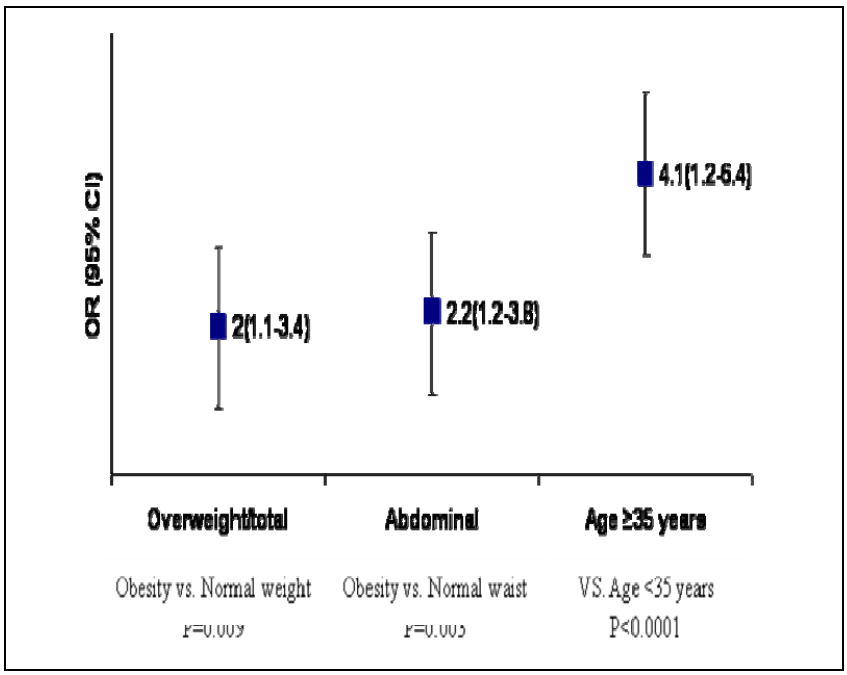
	Factor 1	Factor 2	Factor 3	Factor 4	Factor 5	Factor 6
Variables						
Fat mass	0.892					
ESR 1st H	0.862					
ESR 2nd H	0.854					
BMI	0.670	0.646				
Total nitrogen		0.922				
Total water		0.916				
Wt	0.542	0.728				
Uric acid		0.601				
Fasting glucose			0.808			
TG			0.719			
Tc			0.665	0.529		
LDL-c				0.866		
HDL-c				-0.851		
DBP					0.824	
Heart rate					0.675	
SBP			0.518		0.562	
QTc						0.830
Age						0.557

Factor loadings for original variables with rotated factors in prehypertensive employees.

	Factor 1	Factor 2	Factor 3	Factor 4	Factor 5	Factor 6	Factor 7
Variables							
Total nitrogen	0.902						
Total water	0.891						
Age	-0.647						
BMI		0.967					
Wt		0.849					
Fat mass		0.836					
ESR 1st H			0.894				
ESR 2nd H			0.874				
QTc			-0.646				
LDL-c				0.967			
Tc				0.853			
HDL-c				-0.625			
DBP					0.893		
SBP					0.891		
Heart rate						0.782	
Fasting glucose						-0.567	
Triglycerides							0.893



Relationship between the prevalence of prehypertention and ages of non hypertensive bank employees.



Respective risk of prehypertension conferred by overweight/total obesity, abdominal obesity and age >=35 years.

