

## Prevalence and risk factors of diabetes mellitus in Kinshasa Hinterland

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### Abstract

**Objective:** To estimate the prevalence of diabetes mellitus (DM), impaired fasting glucose (IFG), and impaired glucose tolerance (IGT), and to determine the risk factors of DM among urban and rural areas of Kinshasa Hinterland. **Research, Design and Methods:** Data were collected from a multistage random sample cross-sectional surveys of adult black Africans from Kinshasa region DR Congo with the help of a structured questionnaire, physical examinations and blood samples, using the WHO stepwise approach and the new criteria of WHO to define glucose intolerance. Prevalence rates were adjusted using the standard world population of Waterhouse and the standard population of Kinshasa region. **Results:** A total of 9770 subjects age  $\geq 12$  years participated (response rate of 90.3%) in this study. Age-adjusted rates to world population of IFG, IGT, DM by fasting plasma only, DM by 2h-load test only, and all cases of DM were 9.3%, 9.6%, 16.1%, 8.4% and 25.3%, respectively. Male sex, rural residence, total obesity, abdominal obesity, viral infection, milk intake, and kwashiorkor were the univariate risk factors of all cases of DM. Adjusted for confounders, advancing age, rural-urban migration, physical inactivity, smoking, abstinence of alcohol, low intake of fruits-vegetables, family history of DM, refined sugar intake, high social class, high intake of animal fat and protein, and stress, were the independents determinants of all cases of DM. **Conclusions:** This study observed epidemic rates of glucose intolerance. Primary prevention through lifestyle changes is needed to control DM among Africans under demographic and nutrition transition.

**Keywords:** *Diabetes mellitus, obesity, urbanization, lifestyle, Socioeconomic status, sub-Saharan Africa, Congo.*

### Introduction

Diabetes mellitus (DM), characterized by chronic hyperglycemia is a major global health problem emerging in developing countries. According to the World Health Organization (WHO) Regional Office for Africa, non communicable diseases including DM, will increase so rapidly in Sub-Saharan Africa (SSA) as an epidemic by year 2020.<sup>1</sup> The current prevalence of adjusted DM is estimated to be 14.2% in a small random adult sample (n=250) of Kinshasa Metropolitan area<sup>2</sup> and conflicting with low prevalence rate of 0-9.3% reported earlier in SSA.<sup>3</sup> None of the earlier population-based studies of DM among black Africans confirmed its prevalence with a 75g oral glucose tolerance test according to WHO criteria.<sup>4</sup> Studies reported earlier had limitations such as differing study populations and clinical tests, methodologies and criteria for the diagnosis of DM.

The lack of efficient diagnosis performance in small sizes of populations and information on the relation between sex, age, poverty, environment, lifestyle changes, obesity and

DM, hampers the development and implementation of specific prevention programs.

Indeed, in the Democratic Republic of Congo (DRC), populations are going through demographic change such as aging, better health care and nutrition, sedentary lifestyle and energy rich diet<sup>5,6</sup> after migration from a rural setting to urban regions of Kinshasa, the capital of DRC. In the early stages of the transition, the economically active, urban and richer Africans have carried the highest risk of cardiovascular risk factors (including arterial hypertension and DM).<sup>2</sup> However, as reported by data from Mexico,<sup>7</sup> Brazil,<sup>8</sup> and Chile,<sup>9</sup> there are indications from Kinshasa<sup>2</sup> that the under privileged rural populations will carry the same risks of developing arterial hypertension, a risk factor of DM if they adopt the "bad habits" prevalent in urban areas.

Because DM was determined by fasting capillary blood test, medical history or presence of glycosuria, it is likely that the condition was under diagnosed, in the absence of glucose tolerance test, in SSA and therefore not comparable with global data. What is also highlighted is the relative paucity of information on age-adjusted prevalence rates of DM in both upper-urban, urban and rural settings of SSA.<sup>2,10</sup> The objective of this study was to estimate prevalence rates of

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DM, impaired fasting glycemia (IFG), and impaired glucose tolerance (IGT) among large upper-urban, urban and rural populations of Kinshasa Hinterland. We also examined the association between DM and the consequences of the epidemiologic and nutrition transition. Based on recent information on arterial hypertension (a risk factors of DM) in metropolitan area of Kinshasa,<sup>2</sup> we first hypothesized that in Kinshasa Hinterland (Former Leopoldville Province) including 3 administrative regions of DRC (Kinshasa Metropolitan area, Bas-congo Region and Bandundu Region), a change to a sedentary lifestyle and the recent ethnic and military conflicts with the collapse of economy, might have had a significant effect on the epidemic of DM. We also hypothesized that aging, rural residence, low socioeconomic status, high socioeconomic status, and obesity (total obesity more limited than abdominal obesity) are the risk factors of DM in these communities with different stages of epidemiologic and nutrition transitions.<sup>2,5-9</sup>

## Materials and Methods

### Design of study

This cross-sectional survey was conducted between January and April, 2005 in the Kinshasa Hinterland with 12 million inhabitants.<sup>11</sup> The methodology was based on STEPwise approach<sup>12</sup> which have been described elsewhere.<sup>2,13</sup>

The study protocol was approved by the Kinshasa University School of Medicine Committee of Ethics-confidentiality maintenance and referring screened diabetics to the Primary Care Center. Campaigns of information and sensitization of the general population were delivered during January, 2005, using media (Radio, TV, churches and Tribal Leaders).

### Study population

Classification of the zones and the development status of the Metropolitan and provincial areas was based on a comprehensive development criteria defined by the Congolese government.<sup>14,15</sup> In Metropolitan area with a representative multi-ethnic population including all 432 tribes of DRC, upper urban (westernized subjects) of Gombe zone was selected at random from a list of 12 developed Zones, while rural zone of Kisenso (semi-rural informal housing areas and slums) was selected at random from a list of 12 undeveloped zones. Similarly, in the 2 provincial regions outside of the metropolitan area (regions of Bandundu and Bas-Congo), urban zone of Lukeni in Kikwit city (traditional black locations) was selected at random from a list of 4 developed urban cities (Boma, Matadi, Bandunduville, Kikwit), while deep rural area of Kwilu was selected at random from a list of 12 undeveloped rural zones. Each zone was divided into enumeration districts (ED or localities) which were taken as primary sampling units without a separate selection stage that of selecting the 4 zones.

