

THE UNIVERSITY OF YAOUNDE I

RESEARCH CENTRE AND DOCTORAL
SCHOOL OF LIFE, HEALTH AND
ENVIRONMENTAL SCIENCES

RESEARCH UNIT IN LIFE SCIENCE

DEPARTMENT OF BIOCHEMISTRY



UNIVERSITE DE YAOUNDE I

CENTRE DE RECHERCHE ET DE
FORMATION DOCTORALE EN
SCIENCES DE LA VIE, SANTE ET
ENVIRONNEMENT

UNITE DE RECHERCHE EN SCIENCES
DE LA VIE

DEPARTEMENT DE BIOCHIMIE

LABORATORY OF NUTRITION AND NUTRITIONAL BIOCHEMISTRY
LABORATOIRE DE NUTRITION ET DE BIOCHIMIE NUTRITIONNELLE

**Nutritional Status, Malnutrition and related
conditions among Cameroonian Women of
childbearing age**

THESIS

Presented in Partial fulfillment of the requirements for the award
Doctorate/PhD Degree in Biochemisry,

Speciality: Food Science and Nutrition, **Option:** Nutrition

By:

M'BOBDA MOMDJO Christelle

Matricule 04R083

Masters in Biochemistry

Under the Direction of:

OBEN Julius ENYONG

Professor

Academic Year

2021-2022



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RESEARCH CENTRE AND DOCTORAL
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RESEARCH AND DOCTORATE TRAINING
UNIT IN LIFE SCIENCES
DEPARTMENT OF BIOCHEMISTRY

ATTESTATION DE CORRECTION

Nous soussignés : **Pr. MBANYA Jean Claude, Président** et **Dr. AGBOR Gabriel AGBOR** examinateur du jury, attestons que **Mme M'BOBDA MOMDJO Christelle**, matricule **04R083** a effectué les corrections conformément aux exigences du jury de sa thèse de Doctorat/Ph.D en Biochimie, option Sciences de Aliments et Nutrition, soutenue le **26 Janvier 2022 à 09 heures** dans la salle de Conférence de l'Annexe de la Faculté des Sciences de l'Université de Yaoundé I sur le thème «**Nutritional status, malnutrition and related conditions among cameroonian women of childbearing age**».

En foi de quoi la présente attestation lui est établie pour servir et valoir ce que de droit.

Yaoundé le **02 MARS 2022**

Examineur


Directeur de Recherche

Président du jury


Pr Jean Claude MBANYA
Director Biotechnology Center
Coordonnateur Ecole Doctorale SVSE
University of Yaounde I

Chef de Département



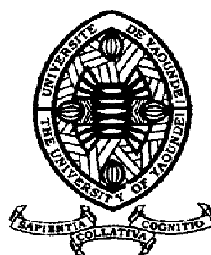
Professor
Toxicology-Toxicology

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PROTOCOL LIST OF LECTURERS OF THE FACULTY OF SCIENCE

THE UNIVERSITY OF YAOUNDE I Faculty of Science Division of Programming and Follow-up of Academic Affairs		UNIVERSITÉ DE YAOUNDÉ I Faculté des Sciences Division de la Programmation et du Suivi des Activités Académiques
LIST OF PERMANENT TEACHING STAFF		LISTE DES ENSEIGNANTS PERMANENTS

ACADEMIC YEAR 2021/2022

(By Department and by Grade)

UPDATE DATE 22 septembre 2021

ADMINISTRATION

DEAN : TCHOUANKEU Jean- Claude, *Associate Professor*

VICE-DEAN / DPSAA : ATCHADE Alex de Théodore, *Associate Professor*

VICE-DEAN / DSSE : NYEGUE Maximilienne Ascension, *Professor*

VICE-DEAN / DRC : ABOSSOLO Monique, *Associate Professor*

Head of the Administrative and Finance Division: NDOYE FOE Marie C. F., *Associate Professor*

Head of Academic Affairs, School and Research Division DAASR: AJEAGAH Gideon AGHAINDUM, *Professor*

1- DEPARTMENT OF BIOCHEMISTRY (BC) (38)

N°	NOMS ET PRÉNOMS	GRADE	OBSERVATIONS
1	BIGOGA DAIGA Jude	Professor	On duty
2	FEKAM BOYOM Fabrice	Professor	On duty
3	FOKOU Elie	Professor	On duty
4	KANSCI Germain	Professor	On duty
5	MBACHAM FON Wilfried	Professor	On duty
6	MOUNDIPA FEWOU Paul	Professor	Head of Department
7	NINTCHOM PENLAP V. épouse BENG	Professor	On duty
8	OBEN Julius ENYONG	Professor	On duty

9	ACHU Merci BIH	Associate Professor	On duty
10	ATOUGHO Barbara Mma	Associate Professor	On duty
11	AZANTSA KINGUE GABIN BORIS	Associate Professor	On duty
12	BELINGA née NDOYE FOE M. C. F.	Associate Professor	Head DAF / FS
13	BOUDJEKO Thaddée	Associate Professor	On duty
14	DJUIDJE NGOUNOUE Marcelline	Associate Professor	On duty
15	EFFA NNOMO Pierre	Associate Professor	On duty
16	EWANE Cécile Anne	Associate Professor	On duty

17	MOFOR née TEUGWA Clotilde	Associate Professor	Inspector of MINESUP Service
18	NANA Louise épouse WAKAM	Associate Professor	On duty
19	NGONDI Judith Laure	Associate Professor	On duty
20	NGUEFACK Julienne	Associate Professor	On duty
21	NJAYOU Frédéric Nico	Associate Professor	On duty
22	TCHANA KOUATCHOUA Angèle	Associate Professor	On duty

23	AKINDEH MBUH NJI	Senior Lecturer	On duty
24	BEBEE Fadimatou	Senior Lecturer	On duty
25	BEBOY EDJENGUELE Sara Nathalie	Senior Lecturer	On duty
25	DAKOLE DABOY Charles	Senior Lecturer	On duty
26	DJUIKWO NKONGA Ruth Viviane	Senior Lecturer	On duty
27	DONGMO LEKAGNE Joseph Blaise	Senior Lecturer	On duty
28	FONKOUA Martin	Senior Lecturer	On duty
29	KOTUE KAPTUE Charles	Senior Lecturer	On duty
30	LUNGA Paul KEILAH	Senior Lecturer	On duty
31	MANANGA Marlyse Joséphine	Senior Lecturer	On duty
32	MBONG ANGIE M. Mary Anne	Senior Lecturer	On duty
33	Palmer MASUMBE NETONGO	Senior Lecturer	On duty
34	PECHANGOU NSANGOU Sylvain	Senior Lecturer	On duty

35	MBOUCHE FANMOE Marceline Joëlle	Assistant Lecturer	On duty
36	OWONA AYISSI Vincent Brice	Assistant Lecturer	On duty
37	WILFRIED ANGIE Abia	Assistant Lecturer	On duty

2- DEPARTMENT OF ANIMAL BIOLOGY AND PHYSIOLOGY (BPA) (46)

1	AJEAGAH Gideon AGHAINDUM	Professor	<i>DAARS/FS</i>
2	BILONG BILONG Charles-Félix	Professor	Head of Department
3	DIMO Théophile	Professor	On duty
4	DJIETO LORDON Champlain	Professor	On duty
5	DZEUFIET DJOMENI Paul Désiré	Professor	On duty
6	ESSOMBA née NTSAMA MBALA	Professor	<i>Vice Dean/FMSB/UII</i>
7	FOMENA Abraham	Professor	On duty
8	KAMTCHOUING Pierre	Professor	On duty
9	KEKEUNOU Sévilor	Professor	On duty
10	NJAMEN Dieudonné	Professor	On duty
11	NJIOKOU Flobert	Professor	On duty
12	NOLA Moïse	Professor	On duty
13	TAN Paul VERNYUY	Professor	On duty
14	TCHUEM TCHUENTE Louis Albert	Professor	<i>Inspector of Coord.Progr./MINSANTE service</i>
15	ZEBAZE TOGOUET Serge Hubert	Professor	On duty

16	BILANDA Danielle Claude	Associate Professor	On duty
17	DJIOGUE Séfirin	Associate Professor	On duty

18	JATSA BOUKENG Hermine épse MEGAPTCHE	Associate Professor	On duty
19	LEKEUFACK FOLEFACK Guy B.	Associate Professor	On duty
20	MEGNEKOU Rosette	Associate Professor	On duty
21	MONY Ruth épse NTONE	Associate Professor	On duty
22	NGUEGUIM TSOFAK Florence	Associate Professor	On duty
23	TOMBI Jeannette	Associate Professor	On duty

24	ALENE Désirée Chantal	Senior Lecturer	On duty
25	ATSAMO Albert Donatien	Senior Lecturer	On duty
26	BELLET EDIMO Oscar Roger	Senior Lecturer	On duty
27	DONFACK Mireille	Senior Lecturer	On duty
28	ETEME ENAMA Serge	Senior Lecturer	On duty
29	GOUNOUE KAMKUMO Raceline	Senior Lecturer	On duty
30	KANDEDA KAVAYE Antoine	Senior Lecturer	On duty
31	MAHOB Raymond Joseph	Senior Lecturer	On duty
32	MBENOUN MASSE Paul Serge	Senior Lecturer	On duty
33	MOUNGANG Luciane Marlyse	Senior Lecturer	On duty
34	MVEYO NDANKEU Yves Patrick	Senior Lecturer	On duty
35	NGOULATEU KENFACK Omer Bébé	Senior Lecturer	On duty
36	NGUEMBOK	Senior Lecturer	On duty
37	NJUA Clarisse Yafi	Senior Lecturer	Head Div. UBA
38	NOAH EWOTI Olive Vivien	Senior Lecturer	On duty
39	TADU Zephyrin	Senior Lecturer	On duty
40	TAMSA ARFAO Antoine	Senior Lecturer	On duty
41	YEDE	Senior Lecturer	On duty

42	BASSOCK BAYIHA Etienne Didier	Assistant Lecturer	On duty
43	ESSAMA MBIDA Désirée Sandrine	Assistant Lecturer	On duty
44	KOGA MANG DOBARA	Assistant Lecturer	On duty
45	LEME BANOCK Lucie	Assistant Lecturer	On duty
46	YOUNOUSSA LAME	Assistant Lecturer	On duty

3- DEPARTMENT OF PLANT BIOLOGY AND PHYSIOLOGY (BPV) (33)

1	AMBANG Zachée	Professor	Head Division/UYII
2	BELL Joseph Martin	Professor	On duty
3	DJOCGOUÉ Pierre François	Professor	On duty
4	MBOLO Marie	Professor	On duty
5	MOSSEBO Dominique Claude	Professor	On duty
6	YOUMBI Emmanuel	Professor	Head of Department
7	ZAPFACK Louis	Professor	On duty

8	ANGONI Hyacinthe	Associate Professor	On duty
9	BIYE Elvire Hortense	Associate Professor	On duty
10	KENGNE NOUMSI Ives Magloire	Associate Professor	On duty
11	MALA Armand William	Associate Professor	On duty
12	MBARGA BINDZI Marie Alain	Associate Professor	CT/ MINESUP
13	NDONGO BEKOLO	Associate Professor	CE / MINRESI

14	NGODO MELINGUI Jean Baptiste	Associate Professor	On duty
15	NGONKEU MAGAPTCHE Eddy L.	Associate Professor	On duty
16	TONFACK Libert Brice	Associate Professor	On duty
17	TSOATA Esaïe	Associate Professor	On duty

18	DJEUANI Astride Carole	Senior Lecturer	On duty
19	GOMANDJE Christelle	Senior Lecturer	On duty
20	MAFFO MAFFO Nicole Liliane	Senior Lecturer	On duty
21	MAHBOU SOMO TOUKAM. Gabriel	Senior Lecturer	On duty
22	NGALLE Hermine BILLE	Senior Lecturer	On duty
23	NGOOU Lucas Vincent	Senior Lecturer	On duty
24	NNANGA MEBENGA Ruth Laure	Senior Lecturer	On duty
25	NOUKEU KOUAKAM Armelle	Senior Lecturer	On duty
26	ONANA JEAN MICHEL	Senior Lecturer	On duty

27	GODSWILL NTSOMBAH NTSEFONG	Assistant Lecturer	On duty
28	KABELONG BANAHOU Louis-Paul-Roger	Assistant Lecturer	On duty
29	KONO Léon Dieudonné	Assistant Lecturer	On duty
30	LIBALAH Moses BAKONCK	Assistant Lecturer	On duty
31	LIKENG-LI-NGUE Benoit C	Assistant Lecturer	On duty
32	TAEDOUNG Evariste Hermann	Assistant Lecturer	On duty
33	TEMEGNE NONO Carine	Assistant Lecturer	On duty

4- DEPARTMENT INORGANIC CHEMISTRY (CI) (33)

1	AGWARA ONDOH Moïse	Professor	<i>Head of Department</i>
2	DJOUFAC WOU MFO Emmanuel	Professor	On duty
3	Florence UFI CHINJE épouse MELO	Professor	<i>Rector Univ.Ngaoundere</i>
4	GHO GOMU Paul MINGO	Professor	<i>Ministre Chargé de Miss.PR</i>
5	NANSEU Njiki Charles Péguy	Professor	On duty
6	NDIFON Peter TEKE	Professor	<i>CT MINRESI</i>
7	NDIKONTAR Maurice KOR	Professor	<i>Vice-Dean Univ. Bamenda</i>
8	NENWA Justin	Professor	On duty
9	NGAMENI Emmanuel	Professor	<i>Dean FS UDs</i>
10	NGOMO Horace MANGA	Professor	<i>Vice Chancellor/UB</i>

11	ACAYANKA Elie	Associate Professor	On duty
12	BABALE née DJAM DOUDOU	Associate Professor	<i>Chargée Mission P.R.</i>
13	EMADACK Alphonse	Associate Professor	On duty
14	KAMGANG YOUNBI Georges	Associate Professor	On duty
15	KEMMEGNE MBOUGUEM Jean C.	Associate Professor	On duty
16	KONG SAKEO	Associate Professor	On duty
17	NDI NSAMI Julius	Associate Professor	On duty
18	NJIOMOU C. épouse DJANGANG	Associate Professor	On duty
19	NJOYA Dayirou	Associate Professor	On duty

20	TCHAKOUTE KOUAMO Hervé	Associate Professor	On duty
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21	BELIBI BELIBI Placide Désiré	Senior Lecturer	CS/ ENS Bertoua
22	CHEUMANI YONA Arnaud M.	Senior Lecturer	On duty
23	KENNE DEDZO GUSTAVE	Senior Lecturer	On duty
24	KOUOTOU DAOUDA	Senior Lecturer	On duty
25	MAKON Thomas Beauregard	Senior Lecturer	On duty
26	MBEY Jean Aime	Senior Lecturer	On duty
27	NCHIMI NONO KATIA	Senior Lecturer	On duty
28	NEBA nee NDOSIRI Bridget NDOYE	Senior Lecturer	CT/ MINFEM
29	NYAMEN Linda Dyorisse	Senior Lecturer	On duty
30	PABOUDAM GBAMBIE A.	Senior Lecturer	On duty

31	NJANKWA NJABONG N. Eric	Assistant Lecturer	On duty
32	PATOUOSSA ISSOFA	Assistant Lecturer	On duty
33	SIEWE Jean Mermoz	Assistant Lecturer	On duty

5- DEPARTMENT ORGANIC CHEMISTRY (CO) (34)			
1	DONGO Etienne	Professor	Vice-Dean/FSE/UYI
2	GHOOGOMU TIH Robert Ralph	Professor	Dir. IBAF/UDA
3	NGOUELA Silvère Augustin	Professor	Head of Department UDS
4	NYASSE Barthélemy	Professor	On duty
5	PEGNYEMB Dieudonné Emmanuel	Professor	<i>Director/ MINESUP/</i> Head of Department
6	WANDJI Jean	Professor	On duty

7	Alex de Théodore ATCHADE	Associate Professor	Vice-Dean / DPSAA
8	AMBASSA Pantaléon	Associate Professor	On duty
9	EYONG Kenneth OBEN	Associate Professor	On duty
10	FOLEFOC Gabriel NGOSONG	Associate Professor	On duty
11	FOTSO WABO Ghislain	Associate Professor	On duty
12	KEUMEDJIO Félix	Associate Professor	On duty
13	KEUMOGNE Marguerite	Associate Professor	On duty
14	KOUAM Jacques	Associate Professor	On duty
15	MBAZOA née DJAMA Céline	Associate Professor	On duty
16	MKOUNGA Pierre	Associate Professor	On duty
17	MVOT AKAK CARINE	Associate Professor	On duty
18	NGO MBING Joséphine	Associate Professor	Under/Direct. MINERESI
19	NGONO BIKOBO Dominique Serge	Associate Professor	C.E/ MINESUP
20	NOTE LOUGBOT Olivier Placide	Associate Professor	C.S/ MINESUP
21	NOUNGOUE TCHAMO Diderot	Associate Professor	On duty
22	TABOPDA KUATE Turibio	Associate Professor	On duty
23	TAGATSING FOTSING Maurice	Associate Professor	On duty
24	TCHOUANKEU Jean-Claude	Associate Professor	<i>Dean /FS/ UYI</i>
25	TIH née NGO BILONG E. Anastasie	Associate Professor	On duty
26	YANKEP Emmanuel	Associate Professor	On duty
27	ZONDEGOUMBA Ernestine	Associate Professor	On duty

28	KAMTO Eutrophe Le Doux	Senior Lecturer	On duty
29	NGNINTEDO Dominique	Senior Lecturer	On duty
30	NGOMO Orléans	Senior Lecturer	On duty
31	OUAHOUE WACHE Blandine M.	Senior Lecturer	On duty
32	SIELINOU TEDJON Valérie	Senior Lecturer	On duty

33	MESSI Angélique Nicolas	Assistant Lecturer	On duty
34	TSEMEUGNE Joseph	Assistant Lecturer	On duty

6- DEPARTMENT OF COMPUTER SCIENCE (IN) (25)

1	ATSA ETOUNDI Roger	Professor	<i>Head Div. MINESUP</i>
2	FOUDA NDJODO Marcel Laurent	Professor	<i>Head Dpt ENS/Head IGA. MINESUP</i>