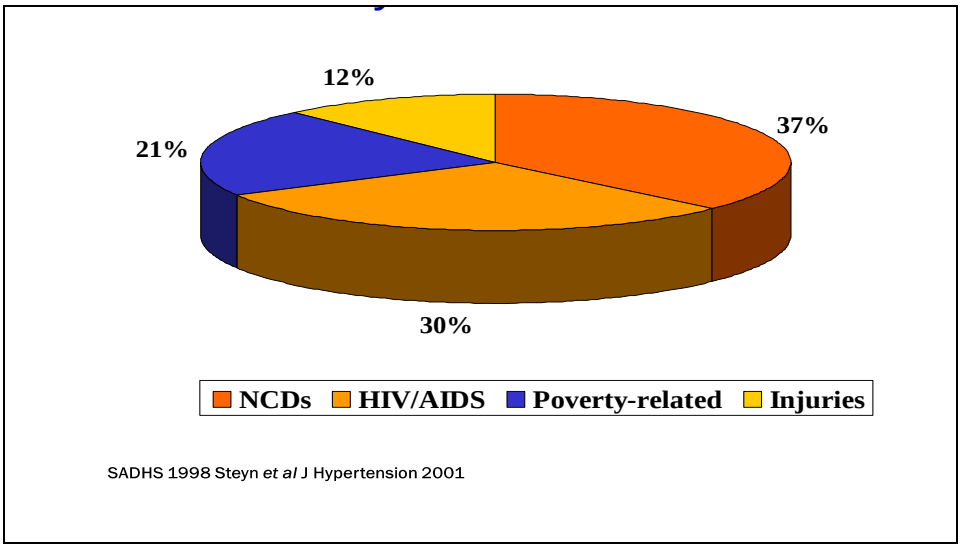
Causes of Death- Global.

World			
Rank	Cause	Deaths (000s)	%
1	Ischaemic heart disease	3371	12.2
2	Stroke	3051	11.1
3	Lower respiratory infections	2014	7.3
4	COPD*	1405	5.1
5	Diarrhoeal diseases	1037	3.8
6	HIV/AIDS	1013	3.7
7	Diabetes mellitus	633	2.3
8	Prematurity and low birth weight	567	2.1
9	Neonatal infections**	546	2.0
10	Hypertensive heart disease	530	1.9

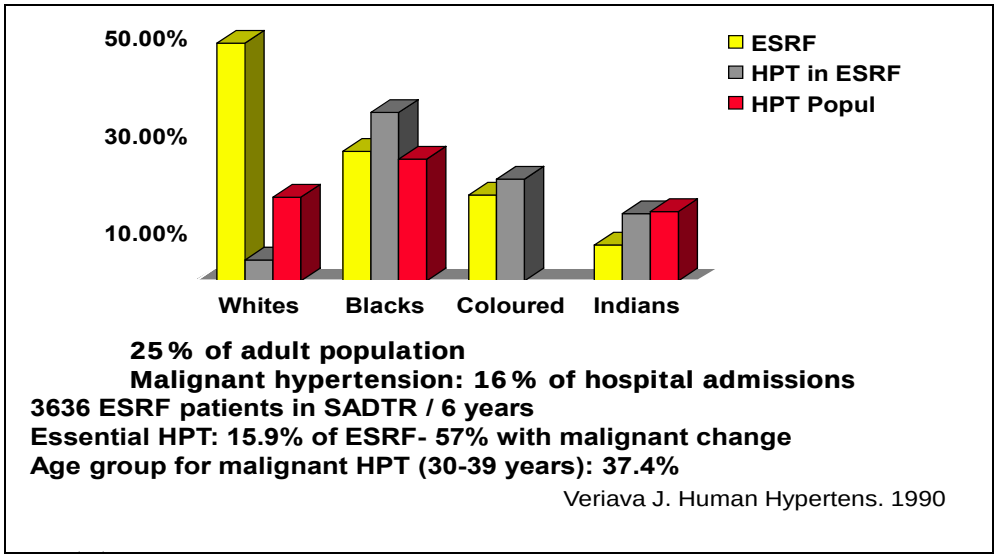
Low-income countries			
Rank	Cause	Deaths (000s)	%
1	Lower respiratory infections	1397	11.4
2	Ischaemic heart disease	1061	8.7
3	Diarrhoeal diseases	851	7.0
4	Stroke	749	6.1
5	HIV/AIDS	742	6.1
6	Maternal conditions	442	3.6
7	Neonatal infections**	426	3.5
8	Prematurity and low birth weight	405	3.3
9	Malaria	404	3.3
10	COPD*	404	3.3