The survey was specifically and extensively designed using a statistical multistage and stratified random model at each level to recruit a study sample (Fig. 1) with similar and representative characteristics of Kinshasa Hinterland

demographic and socioeconomic structure<sup>2,11,14,15</sup> and results comparable with global data on DM ([www.eatlas.org/media](http://www.eatlas.org/media)).

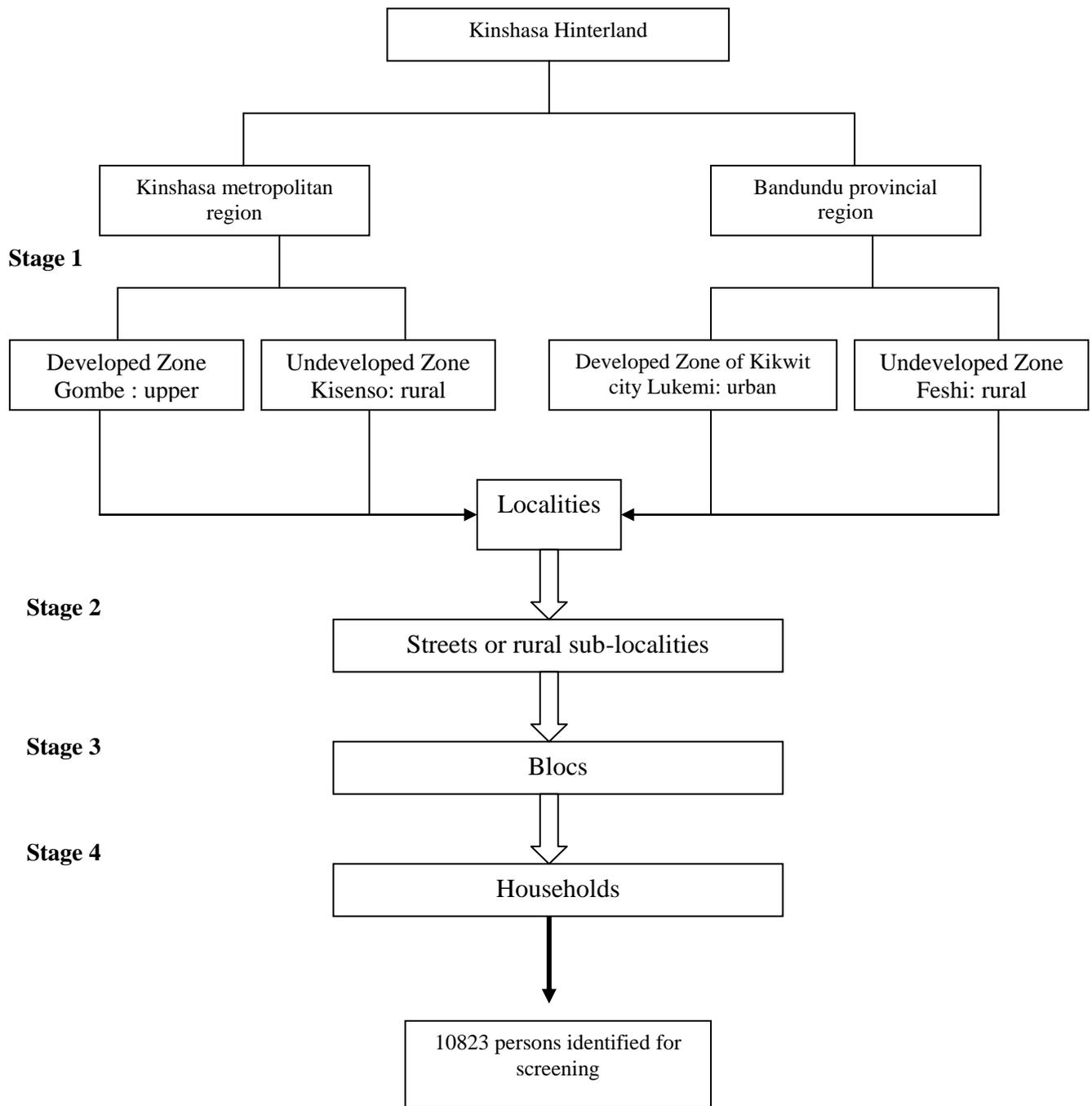
Each region contributed with a number of clusters (localities/location) calculated upon its population number: 185112 inhabitants for upper urban area of Gombe, 161410 inhabitants of rural Kisenso area, 153265 inhabitants for urban Lukemi area and 146034 inhabitants for deepest rural Feshi area. The sample size was calculated ( $Z^2 \times p \times Q \times f/d^2 \times 8.5$  where Z: 1.96 at error risk of 5%; p: expected prevalence of DM in each area; Q: 1-p; d: absolute accuracy of 2%; f: 8.5 to correct design effect). The value of 8.5 for f to correct design effect, a degree to which members of a multistage and complex cluster sample (with design efficiency generally less than that of a simple random sample of the same size, but with other advantages in terms of economy and operational efficiency), was obtained in measuring the intra-class correlation coefficient<sup>16</sup> as follows:

Design Effect (De or f) =  $\text{Varcl}/\text{VarSRS}$  where using Cochran, s formula,  $\text{Varcl}$  (design-based variable =  $126 \times 10 \times \exp 6$ ) is the sampling variance computed using the JRR method,  $\text{VarSRS}$  is the variance computed under the assumption of simple random sampling ( $\text{VarSRS} = 16 \times 10 \times \exp 6$ ), and  $f = 126/16 = 8.5$  the sample size of 6260 subjects was necessary for the metropolitan area at an expected prevalence of 7% and 4563 subjects for those outside of the metropolitan area of 5% according to average results from SSA.<sup>2, 10, 17</sup> At the end of the sampling procedures, all subjects aged  $\geq 12$  years in each selected household and identified by a door-to-door census, were invited to participate for screening between February and April, 2005, the period without intensive agricultural activity.

### Data collection

In the last week of January, 2005, a workshop was held concurrently in Kinshasa city (LOMO MEDICAL Center, Limete) and Kikwit (University of Kikwit) to standardize the protocol and methodology (administering the questionnaires, drawing blood, and taking measurements). Thus, a 3-day training course to use a WHO STEPS approach, specially prepared manual with recommendations for non-communicable diseases survey protocols,<sup>12,18</sup> took place for the investigators (diabetologists, cardiologists, physicians, trained nurses and medicine students) in each setting before the survey.