3	NDOUNDAM René	Associate Professor	On duty
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4	ABESSOLO ALO'O Gislain	Senior Lecturer	On duty
5	AMINOUE Halidou	Senior Lecturer	<i>Head of department</i>
6	DJAM Xaviera YOUH - KIMBI	Senior Lecturer	On duty
7	DOMGA KOMGUEM Rodrigue	Senior Lecturer	On duty
8	EBELE Serge Alain	Senior Lecturer	On duty
9	KOUOKAM KOUOKAM E. A.	Senior Lecturer	On duty
10	MELATAGIA YONTA Paulin	Senior Lecturer	On duty
11	MONTHÉ DJIADEU Valéry M.	Senior Lecturer	On duty
12	MOTO MPONG Serge Alain	Senior Lecturer	On duty
13	OLLE OLLE Daniel Claude Delort	Senior Lecturer	Deputy Director Enset. Ebolowa
14	TAPAMO Hyppolite	Senior Lecturer	On duty
15	TINDO Gilbert	Senior Lecturer	On duty
16	TSOPZE Norbert	Senior Lecturer	On duty
17	WAKU KOUAMOU Jules	Senior Lecturer	On duty

18	BAYEM Jacques Narcisse	Assistant Lecturer	On duty
19	EKODECK Stéphane Gaël Raymond	Assistant Lecturer	On duty
20	HAMZA Adamou	Assistant Lecturer	On duty
21	JIOMEKONG AZANZI Fidel	Assistant Lecturer	On duty
22	MAKEMBE. S . Oswald	Assistant Lecturer	On duty
23	MESSI NGUELE Thomas	Assistant Lecturer	On duty
24	MEYEMDOU Nadège Sylvianne	Assistant Lecturer	On duty
25	NKONDOCK. MI. BAHANACK.N.	Assistant Lecturer	On duty

7- DEPARTMENT OF MATHEMATICS (MA) (30)

1	AYISSI Raoult Domingo	Professor	Head of Department
2	EMVUDU WONO Yves S.	Professor	<i>Inspector MINESUP</i>

3	KIANPI Maurice	Associate Professor	On duty
4	MBANG Joseph	Associate Professor	On duty
5	MBEHOU Mohamed	Associate Professor	On duty
6	MBELE BIDIMA Martin Ledoux	Associate Professor	On duty
7	NKUIMI JUGNIA Célestin	Associate Professor	On duty
8	NOUNDJEU Pierre	Associate Professor	<i>Head of Programs and Degrees/FS/UYI</i>
9	TCHAPNDA NJABO Sophonie B.	Associate Professor	Director/AIMS Rwanda
10	TCHOUNDJA Edgar Landry	Associate Professor	On duty

11	AGHOUKENG JIOFACK Jean Gérard	Senior Lecturer	Cell Head MINPLAMAT
12	CHENDJOU Gilbert	Senior Lecturer	On duty
13	DJIADEU NGAHA Michel	Senior Lecturer	On duty
14	DOUANLA YONTA Herman	Senior Lecturer	On duty
15	FOMEKONG Christophe	Senior Lecturer	On duty
16	KIKI Maxime Armand	Senior Lecturer	On duty
17	MBAKOP Guy Merlin	Senior Lecturer	On duty
18	MENGUE MENGUE David Joe	Senior Lecturer	On duty
19	NGUEFACK Bernard	Senior Lecturer	On duty
20	NIMPA PEFOUKEU Romain	Senior Lecturer	On duty
21	POLA DOUNDOU Emmanuel	Senior Lecturer	On duty
22	TAKAM SOH Patrice	Senior Lecturer	On duty
23	TCHANGANG Roger Duclos	Senior Lecturer	On duty
24	TETSADJIO TCHILEPECK M. E.	Senior Lecturer	On duty
25	TIAYA TSAGUE N. Anne-Marie	Senior Lecturer	On duty

26	BITYE MVONDO Esther Claudine	Assistant Lecturer	On duty
27	MBATAKOU Salomon Joseph	Assistant Lecturer	On duty
28	MBIAKOP Hilaire George	Assistant Lecturer	On duty
29	MEFENZA NOUNTU Thiery	Assistant Lecturer	On duty
30	TCHEUTIA Daniel Duviol	Assistant Lecturer	On duty

8- DEPARTEMENT OF MICROBIOLOGY (MIB) (18)

1	ESSIA NGANG Jean Justin	Professor	<i>Head of department</i>
2	NYEGUE Maximilienne Ascension	Professor	<i>VICE-DEAN / DSSE</i>
3	NWAGA Dieudonné M.	Professor	On duty

4	ASSAM ASSAM Jean Paul	Associate Professor	On duty
5	BOYOMO ONANA	Associate Professor	On duty
6	RIWOM Sara Honorine	Associate Professor	On duty
7	SADO KAMDEM Sylvain Leroy	Associate Professor	On duty

8	BODA Maurice	Senior Lecturer	On duty
9	BOUGNOM Blaise Pascal	Senior Lecturer	On duty
10	ESSONO OBOUGOU Germain G.	Senior Lecturer	On duty
11	NJIKI BIKOÏ Jacky	Senior Lecturer	On duty

12	TCHIKOUA Roger	Senior Lecturer	On duty
13	ESSONO Damien Marie	Assistant Lecturer	On duty
14	LAMYE Glory MOH	Assistant Lecturer	On duty
15	MEYIN A EBONG Solange	Assistant Lecturer	On duty
16	NKOUDOU ZE Nardis	Assistant Lecturer	On duty
17	SAKE NGANE Carole Stéphanie	Assistant Lecturer	On duty
18	TOBOLBAÏ Richard	Assistant Lecturer	On duty

9. DEPARTMENT OF PHYSICS (PHY) (40)

1	BEN- BOLIE Germain Hubert	Professor	On duty
2	DJUIDJE KENMOE épouse ALOYEM	Professor	On duty
3	EKOBENA FOU DA Henri Paul	Professor	<i>Vice-Rector. UN</i>
4	ESSIMBI ZOBO Bernard	Professor	On duty
5	KOFANE Timoléon Crépin	Professor	On duty
6	NANA ENGO Serge Guy	Professor	On duty
7	NANA NBENDJO Blaise	Professor	On duty
8	NDJAKA Jean Marie Bienvenu	Professor	Head of Department
9	NJANDJOCK NOUCK Philippe	Professor	On duty
10	NOUAYOU Robert	Professor	On duty
11	PEMHA Elkana	Professor	On duty
12	TABOD Charles TABOD	Professor	Dean FS Univ/Bda
13	TCHAWOUA Clément	Professor	On duty
14	WOAFO Paul	Professor	On duty
15	ZEKENG Serge Sylvain	Professor	On duty

16	BIYA MOTTO Frédéric	Associate Professor	DG/HYDRO Mekin
17	BODO Bertrand	Associate Professor	On duty
18	ENYEGUE A NYAM épse BELINGA	Associate Professor	On duty
19	EYEBE FOU DA Jean sire	Associate Professor	On duty
20	FEWO Serge Ibraïd	Associate Professor	On duty
21	HONA Jacques	Associate Professor	On duty
22	MBANE BIOUELE César	Associate Professor	On duty
23	MBINACK Clément	Associate Professor	On duty
24	NDOP Joseph	Associate Professor	On duty
25	SAIDOU	Associate Professor	On duty
26	SIEWE SIEWE Martin	Associate Professor	On duty
27	SIMO Elie	Associate Professor	On duty
28	VONDOU Derbetini Appolinaire	Associate Professor	On duty
29	WAKATA née BEYA Annie	Associate Professor	<i>Director/ENS/UYI</i>

30	ABDOURAHIMI	Senior Lecturer	On duty
31	CHAMANI Roméo	Senior Lecturer	On duty
32	EDONGUE HER VAIS	Senior Lecturer	On duty
33	FOUEDJIO David	Senior Lecturer	Cell Head MINADER

34	MBONO SAMBA Yves Christian U.	Senior Lecturer	On duty
35	MELI'I Joelle Larissa	Senior Lecturer	On duty
36	MVOGO ALAIN	Senior Lecturer	On duty
37	OBOUNOU Marcel	Senior Lecturer	DA/Univ Inter Etat/Sangmalima
38	WOULACHE Rosalie Laure	Senior Lecturer	On duty

39	AYISSI EYEBE Guy François Valérie	Assistant Lecturer	On duty
40	TEYOU NGOUPOU Ariel	Assistant Lecturer	On duty

10- DEPARTEMENT OF EARTH SCIENCE (ST) (43)

1	BITOM Dieudonné	Professor	<i>Dean / FASA / UDs</i>
2	FOUATEU Rose épouse YONGUE	Professor	On duty
3	NDAM NGOUPAYOU Jules-Remy	Professor	On duty
4	NDJIGUI Paul Désiré	Professor	Head of Department
5	NGOS III Simon	Professor	On duty
6	NKOUMBOU Charles	Professor	On duty
7	NZENTI Jean-Paul	Professor	On duty

8	ABOSSOLO née ANGUE Monique	Associate Professor	<i>Vice-Dean / DRC</i>
9	BISSO Dieudonné	Associate Professor	<i>Director/Projet Barrage Memve'ele</i>
10	EKOMANE Emile	Associate Professor	On duty
11	GANNO Sylvestre	Associate Professor	On duty
12	GHOGOMU Richard TANWI	Associate Professor	CD/Uma
13	MOUNDI Amidou	Associate Professor	<i>CT/ MINIMDT</i>
14	NGUEUTCHOUA Gabriel	Associate Professor	CEA/MINRESI
15	NJILAH Isaac KONFOR	Associate Professor	On duty
16	NYECK Bruno	Associate Professor	On duty
17	ONANA Vincent Laurent	Associate Professor	<i>Head Service of Maintenance & Material /UYII</i>
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Distribution of the teaching staff of the Faculty of Science of the University of Yaounde I

NUMBER OF TEACHERS					
DEPARTMENT	Professors	Associate Professors	Senior Lecturers	Assistant Lecturers	Total
BCH	8 (01)	14 (10)	13 (05)	3 (02)	38 (18)
BPA	15 (01)	8 (06)	18 (05)	05 (02)	46 (14)
BPV	07 (01)	10 (01)	9 (06)	07 (01)	33 (9)
CI	10 (01)	10 (02)	10 (02)	03 (0)	33 (5)
CO	6 (0)	21 (05)	05 (02)	02 (0)	34(7)
IN	2 (0)	1 (0)	14 (01)	08 (01)	25 (2)
MAT	2 (0)	8 (0)	15 (01)	05 (02)	30 (3)
MIB	3 (0)	4 (02)	05 (01)	06 (02)	18 (5)
PHY	15 (0)	14 (02)	09 (03)	02 (0)	40 (5)
ST	7 (1)	15 (01)	18 (05)	02 (0)	42(7)
Total	75 (5)	105 (29)	116 (31)	43 (10)	339 (75)
A total of		339 (75) of which:			
-	Professors	75 (5)			
-	Associate Professors	105 (29)			
-	Senior Lecturers	116 (31)			
-	Assistant Lecturers	43 (10)			
() = Number of Women		75			

DEDICATION

To:

My Family

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LIST OF ABBREVIATIONS

AIs	:	Adequate Intakes
ANOVA	:	One Way Analysis of Variance
ATP	:	Adenosine triphosphate
BCMRF	:	Biological cardiometabolic risk factors
BMI	:	Body Mass Index
BP	:	Blood pressure
Ca	:	Calcium
Ca-Mg ratio	:	Calcium-to-Magnesium ratio
CI	:	Confidence Interval
CVDs	:	Cardiovascular diseases
DBM	:	Double burden of malnutrition
DBP	:	Diastolic blood pressure
DHS	:	Demographic and Health Survey
DM2	:	Diabetes Miletus Type 2
DNA	:	Deoxyribose nucleic acid
DRI	:	Dietary Reference Intakes
EAR	:	Estimated Average Requirement
F&V	:	Fruits and Vegetables
FCS	:	Food consumption score
Fe	:	Iron
FFQ	:	Food frequency questionnaire
FPG	:	Fasting plasma glucose
GDM	:	Gestational Diabetes Mellitus
HC	::	Hip circumference
HDL-C	:	High density Lipoprotein-Cholesterol
HFIAS	:	Household Food Insecurity Access Scale
HKI	:	Helen Keller International
IDF	:	International Diabetes Federation
IOM	:	Institute of Medicine

IR	:	Insulin resistance
i.e.	:	That is
LDL-C	:	Low density Lipoprotein-Cholesterol
LGA	:	large for gestational age
LSD	:	Least Significant Difference
MetS	:	Metabolic Syndrome
Mg	:	Magnesium
NCDs	:	Non-communicable diseases
NCEP ATP III	:	National Cholesterol Education Program (NCEP) Adult Treatment Panel III (ATP III)
PAL	:	Physical activity level
RDAs	:	Recommended Dietary Allowance
RNA	:	Ribonucleic acid
SAT	:	Subcutaneous adipose tissue
SBP	:	Systolic blood pressure
SEM	:	Standard Error of Mean
SES	:	Socioeconomic status
SPSS	:	Statistical Package for Social Science
SSBs	:	sugar-sweetened beverages
T2DM	:	Type 2 diabetes
TD	:	Traditional Diet
TGs	:	Triglycerides
UNICEF	:	United Nations Children's Emergency Fund
VAT	:	visceral adipose tissue
WC	:	Waist circumference
WFP	:	World Food programme
WHO	:	World Health Organisation
WHR	:	Waist-to- Hip Ratio

ABSTRACT

Malnutrition especially Maternal and Child malnutrition are still serious public health issues in developing countries around the world. Underweight and mineral deficiencies remain significant public health concerns for children and women, but sufficient calories are available such that obesity and cardiometabolic risk factors (CMRFs) begin to emerge in adults in developing countries leading to the phenomenon of the double burden of malnutrition (DBM), and Cameroon is no exception. The general objective of this study was to evaluate the prevalence of undernutrition and overnutrition and their determinants, assess the influence of these forms of malnutrition on the prevalence of CMRFs among this group of Cameroonian women of childbearing age in order to propose interventional strategies. A cross sectional transverse study was carried out from January 2014 to August 2015 during which 608 Cameroonian women of age 14 to 49 years who gave their consent were recruited from the capital city Yaounde in the Center region and four regions of Cameroon: the Western region (Bafoussam, Dschang, Mbouda, Babadjou, Foumban, Bafou, Bafole and Njimom); Littoral (Nkongsamba and ekangté); Northwest (Wum, Mbengwi, Ndu and Nyen) and Far-North (Maroua II and Pont vert). This study was approved by the National Ethic Committee of Research for Human Health (N° 2014/08/488/CE/CNERSH/SP). Information were collected concerning the dietary habits, socio demographic and socioeconomic parameters, food security level and other lifestyle characteristics of the participants using structured questionnaires. Anthropometric measures were collected. Blood sample was collected for the biochemical determination of iron, calcium and magnesium levels, and lipid profile parameters (total cholesterol and triglyceride). Fasting blood glucose level, resting blood pressure, waist-to-hip ratio were equally measured. The biological cardiometabolic risk factors (BCMRFs) were evaluated using the IDF criteria and were used together with deficiency in one or more minerals to establish the eight phenotypes of the DBM and CMRFs. Descriptive statistics were calculated for all variables. Bivariate correlation analysis were performed to check for associations between variables. Logistic regression analyses were performed to assess the various risk factors and the level of statistical significance was $P < 0.05$.

Undernutrition in terms of underweight (2.1%), iron deficiency (11.5%), magnesium deficiency (22.4%) and calcium deficiency (48.3%); and overnutrition in terms of overweight (29.9%) and obesity (37.3%) were all present in the study population. The northwest region followed by the western region presented the highest prevalence of all the forms of malnutrition. Dietary determinants of all these forms included a high intake in a week of ‘cereals and tubers’ food group; and a low intake of ‘pulses’, ‘milk and dairy products’, ‘meat, fish and eggs’, and ‘vegetables’ and ‘fruits’ food groups and an acceptable food consumption score and being in a food secure household. Sociodemographic determinant included a high socioeconomic status. Combined mineral deficiencies existed at the individual level (Ca/Mg, Ca/Fe, Mg/Fe and Ca/Mg/Fe deficiencies). All age groups and a low intake of ‘meat, fish and eggs’ food group were predictors of combined Ca^{2+} and Mg^{2+} deficiencies. A magnesium deficient state was associated to a very high Ca-Mg ratio (8.03 ± 1.57). Higher rates of all BCMRF were associated to a Ca^{2+} deficient state. The overall prevalence of the DBM was 36.7%. Various phenotypes of the DBM and BCMRF existed at the individual level and the most prevalent phenotypes were phenotype I (at least one mineral deficiency and one BCMRF) (49.5%) and phenotype II (overweight or obese and at least one other BCMRF and at least one mineral deficiency) (35.4%). In conclusion, the prevalence of malnutrition among this group of Cameroonian women of childbearing age in terms of Ca^{2+} and Mg^{2+} deficiencies and overweight/obesity were relatively high, and a magnesium deficient state might result in a high Ca-Mg ratio. Various phenotypes of the double burden of malnutrition with cardiometabolic risk factors coexisted in the study population. Therefore, intervention strategies such as bio-fortification of staple foods in Cameroon with Ca^{2+} and Mg^{2+} , reduction of anti-nutrients in potential food sources of Ca^{2+} and Mg^{2+} , nutritional education on dietary diversification, on the reduction of the consumption of processed foods dense in energy and poor in micronutrients should be considered and encouraged especially in the Northwest and West regions with the highest rates of these mineral deficiencies and phenotypes of the double burden of malnutrition and BCMRFs.

Key words: Nutritional status, Malnutrition, Biological cardiometabolic risk factors, Women of childbearing age, Cameroon.