Middle-income countries			
Rank	Cause	Deaths (000s)	%
1	Stroke	1842	16.4
2	Ischaemic heart disease	1659	14.8
3	COPD*	875	7.8
4	Lower respiratory infections	451	.0
5	Hypertensive heart disease	319	2.8
6	Diabetes mellitus	309	2.8
7	HIV/AIDS	264	2.4
8	Breast cancer	231	2.1
9	Stomach cancer	201	1.8
10	Trachea, bronchus and lung cancers	191	1.7

High-income countries			
Rank	Cause	Deaths (000s)	%
1	Ischaemic heart disease	650	15.8
2	Stroke	459	11.2
3	Alzheimer and other dementias	195	4.7
4	Lower respiratory infections	165	4.0
5	Breast cancer	163	4.0
6	Trachea, bronchus and lung cancers	159	3.9
7	Colon and rectum cancers	130	3.2
8	COPD*	126	3.1
9	Diabetes mellitus	123	3.0
10	Hypertensive heart disease	91	2.2

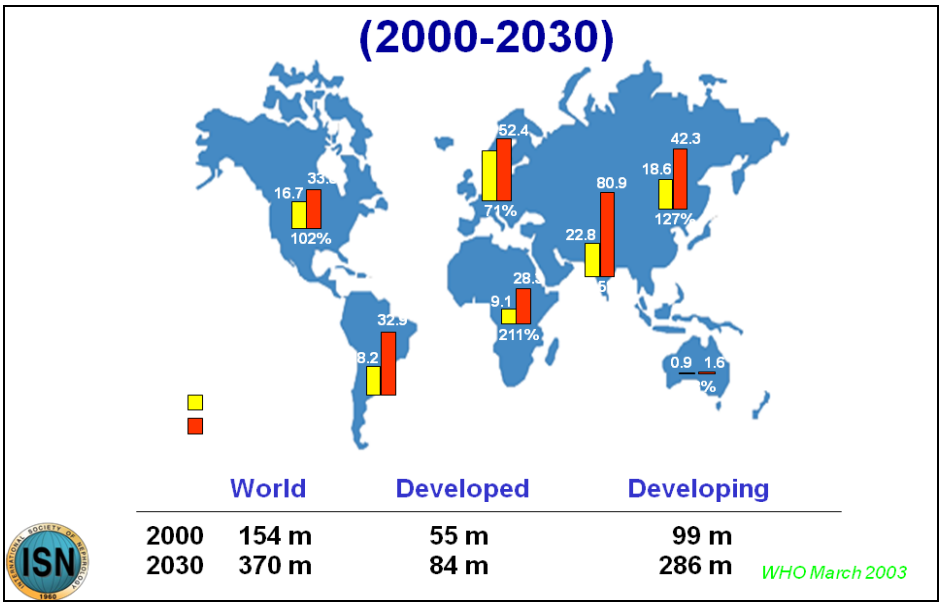


Deaths by cause in South Africa.



Hypertension as a cause of ESRD in South Africa.

The global burden of diabetes (2000-2030).