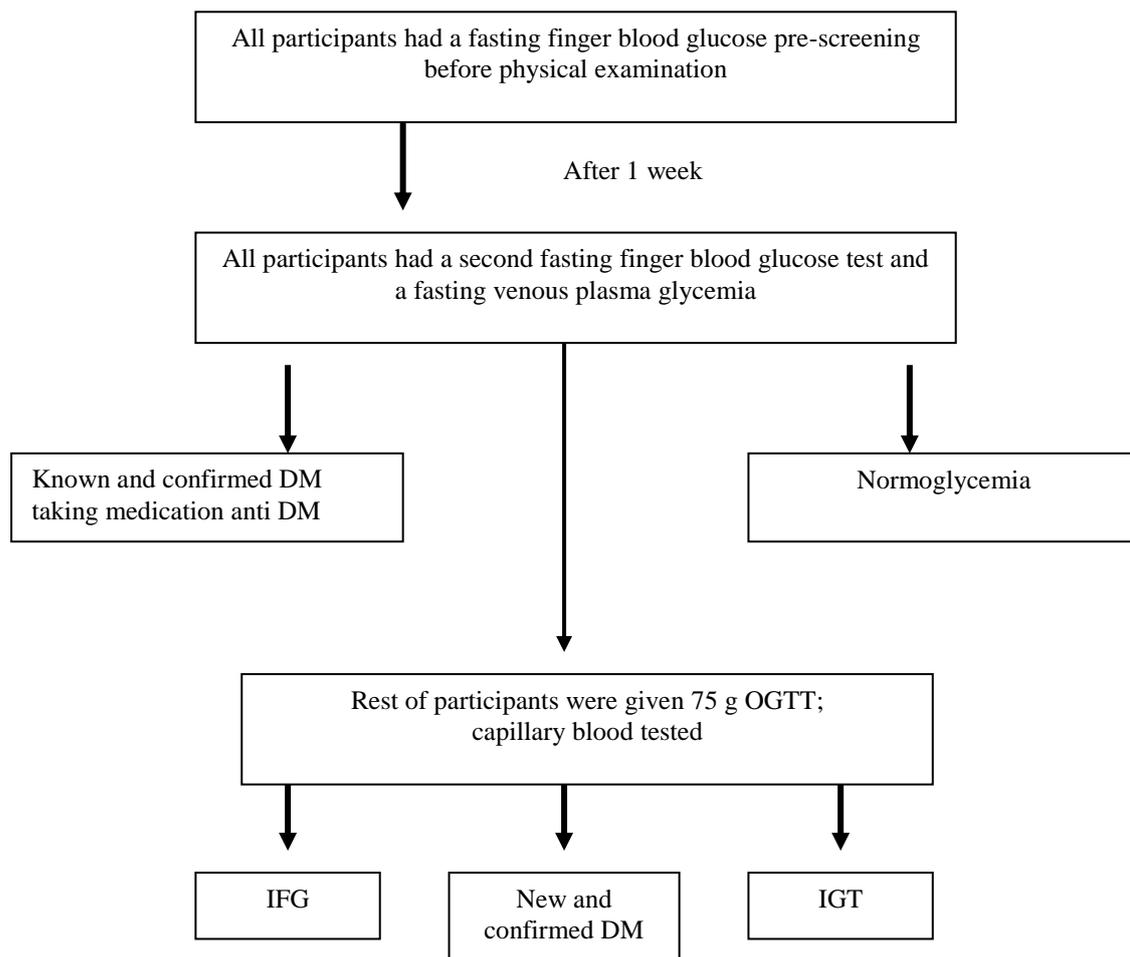
After verbal informed consent was obtained, participants were taken through a structured questionnaire (a 30 min session). The session was recorded the day before physical examination. Information on demographic data (age, sex), smoking habit, excessive alcohol intake, physical activity, residence environment (upper urban, urban, rural, deepest rural), diet (tea and milk, fruits-vegetables protein intake), means of transportation, stress, degree of exercise, health history, education level, monthly income, and time of sitting position in office or watching television.



**Figure 1:** Methodology of probabilistic sampling design.

**Table 1:** World and study populations for calculating adjusted prevalence of diabetes mellitus

Age groups in years	Reference population (N) World N	Proportion of N(a)	Study population (n)	Proportion of n (b)	Adjusting coefficient (a/b)
12 – 19	13500	18.4	420	0.043	427.3
20 – 39	28000	38.1	2510	0.26	148.3
40 – 59	21000	28.6	5250	0.537	53.2
60 – 74	9000	12.2	1260	0.129	95
75 – 98	2000	2.7	330	0.034	80.6
Total	73500	100	9770	1	



**Figure 2:** Design diagram to define impaired glucose regulation during the survey.

#### ***Anthropometric measurements***

Body weight in light clothes was measured to the nearest 100g (0.1 kg) using a Soehnle scale (Soehnle-Waagen GmbH Co, Murrhardt, Germany) and height to the nearest 0.5 cm using portable locally manufactured stadiometers. Participants stood upright on a flat surface without shoes, with the back of the heels and the occiput on the stadiometer. Body mass index (BMI) was calculated as weight divided by height squared ( $\text{kg}/\text{m}^2$ ). Waist circumference (WC) was measured after gentle expiration between the lower rib margins and the iliac crest to the nearest millimetre using a flexible tape, with subjects standing with their heels together. Standardized protocols were used to measure body weight, height and waist circumference<sup>19-21</sup> with appropriate validation and quality control procedures.

#### ***Laboratory methods***

Participants were instructed and encouraged to fast from 19 h: 00 on the evening preceding laboratory measurements according to the design diagram (Fig. 2). Each morning before physical examination, blood samples were collected between 7- 9 h, by finger puncture for all participants, after a 10-12 h of overnight fast. A second capillary whole blood sample drawn from all participants, was obtained one week

later under similar conditions. Both capillary whole blood tests were immediately analyzed using a hemocure blood glucose analyzer (Hemocure<sup>R</sup> AB, 26223 Angelholm, Sweden). After the second capillary whole blood test, fasting blood samples were drawn from the cephalic vein by registered nursing sisters using a sterile butterfly infusion set and disposable syringes. Samples were centrifuged in the field using Universal 16R Hettich centrifuges with cooling facilities. Aliquots of separate plasma from 10 ml venous blood were drawn into a bottle containing the anticoagulant sodium EDTA. The sample was centrifuged immediately in the field using the hexokinase-glucose-6 phosphate dehydrogenase reaction<sup>22</sup> on commercially kits (Biomérieux, Marcy l'Etoile, France) and a Hospitex autoanalyzer (Hospitex Diagnostics, Florence, Italy).