RESUME

La malnutrition, en particulier la malnutrition maternelle et infantile, reste un grave problème de santé publique dans les pays en développement du monde entier. L'insuffisance pondérale et les carences en minéraux restent des problèmes de santé publique importants pour les enfants et les femmes. Mais suffisamment de calories sont disponibles pour que l'obésité et les facteurs de risque cardiométaboliques (FRCMs) commencent à émerger chez les adultes dans les pays en développement conduisant au phénomène du double fardeau de la malnutrition (BFM), et le Cameroun ne fait pas exception. L'objectif général de cette étude était d'évaluer d'une part la prévalence de la dénutrition et de la surnutrition et leurs déterminants, et d'autre part l'influence de ces formes de malnutrition sur la prévalence des FRCMs chez ce groupe de femmes camerounaises en âge de procréer afin de proposer des stratégies interventionnelles. Une étude transversale a été réalisée de janvier 2014 à août 2015 au cours de laquelle 608 femmes camerounaises âgées de 14 à 49 ans ayant donné leur consentement ont été recrutées dans la cite capitale Yaoundé dans la région du Centre et dans quatre régions du Cameroun : la région de l'Ouest (Bafoussam, Dschang, Mbouda, Babadjou, Foumban, Bafou, Bafole et Njimom) ; le Littoral (Nkongsamba et Ekanjé) ; le Nord-Ouest (Wum, Mbengwi, Ndu et Nyen) et l'Extrême-Nord (Maroua II et Pont vert). Cette étude a été approuvée par le Comité National d'Ethique de Recherche pour la Santé Humaine (N° 2014/08/488/CE/CNERSH/SP). Des informations ont été recueillies concernant les habitudes alimentaires, les paramètres socio-démographiques et socio-économiques, le niveau de sécurité alimentaire et d'autres caractéristiques du mode de vie des participants à l'aide de questionnaires structurés. Les mesures anthropométriques ont été recueillies. Un échantillon de sang a été prélevé pour la détermination biochimique des taux de fer, de calcium et de magnésium, et des paramètres du profil lipidique (cholestérol total et triglycérides). La glycémie à jeun, la pression artérielle au repos et le rapport tour de taille/tour de hanche ont également été mesurés. Les facteurs de risque cardiométabolique biologiques (FRCMs) ont été évalués selon les critères de la FID et ont été utilisés conjointement avec la carence en un ou plusieurs minéraux pour établir huit phénotypes du BFM et des FRCMs. Les statistiques descriptives ont été calculées pour toutes les variables. Les analyses de corrélation bivariées ont été

effectuées pour vérifier les associations entre les variables. Les analyses de régression logistique ont été effectuées pour évaluer les différents facteurs de risque et le niveau de signification statistique était $P < 0,05$.

La population étudiée présentait une dénutrition sous la forme d'une insuffisance pondérale (2,1 %), une carence en fer (11,5 %), une carence en magnésium (22,4 %) et une carence en calcium (48,3 %), ainsi qu'une surnutrition sous la forme de surpoids (29,9 %) et d'obésité (37,3 %). La région du Nord-Ouest, suivie de la région de l'Ouest, présentait la plus forte prévalence de toutes les formes de malnutrition. Les déterminants alimentaires de toutes ces formes de malnutrition comprenaient une consommation hebdomadaire élevée de " céréales et tubercules ", une faible consommation de " légumineuses ", de " lait et produits laitiers ", de " viande, poisson et œufs ", de " légumes " et de " fruits ", un score acceptable de consommation alimentaire et la sécurité alimentaire du ménage. Les déterminants sociodémographiques comprenaient un statut socio-économique élevé. Des carences minérales combinées existaient au niveau individuel (carence en Ca/Mg, Ca/Fe, Mg/Fe et Ca/Mg/Fe). Tous les groupes d'âge et une faible consommation du groupe alimentaire " viande, poisson et œufs " étaient des facteurs de risque de carences combinées en Ca et Mg. Un état de carence en magnésium était associé à un rapport Ca-Mg très élevé ($8,03 \pm 1,57$). Des taux plus élevés de tous les FRCMs étaient associés à un état de carence en calcium. La prévalence globale du double fardeau de la malnutrition était de 36,7 %. Différents phénotypes DFM et des FRCMs existaient au niveau individuel et les phénotypes les plus prévalents étaient le phénotype I (au moins une carence minérale et un FRCM) (49,5%) et le phénotype II (surpoids ou obésité et au moins un autre FRCM et au moins une carence minérale) (35,4%). En conclusion, la prévalence de la malnutrition chez ce groupe de femmes camerounaises en âge de procréer en termes de carences en calcium et magnésium et de surpoids/obésité était relativement élevée, et un état de carence en magnésium pourrait entraîner un rapport Ca-Mg élevé. Divers phénotypes du double fardeau de la malnutrition et des facteurs de risque cardiométaboliques coexistaient dans la population étudiée. Par conséquent, des stratégies d'intervention telles que la bio-fortification des aliments de base au Cameroun avec le calcium et le magnésium, la réduction des anti-nutriments dans les sources alimentaires potentielles de Ca^{2+} et Mg^{2+} , l'éducation

nutritionnelle sur la diversification alimentaire, sur la réduction de la consommation d'aliments transformés denses en énergie et pauvres en micronutriments devraient être considérées et encouragées, en particulier dans les régions du Nord-Ouest et de l'Ouest avec les taux les plus élevés de ces carences minérales et des phénotypes du double fardeau de la malnutrition et des facteurs de risque cardiométaboliques.

Mots clés : Statut nutritionnel, Malnutrition, Facteurs de risque biologiques cardiométaboliques, Femmes en âge de procréer, Cameroun.

INTRODUCTION

INTRODUCTION

Malnutrition is still a serious public health issue in developing countries around the world. This condition (which comprises of under nutrition and over nutrition) is a health impairment resulting from a deficiency, excess or imbalance of nutrients (Bailey *et al.*, 2015). Most developing countries and Africa in particular are faced with persisting under nutrition as well as the growing epidemic of obesity (Prentice, 2018), diabetes and non-communicable diseases, and Cameroon is no exception (Kouebou *et al.*, 2013). Maternal including child malnutrition, encompassing both undernutrition and overweight, are global problems with important consequences for survival, incidence of acute and chronic diseases, healthy development, and the economic productivity of individuals and societies. Prevalence of low BMI ($<18.5 \text{ kg/m}^2$) in adult women has decreased in Africa and Asia since 1980, but remains higher than 10% in these two large developing regions. During the same period, the prevalence of overweight (BMI $\geq 25 \text{ kg/m}^2$) and obesity (BMI $\geq 30 \text{ kg/m}^2$) has been rising in all regions (Black *et al.*, 2013; Young & Ramakrishnan, 2020). Maternal malnutrition leads to poor foetal growth and low birthweight of the child and has many other health outcomes both for the mother and her child (UNICEF, 2013; Young & Ramakrishnan, 2020). In addition to the reproductive risks associated with these symptoms of overnutrition, it is well-established that certain micronutrient deficiencies also carry risks for reproductive outcomes (Hwalla *et al.*, 2017). Multiple micronutrient deficiencies often occur as part of a cycle of malnutrition and may be coupled with protein or energy malnutrition (Bailey *et al.*, 2015). Micronutrient deficiencies remain major public health concerns in many developed and developing countries around the world especially among reproductive women (Neufeld and Cameron, 2012) amongst whom their effects are devastating and sometimes irreversible. For example, iron deficiency contributes substantially to maternal deaths. Iron deficiency is a primary cause of anemia (Jones *et al.*, 2016a) especially maternal anemia (Black *et al.*, 2013).

The importance of coexisting micronutrient deficiencies in developing countries is gaining recognition, prompted by the disappointing responses often observed with single micronutrient supplements. It has been reported that their aetiology is multi-

factorial: inadequate intakes and genetic, parasitic and infectious diseases may all play a role (Gibson, 2011). Also, micronutrient deficiencies may also result from the presence of anti-nutrients in potential food sources. Phytates for example, have been reported to inhibit the intestinal absorption of calcium, iron, zinc and magnesium (Joy *et al.*, 2014). Micronutrient deficiencies can have major adverse health consequences that cannot always be reversed by nutrition interventions. Clearly, there is an urgent need for programmes to alleviate micronutrient deficiencies in developing countries (Gibson, 2011). But such programmes exist for the most common deficiencies including vitamin A, folate, iron, iodine, and zinc; however, several other micronutrient deficiency disorders exist (Bailey *et al.*, 2015). Intervention strategies have been proposed and are implemented in order to reduce the prevalence of these micronutrient deficiencies. Strategies commonly used are supplementation and food-based approaches, preferably in conjunction with public health interventions such as promotion and support of breastfeeding and control of infectious and parasitic diseases (Gibson, 2011). For example, Routine iron supplementation in pregnancy and infancy is recommended in areas without endemic malaria (WHO, 2007). Fortification programs with iron exist in several countries with the food vehicles of choice ranging from flours, dairy products, condiments, sugar, and salt to infant formulas (Bailey *et al.*, 2015). Also universal salt iodization is the most practical strategy to reduce iodine deficiency globally and zinc supplementation during pregnancy is associated with a significant reduction in preterm births without an effect on infant birth weight (Bailey *et al.*, 2015).

Despite the reduction in the prevalence of maternal underweight in middle and low income countries, mineral deficiencies including iron, iodine and zinc remain prevalent (Stephenson *et al.*, 2018). National and within-country representative data for micronutrients beyond vitamin A and iodine and anaemia remain scarce for most countries. While other micronutrient deficiencies such as calcium deficiency for example have also been reported to contribute substantially to maternal deaths (Black *et al.*, 2013). Dietary calcium is known to be an essential nutrient for bone accretion and bone health (Yamborisut *et al.*, 2015) and low calcium intakes has been reported as a risk factor for hypertension (Martinez, 1998), type 2 diabetes (Ojuka, 2004), osteoporosis (Filip *et al.*, 2005). Calcium supplementation has been observed to reduce

bone mass loss (Nieves, 2005). Also, low serum calcium may predispose women to pre-eclampsia (Richards *et al.*, 2015). Calcium interaction with magnesium has been reported to have many health outcomes (Rosanoff *et al.*, 2016). Magnesium alone has been reported to be associated with critical health issues, including cardiovascular disease (CVD), type 2 diabetes (T2DM), metabolic syndrome (Rosanoff *et al.*, 2012; Rosique-Esteban *et al.*, 2018; Piuri *et al.*, 2021) and osteoporosis (Rosanoff *et al.*, 2012; Piuri *et al.*, 2021). With regards to calcium, increased urinary calcium excretion and abundance of calcium-regulating hormones such as parathyroid hormone and calcitriol have been observed to cause a decrease in bone mineral content and increase intracellular calcium in vascular smooth muscle which produces contraction and therefore vasoconstriction leading to high blood pressure (Fong and Khan, 2012). Low intracellular levels of magnesium have also been proposed to affect glucose and insulin homeostasis through decreasing tyrosine kinase activity at insulin receptors (Suarez *et al.*, 1995) and to increase intracellular calcium levels (Barbagallo *et al.*, 2003); leading to an impairment in insulin signalling thereby reducing insulin sensitivity and secretion. These in association with other metabolic effects (increased dyslipidemia, inflammation, oxidative stress, endothelial dysfunction, blood pressure) also influenced by low intracellular magnesium levels lead to type 2 diabetes (T2DM) (Song *et al.*, 2013).

Therefore, as famine recedes, underweight and mineral deficiencies remain significant public health concerns for children and adults, but sufficient calories are available such that obesity and cardiometabolic risk factors begin to emerge in adults (FAO, 2006). Even at the individual level, obesity can coexist with micronutrients deficiency (Provo, 2013) leading to a double burden of malnutrition. Except in cases of extreme food insecurity, diets of the poor are energy dense, providing sufficient macronutrients but lacking micronutrients (Delisle, 2008).

Problematic and Justification of the Study

It has been shown that a child's future nutrition status is affected before conception and is greatly dependent on the mother's nutrition status prior to, and during pregnancy. A chronically undernourished woman is likely to give birth to a baby who is

also likely to be undernourished as a child, causing the cycle of undernutrition to be repeated over generations (UNICEF, 2013); leading to a vicious cycle of malnutrition from generation to generation. In order to break this cycle, women of childbearing age who constitute the very beginning of this vicious cycle and who have been cited by the World Health Organisation (WHO) as belonging to the vulnerable group, were targeted in this study. This was done in order to identify, prevent, reduce or even eradicate the presence of malnourished women or those at risk of becoming malnourished or developing the consequences of malnutrition in an attempt to break this vicious cycle of malnutrition. Equally, maternal nutrition was targeted because certain mineral deficiencies which have great consequences for the mother and her child are neglected and still persist.

Iron deficiency and its related condition (anemia) together with other micronutrients like Vitamin A, iodine, Zinc, the four most widespread micronutrient deficiencies (Bailey *et al.*, 2015), have received a lot of attention in Cameroon; through nutritional interventions. For example, the fortification of staple food like vegetable oil, bouillon cube, salt using the FRAT (Hess *et al.*, 2013) and mass supplementation of vulnerable groups (children under five years of age and women of childbearing age) in order to reduce their deficiencies (Tanya *et al.*, 2011). Despite the reduction of underweight and the existence of programmes earlier stated to alleviate micronutrient deficiencies (MNDs), hidden malnutrition in terms of mineral deficiencies still exist in Cameroon. In addition to these micronutrients earlier mentioned that remain prevalent especially among vulnerable groups, other important minerals like Calcium and Magnesium have received less attention in Cameroon while their low intakes can severely impede the health of a population especially women of childbearing age since they both play important roles in lipid and glucose metabolism as it has been earlier illustrated. In addition, national data on the prevalence of these minerals among women of childbearing age are scarce or inexistent. Also, the country seems to be facing a rapid urbanization of her society and the major consequence of this situation is the coexistence of nutritional double burden, with an important increase of non-communicable diseases (Ntentie *et al.*, 2014). For example the prevalence of hypertensive disorders of pregnancy has been reported to be high in Sub-Saharan Africa and pregnant or

postpartum women with this disorders have increased risk of maternal mortality (Gemechu *et al.*, 2020), which may be due to increasing rates of biological cardiometabolic risk factors associated to the persistant presence of hidden malnutrition (mineral deficiencies) as earlier mentioned.

This study was therefore carried out among Cameroonian women of childbearing age (WCB) for two reasons: firstly, in order to try to break the vicious cycle of malnutrition and its consequences among this vulnerable group through the nutritional assessment of crucial minerals including: iron to verify wether all the nutritional interventions earlier stated are helping to ameliorate the iron status among this group of Cameroonian women; and calcuim and magnesium, two essential elements in lipid and glucose metabolism as previously stated and whose deficiencies can have severe consequences on the cardiometabolic health of WCB; and secondly, the fact that national data for these two minerals are inexistence since the assessment of the status of these minerals in the Cameroonian population in general and women of childbearing age in particular have been neglected.

Study Hypothesis

There is a high prevalence of malnutrition in women of childbearing of the study population; and the coexistence of overweight/obesity and one or more mineral deficiencies at the individual level, is associated with high rates of biological cardiometabolic risk factors in the study.

Main objective

The general objective of this study was to evaluate the prevalence of malnutrition and its determinants, assess the influence of various forms malnutrition on the prevalence of biological cardiometabolic risk factors among this group of Cameroonian women of childbearing age in order to propose intervention strategies.

Scientific Interest

This study provides baseline statistics on the prevalence of the various forms of malnutrition and their determinants among women of childbearing age. It provides a

preliminary in the understanding of mineral-mineral interactions and the influence of such interactions on the occurrence of mineral deficiencies, thereby aiding in better addressing issues concerning maternal nutrition especially mineral status.

Also, it provides an understanding of the effect of multiple mineral deficiencies on individual cardiometabolic risk factors (observed in this study as probable consequences of overweight/obesity or even overweight/obesity combined to mineral deficiencies) thereby providing the grounds for strategies to prevent both mineral deficiencies and individual biological cardiometabolic risk factors at the individual level; so as to limit the consequences of undernutrition and overnutrition especially on maternal health and on the development and health of off springs.

Finally, it provides interventional strategies addressing both issues of nutritional deficiencies and overnutrition at the individual, household, community and population level.

Research questions

1. Is the nutritional status of Cameroonian women of childbearing age good in general, if not, is malnutrition present in this group of women and what category of malnutrition is most present and what are the determinants of this poor nutritional status?
2. If actually malnutrition is present, are increasing rates of biological cardiometabolic risk factors consequences of this poor nutritional status and how can these informations be used to break the vicious cycle of malnutrition and its consequences among this group of Cameroonian women?

Specific objectives of the study

1. Determine nutritional status and its determinants in the study population
2. Study combined mineral deficiencies and the influence of specific mineral deficiencies on abnormal weight status.
3. Determine the influence of mineral deficient states on biological cardiometabolic risk factors and the effects of the double burden of malnutrition (DBM) on BCMRFs.

**CHAPTER I: LITERATURE
REVIEW**

CHAPTER I: LITERATURE REVIEW

I.1. Nutritional status among women of childbearing age

The term reproductive age also called childbearing age is usually referred by WHO as the active reproductive years in women starting with menarche around 12-14 years and ending with menopause around 45-49 years. For demographic purposes, reproductive age group is usually defined as 15-49 years (WHO, 1998).

With regards to women of childbearing age, nutritional status is an important aspect of health and wellness before and during pregnancy. A child's future nutrition status is affected before conception and is greatly dependent on the mother's nutrition status prior to, and during pregnancy (UNICEF, 2013). Maternal nutritional status is important for the health and quality of life of women and for the health of their newborns (Mastiholi *et al.*, 2018), and pre-pregnancy weight is a common indicator of a woman's nutritional status (Ward and Siega-Riz, 2012).

Several factors have been reported to contribute to marginal or low nutrient status including poor dietary quantity or quality, increased requirements, increased metabolic losses, or impaired gastrointestinal digestion or absorption. Also the long-term consumption of poor dietary quantity or quality (e.g. restrictive, unbalanced, or low-nutrient dense diets) also referred to as poor nutrition or malnutrition increases the risk of poor nutritional status (Bruins *et al.*, 2018). Malnutrition, which is defined as either undernutrition (underweight, wasting and stunting) or overnutrition (overweight and obesity) is a public health challenge (Tathiah *et al.*, 2013). According to the World Health Organisation, malnutrition can be defined as a nutritional disorder in all its form (including imbalances in energy, specific macronutrients and micronutrients, and dietary patterns).

Conventionally, the emphasis has been on inadequacy, but malnutrition also applies to excess and imbalanced intakes. It occurs when the intake of essential macronutrients and micronutrients does not meet or exceeds the metabolic demands for those nutrients. Metabolic demands vary with age and other physiological conditions, they are also

affected by environmental conditions, including poor hygiene and sanitation, which lead to diarrhoea, both foodborne and waterborne (WHO, 2013).

I.1.1. Prevalence, causes and consequences of undernutrition among women of childbearing age

I.1.1.1. Definition of Undernutrition

Undernutrition is a situation in which the body's requirements are not met, due to under consumption, or to impaired absorption and use of nutrients. Undernutrition commonly refers to a deficit in energy intake, but can also refer to deficiencies of specific nutrients, and can be either acute or chronic (WHO, 2013).