	World	Developed	Developing
2000	154 m	55 m	99 m
2030	370 m	84 m	286 m

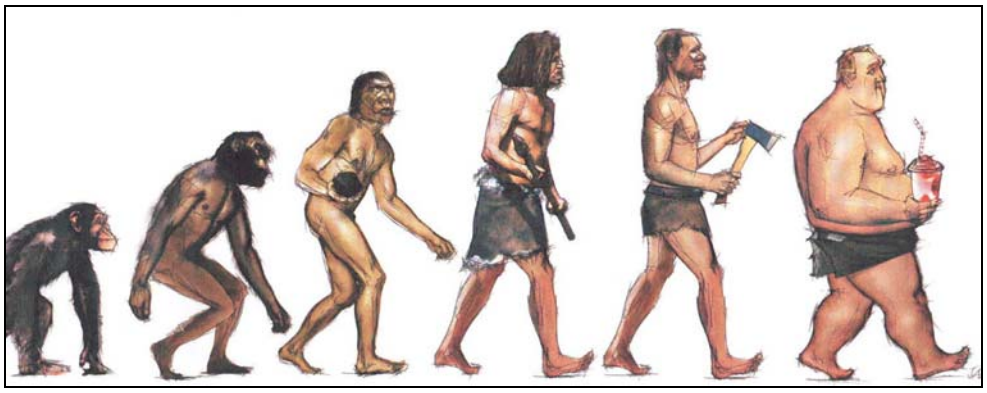
WHO March 2003

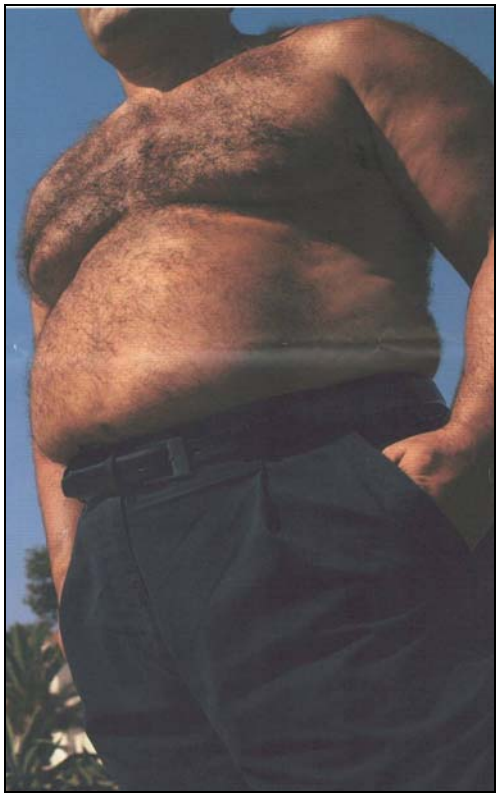
The world health is in transition.

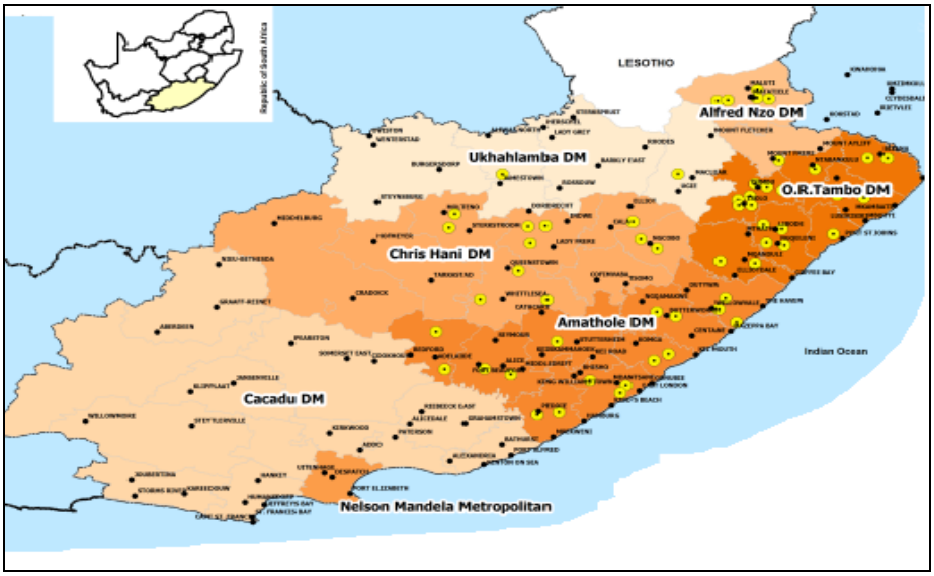
Epidemiological:	**NCD overriding CD, & double burden of diseases in many developing countries**
Demographic:	**Population ageing**
Lifestyles:	**Diets are rapidly changing** **Physical activity reducing** **Tobacco use increasing**
Urbanization:	**Growing cities**
Globalisation:	**Increasing global influences**