Participants with known DM (medical history of DM confirmed by a record review conducted at the offices of their Primary Care Centers) not taking insulin or oral diabetic medication and those with one fasting blood glucose  $> 5.6$  mmol/L among the 3 fasting tests (2 capillary tests and 1 plasma test) underwent a 2 h, 75g oral glucose tolerance test (OGTT) using a fingerstick and capillary blood analyzed by the same Hemocure<sup>R</sup> AB devices.

**Table 2:** Crude and age-standardized prevalence rates of impaired glucose regulation (IGR) in the study population

IGR	n	Crude rate % (95%CI)	Age-adjusted rate to Kinshasa population % (95%CI)	Age-adjusted rate to World population % (95%CI)
IFG	1030/9770	10 (9.9 – 10.1)	8.3 (8.2 – 8.5)	9.3 (9.2 – 9.4)
IGT	670/8040	8.3 (6.6 – 10.5)	7.4 (7.2 – 7.6)	9.6 (9.3 – 9.8)
DM by fasting plasma	1590/9770	16.3 (14.1 – 18.9)	12.4 (12.3 – 12.5)	16.1 (15.8 – 16.3)
DM by OGTT only	850/8040	10.6 (8.6 – 13)	5.8 (5.7 – 5.9)	8.4 (8.2 – 8.6)
All diabetics	2600/9770	26.6 (23.9 – 29.5)	20.5 (20.4 – 20.6)	25.3 (25 – 25.6)

The intra-assay coefficient of variation (CV) for glucose measurements was 1.3% and an intra-assay CV of 1.6%.

### Definitions

Absence of DM (normoglycemia) was defined in participants with concordant 3 fasting tests (2 capillary tests and 1 plasma test)  $\leq 5.6$  mmol (100 mg/dL) or those with 2 h post-glucose load in whole capillary blood  $< 7.8$  mmol/L ( $< 140$  mg/dL). Impaired fasting glucose (IFG) with concordant capillary and plasma glycemia  $\geq 5.6$  mmol/L (100 mg/dL) and  $< 7$  mmol/L ( $< 126$  mg/dL), impaired glucose tolerance (IGT) with both concordant capillary and plasma glycemia  $\geq 5.6$  mmol/L and  $< 7$  mmol/L and 2 h post-glucose load in capillary blood  $\geq 7.8$  mmol/L ( $\geq 140$  mg/dL), not confirmed new DM with concordant fasting capillary and plasma glycemia  $\geq 7$  mmol/L ( $\geq 126$  mg/dL) but 2 h post-glucose load in capillary blood  $< 11.1$  mmol/L,

**Table 3:** Age-specific and age-standardized to World population of all cases of DM in all participants, men and women

Age-group (years)	All %	Men %	Women %
12 – 14	16	28.5	0
15 – 24	18.5	23.5	13.5
25 – 34	13.1	13.4	12.9
35 – 44	28.6	29.9	27.5
45 – 54	18.3	18.5	18.1
55 -64	41.4	37.7	44.5
65 - 98	33.4	36.1	30.6
P for trend	$< 0.0001$	$< 0.0001$	$< 0.0001$

**Table 4:** Age-specific and age-adjusted to world population rates of IGT, DM by fasting plasma glucose, and DM by OGTT

Age-group (years)	IGT%	DM by OGTT	DM by OGTT %
12 – 14	0	0	0
15 – 24	9.6	12	1.5
25 – 34	3.9	7.5	6.1
35 – 44	9.2	18.5	10.8
45 – 54	4.1	11.5	6.9
55 -64	15	26	18.5
65 - 98	15.5	17	13.5
P for trend	$< 0.0001$	$< 0.0001$	$< 0.0001$

confirmed new DM by but 2 h post-glucose load in capillary blood  $\geq 11.1$  mmol/L ( $\geq 200$  mg/dL), and known DM defined impaired glucose regulation (IGR).<sup>23</sup>

Potential risk factors of DM were defined as sex (men vs. women), aging (age  $\geq 40$  years vs.  $< 40$  years), rural-urban migration, physical inactivity,<sup>18,24</sup> sedentary lifestyle such as motorised means of transportation to work, sitting position in office/house  $\geq 9$  hours, physical exercise times/week x hours each time x intensity in METS x body weight  $< 600$  kcal/week), urban vs. rural residence environment, education (low for  $< 12$  years), monthly income from all sources (low for  $< 600$  USD), low intake of fruits-vegetables for  $<$  consumption of 4 portions of fruits-vegetables/week,<sup>18</sup> stress,<sup>18</sup> tea and milk intake at breakfast vs. other (coffee, traditional habitual cassava or maize meal), social inequalities in terms of socioeconomic status (SES), animal fat and protein intake vs. no intake, current cigarette smoking (yes vs. no), personal histories of malnutrition/kwashiorkor or viral parotiditis in childhood, current malnutrition (BMI  $< 18.5$  kg/m<sup>2</sup>) or total obesity (BMI  $\geq 30$  kg/m<sup>2</sup>),<sup>21</sup> abdominal obesity defined by WC  $\geq 102$  cm for men and WC  $\geq 88$  cm for women according to NCEP-ATPIII thresholds<sup>25</sup> and arterial hypertension (blood pressure  $\geq 140/90$  mmHg)<sup>26</sup> from the average of three readings separated by 2 min each, and/or medical history of hypertension.

The levels of SES (high for Tertile 3, moderate for Tertile 2, and low for Tertile 1) were defined using a composite scale value scores<sup>2</sup> according to the stage of urbanization (westernization, electricity, water supply, types of houses), income, and education level. Globally, urban residence was defined for the participants living in Kinshasa Metropolitan area vs. rural residence in those living outside the Metropolitan area for more than 1 year, respectively.

### Statistical analysis

Data were as means  $\pm$  standard deviation for continuous variables and proportions (%) with 95% confidence interval (95%CI) for qualitative variables.