Undernutrition is considered as one of the world's most serious but least addressed health problems (SUN Movement, 2011). Undernutrition encompasses stunting, wasting, underweight and deficiencies of essential vitamins and minerals, collectively referred to as micronutrient deficiencies. Undernutrition is an important determinant of maternal and child health (Black *et al.*, 2008).

I.1.1.2. Prevalence of Undernutrition

In the world, the prevalence of undernutrition among women of childbearing age has been reported in WHO African regions (WHO, 2017). According to this WHO report, maternal undernutrition rates i.e. women with a BMI <18.5kg/m² with recent data from 2000 to 2015 were 18% in Nepal in 2011; 18% in Pakistan in 2011; 27% in Timor-Leste in 2009 to 2010 (WHO, 2017). As earlier stated, undernutrition may also be reflected by micronutrient deficiencies. Anemia, a micronutrient deficiency associated disorder remains a considerable public health concern in most LMICs, especially among women (Jones *et al.*, 2016a). The population group in which the largest number of individuals is affected by anemia is non-pregnant women (468.4 million) (WHO, 2013). The prevalence of anemia (<12g/dl) in non-pregnant women according to the UNICEF report of 2013 were as follows: 40% in Bangladesh in 2011; 21% in Guatemala from 2008 to 2009; 53% in India from 2005 to 2006; 33% in Nepal in 2011; 19% in Timor-Leste in 2009 to 2010 (UNICEF, 2013).

In Africa, the prevalence rates of undernutrition compared to the WHO cut-off values for the public health significance of adults with a BMI <18.5 for women 15 – 49 years in some countries of the WHO African regions reported with data from 2000 to 2015 are shown in the figure below: (WHO, 2017).

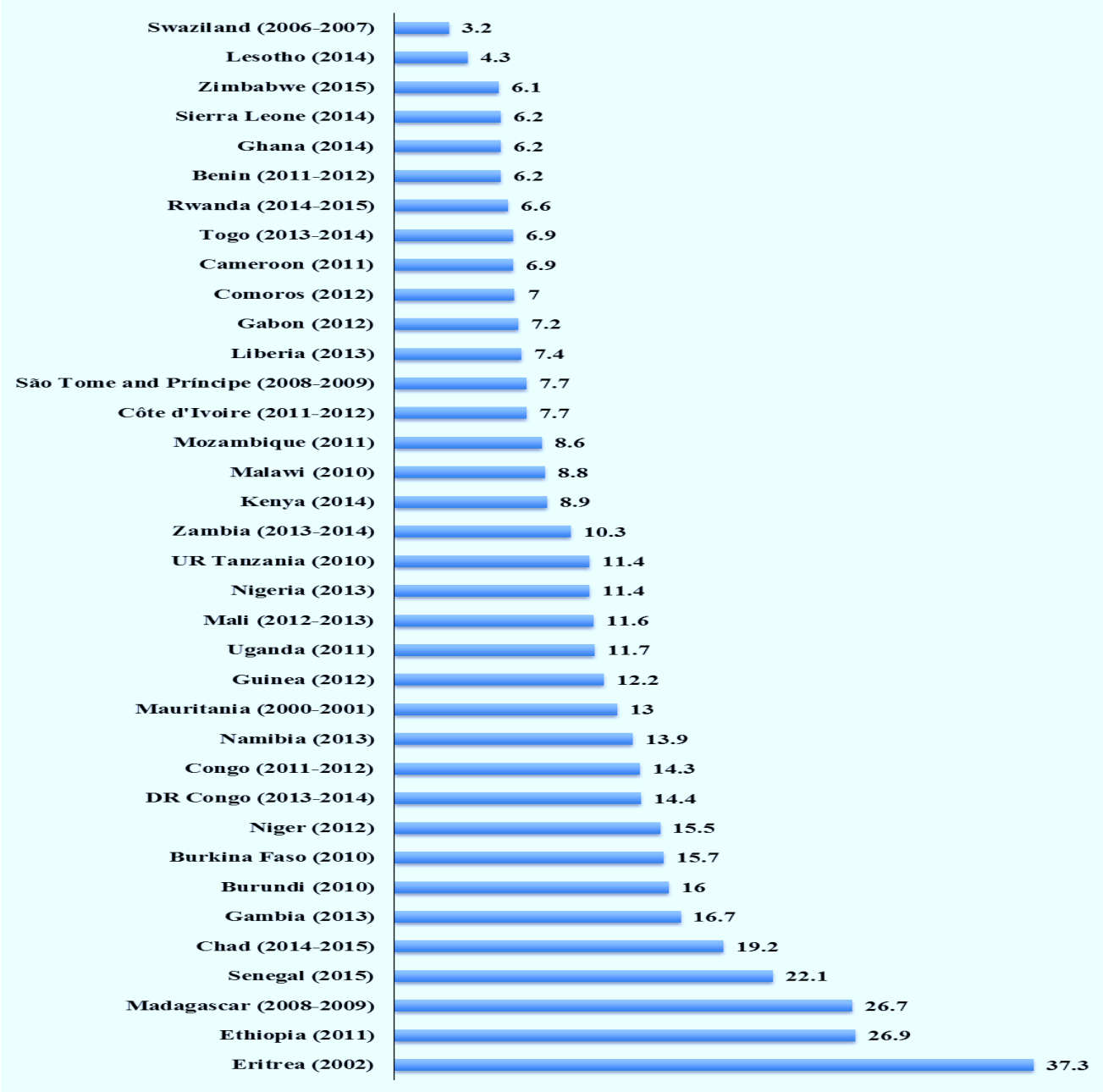


Figure 1: Percentage of women aged 15–49 who were underweight (BMI<18.5 kg/m²) in the WHO African Region. Most recent data: 2000–2015 (WHO, 2017)

According to the same WHO report of 2017, the prevalence of anaemia among non-pregnant, non-lactating women in the WHO African Region are shown in the figure below:

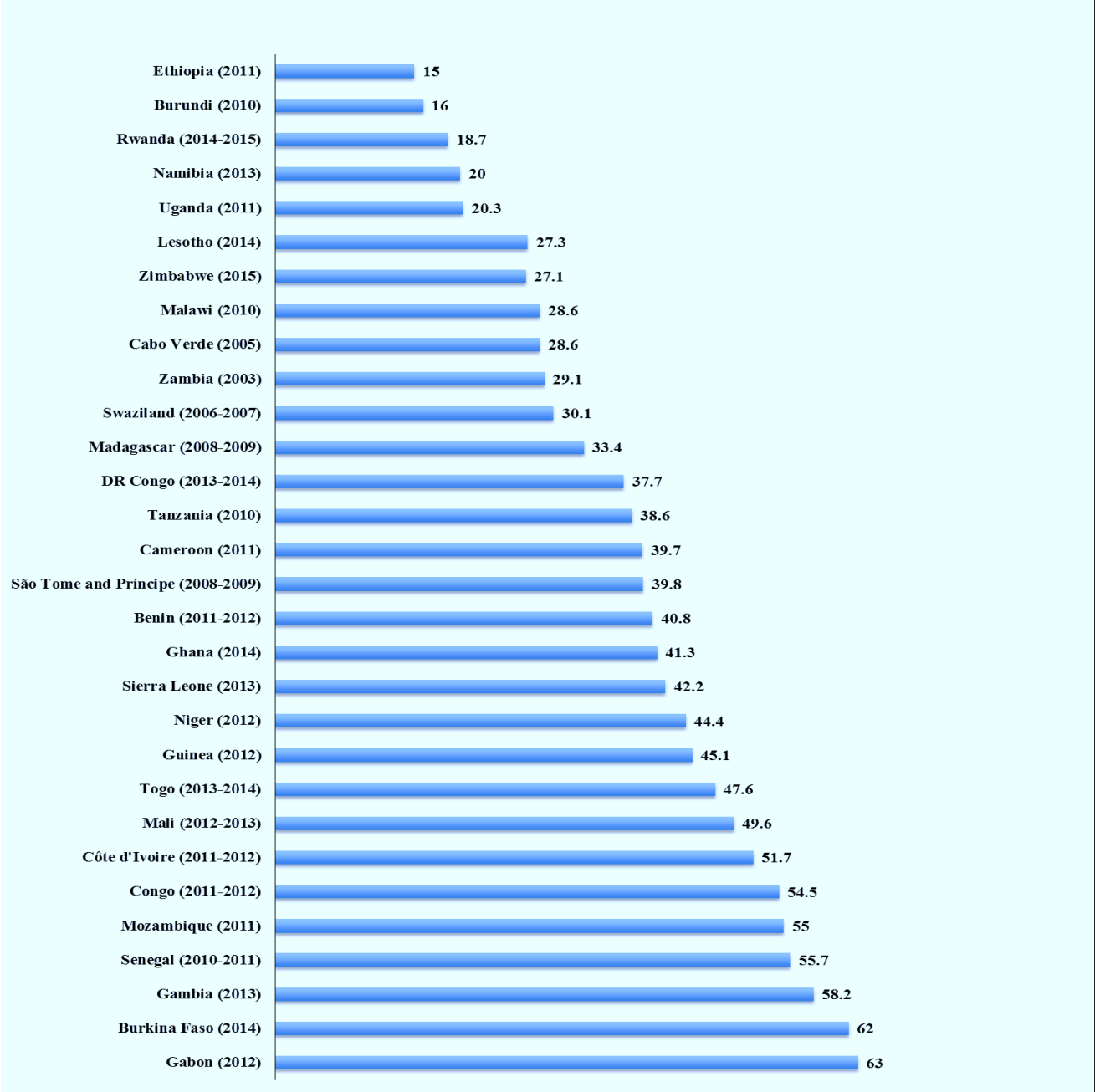


Figure 2: Prevalence of anaemia among non-pregnant, non-lactating women in the WHO African Region. Most recent data: 2005–2015. (WHO, 2017).

In Cameroon, seven percent of mothers of children under the age of five were undernourished according to the demographic and health survey of 2004. The highest level of maternal undernutrition was in the Extreme-North region (15 percent). None of the mothers were found to be undernourished in Douala region. The undernutrition rate (chronic energy deficiency) for mothers of children under five was 2 percent in Yaounde/Douala, 8 percent in other urban areas, and 7 percent in the rural areas (DHS and NIS, 2004). In the recent WHO 2017 report, in Cameroon, the rate of underweight among women of childbearing age was 6.9%. This rate was the lowest rate of underweight in the WHO African regions among women together with the rates found in Benin, Ghana, Lesotho, Rwanda, Swaziland and Togo. Whereas, in the 2018 DHS, only 6 % of Cameroonian women were categorised as thin (NIS & ICF, 2020). The prevalence of anaemia among non-pregnant and non-lactating women in Cameroon reported in this same 2017 WHO report was 39.7% in 2011 (WHO, 2017) and this prevalence has been reported not to have changed in the 2018 DHS (NIS & ICF, 2020).

I.1.1.3. Causes and consequences of Undernutrition among women of childbearing age

I.1.1.3.1. Causes of Undernutrition

The causes of malnutrition are complex, multidimensional and interrelated. They range from broad factors such as political instability and slow economic growth, to immediate determinants - dietary intake (energy, protein, fat, and micro nutrients) and health status. These factors are interdependent on each other. In turn, these conditions are closely linked to the overall standard of living of people, and whether a population can meet its basic needs such as access to food, housing, and health care. Furthermore, there is recognition that malnutrition is frequently part of a vicious cycle that includes dietary intake, poverty and disease, and these three factors are interlinked. Deficiencies are not solely the result of inadequate dietary intake. Disease can impair absorption of nutrients and reduce appetite, and environmental and psychosocial stress affecting the mother can contribute to child undernutrition (Walker *et al.*, 2011). Socio-economic and political changes that improve health and nutrition can break the cycle (Priyanka, 2014).

Increased income usually enables poor families to get better access to the things that enhance the nutrition: sufficient quantity and quality of food, better child feeding and hygiene practices, adequate supplies of clean water, and access to good quality preventive and curative health care. However, if families do not spend their increased income on these factors which determine good nutrition; then malnutrition rates are unlikely to decrease (Priyanka, 2014). In this context women are the key actors in utilizing available resources for good nutrition and household food security. Women with more control over resources are also in a better position to provide care to children and achieve better birth weights. As explained by Smith *et al.* (2003) women's status affects the quality of care for children in a direct manner, but also indirectly through the quality of the care women themselves receive (Smith *et al.*, 2003).

The cultural and socio-economic environment can adversely affect women's life style, and in particular result in inadequate diet. From infancy in many parts of the world women receive less and lower quality food and are treated less often when sick and then only at a more advanced stage of disease. Also when women are less educated, they receive less information than men and have less control over decision making and family resources, they are also less apt to admit to health problems or to seek care. A life cycle approach to women's health takes into account both the specific and cumulative effects of poor health and nutrition. Many of the health problems that affect women of reproductive age, their newborns and older women begin in childhood and adolescence (World Bank, 1994). In many developing countries, the low status of women is considered to be one of the primary determinants of undernutrition across the life cycle. Women's low status can result in their own health outcomes being compromised (Shroff *et al.*, 2009).

It has been stated that many factors are linked to malnutrition in Cameroon with the most important being the problem of food availability. Also, the same author reported that the problem of food availability is coupled with the problem of accessibility, affordability and transformation especially in the northern and eastern parts of the country, marked by the entry of refugees and other internally displaced persons due to transnational instability. Poor nutritional habits, on the other hand, ironically make

malnourished persons out of those having plenty. The situation worsens in pregnant and breastfeeding women, thus affecting the nutritional status of their babies (Ndenkeh & Cumber, 2016).

Therefore, nutritional status is influenced by three broad factors: food, health and care. Optimal nutritional status results when women have access to affordable, diverse, nutrient-rich food; appropriate maternal practices; adequate health services; and a healthy environment including safe water, sanitation and good hygiene practices. These factors directly influence nutrient intake and undernutrition, and infection creates a potentially lethal cycle of worsening illness and deteriorating nutritional status. Food, health and care are affected by social, economic and political factors. The combination and relative importance of these factors differ from country to country. Understanding the immediate and underlying causes of undernutrition in a given context is critical to delivering appropriate, effective and sustainable solutions and adequately meeting the needs of the most vulnerable people (UNICEF, 2013).

I.1.1.3.2. Consequences of Undernutrition

Maternal undernutrition leads to poor fetal growth and low birthweight of the child and has many other health outcomes both for the mother and her child (UNICEF, 2013).

1-) Low birthweight child

Poor maternal nutrition impairs foetal development and contributes to low birthweight, subsequent stunting and other forms of undernutrition (Özaltın *et al.*, 2010). As stated, an intergenerational cycle of malnutrition exists whereby a mother who has anaemia, for example, is likely to have a baby with a low birth weight. Reduced birthweight babies are more likely to be wasted or stunted who may also become later in life malnourished adults who give birth to low birthweight babies; or may have a higher risk of morbidity and mortality and of developing non-communicable diseases later in life (Branca *et al.*, 2015). This vicious cycle of malnutrition continues if no measure actions are undertaken. Short intervals between pregnancies and having several children may accumulate or exacerbate nutrition deficits, passing these deficiencies on to the children.

Low birthweight is associated with increased morbidity and mortality (Lawn *et al.*, 2005). Undernourished mothers do not only have a higher frequency of low-birth weight babies, but also a higher frequency of long term economic disadvantage (UNICEF, 2009). Evidence shows that the improved status and education of women dramatically reduces the percentage of underweight children (Gakidou *et al.*, 2010)

2-) Unhealthy pregnancy outcome

The nutritional status of a woman before and during pregnancy is important for a healthy pregnancy outcome (Kramer and Victoria, 2001). Maternal short stature is a risk factor for caesarean delivery, largely related to cephalopelvic disproportion. A meta-analysis of epidemiological studies found a 60% (95% CI 50-70) increased need for assisted delivery among women in the lowest quartile of stature (146 cm to 157 cm, depending on the region) compared with women in the highest quartile. If operative delivery to ensure a healthy birth is not available to women who need it, both mother and baby are at risk (Ronsmans *et al.*, 2006). Even if operative delivery is accessible, affordable, and safe, anaesthesia and laparotomy increase the risk of maternal morbidity (Villar *et al.*, 2006). Low maternal body-mass index does not seem to increase the risk of pregnancy complications and assisted delivery. Rather, there seems to be a synergistic positive effect of short stature and higher maternal body-mass index on increasing these complications (Dempsey *et al.*, 2005)

3-) Intrauterine growth restriction

Attained height is affected by genetic and environmental factors throughout the growth period. Linear growth failure is largely confined to the intrauterine period and the first few years of life, and is caused by inadequate diets and frequent infections (Shrimpton *et al.*, 2001) Short stature of the mother and poor maternal nutrition stores are associated with increased risk of intrauterine growth retardation (Black *et al.*, 2008).