World Health Organization

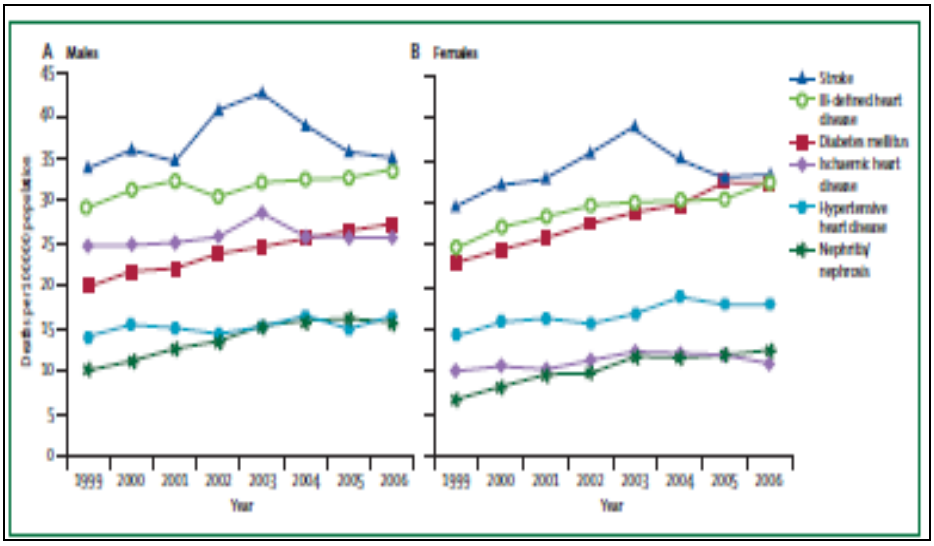
3rd Global Forum on NCD Prevention and Control,
Rio de Janeiro, 9-12 November 2003





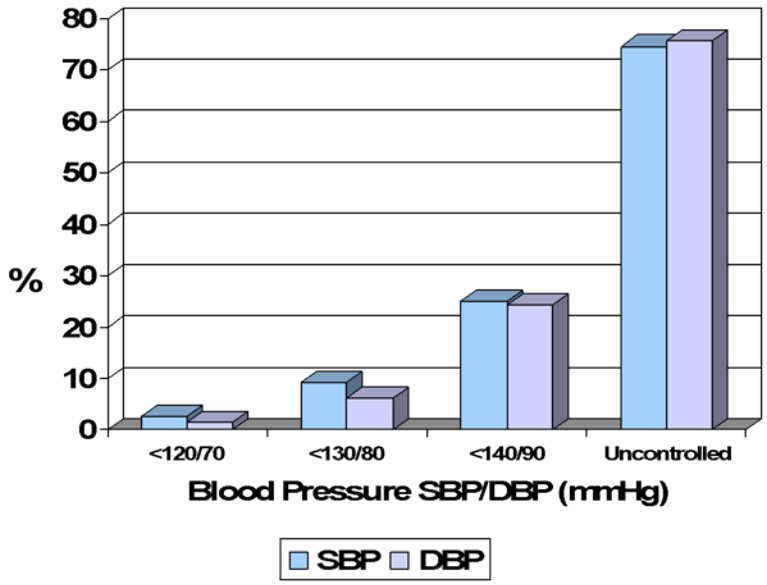


Map of Eastern Cape Province in South Africa with districts.



Mayosi et al. Lancet 2009;374:934-947

Death rate per 100 000 population from cardiovascular and related diseases in 15-64 year old male (A) and female (B) South Africans 1999-2006.



BP control at baseline.

Prevention

The usefulness of these and other data on CVD include planning for primordial, primary, secondary and tertiary prevention in Africa

Additional long term surveillance data to define the burden and distribution of etiologies are necessary in Africa. Education and advocacy to transfer the present results to policymakers, Administrator and Community of WSU, researchers, and population is required.

Africa can overcome its slow response to the CVD epidemic and curb further deterioration by reducing metabolic syndrome (obesity as the perquisites) and, thus, inheritance and clustering of risk factors. This can be achieved via multilayered awareness and intensive parental and familial involvement.

CONCLUSION

Honorable Administrator, I hope that I attempted to present perspectives for the future. As a species, we have ventured well beyond our evolutionary Africa, and the burden of CVD imposed by these environmentally mediated anomalies in our society are the principal challenges for Prevention.

Reasoning in the opposite direction, it is clear that some CVD allows an evolutionary solution while others do not.