The prevalence estimates for DM were age-standardized to the Kinshasa Hinterland population<sup>11</sup> using the adjusted sample weight, the non response weight and the population

**Table 5:** Determinants of the prevalence of all cases of DM: Multivariate analysis

Independent variables	Standard error	OR (95%CI)	P value
Rural-urban migration	0.226	4.2 (2.7 – 6.5)	<0.0001
Stress	0.273	2.8 (1.6 – 4.7)	<0.0001
Physical inactivity	0.188	1.6 (1.1 – 2.3)	0.011
Alcohol intake	0.193	0.6 (0.4 – 0.9)	0.013
Low intake of fruits/V	0.241	1.7 (1.04 – 2.7)	0.034
Refined sugar intake	0.205	2.5 (1.7 – 3.7)	<0.0001
Cigarette smoking	0.201	1.6 (1.1 – 2.4)	0.020
Family history of DM	0.233	1.8 (1.2 – 2.9)	0.009
High intake of animal fat and protein	0.365	2 (1.01 – 4.2)	0.05
Affluent highest social class	0.311	2.8 (1.5 – 5.2)	<0.001
Age – group			
≥ 60 years		1 (Referent)	<0.0001
< 20 years	0.539	3.4 (1.2 – 9.8)	0.022
20 – 39 years	0.290	3.8 (2.2 – 6.8)	<0.0001
40-59 years	0.228	2.6 (1.7 – 4.1)	<0.0001
Constant	0.433		<0.0001

weight as recommended by the WHO STEPS surveillance<sup>12</sup> and published in detail elsewhere<sup>2</sup>. Crude prevalence of DM was also age-adjusted to the World population<sup>27</sup> using the direct method shown in Table 1.

The Chi-squared tests were used to test for significance of observed univariate associations. The Cochran-Mantzel-Haenszel statistic and the estimates of Odds ratios (OR) were calculated with their 95%CI CIs. The adjusted logistic regression considered four categories of ages (<20 years, 20-39 years, 40-59 years, and ≥ 60 years) among all the potential risk factors.

A value of P<0.05 was considered significant. All data analyses were performed with SPSS for Windows version 13 (SPSS Inc, Chicago, IL, USA).

## Results

A total of 9770 individuals, aged 12 years and above, among the 10823 selected subjects, agreed to participate and provided informed consent (response rate of 90.3%). The sex ratio was 1 male: 1.1 female (4580 men and 5190 women) and the mean age was 46 ± 15 years (range 12 to 98 years). The representation rate was higher in urban residents (71.5%; n=6990) than in rural residents (28.5%; n=2780).

Table 2 presents crude, age-adjusted to Kinshasa Hinterland population and age-adjusted to World population prevalence rates of IFG, IGT, DM confirmed only fasting plasma glucose, DM diagnosed only by 2h, 75g OGTT, and all diabetics (fasting plasma glucose and/or 2h, 75g OGTT).

Compared to women, men had higher age-adjusted to World population rates of all diabetics (23.7% vs. 17.7%; P<0.0001), DM by fasting plasma glucose (12.8% vs. 12.1%; P<0.0001), and DM by OGT only (7.1% vs. 4.6%; P<0.0001). However, women had higher age-adjusted to World population rate of IGT (8.2%; P<0.0001) than men (6.4%). Age-adjusted to World population rate of IFG of men (9.5%) was similar (P>0.05) with that of women (9.2%) and not influenced by the majority of variables.

Age-adjusted to World population rates of all cases of DM increased significantly with advancing age in all participants, men and women, respectively (Table 3). All cases of DM were peaked at 37.7 - 44.5% among participants age of 55-64 years. All cases of DM were less common in women aged 12 – 54 years, whereas women aged ≥ 55 years had higher rates of all cases of DM than men.

Both age-adjusted to World population rates of IGT, DM by fasting plasma glucose, and DM by OGTT were significantly increased with advancing ages of the study population (Table 4).

Compared to urban residents, rural resident had higher age-adjusted rates of all cases of DM (27.3% vs. 19.5%; P<0.0001), DM by fasting plasma glucose (17.2% vs. 11.7%; P<0.0001), and DM by OGTT (12.1% vs. 4.8%; P<0.0001), respectively. However, IGT was more prevalent in urban area (7.5%; P<0.0001) than in rural area (7%).

There was a significant and negative association between IGT and SES (5% in high SES, 7% in moderate SES, and 8.6% in low SES; P for trend <0.0001), whereas DM by fasting plasma glucose (17.2% in high SES, 15.4% in moderate SES, and 10% in low SES; P for trend <0.0001), DM by OGTT (12.1% in high SES, 7.4% in moderate SES and 3.7% in low SES; P for trend <0.0001), and all cases of DM (27.3% in high SES, 21.5% in moderate SES, and 18.6% in low SES; P for trend <0.0001) were positively associated with SES, respectively.

Both total obesity and abdominal obesity were positively associated with DM: all cases of DM (32% in total obesity vs. 18.9% in non obese; P<0.0001 and 90.4% in abdominal obesity vs. 18.1% in non obese; P<0.0001), DM by fasting plasma glucose (25% in total obesity vs. 10.6% in non obese; P<0.0001 and 59.2% in abdominal obesity vs. 10.8% in non obese; P<0.0001) and DM by OGTT (16.7% in total obesity vs. 5.4% in non obese; P<0.0001 and 74.4% in abdominal obesity vs. 4.6% in non obese; P<0.0001).

However, absence of total obesity (7.5 vs. 6.7% in obese;  $P < 0.0001$ ) and abdominal obesity (9.5% vs. 7.4% in non obese;  $P < 0.0001$ ) were positively associated with IGT, respectively.

Age  $\geq 40$  years, stress, physical inactivity, smoking, low intake of fruits-vegetables, refined sugar intake, tea and milk intake, high animal fat and protein intake, family history of DM, personal history of viral parotiditis and protein malnutrition/Kwashiorkor, but not hypertension and malnutrition ( $BMI < 18.5 \text{ kg/m}^2$ ), were also identified as univariate risk factors of all cases of DM.

The multivariate analysis with logistic regression model showed that Metropolitan and westernized area, stress, physical inactivity, low intake of fruits-vegetables, refined sugar consumption, cigarette smoking, family history of DM, high intake of animal fat and protein, affluent and highest social class (combined high and moderate SES), age  $< 20$  years, age 20-39 years, and age 40-59 years were significantly and independently associated with the presence of all cases of DM, whereas alcohol intake was significantly and independently associated with protection against DM after adjusting for sex, arterial hypertension, total obesity, abdominal obesity, tea and milk, personal history of viral parotiditis and kwashiorkor (Table 5).