Low maternal body-mass index is associated with intrauterine growth restriction. Previous analyses estimated the disease burden of low maternal body-mass index as a risk factor for perinatal conditions, (Fishman *et al.*, 2004) whereas the estimates presented in this paper consider intrauterine growth restriction to be the risk factor for

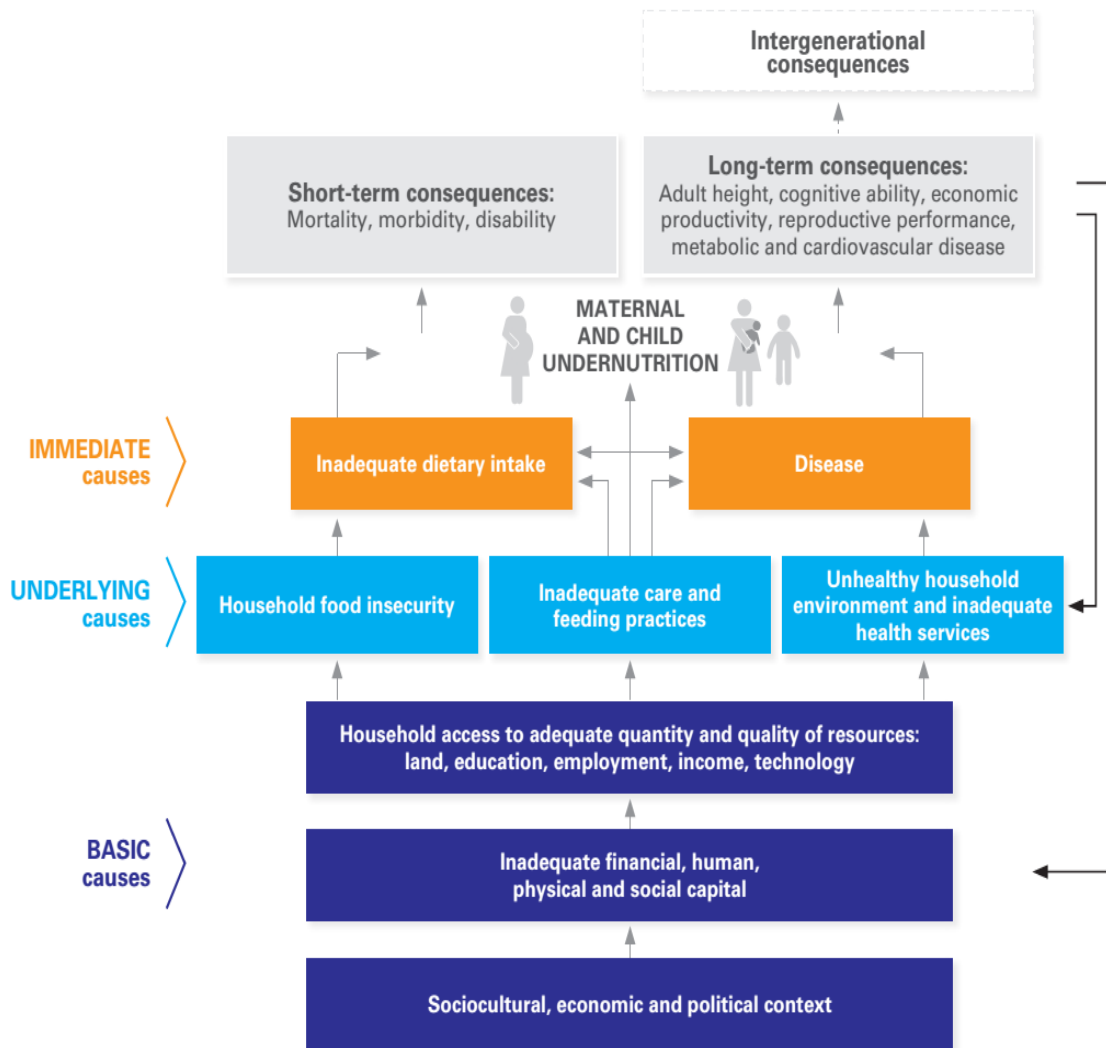
neonatal conditions. Additional work is needed to quantify the relative effects of low maternal body-mass index, extent of weight gain in pregnancy, and maternal micronutrient deficiencies on the occurrence and severity of intrauterine growth restriction (Black *et al.*, 2008).

4-) Effect on the volume or composition of breastmilk

Maternal undernutrition has little effect on the volume or composition of breast milk unless malnutrition is severe. The concentration of some micronutrients (vitamin A, iodine, thiamin, riboflavin, pyridoxine, and cobalamin) in breast milk is dependent on maternal status and intake, so the risk of infant depletion is increased by maternal deficiency (Allen, 1994). This factor is most evident in the case of vitamin A, where the content in breast milk is the main determinant of infant status because stores are low at birth. Maternal supplementation with these micronutrients increases the amount secreted in breast milk, which can improve infant status (Black *et al.*, 2008).

5-) Infertility

Infertility is defined as at least 12 months unsuccessful attempt to conceive for women younger than 35 years or at least 6 months for a women older than 35 years old (Hosseini and Eslamian, 2015). Undernutrition and underweight can exert an inhibitory effect on the hypothalamo–pituitary– ovarian axis, thereby suppressing ovulation and causing infertility. Although underweight does not seem to adversely affect the pregnancy rate in fertility treatment, underweight or malnourished women who conceive have higher risks of obstetric complications, such as hyperemesis gravidarum, anaemia, fetal growth restriction and premature delivery (Li and Ng, 2012). Body mass index (BMI) and weight are closely related to reproductive function. In a study investigating lifestyle factors, time to conception increased in both overweight (BMI >35 kg/m²) and underweight (BMI <19kg/m²) individuals. After adjusting for age, menstrual status and other lifestyle variables, compared to women with a normal weight, women with a BMI<19 or ≥ 25- 39 kg/m² had a relative risk of time to conception >12 months of 2.2 (95 % CI 1.6-3.2) (Hassan and Killick, 2004). Given this, it was recommended that women who were overweight or obese lose weight and women who are underweight should gain weight to improve fertility (Collins and Rossi, 2015).



The black arrows show that the consequences of undernutrition can feed back to the underlying and basic causes of undernutrition, perpetuating the cycle of undernutrition, poverty and inequities.

Figure 3: Conceptual framework of UNICEF for child malnutrition applicable to malnutrition of women with some modification. Source: UNICEF, 2013

I.1.1.4. Efforts or interventions to reduce undernutrition among women of childbearing age in Cameroon

A lot of efforts are being carried or have been carried out concerning nutrition interventions to reduce maternal undernutrition in Cameroon. The government and many organizations are greatly working to reduce undernutrition in women. For example, the Helen Keller International (HKI) is carrying out essential actions in nutrition such as promotion of good nutrition for women; waging a war and fighting

against vitamin A, iron and iodine deficiencies; providing appropriate nutrition for vulnerable groups who are sero-positive for HIV/AIDS among which sero-positive women. In health care systems, essential actions in nutrition can be applied during pregnancy, birth, immunization, child welfare service and when a child is sick. Also, strategies for fighting against vitamin deficiencies have been adopted by the Ministry of public health in Cameroon amongst which: the consumption of foods rich in vitamin A (red palm oil (not bleached), tomatoes, fruits, carrots, green vegetables, eggs, margarine and butter); vitamin A supplementation to all the women (where possible) within eight weeks post-delivery to enrich maternal milk; Food fortification with vitamin A through industries producing flour, oils, salt and other products (Tanya *et al.*, 2011).

I.1.2. Prevalence, causes and consequences of Overnutrition among women of childbearing age

I.1.2.1. Definition of over nutrition (overweight and obesity)

Overnutrition results in overweight and obesity, which are conditions of abnormal or excessive body fat accumulation in adipose tissue, to the extent that health may be impaired (WHO, 2000). Obesity is a medical condition in which excess body fat has accumulated to the extent that it may have an adverse effect on health leading to reduced life expectancy and or increased health problems (Ojiegbe, 2016). It is also defined as a body mass index equal or greater than $30\text{kg}/\text{m}^2$. In absolute terms, obesity is an increase of body adipose (fat tissue) mass. Overweight can be defined as a body mass index (BMI) equal to or more than $25\text{kg}/\text{m}^2$ (WHO, 2005). When a person's caloric intake exceeds his/her energy expenditure, the body stores the extra calories in the fat cells present in adipose tissue. These adipose cells act as energy reservoirs, and they enlarge or contract depending on how people use this energy. If people do not balance energy input and output by adopting healthy eating habits and regular exercise, then fat builds up, and they may become overweight and eventually obese (Mbochi, 2010).

I.1.2.2. Prevalence of over nutrition (overweight and obesity)

In the modern society, obesity represents an important health issue, because of its increasing prevalence in the last decades in the developed and developing countries on

one hand, and the complications that occur in obese patients, accompanied by an increased risk for cardiometabolic diseases on the other hand (Bodea & Popa, 2015). Globally, in 2008, one in three adults (35% of women) was overweight; that is, they had a body mass index (BMI) equal to or greater than 25 kg/m². Also, more than one in 10 women (14%) were obese; that is, they had a BMI equal to or greater than 30 kg/m². The prevalence of overweight and obesity in adults varied considerably among regions and income groups. It has been reported that, in the past three decades, global overweight increased from 29.8% to 38.0% in women. Also, it has been pointed out that more than 20% of women are obese in 14 Latin American countries, and more than 30% of women in several countries in the Middle East and in North and southern Africa are obese. Rates of overweight and obesity in adolescent girls (15–19 years) follow the same regional patterning as is seen in adult women (Ford *et al.*, 2017). According to a UNICEF report of 2013, the proportion of women of childbearing age with a BMI ≥ 25 kg/m² was 17% in Bangladesh in 2011, 51% in Guatemala from 2008 to 2009, 13% in India from 2005 to 2006, 14% in Nepal in 2011, 29% in Pakistan in the same year. The proportion was 5% among women in Timor-Leste from 2009 to 2010 (UNICEF, 2013).

In Africa, various studies on the prevalence of obesity among women of reproductive age have been carried out. A study carried out in Nigeria reported the prevalence of obesity to be 33.1% among pregnant women who registered for antenatal care within the first trimester (Ajen *et al.*, 2014). In a study in Gabon, an increase in the proportion of overweight among women was noted to be nearly 13.22% between 2000 and 2012 (Yongsi and Ngwa, 2015). A study carried out in Ghana observed the prevalence of overweight to be about 34% among female teachers of child-bearing age while 27% of them were found to be obese with 17.8% centrally-obese (Pobee *et al.*, 2013). The overall prevalence of maternal overweight and obesity as reported in 27 demographic and health survey (DHS) countries surveyed in sub-Saharan Africa was 16.5% (95% CI 16.1-17.0%) (Wojcicki, 2014). According to a recent WHO report, the percentage of women of childbearing age classified as overweight (including obesity) in WHO African Regions is at a median of 23.8%. The range is from 5.7% in Ethiopia to 50.6% in Swaziland. In all, 12 countries have a prevalence rate of over 30% (Gabon, Ghana

and Lesotho have over 40% overweight prevalence). Burkina Faso, Burundi, Chad, Eritrea, Ethiopia and Madagascar have prevalence rates for overweight including obesity below 15% (WHO, 2017).

In Cameroon, the estimated prevalence of obesity, based on body mass index (BMI), was 17.1% in women in urban Cameroon by 2002 (Sobngwi *et al.*, 2002). A study carried out in Cameroon using the Cameroon Burden of Diabetes Baseline Survey revealed the prevalence of obesity among women to be 19.5% using BMI (Kamadjeu *et al.*, 2006). It has been reported that, the prevalence of obese women among women of childbearing age was 29.40% in 2004 as compared to 32.17% in 2011 (DHS, 2004; DHS, INS & ORC Macro, 2011); an increase in the proportion of overweight among women of childbearing age was noted to be nearly 2.77% between 2004 and 2011 (Yongsi and Ngwa, 2015). In the recent 2018 Cameroon DHS report, 37 % of women were overweight or obese. In this report, overweight/obesity was twice as common among women in urban areas as women in rural areas (NIS & ICF, 2020).

I.1.2.3. Causes and consequences of over nutrition among women of childbearing age

I.1.2.3.1. Causes of overnutrition

Adult obesity results from long-term positive energy balance or simply storing more energy than your body requires for normal day-to-day functions. However, this over simplifies the many causal factors responsible for obesity in modern society. Obesity may be caused by one or more of a combination of factors, including but not limited to, genetic predisposition, in utero determinants related to prenatal health, lack of physical activity, poor nutrition (namely excessive caloric consumption), socio-economic drivers, gender, lack of sleep, medication use and/or underlying medical problems that promote weight gain (Health Nexus Santé, 2013). It has been reported that physical activity level and diet are major variables of obesity although researchers believe that obesity has multiple possible determinants (Mehrabani and Ganjifar, 2018). This study focuses on causes such as changing lifestyle implicating poor nutrition and lack of physical activity, food insecurity level, socioeconomic factors and genetic predisposition.

1. Changing lifestyle

Lifestyle changes characterized by the high availability of energy-dense foods and a high level of physical inactivity together with genetic predisposition play an important role in the obesity problem (Erik *et al.*, 2011). Nutrition transition characterized by changing lifestyles and diet composition has been defined as dietary and physical-activity changes, reflected in nutritional outcomes such as changes in average stature and body composition. Relevantly, nutrition transition refers to the shift from traditional diets composed of whole foods, such as pulses and whole grains, and that are low in animal-source foods, salt, and refined oils, sugars, and flours, to an energy-dense and nutrient-poor diet composed of refined carbohydrates, high fat intake, and processed foods. Relative to traditional diets, both total caloric intake and energy density tend to be higher in modern diets owing to increased consumption of processed foods and fats facilitated by increased income, urbanization, and shifts in food availability and pricing (Ford *et al.*, 2017); which has been associated with the documented increases in Non-communicable diseases (NCDs) like obesity and degenerative diseases (Habib-Mourad, 2013).

1. Poor Dietary habits

a.) Low or lack of consumption of Fruits and vegetables

Fruit and vegetables tend to have low energy density and are high in fibre, which may enhance satiety. Sufficient intake of fruit and vegetables (F&V) has been related epidemiologically with reduced risk of many non-communicable diseases (Pem and Jeewon, 2015) including obesity. A high intake of fruits and vegetables have been shown to protect against excess weight due to the fact that fruits and non-starchy vegetables are very low in energy, since they contain high amount of water and fiber and can be consumed in a relatively larger amount contributing to increased satiety to maintain normal weight (Tohill *et al.*, 2004). Fibers also form a gel-like environment in the small intestine, resulting in reduced activity of the enzymes involved in the digestion of fat, protein and carbohydrates (Alinia *et al.*, 2009; Pem and Jeewon, 2015). One large prospective study among middle aged women observed that increasing fruit and/or

vegetable intake was associated with a reduced risk of major weight gain (≥ 25 kg) or becoming obese ($\text{BMI} \geq 30 \text{ kg/m}^2$) (He *et al.*, 2004).

b.) Consumption of Carbohydrates

Over the years, the diets of populations have been changing towards diets that favour the weight gain as traditional diets are gradually replaced with modern diets. The role that the increase intake of carbohydrates play in the onset of obesity tend to be linked to the type of carbohydrates (Mbochi, 2010). For instance, an increase intake of whole grains, primarily from food such as wholegrain breads and breakfast cereals, have also been associated with reductions in weight gain (Koh-Banerjee *et al.*, 2004). This effect may be partly mediated by the fibre content of wholegrain food (Branca *et al.*, 2007). Also, it has been shown that traditional diet characterized by low-energy, nutrient-dense with high-quality carbohydrates may be beneficial for reducing the risk of many chronic diseases including obesity. Indeed, a low–energy-density diet may have a higher capacity to prevent nutritional deficiency, despite the lower energy content, than a high–energy-density diet (Willcox *et al.*, 2009). In addition, it has been reported that, staple food-based diets typically lack dietary diversity and have been associated with micronutrient deficiencies. To add to this, diets high in carbohydrates and low in fat are associated with increased plasma triglycerides and decreased high-density lipoprotein cholesterol (HDL-c), both of which are associated with cardiometabolic disease (Ford *et al.*, 2017).

c.) High Consumption of fats and oils

Dietary fat provides about one third of total energy intake in most high-income countries, and there has been considerable debate on its role in causing obesity (Bray and Popkin, 1998; Willett, 1998). Dietary fat is readily stored as body fat with minimal energy costs of conversion. Fat is less satiating than isoenergetic quantities of other nutrients, and habitual consumption of a high-fat diet may downregulate some elements of the appetite-control system, favouring decreased satiety. Early mechanistic studies noted a phenomenon described as high-fat hyperphagia, in which people on high-fat diets tended to consume excess energy (Stubbs *et al.*, 1995). This effect is usually ascribed to the higher energy density of these diets relative to carbohydrate or protein, rather than the fat content per se (Branca *et al.*, 2007). Different types of fat have

different metabolic effects, and this may extend to differences in the risk of weight gain (Storlien *et al.*, 2001).

d.) Sugar-rich drinks

Consumption of sugar-sweetened beverages (SSB's) is often linked to an excess in caloric intake and the increasing prevalence of obesity. It is hypothesized that the calories in SSB's have little effect on satiety and therefore easily lead to over-consumption. SSB's are defined as drinks with added sugars, excluding milk and pure fruit juices (Erik *et al.*, 2011). A review of the literature between 1966 and 2006 on the relationship between SSB's and weight gain by Wolff and Dansinger (Wolff and Dansinger, 2008) revealed that six of 15 cross-sectional and six of 10 prospective cohort studies identified statistically significant associations between soft drink consumption and increased body weight (Erik *et al.*, 2011). Also, it has been pointed out that added sugars are a dietary driver of obesity worldwide, especially when consumed in beverages such as soft drinks, sweetened coffee and tea, juices, and alcoholic beverages. In most LMICs, sugar-sweetened beverage (SSB) sales are increasing (in daily calories per person) and represent an important source of caloric intake in many parts of the developing world (Ford *et al.*, 2017).

e.) Consumption of Alcohol

Although moderate alcohol use is recommended, excessive alcohol consumption is the third leading cause of premature death in the United States (behind smoking and obesity). Based on the fact that 1 gram of alcohol provides 7.1 kcal (29 kJ) and studies showing that energy consumed as alcohol is additive to that from other dietary sources, increased energy intake with alcohol use can certainly promote a positive energy balance and ultimately weight gain. However, a clear cause-and-effect association between alcohol intake and weight gain is not apparent based on the mixed and conflicting available evidence on the topic (Traversy and Chaput, 2015). For instance, it has been pointed out that, several studies in adults have found that the amount or intensity of drinking per drinking occasion is positively correlated with BMI, while the frequency of drinking is negatively correlated, suggesting that frequent light drinking might offer a protective effect. Despite the recent body of cross-sectional evidence suggesting the

benign or potentially protective effect of frequent light drinking on body weight and obesity, several studies have found conflicting results (Traversy and Chaput, 2015).

2. Physical inactivity and sedentary lifestyle

Physical inactivity is one of the four common risk factors of this non-communicable diseases burden, together with unhealthy diet, smoking and excessive consumption of alcohol (Assah *et al.*, 2015). Inactivity on the other hand is one of the most important factors that have been known to fuel overweight and obesity (Mbochi, 2010). It has been brought out that, population physical activity levels have been reported to be declining in developed and developing countries. The decline in developing countries may be due to urbanisation as demonstrated by a rural-to-urban negative gradient of physical activity levels (Assah *et al.*, 2015). The amount of physical activity needed to prevent gaining or regaining weight obviously depends on the habitual food intake among the population in question. This means that the nutritional context must be considered (Branca *et al.*, 2007). In Cameroon, only 3.20% of women are engaged in regular physical activities (DHS, INS & ORC Marco, 2011). This sedentary condition and lack of physical exercises affect many women who share "secondary or higher" educational level, and performing other activities that requires little physical effort. Due to their occupational status, they are predisposed to adopt new eating habits (nibbling between meals, meals at irregular intervals, ...) associated with new life styles which contribute to reduce physical activities (using a car for short distances, long hours spent sitting in front of a computer or TV screen) (Yongsi and Ngwa, 2015) thereby promoting weight gain and obesity. Also, a research carried out in Cameroon found out that obesity was strongly inversely associated with physical activity energy expenditure (PAEE) in rural and urban dwellers (Assah *et al.*, 2015).