Our results and other African research are significantly associated with the emergence of CVD

Among these risk factors, aging, female sex, inappropriate diet, physical inactivity, obesity, HIV infection, smoking, excessive alcohol intake, Helicobacter pylori infection, inflammation, imbalance of oxidative stress, seasons/climate change, malnutrition and low birth weight are suggested as causes of CVD.

Unfortunately, no similar solutions exist for obesity, hypertension and diabetes mellitus. On average, we are programmed from fetal life to adult hood as a species to experience an increase in body weight, blood pressure and blood gloose with age under the conditions of Coca colonization due to modern and Western life.

In this perspective the challenges of CVD in Africa are no different from any other modernized segments of the world of races, but of one Human race with ethnic groups.
While the general biological principles may be surely the same, the social conditions under which they are operating are different. African modifications of preventive strategies will be necessary.

Long-term, Africa-based surveillance projects are needed to define the burden of CVD-both the morbidity and the mortality.

Availability of new tools, including molecular biology, will provide important resources for African researchers in areas from epidemiological approaches and diagnostics to etiological research.

The increase in CVD and its impact has gained paid attention in recent years. Theories of developmental and degenerative determinants of CVD are discussed to provide strong evidence for a causally informed approach to prevention.
Finally, in a renewed research environment of WSU, a developmental University, technological, scientific, innovative and responsive, as the essential long-term goal of my Professorial Inaugural Lecture integrated research on human biology must be successful if we are to construct either evolutionary or non-evolutionary preventive strategies.

GOD BLESS WITH LOVE

ACKNOWLEDGEMENT

As the sun is shining on this day of the Professorial Inaugural Lecture, I would like to thank Walter Sisulu University for giving me the opportunity to serve as Research Professor within the Faculty of Health Sciences. I would also like to thank particularly the Administrator of the Alma Mater, Professor Lourens van Staden for his able and generous Leadership; the Deputy Vice chancellor for Academic Affairs and Research, Professor C.L. Obi for creating a revolutionary conducive academic and Research environment; the Deputy Vice Chancellor for Quality, Professor Buijs for her encouraging and abundant smile during the Coffee Day of the interview for the candidates from the short list; the Director of Research, Professor G. Ekosse, an eminent scientist and physicist for making this special day possible and for his contributions to the success of this Professorial Inaugural Lecture.

I would like to thank the Executive Dean, Faculty of Health Sciences, Professor K. Mfenyana for his Humanitarian Leadership.

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Post-Conference Workshop on Treatment of Tobacco Dependency, Hong-Kong, 27th and 28th November 2006

Panafrican Meeting on Hypertension (AMHY). Yaoundé, Cameroun, 2 – 5 Décembre 2005.

3rd World Assembly on tobacco Counters Health (WATCH). New Delhi, India. 7th – 11th March, 2004.

13ème Conférence Internationale sur le SIDA et les MST en Afrique (CISMA). Nairobi, Kenya, du 21 au 26 Septembre 2003.

12th World Conference on Tobacco or Health. Helsinki, Finland, August 3 - 8, 2003.

Communications:
Cigarette smoking, gender menopause and coronary heart disease in sub-Saharan Africa: a clinic based study. **Longo-Mbenza B**.

Stroke incidence and cigarette smoking in hypertensive Africans. **Longo-Mbenza B.**

Tobacco smoking and other risk factors for coronary heart disease in sub-Saharan Africa: a clinic based study. **Longo-Mbenza B**, Lukoki Luila E.

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Smoking prevalence among Africans workplace:socio-demographic inegalities and cardiovascular risk. **Longo-Mbenza B**.

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14ème World Congress of Cardiology. Sydney, Australia, May 5-9, 2002.

11th World Conference on Tobacco or Health. Chicago, Illinois, USA, August 6-11, 2000.

Global Tobacco Control Scholarship Program, Pre-World Conference Training. Lisle, Illinois, August 2 – 5, 2000.

9th Course of the European School of Medical Genetics. Genoa, Italia, March 24-31, 1996.

African Regional Conference on Medical Education. Cape town, South Africa, 2 – 5 April 1995.

Joint 12th World Congress of Cardiology and 16th Congress of the European Society of Cardiology. Berlin, Germany, September 10-14, 1994.