Out of all diabetics, only 6.5% ( $n=170$ ) were aware of their DM. The majority of participants with known DM (78.8%) were not treated diabetics (100%) had poor control of fasting glycemia ( $\geq 126 \text{ mg/dL}$  or  $\geq 7 \text{ mmol/L}$ ).

## Discussion

To our knowledge, this is the first African study to examine the prevalence rates of DM, IFG, and IGT based on standardized WHO criteria for glucose tolerance,<sup>23</sup> World standard population,<sup>27</sup> and Kinshasa Hinterland population in 2001.<sup>11</sup> Thus, this survey has documented much higher prevalence rates of DM, IFG and INGT within a representative sample (similar to demographic/socioeconomic structure to that of 2001 census for Kinshasa Hinterland province). Univariate and multivariate (determinants) risk factors of DM were also identified.

### *Prevalence rates of DM, IFG, and IGT*

The study ensured reliable estimates of prevalence rates of DM, IFG and IGT, which were highly epidemic in the adult African from Kinshasa Hinterland population compared with previous studies conducted in SSA.<sup>2,3,10,17</sup> The diagnosis of IGT was characterized by introducing repeated capillary glucose pre-screening before the application of fasting plasma glucose test and an OGTT using capillary whole blood. These techniques have many advantages: cases of DM undetected by capillary and plasma fasting tests were captured by OGTT. The yield of IGT at OGTT was maximized in a large population-based, high-risk sample identified by raised postprandial glucose. However, previous African studies<sup>2,10,17,28</sup> reported lower prevalence rates because of differences in the methodology in terms of

age, group examined, the use of only fasting blood glucose, and the use of reflectance meters as opposed to formal laboratory blood glucose measurements.

The present study has shown that the prevalence of abnormal glucose tolerance has tripled in 10 years. Therefore, the question “Is the prevalence of diabetes increasing in Africa?” raised by South African researchers Motala Ayesha, Omar Mahomed, and Fraser Pirie<sup>29</sup> is easy to answer using the present data. Our data shows that the degree of urbanization/westernization and lifestyle changes is a clear determinant factor in the increase in the number of diabetic patients. These findings will provide baseline data for the planning of health care, the establishment of medical priorities and the preparedness for tracking the present diabetes epidemic. In terms of a global threat of DM in the world, these findings confirm that the major increase of the burden of DM will be in developing countries, which will contribute to  $>75\%$  of the world’s diabetic population.<sup>30</sup> The DRC should be included among the top 10 countries in prevalence of DM worldwide and after Nauru with 30.7% of diabetes ([www.eatls.idf.org/media](http://www.eatls.idf.org/media)).

The changes in the absolute levels of DM, IFG, arterial hypertension, obesity and atherosclerotic diseases in Kinshasa region<sup>2,31</sup> are likely to have been driven by major sociodemographic (collapse of economy, civil wars), nutritional and lifestyle changes over the last two decades, particularly, aging population (demographic transition),<sup>6,7</sup> high salt, refined sugar and animal fat and protein intake (nutritional transition),<sup>5,7-9</sup> sedentary lifestyle, obesity, westernization (rural-urban migration) and psychosocial risk factors.<sup>13-15,31</sup> A significant interaction between genetic (non modifiable) and modifiable environmental factors in the initiation and progression of DM in this survey and other studies<sup>10</sup> has been observed.

### *Univariate risk factors of DM*

Only univariate analyses showed a significant and positive association between male, sex, rural residence, SES gradient (dose-response relationship), total obesity, abdominal obesity, personal history of viral parotiditis in childhood, personal history of malnutrition/Kwashiorkor in childhood, and DM diagnosed by fasting plasma glucose and OGTT only as well as confirmed by OGTT (all cases of DM). They showed however, a significant and positive relationship between female sex, urban residence, abdominal obesity, absence of total and IGT, but a significant and negative association between SES and IGT.

The apparent male preponderance in DM reported from this study confirms those observed in rural and Cameroonians<sup>32</sup> and rural Tanzanians.<sup>33</sup> A female vulnerability was found in South Africa (urban), Cameroon (urban)<sup>32</sup> and Tanzania (rural).<sup>35</sup> Discernible trend in gender distribution of DM (equal distribution) was reported from South Africa (urban Cape town),<sup>36</sup> Tanzania (urban)<sup>35</sup> and global estimates (37). Menopause may explain the peak of DM observed in women aged  $\geq 55$  years in comparison with men aged  $\geq 55$  years.

The higher risk of DM for rural residents and that of IGT for low SES (including rural area in its definition) accords with the reported vicious cycle interconnecting chronic non communicable diseases (including DM and arterial hypertension) and poverty,<sup>2</sup> as the chronic disease burden is concentrated among the poor.<sup>38,39</sup> Furthermore, this study indicates that as the nutrition transition progresses, the burden of cardiovascular disease risk factors (including DM, hypertension, obesity, IGT, smoking) shifts to the poor rural residents.<sup>2, 7-9</sup>

Viruses (Coxsackie-B variant, congenital cytomegalovirus or rubella infection) have long been implicated as possible environmental determinants in Type 1 DM through beta-cell destruction,<sup>40,41</sup> immunological abnormalities,<sup>42</sup> molecular mimicry limited to the HLA-DR3 positive subpopulation of type 1 diabetic patients,<sup>43</sup> virus-induced immune reactions with expression of HLA class II molecules with subsequent antigen presentation.<sup>44</sup> However, a protective effect of measles vaccination has also been described.<sup>45</sup>

One of the major changes in the provisional World Health Organization Consultation report was the disappearance of the Malnutrition Related Diabetes Mellitus (MRDM) as a major category.<sup>46</sup> Whilst the protein-deficient pancreatic diabetes (PDPD) variant of MRDM has been dropped, in our tropical regions protein malnutrition/kwashiorkor in childhood or excessive alcohol intake/denutrition may be associated with fibrocalculous pancreatic diabetes (FCPD) variant, a current part of the other types category. This is clear through the lack of significant association between current malnutrition (BMI < 18.5 kg/m<sup>2</sup>) and DM in the present study. However, our previous Kinshasa population-based studies<sup>47,48</sup> have shown that low birth weight (fetal malnutrition) low SES, and current protein malnutrition were significantly associated with arterial hypertension and insulin resistance, risk factors of type 2 DM.