3. Food insecurity and security

Food insecurity has been viewed as one of the potential mechanisms underlying the relationship between poverty with obesity and other adverse health outcomes (Finney *et al.*, 2010). Kaiser and colleagues found that food-insecure Hispanic/Latino women were 98 % more likely to be obese (BMI.30.0 kg/m²) than their food-secure counterparts in a convenience sample of Hispanic/Latino women (Kaiser *et al.*, 2004). Increasing food

insecurity in the developing world paradoxically has resulted in increasing numbers of overweight (Keino *et al.*, 2014). Women and children are vulnerable to malnutrition and food insecurity. Further, women globally have higher rates of obesity than men. The results from a study on food insecurity among women of childbearing age carried out by Keino and collaborators in Kenya indicated that household food insecurity was a predictor of overweight but not a statistically significant predictor of underweight among these women (Keino *et al.*, 2014). A possible mediator of the association between food insecurity and obesity according to some authors is dietary behaviour, where food-insecure individuals may reduce the quality and/or quantity of foods consumed (Leung *et al.*, 2012). Another study carried out in Malaysia by Shariff and collaborators found out that after controlling for demographic and socioeconomic covariates, women in food insecure households were less likely to have MetS (individual food insecure and child hunger), abdominal obesity (individual food insecure and child hunger), elevated glucose (household food insecure), total cholesterol (child hunger) and LDL-cholesterol (household food insecure and child hunger) compared to food secure women. The same authors stated that for food secure households, despite the access to a variety of food choices, the increasing food prices could also force them to avoid relatively expensive foods (ie fruits, vegetables, fish and lean meats) in order to maintain food variety and higher energy intake at a lower cost (Shariff *et al.*, 2014).

4. Socioeconomic status and obesity

As pointed out by Drewnowski and collaborators, Socioeconomic status (SES), urban form and the food environment can exert a powerful influence on body weights and health (Drewnowski *et al.*, 2013). Multiple mechanisms linking SES to obesity have been proposed; including the relationship of education levels, income, and other markers of SES to lower levels of recreational physical activity, poor nutrition, and certain psychosocial factors. Both in France and in the United States, higher obesity rates are associated with lower education and incomes, lower occupational status (Drewnowski & Specter, 2004; McLaren, 2007) and with lower-quality diets (Mendoza *et al.*, 2007; Méjean *et al.*, 2011). New geospatial analyses of residential neighborhoods in the United States have found higher obesity rates in more deprived and underserved areas (Bodor

et al., 2010; Rundle *et al.*, 2009). In developing countries, however, the level of obesity is greater in the higher socioeconomic status segments of society (Wang, 2001). Evidence of this exists in Brazil, (Montiero *et al.*, 2004), Cameroon (Fezeu *et al.*, 2005), India (Reddy, 2002) Jordan (Montiero *et al.*, 2004) and Madagascar (Montiero *et al.*, 2004). A study carried out in Cameroon observed that women, who lived in households with an average or a higher standard of living, were 1.37 and 1.65 times more likely to be obese respectively as compared to those living in household with a lower standard of living (Yongsi and Ngwa, 2015). A recent study carried out in urban Cameroon among children aged 3-13 years found out that after adjustment for only age and gender, a high socioeconomic status was associated with a 2.40 higher risk of overweight/obesity compared to a low socioeconomic status (Choukem *et al.*, 2017).

5. Individual/ biological susceptibility and Genetic predisposition to obesity

Individual/ biological susceptibility to overweight and obesity research has revealed the important role of biological factors in the regulation of body weight. Some people are more susceptible than others to becoming overweight and obese. For instance basal metabolic rate (BMR) affects body weight and weight loss because some individuals naturally use more calories to sustain basic body processes. The size and number of an individual's fat cells also help determine the amount of weight loss that is possible. Obesity is also partially determined by a person's genetic make-up. Genes involved in weight gain increase the risk of susceptibility of an individual to the development of obesity when exposed to an adverse environment. Genetics plays an important role in determining a person's susceptibility to obesity (Mbochi, 2010). Genes can favor fat accumulation in a given environment by increased desire to overeat; the tendency to be sedentary; a diminished ability to utilize dietary fats as fuel; an enlarged, easily stimulated capacity to store body fat. The variation in how people respond to the same environmental conditions is an additional indication that genes play an important role in the development of obesity (Via & Hawthorne, 2005). The influence of genes ranges from polygenic genetic predisposition with impact on appetite, metabolism, and the deposition of fat, to rare monogenic disorders where obesity is the primary feature (Puiu *et al.*, 2013). Even if genes do cause obesity, genetic factors may influence the food

intake and activity patterns that lead to it and metabolic pathways that maintain it (Rolfes *et al.*, 2006). For example, Leptin gene is also an important determinant of overweight and obesity. It is produced in the adipose tissue and secreted into the blood in relation to the amount of body fat. Leptin-deficient individuals are massively obese and when leptin is administered, food intake falls and body fat is mobilized until body weight is nearly normalized, indicating that important metabolic-genetic pathways exist that can control body fat (Bray & Champaigne, 2005; Mbochi, 2010).

I.1.2.3.2. Consequences of over nutrition (overweight and obesity) in women of childbearing and offsprings

There is compelling evidence that overweight or obese individuals are at increased risk of a variety of health problems. Women who are obese during their childbearing years are at risk for conditions such as polycystic ovary syndrome (Motta, 2012), menstrual irregularities, as well as poor cardiometabolic health (the clustering of various risk factors, namely insulin resistance, high triglycerides, high blood pressure and low HDL cholesterol putting an individual at high risk of developing type 2 diabetes) and cardiovascular disease. Women who are obese are also at higher risk for hyper tension, type 2 diabetes mellitus (Ovesen *et al.*, 2011), respiratory issues (sleep apnea, asthma), thromboembolic disease, mental health issues such as depression and osteoarthritis (Dietl, 2005; Smith *et al.*, 2008).

BMI above the normal range of 19.8 to 26.1 (IOM, 1990) is also associated with a number of adverse reproductive health outcomes. For example, infertility, gestational diabetes and type 2 diabetes (Hedderson *et al.*, 2008; Chu *et al.*, 2007), pregnancy induced hypertension and pre-eclampsia, birth defects, large for gestational age (LGA) or macrosomia (>4500 g), cesarean sections, prolonged labor, and recently postpartum anemia have all been associated with maternal overweight yet the exact mechanisms have not been identified (Siega-Riz & Laraia, 2006).

With regards to fetal outcome, maternal pre-pregnancy obesity and excessive gestational weight gain are important risk factors for multiple adverse fetal outcome. A review article, pointed out that large meta-analyses have shown that a higher maternal pre-

pregnancy or early pregnancy BMI is associated with increased risks of fetal death, stillbirth, neonatal death and the development of various congenital anomalies (Gaillard *et al.*, 2016). Also, maternal pre-pregnancy obesity has been reported to be associated with a 2-fold higher risk of delivering larger sized gestational age infants (Gaudet *et al.*, 2014)

I.1.2.4. Impacts of obesity on the reproductive health of women of childbearing age

It has been reported that although studies have shown that the mechanisms of impacts of obesity on fertility are not well understood, yet obesity has been shown to be associated with several reproductive disturbances especially abdominal phenotype of obesity (Ojiegbe, 2016). It has been observed that a good number of women who suffered from different menstrual disorders, infertility and recurrent miscarriages were either overweight or obese (Rogers & Mitchell, 1952; Ojiegbe, 2016). Findings of another study also showed that the incidence of anovulatory cycles, oligomenorrhoea and hirsutism are higher in obese women than in normal-weight women; there was also higher incidence of infertility among women (adult married women without children) who were obese during puberty and early adolescence than in those who were not obese (Rogers & Mitchell, 1952; Ojiegbe, 2016). Obesity could lead to functional hyperandrogenism and hyperinsulinaemia which accompanies the insulin-resistant state. In women with the polycystic ovary syndrome, abdominal obesity may be co-responsible for the development of hyperandrogenism and the associated chronic anovulation, through mechanisms primarily involving the insulin mediated overstimulation of ovarian steroidogenesis and decreased sex hormone binding globulin blood concentrations (Pasquali *et al.*, 2003; Ojiegbe, 2016). In obese women and/or those with polycystic ovary syndrome (PCOS), an increase in the number of fat cells results in increased leptin and insulin levels and an increase in luteinizing hormone (LH), but not follicle stimulating hormone (FSH) levels, thereby stimulating the development of follicles that secrete supranormal levels of testosterone, which rarely ovulate (anovulation), hence low progesterone. These could be aggravated by insulin-induced reduction in sex-hormone binding globulin (SHBG) from the liver, which

increases ovarian testosterone production/action (Sharpe and Franks, 2002; Ojiegbe, 2016).

I.1.2.5. Efforts to reduce obesity and its consequences in women of childbearing age in Cameroon

Weight loss can be achieved by lifestyle modification, dietary restriction, physical activity and pharmacotherapy with varied results. In developing regions, this may be even more of a challenge as they are not accustomed to dealing with overweight and obesity. In addition, these countries have more pressing needs like food insecurity and infectious diseases (Mbochi, 2010).

In Cameroon, the government has started to prioritize chronic non-communicable diseases in his health agenda. However, this is mostly happening for diabetes, probably because the vast majority of the available evidence to formulate policies originates from the local diabetes research community. In the country since 2001, a number of health policies relevant to chronic diseases management, as well as those addressing standards and norms for a primary health care package and community care workers, have been formulated and adopted by the Cameroonian Ministry of public health. The baseline Cameroon Burden of Diabetes (CAMBoD) survey provided new scientific knowledge that guided health policy and the implementation of a diabetes and hypertension program (Njamnshi *et al.*, 2006).

Diabetes and hypertension were recognized as emerging public-health problems, and incorporated into a national 10-year plan for health promotion, with this leading to the creation of two bodies within the Ministry of Public Health: the Department of Applied Research; and the Department of Disease Control that focuses on non-communicable diseases. This also occurred as result of an increasing political will to develop policies and national programs to prevent and control non-communicable chronic diseases (Njamnshi *et al.*, 2006). The aim of the nationwide diabetes-hypertension control program is to promote equitable access to quality health services in order to reduce the morbidity and mortality linked to these conditions. With regards to dietary interventions, there is no national nutritional policy in Cameroon. Although there is a law on food labeling, which mandate that information on the nutritional value of foods, microbial

content and additives be clearly displayed on packaging, it is too recent to have been effectively implemented (Echouffo-Tcheugui & Kengne, 2011).

A recent study on the prevalence and time trends in overweight and obesity among urban women using DHS data from 24 African countries suggested that interventions and strategies on reducing overweight and obesity should focus on healthy diet, physical activity, weight reduction and maintenance strategies in African countries, particularly in urban areas to curb the growing proportion of unhealthy weight women of childbearing age in urban Africa. Strategies should include measures such as price reduction for healthy foods (eg, fruits and vegetables) and promotion of physical activity (Amugsi *et al.*, 2017).

I.2. Lifestyle practices among women of childbearing age

Reproductive years represent a major proportion of women's life, therefore adequate nutrition and healthy lifestyle behaviors such as physical activity and alcohol consumption are to be given significant consideration among women of reproductive age to optimize the health of their babies as well as their own health. An adequate nutrition before the reproductive years helps to ensure achievement of proper adolescent growth, sufficient nutrient store during reproductive years for a healthy pregnancy and an appropriate nutritional status specially to maintain skeletal health during the postmenopausal period (Dunneram & Jeewon, 2015). Women of childbearing age are at greater risk of adverse health outcomes than other population groups because of the increased physiological demands of pregnancy and lactation (Rai *et al.*, 2015). However, nutrition during the childbearing years is frequently neglected. It has been reported that women of reproductive age living in the Philippines, Bangladesh, Burkina Faso, Mali and Mozambique had significant deficiencies across a range of micronutrients including calcium, iron, niacin, folate, riboflavin and vitamin B (Arimond *et al.*, 2010; Rai *et al.*, 2015). In Cameroon, 82% women of childbearing age are being found with a low adjusted plasma zinc concentration (PZC) (Engle-Stone *et al.*, 2014).

I.2.1. Healthy lifestyle practices among women of childbearing age

Along with proper diet, other lifestyle patterns such as regular physical activity (PA) and reduced alcohol consumption are also indispensable among women of reproductive age (Dunneeram & Jeewon, 2015).

It is recommended that regular PA has to be established pre-conceptionally so as to prevent several complications of pregnancy (Berghella *et al.*, 2010). Women of childbearing age, especially those planning a pregnancy, should be encouraged to adopt a healthy lifestyle that includes exercise before conception (Nascimento *et al.*, 2015). Women who exercise regularly before pregnancy have been found more likely to continue to exercise during pregnancy (Hegaard *et al.*, 2010; Owe *et al.*, 2009). In addition, exercise prior to pregnancy help woman to control weight and prevent mood swings during pregnancy as well as minimize the likelihood of depression post pregnancy. Women are recommended to practice regular PA for 30 to 60 minutes per day for 5 or more days per week (Berghella *et al.*, 2010; Dunneeram and Jeewon, 2015). The US Centers for Disease Control and Prevention published preconception references to promote knowledge of healthy lifestyles practices and to discourage health risk behaviors (e.g., cigarette smoking and alcohol consumption) (Dunneeram & Jeewon, 2015).

Alcohol exposure during the prenatal period is one of the main sources of neurodevelopmental deficits among children, as well as those of fetal alcohol spectrum disorder (FASD) (Dunneeram and Jeewon, 2015). Due to the unique physiological characteristics, women are also more prone to several negative sequelae associated with alcohol use such as osteoporosis, reproductive problems, heart disease, stroke, breast cancer, brain damage and rapid progression to dependency, and liver cirrhosis, when compared to men (Machado *et al.*, 2013). There is no safe limit for alcohol in pregnancy and even small amounts of alcohol can lead to adverse effects on the fetus. There is a general consensus including a WHO policy statement that women must totally abstain from alcohol during pregnancy (Nilsen, 2009; Sharma, 2014).

Women intending a pregnancy should be advised to adopt healthy behaviors like smoking cessation. For women of childbearing age, active and passive smoking is linked

to reduced fertility (Murin *et al.*, 2011). Several studies concluded that maternal prenatal cigarette smoking disturbs the equilibrium among the oxidant and antioxidant system, thus causing additional oxidative stress and augmenting lipid peroxidation. Smoking during pregnancy increases the free radical damage to the unborn fetus as well as to the mother (Chelchowska *et al.*, 2011; Sahinli *et al.*, 2012). Intrauterine growth retardation of the unborn child is the most important smoking-induced pathology (Mund *et al.*, 2013).

I.2.2. Nutrition requirements for women of childbearing age

Women of reproductive age have the same dietary requirements as the general population whereas pregnant and lactating women have additional nutritional recommendations (Inskip *et al.*, 2009). For example; another 100 kcal/day in addition to dietary intake that allows a constant pre-pregnancy weight is generally satisfactory (Dunneram & Jeewon, 2015). In order to meet these guidelines, it is recommended that simple carbohydrates should be restricted while complex carbohydrates in the form of starches, legumes, seeds and bread should be limited to reasonable quantities. Useful protein sources can be meat, fish, cheese and dairy products (source of calcium), supplemented with small amounts of butter and vegetable fats (Dunneram & Jeewon, 2015).

Dietary Reference Intakes (DRIs) have been set for fat, carbohydrates (including sugars and dietary fibre) and proteins for adults. DRIs for total fat, saturated fat, total carbohydrates and sugars are given as a percentage of daily energy intake (British Nutrition Foundation (BNF), 2015).

Table 1 : Dietary Reference Intakes (DRIs) for fat, carbohydrates and proteins for adults

Macronutrients	% Daily Food Energy
Total Carbohydrate*	45-65%
<i>of which free sugars*</i>	Not more than 5%
Proteins	10-35%
Total Fat	20-35%
<i>of which Saturated Fat</i>	Not more than 11%
<i>n-6 polyunsaturated fatty acids a (linoleic acid)</i>	5-10%
<i>n-3 polyunsaturated fatty acidsa (α-linolenic acid)</i>	0.6-1.2%

SOURCE: Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids (2002/2005); British Nutrition Foundation, 2015

The table below presents the Recommended Dietary Allowances (RDAs) for Macronutrients and Micronutrients and Adequate Intakes (AIs) for total Water recommended by the Food and Nutrition Board, Institute of Medicine, National Academies for women of childbearing age.

Table 2: Recommended Dietary Allowances (RDAs) for Macronutrients and Micronutrients and Adequate Intakes (AIs) for total Water recommended

Age group	14-18 years	19-30 years	31-49 years
Total water (L/d)	2.3	2.7	2.7
Carbohydrate (g/d)	130	130	130
Total fiber (g/d)	26	25	25
Fats (g/d)	ND	ND	ND
Linoleic Acid (g/d)	11	12	12
α -Linolenic Acid (g/d)	1.1	1.1	1.1
Protein (g/d)	46	46	46
Vitamin A (μ g/d)	700	700	700
Vitamin C (mg/d)	65	75	75
Vitamin D (μ g/d)	15	15	15
Vitamin E (mg/d)	15	15	15
Thiamin (mg/d)	1.0	1.1	1.1
Riboflavin (mg/d)	1.0	1.1	1.1
Niacin (mg/d)	14	14	14
Vitamin B ₆ (mg/d)	1.2	1.3	1.3
Folate (μ g/d)	400*	400*	400*
Vitamin B ₁₂ (μ g/d)	2.4	2.4	2.4
Calcium (mg/d)	1300	1000	1000
Copper (μ g/d)	890	900	900
Iodine (μ g/d)	150	150	150
Iron (mg/d)	15	18	18
Magnesium (mg/d)	360	310	320
Molybdenum (μ g/d)	43	45	45
Phosphorus (mg/d)	1250	700	700
Selenium (μ g/d)	55	55	55
Zinc (mg/d)	9	8	8

* It is assumed that women will continue consuming 400 μ g from supplements or fortified food until their pregnancy is confirmed and they enter prenatal care, which ordinarily occurs after the end of the periconceptual period—the critical time for formation of the neural tube.