Eight International Interdisciplinary Conference on Hypertension in Blacks. Cardiovascular Disease on Populations of the African Diaspora. Yaounde, Cameroon, April 7-10, 1993.

The fifth Congress of the Pan African Society of Cardiology (PASCAR). Preventive Cardiology in Africa. Yaounde Cameroon, April 4-9, 1993

Staff Meeting, Département de gynécologie-obstétrique, Cliniques Universitaires de Kinshasa, le 14 sept 1992.

Communication : - Apport de l'échographie et de la vélocimétrie en gynéco-obstétrique. **Longo-Mbenza B.**

Symposium: Meeting of the Working Group Epidemiology and Prevention of European Society of cardiology. Ghent, Belgium, December 11th – 12th, 1992.

1ère soirée d'échographie de Médicis, Kinshasa, le 30 Oct 1992. Communication :- Le Doppler en obstétrique : historique, Intérêt : Ponts de vue du cardiovasculaire. **Longo-Mbenza B**.

Seventh International Interdisciplinary Conference on Hypertension in Blacks.
Clinical trials in Minority populations. Atlanta, Georgia. USA, May 26th – 31th 1992.

Journées scientifiques sur le Président Kasa-vubu.
Thème : Aide à la démocratie. Kinshasa, le 30 juin 1991.
Communication : Méditation sur Kasa-vubu, le sens d'un souvenir. **Longo-Mbenza B**.

National Meeting. Practical echocardiography in 1990. Interactive training sessions on interpretation of Echo-Doppler recordings. Belgian Working Group on Echocardiography and Doppler. University clinics of Mont-Godine, December 8th, 1990.

Activités cardiologiques. BAYER-Forum. Sous les auspices de la Société Belge de cardiologie. Genval, Belgique le 24 Novembre 1990.

Second BALPPM annual Forum Joint Meeting on Psycho-cardiology. Brussels, Belgium, November 17th, 1990.

Troisième journée zaïroise de l'hypertension. Ligue zaïroise contre l'hypertension.
Thèmes : - Education du patient
- Accident vasculaire cérébral
Kinshasa, le 28 avril 1990.

Journées scientifiques de la Faculté de Pharmacie de l'Université de Kinshasa.
Thème : Faculté de Pharmacie 12 ans après, Perspectives d'avenir.
Kinshasa, 27-29 avril 1989.

Deuxième Journée zaïroise
Thème : L'éducation pour la lutte et le contrôle de l'hypertension. Kinshasa, Institut Goethe, le 25 mars 1989.

Communication : Qualité de vie et observance du traitement. **Longo-Mbenza B** et M'Buyamba Kabangu.

Cinquième Séminaire de l'Institut de Cardiologie d'Abidjan. Du 20 au 23 Novembre 1989.

1er Congrès Européen RCP, Anvers, le 3 et 4 Novembre 1989.

15ème Congrès de Cardiologie de langue française. Abidjan, Côte d'Ivoire, 18, 19 et 20 avril 1988.

14ème Congrès de Cardiologie de langue française. Bruxelles, Juin 1986.

International Conference on Preventive Vardiology. Moscow, June 23th – 26th, 1985.
Poster: Prevalence of sickle-cell cardiomyopathy in Zairean adults. **Longo-Mbenza B.**

CITATION

Professor Benjamin Longo-Mbenza was born on 5 January 1951 in Konde-vinda, South-Western Democratic Republic of Congo. He was the sixth child of eight children of Ernest and Elisabeth. He became an orphan at 3 years. His father, an assistant physician in colonial Belgian Congo, died in a road accident. At the liberation of Congo in 1960, his uncle, Mr Joseph Kasa-Vubu became the first President of Congo.

Longo-Mbenza started formal schooling in 1956 at Mbata Ntombo Tshela Catholic Primary School. He wanted to become a Catholic Priest and attended the Seminary High School of Mbata Kiela, where Mr Joseph Kasavuba, the first President of Congo and Josephl Malula of Congo were also trained. When the Matric results were released in 1970, Longo-Mbenza was the top matriculant nationwide with distinction in all subjects. In 1970, he was accepted at the Faculty of Medicine, University of Kinshasa, DRC.