#### **Multivariate analyses**

The logistic regression analysis showed that several urbanization/westernization/globalization consequences, aging, and family history of DM could account as independent risk factors for the present epidemic rise in prevalence of DM in general (and for type 2 DM in particular), while alcohol intake in social moderation (median intake 3 glasses of beer/day) could have a protective effect.

As reported in other regions of the world including Africa,<sup>17,30,36,37</sup> the present prevalence of DM increased with age, both in men and women. The peak prevalence of DM occurred at age 55 – 64 years with a decline after age 65 years, probably because of greater mortality amongst diabetes subjects or the underrepresentation of subjects in the sixth-seventh decades for young populations in developing countries. The present findings suggest that the examined population is under demographic transition.<sup>6,14</sup>

The significant and positive association between family history of DM and DM prevalence has been reported in

other African studies<sup>17,36</sup> specifically in urban Xhosa ethnic group from Cape town, South Africa.<sup>36</sup>

The present survey highlighted the consequences of urbanization, industrialization, globalization, acculturation/westernization, nutrition transitions<sup>5-9</sup> in terms of rural-urban migration, social inequalities, stress and lifestyle changes (sedentary live/physical inactivity, low intake of fruits-vegetables, refined sugar intake, cigarette smoking, high intake of animal fat and protein) on the epidemiology of Type 2 diabetes. There is now a considerable amount of evidence to suggest that rapid acculturation, sedentary lifestyle, and moving away from the traditional carbohydrate-based diet to one that is rich in fat and protein content are associated with increased rates of type 2 DM.<sup>7,10,29</sup> The high rates of DM in subjects with rural-urban migration suggest that environmental factors that accompany migration might also be important. This is well established in migrant Asian Indians.<sup>29,49</sup>

A light to moderate intake of alcohol is associated with enhanced insulin sensitivity.<sup>50</sup>

As reported from our previous survey in the Metropolitan area of Kinshasa region,<sup>2</sup> there is no significant association between DM and arterial hypertension in these Central Africans from Kinshasa Hinterland (including Metropolitan and outside of Metropolitan areas of Kinshasa province). This probably reflects the significant role of other risk factors of DM in these black Africans as incidence of type 2 DM was elevated 2-fold in white Italians with hypertension after univariate analyses but not after multivariate analyses.<sup>51</sup>

#### **Implications**

The ultimate aim of identifying modifiable environmental risk factors for DM lies in the hope of preventing the disease.

There is an urgent need for health leaders to modify lifestyle changes including exercise and diet which may have important public health significance in reducing adult-onset DM. There are clear health benefits to many interventions that do not save money.

The application of the present information could make a major, rapid, and cost-effective contribution to the prevention and control of DM.

#### **Limitations**

Despite its cross-sectional design, this study avoided many limitations through the large sample size representative of a large survey area, higher response rate, sensitization, and 2-h load glucose test to define DM. However, it was difficult to define urbanization, the amount of alcohol intake, the nutrient intake, and the close associations between different dietary characteristics.

The difficulties are recommended by the fact that many of the lifestyle-related factors are linked with the development

of obesity and arterial hypertension, which in turn are determinants of the risk of developing DM.

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### References

1. World Health Organization. Non communicable diseases: A strategy for the African region. WHO Regional Office for Africa, Harare, 2000
2. Longo-Mbenza B, Vangu Ngoma D, Nahimana D, et al. Screen detected and the WHO Stepwise approach to the prevalence and risk factors of arterial hypertension in Kinshasa. *Eur J Cardiovasc Prev Rehabil* 2008 ; 5:503-508.
3. Motala AA. Diabetes trends in Africa. *Diabetes Metab Res Rev* 2002;18 (Suppl.3): S14-20.
4. World Health Organization. Diabetes mellitus: Report of a Study group. Geneva, WHO (Tech. rep. ser. No. 727), 1985.
5. Popkin BM. An overview on the nutrition transition and its health implications: the Bellagio meeting. *Pub Health Nutr* 2002; 5: 93-103.
6. Omran AR. The epidemiologic transition, the key of the epidemiology population change. *Milbank memorial Fund* 1971;49: 509-538.
7. Riviera JA, Barquera S, Camirano F, et al. Epidemiological and nutritional transition in Mexico: rapid increase of non-communicable chronic diseases and obesity. *Pub Health Nutr* 2002;1: 113-122.
8. Monteiro CA, Conde WL, Popkin BM. Is obesity replacing or adding to under nutrition? Evidence from different social classes in Brazil. *Pub Health Nutr* 2002; 5:105-112.
9. Albala C, Vio F, Kain J, Uauy R. Nutrition transition in Chile: determinants and consequences. *Pub Health Nutr* 2002; 5: 123-128.
10. Balde NM, Diallo I, Balde MD, et al. Diabetes and impaired fasting glucose in rural and urban populations in Futa Jallon (Guinea): prevalence and associated risk factors. *Diabetes Metab* 2007; 33: 114-120.
11. Democratic Republic Of Congo. National survey of children and women situation. MICS2 2001. Say yes for children, Kinshasa, USAID/WHO/UNICEF 2002; 40-46.
12. WHO: WHO/NMH/CCS/03.04. STEPS planning and implementation. The WHO STEP-wise approach in the management of non communicable diseases and mental health, 2006
13. Kasiam Lasi On’Kin JB, Longo-Mbenza B, et al. Survey of abdominal obesities in an adult urban population of Kinshasa, Democratic Republic of Congo. *Cardiovasc J Afr* 2007; 18: 300-307
14. Ngondo A, Pitshandenge S. Demographic perspectives of Zaire during 1984 – 1999. *Cedas* 1984; 1: 31-32.
15. National Program of Nutrition. Nutrition survey in Kinshasa Hinterland region, March 2004. Democratic Republic of Congo, Ministry of health 2004; 100-106.
16. Kish L, Frankel MR. Inference from complex samples. *J Royal Stat Soc* 1974; 36:1-37.
17. Elbagir MN, Eltom MA, Elmahadi EMA, et al. A population-based study of the prevalence of diabetes and impaired glucose tolerance in adults in Northern Sudan. *Diabetes Care* 1996; 19: 1126-1128.
18. World Health Organization. The WHO STEPwise approach to surveillance of non communicable diseases (STEPS)-A framework for surveillance. World Health Organization, Geneva 27, 2003.
19. Lohman TG, Roche AF, Martorell R. Anthropometric standardization reference Manual Champaign, IL, Human kinetics 1998; 3: 39-70.
20. Wang J, Thornton JC, Bari S, et al. Comparison of waist circumferences measured at four sites. *Am J Clin Nutr* 2003; 77:379-384.
21. WHO: OBESITY. Preventing and managing the global epidemic: report of WHO consultation on obesity, 3-5 June, 1997, Geneva: WHO, 2006.
22. Kunst A, Draeger B, Ziegenhorn J. UV-methods with hexokinase and glucose-6-phosphate dehydrogenase. In: Bergmeyer H (Editor): *Methods of enzymatic analysis*. Durfield: Verlag Chemie, 1983.
23. The expert committee on the diagnosis and classification of diabetes mellitus. Report of the expert committee on the diagnosis and classification of diabetes mellitus. *Diabetes care* 2003; 26 (Suppl 1): S5 -S20.
24. Craig CL, Marshall AL, Sjostrom M, et al. International physical activity questionnaire: 12-country reliability and validity. *Med Sci Sports Exerc* 2003; 35:1381-1395.
25. Adult Treatment Panel III. Executive summary of the third report of the National (Cholesterol Education Program (NCEP) Expert panel on detection, evaluation and treatment of high blood cholesterol in adults. *J Am Med Assoc* 2001; 285:2486 -2497.
26. Guidelines Sub-Committee 1999. World Health Organization: International Society of Hypertension. Guidelines for the Management of Hypertension. *J Hypertens* 1999; 17: 151-183.
27. Waterhouse J, Muir C, Correa P, Powell J, (eds). *Cancer incidence in five continents. Vol III*, Lyon, France: IARC, 1976.
28. McLarty DG, Pollitt C, Swai ABM. Diabetes in Africa. *Diabetic Med* 1990; 7: 670-684.
29. Ekoe JM, Zimmet P, Williams P, The epidemiology of diabetes mellitus - an international perspective. New York: John Wiley & Sons 2001 p. 181- 204.
30. King H, Aubert RE, Herman WH. Global burden of diabetes, 1995-2025: prevalence, numerical estimates, and projections. *Diabetes Care* 1998; 21: 1414-1431.
31. Longo-Mbenza B, Lukoki Luila E, Mbete P, Kintoki Vita E. Is hyperuricemia a risk factor of stroke and coronary heart disease among Africans? *Int J cardiol* 1999; 71:17-22.