SOURCES: Institute of Medicine (US) Standing Committee on the Scientific Evaluation of Dietary Reference Intakes (1997). *Dietary Reference Intakes for Calcium, Phosphorous, Magnesium, Vitamin D, and Fluoride* (1997); *Dietary Reference Intakes for Thiamin, Riboflavin, Niacin, Vitamin B₆, Folate, Vitamin B₁₂, Pantothenic Acid, Biotin, and Choline* (1998); *Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium, and Carotenoids* (2000); and *Dietary Reference Intakes for Vitamin A,*

Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc (2001); Dietary Reference Intakes for Water, Potassium, Sodium, Chloride, and Sulfate (2005); and Dietary Reference Intakes for Calcium and Vitamin D (2011).

A Recommended Dietary Allowances (RDAs) is the average daily dietary intake level; sufficient to meet the nutrient requirements of nearly all (97-98 percent) healthy individuals in a group. It is calculated from an Estimated Average Requirement (EAR). If sufficient scientific evidence is not available to establish an EAR, and thus calculate an RDA, an AI is usually developed (www.nap.edu).

I.2.2.1 Micronutrients and their impact on women of childbearing age health

Micronutrients are critical for women's health during reproductive years and during pregnancy as well as during adolescence and post-menopausal period (Bartley *et al.*, 2005). Although poor dietary intake is a common cause of micronutrient deficiencies, other individual causes including genetics, nutrient and drugs interactions, poor absorption, as well as certain diseases can lead to such deficits. In particular, deficiencies of calcium, iron, folate, zinc, thiamine, riboflavin, and vitamins A, D, B-6 and B-12 are very frequent and of concern among women of reproductive age. Possible reasons for these micronutrients deficiencies are low income level and lack of education about healthy practices like healthy eating patterns (Dunneram & Jeewon, 2015). Deficiencies in these indispensable nutrients increase the predisposal to adverse pregnancy outcomes such as neural tube defects, early fetal loss, preeclampsia, gestational diabetes mellitus and maternal mortality (Bartley *et al.*, 2005; Becquey and Martin-Prevel, 2010; Dunneram & Jeewon, 2015).

I.2.2.2. Crucial minerals for the health of women of childbearing age

I.2.2.2.1. Magnesium: Functions, Absorption, excretion, Food sources, deficiency-causes and health consequences

Magnesium is an essential electrolyte for living organisms and is the fourth most abundant mineral in the human body. Humans need to consume magnesium regularly to prevent magnesium deficiency, but as the recommended daily allowance for magnesium varies; Based on magnesium's many functions within the human body, it plays an

important role in prevention and treatment of many diseases. Low levels of magnesium have been associated with a number of chronic and inflammatory diseases, such as Alzheimer's disease, asthma, attention deficit hyperactivity disorder (ADHD), insulin resistance, type-2 diabetes mellitus, hypertension, cardiovascular disease (e.g., stroke), migraine headaches, and osteoporosis (Song *et al.*, 2005; Gröber *et al.*, 2015). Also, it has been reported that, the high intracellular calcium induced by magnesium deficiency may induce both insulin resistance and hypertension (DiNicolantonio *et al.*, 2018).

Functions of Magnesium

Over 600 enzymes with magnesium as a cofactor are currently listed by the enzymatic databases, while an additional 200 are listed in which Mg²⁺ may act as an activator. More specifically, it mainly interacts directly with the substrate, rather than acting as a real cofactor (Fiorentini *et al.*, 2021). Magnesium (Mg) as a cofactor for many enzyme systems regulates diverse biochemical reactions in the body, including protein synthesis, muscle and nerve transmission, neuromuscular conduction, signal transduction, blood glucose control, hormone receptor binding, gating of calcium channels and blood pressure regulation. Some magnesium dependent enzymes are Na⁺/K⁺-ATPase, hexokinase, creatine kinase, protein kinase, and cyclases. Stores of Magnesium within the cell are found in high concentration in mitochondria, where magnesium plays a vital role in the synthesis of ATP (adenosine triphosphate) from ADP (adenosine diphosphate) and inorganic phosphate (Schwalfenberg & Genui, 2017). Magnesium is also necessary for structural function of proteins, nucleic acids or mitochondria. It is required for DNA and RNA synthesis, and for both aerobic and anaerobic energy production—oxidative phosphorylation and glycolysis—either indirectly as a part of magnesium-ATP complex, or directly as an enzyme activator (Gröber *et al.*, 2015; Schwalfenberg & Genui, 2017).

Magnesium maintains ionic gradients by keeping intracellular sodium and calcium low and potassium high (DiNicolantonio *et al.*, 2018). Magnesium plays a key role in the active transport of calcium and potassium ions across cell membranes, a process that is important for nerve impulse conduction, muscle contraction, vasomotor tone and normal heart rhythm. Also, it contributes to the structural development of bone and is required

for the adenosine triphosphate-dependent synthesis of the most important intracellular antioxidant glutathione (Castiglioni *et al.*, 2013; Gröber *et al.*, 2015; Fiorentini *et al.*, 2021).

Distribution of Magnesium in the Body

About 99% of total body magnesium is located in bone, muscles and non-muscular soft tissue (Classen & Nowitzki, 1990). Approximately 50%–60% of magnesium resides as surface substituents of the hydroxyapatite mineral component of bone. Most of the remaining magnesium is contained in skeletal muscle and soft tissue. The magnesium content of bone decreases with age, and magnesium that is stored in this way is not completely bioavailable during magnesium deprivation. Intracellular magnesium concentrations range from 5–20 mmol/L; 1%–5% is ionized, the remainder is bound to proteins, negatively charged molecules and adenosine triphosphate (ATP). Magnesium is categorized into three fractions. It is either ionized (55%–70%), bound to protein (20%–30%) or complexed with anions (5%–15%) such as phosphate, bicarbonate and citrate or sulphate. Red blood cells/serum magnesium ratio is about 2.8. Extracellular magnesium accounts for about 1%–3% of total body magnesium (Jahnen-Dechent & Ketteler, 2012) which is primarily found in serum and red blood cells. Normal serum magnesium concentration is about 0.76–1.15 mmol/L (Ismail & Ismail, 2010; Gröber *et al.*, 2015; Fiorentini *et al.*, 2021)

Absorption and Excretion of Magnesium

Magnesium homeostasis is maintained by the intestine, the bone and the kidneys. Magnesium is mainly absorbed in the small intestine. Of the total dietary magnesium consumed, only about 24%–76% is absorbed in the gut the rest is eliminated in the faeces (Jahnen-Dechent & Ketteler, 2012). A minor, yet important, regulatory fraction of magnesium is transported via the transcellular transporter transient receptor potential channel melastatin member TRPM 6 and TRPM 7—members of the long transient receptor potential channel family—which also play an important role in intestinal calcium absorption (Van der Wijst *et al.*, 2014; Fiorentini *et al.*, 2021). It is worth noting that intestinal absorption is not directly proportional to magnesium intake but is dependent mainly on magnesium status. The lower the magnesium level, the more of

the mineral is absorbed in the gut, thus relative magnesium absorption is high when intake is low and vice versa. Magnesium absorption and excretion is influenced by different hormones. It has been shown that 1, 25-dihydroxyvitamin D [1,25(OH)₂D] can stimulate intestinal magnesium absorption. Magnesium deficiency, which leads to reduced 1, 25(OH)₂D and impaired parathyroid hormone response, has been implicated in “magnesium-dependent vitamin-D-resistant rickets” (Zittermann, 2013). Several other factors, such as oestrogen or parathyroid hormone (PTH), in addition to 1, 25(OH)₂D, are involved in magnesium excretion. Of special importance is PTH. Absorption of both magnesium and calcium appears to be inter-related, with concomitant deficiencies of both ions well described. For example, the stimulation of PTH secretion in response to hypocalcemia acts to restore the serum calcium concentration to normal. Hypomagnesemia impairs hypocalcemic-induced PTH release, which is corrected within minutes after infusion of magnesium (Gröber *et al.*, 2015).

Magnesium food sources and intakes

It has been found that ‘Cereals’ and ‘Roots and Tubers’ contributed 65 and 11%, respectively, of the Mg supply across the African continent (Joy *et al.*, 2014). Water accounts for ~10% of daily magnesium intake (Marx and Neutra, 1997). Nuts, seeds, unprocessed cereals or unrefined whole grains, nuts and unrefined dark chocolate, bananas are also rich in magnesium. Tap, mineral and bottled water can also be sources of Mg (Fiorentini *et al.*, 2021). Legumes, fruit, fish and meat have an intermediate magnesium concentration. Some types of food processing, such as refining grains in ways that remove the nutrient-rich germ and bran, lower magnesium content substantially. Low magnesium concentrations are found in dairy products, except milk (Altura *et al.*, 1994; Gröber *et al.*, 2015; Fiorentini *et al.*, 2021).

The Food and Nutrition Board (FNB) of the Institute of Medicine (IOM) increased the dietary references intakes (RDA) for magnesium, based on the results of controlled balance studies. The new RDA ranges from 80 mg/day for children 1–3 year of age to 130 mg/day for children 4–8 year of age. For older males, the RDA for magnesium ranges from as low as 240 mg/day (range, 9–13 year of age) and increases to 420 mg/day for males 31–70 year of age and older. For females, the RDA for magnesium ranges

from 240 mg/day (9–13 year of age) to 360 mg/day for females 14–18 year of age. The RDA for females 31–70 year of age and older is 320 mg/day (FNB, IOM, 1997).

Magnesium deficiency and its causes

Magnesium deficiency is not uncommon among the general population: its intake has decreased over the years especially in the Western world. Hypomagnesaemia is defined as serum magnesium concentration <0.75 mmol/L. Early signs of magnesium deficiency are non-specific and include loss of appetite, lethargy, nausea, vomiting, fatigue, and weakness. More pronounced magnesium deficiency presents with symptoms of increased neuromuscular excitability such as tremor, carpopedal spasm, muscle cramps, tetany and generalized seizures. Hypomagnesaemia is frequently associated with other electrolyte abnormalities such as hypokalemia and hypocalcaemia (Gröber *et al.*, 2015). There are several causes of hypomagnesemia and one of the most relevant is an insufficient dietary intake (Piuri *et al.*, 2021). Conditions that may lead to hypomagnesemia include alcoholism, poorly-controlled diabetes, malabsorption (e.g., Crohn's disease, ulcerative colitis, coeliac disease, short bowel syndrome, Whipple's disease), endocrine causes (e.g., aldosteronism, hyperparathyroidism, hyperthyroidism), renal disease (e.g., chronic renal failure, dialysis, Gitelman's syndrome) and medication use. A variety of drugs including antibiotics, chemotherapeutic agents, diuretics and proton pump inhibitors can cause magnesium loss and hypomagnesemia (Gröber *et al.*, 2015; Piuri *et al.*, 2021). The risk of dietary magnesium deficiency is estimated at 0-10 % for magnesium in Cameroon due to inadequate intakes (Joy *et al.*, 2014). Ca supplementation may accentuate the problem of reduced Mg levels by impairing the retention of Mg leading to its excretion and consequently to Mg deficiency (Abrams & Atkinson, 2003).

Consequences of Magnesium deficiency

It has been reported that, magnesium deficiency has been linked to atherosclerosis, alterations in blood lipids and blood sugar, type 2 diabetes, myocardial infarction, hypertension, kidney stones, premenstrual syndrome and psychiatric disorders (Gröber *et al.*, 2015). In addition, it has been reported that, low Mg contributes to vascular calcification, accumulation of connective tissue in the vessel wall, altered lipid exchange

between the vessel walls and blood, increased triglycerides, accumulation of oxalate in vessel walls, and reduced cholesterol transport by HDL (Schwalfenberg & Genui, 2017). Also in humans Mg deficiency contributes to osteoporosis. Low serum Mg is a co-contributing factor to osteopenia in adults with sickle cell anemia (Elshal *et al.*, 2012). It has been reported that an association between serum Mg and bone density has been reported in pre and post-menopausal women. With respect to various results obtained from various research studies, it has been concluded that, Mg supplementation is beneficial in osteoporotic women (Castiglioni *et al.*, 2013).

I.2.2.2.2. Calcium: Distribution, Function, Homeostasis, Food sources and intakes, nutritional deficiency-causes and consequences

Calcium is the fifth most abundant element in the human body, with key role in skeletal mineralization, as well as a wide 1000 g present in adults. It plays a range of biologic functions. Calcium is an essential element that is only available to the body through dietary sources. Current dietary calcium recommendations range from 1000 to 1500 mg/d, depending on age. In some individuals, particularly the elderly, calcium supplements may be needed to achieve the recommended dietary calcium intake (Peacock, 2010). The body's calcium supply is stored in the bones and teeth where it supports their structure and function. Bone undergoes continuous remodelling, with constant resorption and deposition of calcium into new bone. The balance between bone resorption and deposition changes with age. Bone formation exceeds resorption in periods of growth in children and adolescents, whereas in early and middle adulthood both processes are relatively equal. In ageing adults, particularly among postmenopausal women, bone breakdown exceeds formation, resulting in bone loss that increases the risk of osteoporosis over time (IOM, 2010).

Distribution and functions of Calcium in the Body

The average adult skeleton contains 1200 g of calcium, present in the form of hydroxyapatite, an inorganic crystalline structure made up of calcium and phosphorus [$\text{Ca}_{10}(\text{PO}_4)_6(\text{OH})_2$], which provides rigidity (Theobald, 2005). In the circulation, Ca^{2+} exists in three forms: ionized (~51%, Ca^{2+}), protein-bound (~40%, primarily albumin-

bound) and complexed (~10%); the ionized portion is functional (Peacock, 2010). The protein-bound portion can be influenced by blood pH: increased by alkalemia and reduced by acidemia. Bone mineral metabolism influences Ca^{2+} concentration by releasing or absorbing circulating Ca^{2+} . When in balance, bone Ca^{2+} absorption equals bone Ca^{2+} resorption; hence, absorbed dietary Ca^{2+} is excreted by both the colon (~100–150 mg/day) and kidneys (~150–200 mg/day) (Tejwani & Qian, 2013).

Calcium plays a key role in a wide range of biologic functions, either in the form of its free ion or bound complexes. As reported, calcium is involved in muscle contraction, enzyme activation, cell differentiation, immune response, programmed cell death and neuronal activity. Such broad functions are maintained by tightly controlled calcium concentration in extracellular fluid and cellular compartments (Pu *et al.*, 2016). One of the most important functions as bound calcium is in skeletal mineralization. The vast majority of total body calcium (99%) is present in the skeleton as calcium-phosphate complexes, primarily as hydroxyapatite, which is responsible for much of the material properties of bone (Wang *et al.*, 2006). In bone, calcium serves two main purposes: it provides skeletal strength and, concurrently, provides a dynamic store to maintain the intra- and extracellular calcium pools. Non-bone calcium represents 1% of total body calcium (10 g in an adult). However, it is in constant and rapid exchange within the various calcium pools and is responsible for a wide range of essential functions, including extra- and intracellular signaling, nerve impulse transmission, and muscle contraction (Campbell, 1990; Bootman *et al.*, 2001). Serum calcium ranges from 8.8 to 10.4 mg/dl (2.2 to 2.6 mM) in healthy subjects and comprises free ions (51%), protein-bound complexes (40%), and ionic complexes (9%) (Robertson & Marshall, 1979).

To avoid calcium toxicity, the concentration of serum ionized calcium is tightly maintained within a physiologic range of 4.4 to 5.4 mg/dl (1.10 to 1.35 mM). Non-ionized calcium is bound to a variety of proteins and anions in both the extra- and intracellular pools. The main calcium binding proteins include albumin and globulin in serum and calmodulin and other calcium-binding proteins in the cell. The major ionic complexes in serum are calcium phosphate, calcium carbonate, and calcium oxalate (Peacock, 2010).

Calcium Homeostasis

As reported, extracellular calcium homeostasis is mainly controlled by three physiological modes, including intestinal calcium absorption, renal calcium reabsorption, and bone formation/resorption, which is mainly regulated by CaSR through the modulation of parathyroid hormone (PTH), calcitonin and 1,25-dihydroxyvitamin D₃ secretion (Pu *et al.*, 2016). Calcium homeostasis is largely regulated through an integrated hormonal system that controls calcium transport in the gut, kidney, and bone. It involves two major calcium-regulating hormones and their receptors—parathyroid hormone (PTH) and the PTH receptor (PTHrP) (Potts & Gardella, 2007) and 1, 25-dihydroxyvitamin D (1, 25(OH)₂D) and the vitamin D receptor (VDR) (Jurutka *et al.*, 2001)—as well as serum ionized calcium and the calcium-sensing receptor (CaR) (Brown, 2007). Serum calcium homeostasis has evolved to simultaneously maintain extracellular ionized calcium levels in the physiologic range while allowing the flow of calcium to and from essential stores. A decrease in serum or plasma calcium inactivates the CaR in the parathyroid glands to increase PTH secretion which has an effect on bones by acting on an osteoblast cell membrane receptor, activating adenylate cyclase and increasing intracellular cAMP, which increases the cell permeability to calcium. The increase in cytosolic calcium activates a pump that drives calcium from the bone to the extracellular fluid. The pump is enhanced by 1, 25 (OH)₂D₃ (Reichel *et al.*, 1989; Baker & Worthley, 2002). PTH secretion also acts on the PTH receptors in kidney to increase tubular calcium reabsorption and therefore, decrease urinary excretion; and in bone to increase net bone resorption. The increased PTH also stimulates the kidney to increase secretion of 1,25(OH)₂D, which activates the vitamin D receptor in gut to increase calcium absorption, in the parathyroid glands to decrease PTH secretion, and in bone to increase resorption. The decrease in serum calcium probably also inactivates the CaR in kidney to increase calcium reabsorption and potentiate the effect of PTH (Peacock, 2010).