Between 1970 and 1973, Longo-Mbenza obtained a diploma of bachelor of Natural and Medical Sciences at University of Kinshasa with distinction.

The Congolese government granted a scholarship to Longo-Mbenza to study towards a Doctorate in Medicine between 1974 and 1977 at the University of Bucharest, Romania, where he received his degree in 1977. There, he continued his specialization and PhD in pathophysiology between 1977 and 1979 at the University of Bucharest, Romania.

Between 1979 and 1981, he was awarded the Degree of Master of Science in Cardiology at the Free University of Brussels, Belgium.

He attended several training courses in molecular biology and epidemiology at the Catholic University in Leuven, Belgium. He passed the final examination for the Certificate in Molecular and Medical Genetics in 1996 at the European School, Genoa, Italy. Dr Longo-Mbenza collaborated with the Catholic University of Leuven, Belgium, to discover a gene responsible for deafness among Congolese patients.

Professor Longo-Mbenza registered at the Free University of Brussels, Belgium, between 1994 and 1998, presented two theses on cardiovascular diseases in HIV-patients and in rheumatic heart diseases among children. He was awarded the degree of DSc in Cardiovascular Sciences.

Dr Longo-Mbenza's professional working life started as a lecturer from 1981, till 2007 rising through Senior Lecturer, Associate Professor, Full Professor and Outstanding Professor of Medicine at the University of Kinshasa, DR Congo. He served at the University of Kinshasa as Dean of the Faculty of Medicine, the deputy Vice Chancellor, Academic Affairs and the Vice Chancellor.

At the University of Kinshasa, Professor Longo-Mbenza taught general Human Physiology, Anatomy Physiology, Cardiolovascular Physiology, Pathophysiology, renal Physiology, Cardiology, Rehabilitation and Hematology.

In 1980, Professor Longo-Mbenza detected symptoms of Immunodepression among Congolese patients with chronic diarrhea when later Ugandan colleagues spoke about Slim Disease with Trypanosoma, and US CDC discovered AIDS in 1981. Because of the merit of Professor Longo-Mbenza who was awarded a Fulbright Scholarship, he worked in 1985 as a Visiting Professor in Clinical Pharmacology, Hypertension and Lipids at Baylor College of Medicine, Houston, Texas, USA. He worked on Beta-receptors research.

Professor Longo-Mbenza served as a consultant to the World Health Organization and the United Nations for Climate Change. He is a member of New York Academy of Sciences, the American Heart Association, the Belgium Cardiac Society, the French Cardiac Society, the Egyptian Cardiac Society, the Congolese cardiac Society, the Pan African Society of Cardiology, the Human Genome Organization, the International Society on Hypertension in Blacks, the American Society of Blacks cardiologists, the International Hypertension Society and other scientific societies.

Professor Longo-Mbenza's original and prolific research, and accomplished clinical practice were focused on elucidating the pathophysiological mechanisms of toxic myocarditis and immune-allergic cardiomyopathies.

From 1980s until present, most of his research works are related to molecular biology in intestinal parasites in HIV/AIDS, inflammatory states, oxidative stress, genetics, biomarkers and environmental impacts in atherosclerosis, Geophagia, Sickle Cell Disease, HIV/AIDS-related cardiac lesions, diabetes mellitus, and co-expression of genes in pancreas.

In addition to his role as clinician, lecturer and academic leader, Professor Longo-Mbenza has been a very productive scientist: Editorial-Board member of more than 30 International Journals such as Stroke, supervisor of more than 10 PhD theses, 25 MSc theses, 100 mini-dissertations, 6 books, and over 300 papers published in Romanian, Spanish, French, and English languages.

Professor Longo-Mbenza speaks 11 languages and participated in more than 50 international conferences and travel studies on all the continents. He is currently a Research Professor at the Faculty of Health sciences, Walter Sisulu University, Mthatha, South Africa.

With his valuable input, he will learn more about that he thinks.

In the future, Professor Longo-Mbenza will continue to expand his sphere of collaboration with researchers in various disciplines in order to enhance the profile of WSU.

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