32. Mbanya JC, Ngogang J, Salah JN, et al. Prevalence of NIDDM and impaired glucose tolerance in a rural and an urban population in Cameroon. *Diabetologia* 1997; 40: 824-829.
33. McLarty DG, Swai AB, Kitange HM, et al. Prevalence of diabetes and impaired glucose tolerance in rural Tanzania. *Lancet* 1989;1(8643): 871-875.
34. Omar MAK, Seedat MA, Motala AA, et al. The prevalence of diabetes mellitus and impaired glucose tolerance in a group of urban South African Blacks. *S Afr Med J* 1993; 83: 641-643.
35. Ahren B, Corrigan CB. Prevalence of diabetes mellitus in North Western Tanzania. *Diabetologia* 1984; 26: 333-336.
36. Levitt NS, Katzenellenbogen JM, Bradshaw D, et al. The prevalence and identification of risk factors for NIDDM in urban Africans in Cape Town, South Africa. *Diabetes Care* 1993; 16: 601-607.
37. King H, Rewers M, WHO AD HOC Diabetes Reporting group. Global estimates for Prevalence of Diabetes Mellitus and Impaired Glucose Tolerance in Adults. *Diabetes Care* 1993; 16: 157-176.
38. Jon-Wook L. The cost of inaction is clear and unacceptable. In: World Health Organization. *Preventing Chronic Diseases: a vital investment: WHO global report*. World Health Organization, Geneva, 2005.
39. Moore M, Gould O, Keary BS. Global urbanization and impact on health. *Int J Hyg Environ Health* 2003; 206: 269-278.
40. Jenson AB, Roseberg HS, Notkins AL. Pancreatic islet cell damage in children with fatal viral infections. *Lancet* 1980; 2(8190): 354-358.
41. Dahlquist GG, Ivarsson S, Lindberg B, Forsgren M. Maternal enteroviral infection during pregnancy as a risk factor for childhood IDDM. A population-based case-control study. *Diabetes* 1995; 44: 408-413.
42. Rubinstein P, Walker ME, Fedun, B, et al. The HLA system in congenital rubella patients with and without diabetes. *Diabetes* 1982; 31:1088-1091.
43. Honeyman MC, Stone NL, Harrison LC. T-cell epitopes in Type 1 diabetes autoantigen tyrosine phosphatase IA-2: potential for mimicry with rotavirus and other environmental agents. *Mol Med* 1998; 4: 231-239.
44. Schattner A, Rager-Zisman B. Virus-induced autoimmunity. *Infect Dis* 1990; 12: 204-222.
45. Blom L, Nystrom L, Dahlquist G. The Swedish childhood diabetes study: Vaccinations and infections as risk determinants for diabetes in childhood. *Diabetologia* 1991; 34:176-181.
46. Alberti KGMM, Zimmet P, for the Who consultation. Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: Diagnosis and classification of diabetes mellitus, provisional report of a WHO consultation. *Diabet Med* 1998; 15: 539-553.
47. Longo-Mbenza B, Ngiyulu R, Bayekula M, et al. Low birth weight and risk of hypertension in African school children. *J Cardiovasc Risk* 1999 ; 6: 311-314.
48. Longo-Mbenza B, Lukoki Luila E, M'buyamba-Kabangu JR. Nutritional status, socio-economic status, heart rate, and blood pressure in African school children and adolescents. *Int J cardiol* 2007; 121:171-177.
49. McKeigue PM, Shah B, MARMOT MG. Relation of central obesity and insulin resistance with high diabetes prevalence and cardiovascular risk in South Asians. *Lancet* 1991; 337:382-386.
50. Facchini F, Chen J, Reaven GM. Light-to-moderate alcohol intake is associated with enhanced insulin sensitivity. *Diabetes Care* 1994 ; 17: 115-119.
51. Bonora E, Kiechl S, Willeit J, et al. Population-based incidence rates and risk factors for type 2 diabetes in white individuals. The Bruneck study. *Diabetes* 2004; 53:1782-1789.