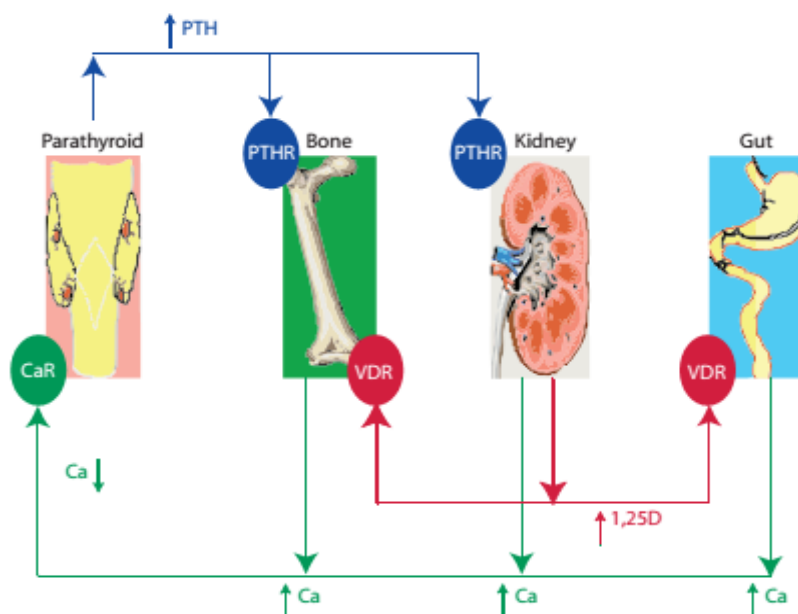


Figure 4 : Regulation of serum calcium homeostasis.

Serum calcium homeostasis is regulated by a rapid negative feedback hormonal pathway involving the concentration of ionized calcium in serum (Ca, green arrows) and the secretion of parathyroid hormone (PTH, blue arrows) from the parathyroid. A fall in serum calcium (2 Ca) inactivates the calcium receptor in the parathyroid cell (CaR; green circle) and increases PTH secretion (1 PTH), which restores serum calcium (1 Ca) by activating the parathyroid receptor (PTHR; blue circles) in bone, to increase calcium resorption, and in kidney, to increase tubular calcium reabsorption. In kidney, the increased PTH secretion augments its calcium-restorative effect by increasing secretion of 1,25-dihydroxyvitamin D (1,25D; red arrows), which, acting on the vitamin D receptor (VDR, red circles) in gut, increases active calcium absorption and increases calcium resorption in bone (Peacock, 2010).

Calcium food sources in Africa and Calcium Intakes

A wide range of foods contain calcium; with the amount of calcium provided on a per 100 g or serving basis and its bioavailability varying considerably (Theobald, 2005). In Africa, ‘Animal Products’ provided the greatest single contribution to Ca supply, ranging from 19 (Cote d’Ivoire) to 88% (Mauritania). Dairy products has been shown to be especially important sources of Ca in the Northern and Southern regions, supplying greater than 50% of Ca, while fish supplied 40, 38 and 30% of Ca in the Eastern, Middle and Western regions, respectively. ‘Roots and Tubers’ (notably cassava) have been reported as important sources of Ca in the Western and Middle regions, e.g. supplying 51% of Ca in Cote d’Ivoire. ‘Fruits and Vegetables’ are important sources of Ca, particularly in Northern Africa, e.g. supplying 39% of Ca in Egypt and 36% in Morocco (Joy *et al.*, 2014). To assure optimal whole body calcium retention and consequently adequate development and maintenance of bone mass and mineral density, different intake levels for calcium are recommended by FAO/WHO experts for infants,

children and adults. For children and adolescents between 10–18 years of age, consumption of 1,300 mg per day is recommended, while 1,000 mg per day apply for men and women between 19–50 years of age. Higher intake is necessary during pregnancy or after menopause. Recommended calcium allowance per day for males over 65 years and postmenopausal women is 1,300 mg (Peterlik *et al.*, 2009). Well established calcium rich-foods include: milk, yogurt, cheese, soybean, soy milk, tofu, broccoli, orange and others. The major forms of calcium supplements are calcium carbonate and calcium citrate (Pu *et al.*, 2016).

Calcium deficiency and its causes

Hypocalcemia is a term used clinically to refer to abnormally low serum or plasma calcium concentrations. Hypocalcemia indicate serious disruption of calcium homeostasis but does not on its own reflect calcium balance. The estimated mean risk of dietary Ca deficiency throughout Africa has been estimated to be 54%, the greatest deficiency risk of all micronutrients studied by these authors; this deficiency was >95% in 16 of the 46 countries examined. The risk of Ca deficiency was greatest in the Southern region (99%), followed by Eastern (69%), Northern (62%), Western (36%) and Middle (31%) regions of Africa. In Cameroon, the risk of dietary calcium deficiency is estimated at 11-25 percentage (Joy *et al.*, 2014).

Causes of Nutritional calcium deficiency

1.) Intestinal Calcium malabsorption

Dietary intake and absorption are essential to provide sufficient calcium to maintain healthy body stores. Approximately 30% of dietary calcium ingested in a healthy adult is absorbed by the small intestine. Calcium absorption is a function of active transport that is controlled by 1, 25(OH) 2D, which is particularly important at low calcium intakes, and passive diffusion, which dominates at high calcium intakes. Typically, at normal calcium intake, 1, 25(OH) 2D-dependent transport accounts for the majority of absorption, whereas as little as 8 to 23% of overall calcium absorption is caused by passive diffusion (McCormick, 2002). Calcium absorption is lowered if the bioavailability of dietary calcium is lowered by calcium-binding agents such as

cellulose, phosphate, oxalate (Peacock, 2010) and phytate (Joy *et al.*, 2014). A variety of diseases of the small bowel, including sprue and short bowel syndrome, can result in severe calcium malabsorption resulting in calcium deficiency at the level of the serum or plasma (Peacock, 2010).

2.) Absorptive hypocalcemia

Caused solely by a low dietary calcium intake is rare, because the homeostatic mechanisms are highly efficient and maintain serum calcium in the low physiologic range at the expense of calcium stores in bone. However, absorptive hypocalcemia is common in states of low, or inappropriately low, serum 1, 25(OH) 2D as occurs in chronic vitamin D deficiency, osteomalacia, and rickets or in impaired 1, 25(OH) 2D production as occurs in chronic kidney diseases (Peacock, 2010).

3.) Tubular reabsorptive hypocalcemia

This arises from a sustained decrease in tubular calcium reabsorption as occurs in postsurgical hypoparathyroidism, abnormalities in the parathyroid hormone receptor (PTHr) complex, and activating CaR mutations (Peacock, 2010) resulting in its excretion.

Consequences of Nutritional Calcium deficiency

It has been reported that, low dietary calcium causes hyperparathyroidism by impairment of CaR activity and, by the same token, can be linked to the development of not only osteoporosis and various malignancies, but possibly other calcium-insufficiency-related chronic diseases which are supported by evidence from different kinds of studies as shown below (Peterlik *et al.*, 2009).

Table 3 : Calcium deficiency related chronic diseases with respect to types of studies

Types of studies	Calcium-insufficiency-related chronic diseases
Convincing evidence from multiple epidemiological (prospective, cross-sectional, retrospective) large cohort studies, interventional trials and experimental studies	-Osteoporosis -Cancer (colorectal, breast)
Good evidence from >3 observational studies and/or interventional trials	-Cancer (renal) -Cardiovascular disease -Hypertension -Neuromuscular dysfunctions -Periodontal disease, tooth loss
Emerging evidence from observational studies	-Metabolic Syndrome -Diabetes mellitus Type II
Evidence mainly from studies with animal models of the respective human disease	-Inflammatory bowel disease -Multiple Sclerosis

Source: Peterlik *et al.*, 2009

I.2.2.2.3. Calcium to Magnesium Ratio and Calcium to Magnesium interaction

Dietary Calcium to magnesium ratios close to 2.0 had been advised for optimal health outcomes in humans. Just as a calcium to magnesium ratio $> 2.6 - 2.8$ can result in a detrimental effect, baseline calcium to magnesium ratios < 2.0 may also have a detrimental effect (Rosanoff *et al.*, 2016).

Increasing calcium to magnesium ratio in the USA population has been associated to higher calcium intake via food selections, the rising calcium content of food, or both (Rosanoff *et al.*, 2012). The importance of the cellular calcium-to-magnesium ratio for the physiological function of several tissues has been largely elucidated. It has been shown that a strong physiological/cellular link exist between a rising intracellular ratio of calcium to magnesium and aspects of metabolic syndrome, including hypertension, hyperinsulinemia, insulin resistance, and left ventricular cardiac hypertrophy. Inflammatory syndrome can also be added to the effects of possible cytosolic calcium activation as a result of magnesium deficit (Rayssiguier *et al.*, 2010) and its concomitant high calcium-to-magnesium ratio within cells (Rosanoff *et al.*, 2012). Magnesium deficiency may be a common link between stress, inflammation, and metabolic syndrome because magnesium deficiency at the cellular level can elicit calcium activation in an inappropriate response, i.e., the calcium-activated cascade is not triggered by an environmental injury or pathogen but rather as a result of a magnesium deficit that manifests in various tissues as aspects of CVD, type 2 diabetes miletus

(T2DM), and other health conditions associated with low magnesium. Active research on the discovered TRPM channels, which regulate both calcium and magnesium ion transport and calcium binding proteins such as those with the EF-hand motif that depend upon adequate Mg^{2+} to remain “at rest,” may lead to an understanding of possible mechanisms to explain how rising calcium-to-magnesium ratios at the cellular level may be among the root causes of metabolic syndrome and its links to T2DM, CVD, osteoporosis, and other diseases. Other proteins important in the cellular transport of magnesium may yet be found (Meyer *et al.*, 2010) in the complex dynamics of magnesium homeostasis. It is possible that the cellular calcium activation phenomenon is part of the pathology of a dietary magnesium deficit caused by low dietary magnesium, which can be exacerbated by a high dietary calcium-to-magnesium ratio, and this inappropriate calcium activation at the cellular level can lead to T2DM, CVD, or other manifestations of magnesium deficiency if the magnesium inadequacy is not corrected (Rosanoff *et al.*, 2012). According to Rosanoff and collaborators, high calcium intakes can exacerbate the onset of low magnesium status and vice versa. Studies showed that a calcium to magnesium intake ratio <2.8 is critical for optimal health, supporting a long-held but non-evidence-based recommendation that the calcium to magnesium ratio should be close to 2 (Rosanoff *et al.*, 2016).

I.2.2.2.4. Iron, its functions and metabolism

Given its central role in key biological processes, iron is one of the most important micronutrients for human populations. One key process is that of tissue oxygenation, which is accomplished by red blood cells (RBCs); generation of RBCs requires hemoglobin, of which iron is a key component. New RBCs are also created to replace RBCs that are lost from normal turnover, shedding (of skin cells or from the intestinal lining) or via hemorrhage (Miller, 2013; Burke *et al.*, 2014). Situations that require an increase in RBCs (such as the increased tissue mass of a growing fetus or infant) will consequently increase iron requirements. Iron homeostasis is tightly regulated because, absorption of iron is primarily regulated within the intestine, and once iron has been absorbed, there is no mechanism of excretion from the kidneys or liver. After absorption, iron is either stored within ferritin, which keeps the iron in a

nonreactive state within cells, or within transferrin, which also keeps the iron in a nonreactive state, but maintains it in aqueous circulation, so that it can be delivered to cells (Andrews, 2008). Stored iron (as ferritin) within cells, can be located in the cytoplasm, nucleus or mitochondria. With the assistance of divalent metal transporter 1 (DMT1; importing function) and ferroportin (exporting function), iron is transported across cell membranes. Ferroportin can be bound by the protein hepcidin, preventing ferroportin's export function and, thus, decreasing levels of serum iron (Figure 3) (Andrews, 2008). Most stored iron is present as ferritin whether in serum or within cells, (WHO, 2004; Burke *et al.*, 2014).

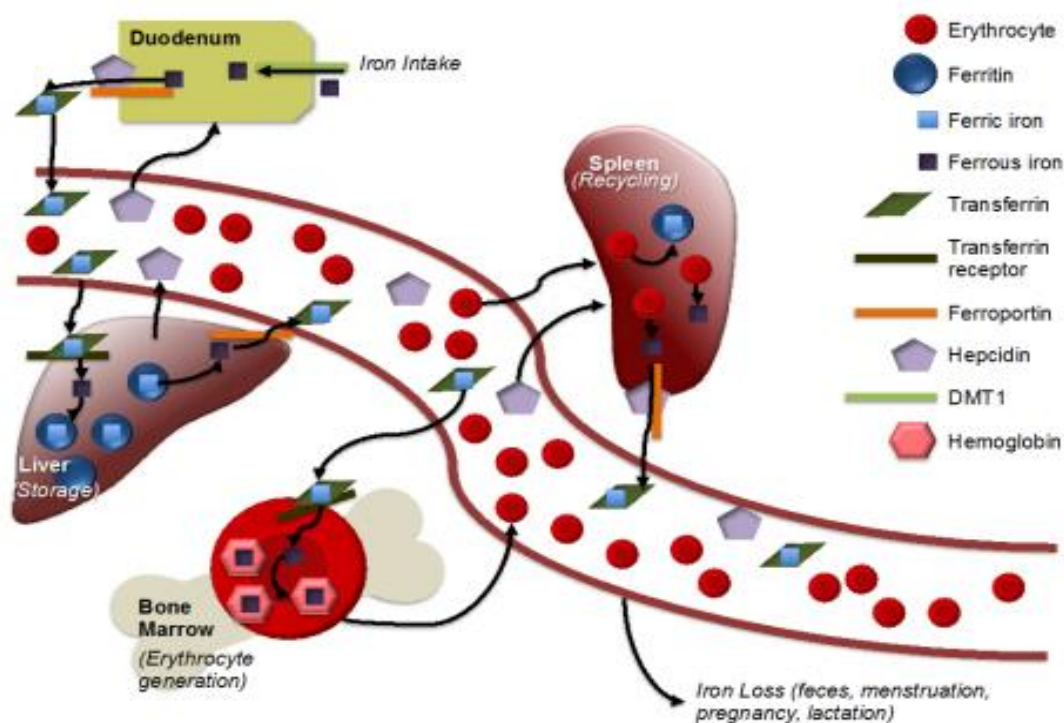


Figure 5 : Simplified representation of iron metabolism.

Iron is absorbed in the intestine, with non-heme iron being imported by divalent metal transporter 1 (DMT1). Ferrous iron is reduced to ferric iron and then exported by ferroportin. Within an aqueous solution, iron is stored within transferrin. Transferrin-bound iron is imported with the help of the transferrin receptor into the liver, heart and other storage areas, where it is stored within ferritin. Hepcidin, produced by the liver, helps to regulate iron metabolism by binding to ferroportin and, thus, inhibiting iron export. Within the bone marrow, iron is incorporated into hemoglobin for incorporation into erythrocytes. Macrophages recycle iron from erythrocytes, largely in the spleen. There is no mechanism for iron excretion by the kidneys or liver, though small amounts are lost via feces. Menstruation, pregnancy and lactation result in iron loss in women (Escobar-Morreale, 2012, Burke *et al.*, 2014).

Iron requirements for women and food source

Iron recommendations during pregnancy (27 mg per day) far exceed those for non-pregnant, non-lactating women (18 mg per day). While iron recommendations for lactating women are much lower than those for non-pregnant, non-lactating women (9 mg vs. 18 mg), this number is based on the assumption of lactation-induced amenorrhea and does not take into account that many women enter or conclude pregnancy with iron insufficiency or deficiency (Picciano, 2003). The Reference Nutrient Intake (RNI) recommended for non-pregnant and non-lactating women aged 19-50 years is 29.4 mg (FAO/WHO, 2004). Food sources of iron include meat, fruits and vegetables and an enhancer of iron absorption like ascorbic acid (Beck *et al.*, 2014).

Iron deficiency and its causes

The spectrum of iron deficiency is typically characterized by three phases: iron depletion, iron deficiency (iron deficient erythropoiesis or non-anemic iron deficiency), and iron deficiency anemia. The terminology and cut-off values for the biochemical measures of each of these stages often vary. In the first phase (iron depletion), the body's stores of iron decrease, reflected by a reduction in serum ferritin concentration. During the second phase (iron deficiency), tissue iron stores also decrease, and some iron-dependent functions are compromised. Serum ferritin concentrations are further reduced, serum iron decreases and total iron-binding capacity increases, resulting in a fall in transferrin saturation (Gibson, 2005; Beck *et al.*, 2014). In this phase, soluble transferrin receptor and zinc protoporphyrin concentrations increase. In the final phase (iron deficiency anemia), oxygen supply to the tissues becomes impaired, which is reflected by a decrease in hemoglobin concentrations (Gibson, 2005; Beck *et al.*, 2014).

Iron deficiency is associated with impaired physical work capacity (Brownlie *et al.*, 2004), reduced mood and cognitive function (Murray-Kolb *et al.*, 2011), and poor pregnancy related outcomes (Scholl *et al.*, 2005). An individual's iron status falls on a continuum, ranging from replete iron stores, through to depleted iron stores, iron deficiency and iron deficiency anemia (Gibson, 2005). Individuals with iron deficiency are, therefore, at increased risk of developing iron deficiency anemia. As reported, analyzing data from 1993 to 2005, it was estimated that about 1.6 billion people (a

quarter of the world's population) suffered from anemia, with the highest prevalence in pre-school children and women of reproductive age in Africa and south East Asia. Data from 1990 to 2011 still show that pre-school children and women of reproductive age have the highest burden of anemia. Dietary iron deficiency, inherited blood disorders (sickle cell anemia), malaria, hookworm infestation, and schistosomiasis are the most frequent causes of anemia (Petry *et al.*, 2016).

Despite advances in healthcare, iron deficiency remains a major public health concern in both industrialized and non-industrialized (developing) countries, with young women being particularly vulnerable (Beck *et al.*, 2014).

Pregnant women are especially vulnerable to iron deficiency, not only because of the large quantities of iron required for fetal and placental growth (825 mg for fetus, placenta and increased blood volume (Bothwell, 2000)), but also due to the fact that so many enter pregnancy without adequate iron stores, especially in developing countries (Milman, 2011; Burke *et al.*, 2014). Iron status can also be affected by the intake of other nutrients that may inhibit (e.g., calcium, phytates) or promote (e.g., vitamin C) iron absorption (Collings *et al.*, 2013). Other proximal risk factors for low iron status during pregnancy include low intake of bioavailable iron, infections (e.g., intestinal helminthic infections, malaria), multiple pregnancies and adolescent pregnancy, while intermediary factors include low socioeconomic status and membership in certain ethnic groups, depending on the country of residence (Milman, 2011; Burke *et al.*, 2014). Obesity has also been associated with iron deficiency in adult populations, with dietary deficiency, elevated blood volume and subclinical inflammation as the suggested mechanisms (Nikonorov *et al.*, 2014; Burke *et al.*, 2014). The prevalence of iron deficiency and iron deficiency anemia is higher in developing countries than in industrialized countries, due to factors, such as infections (hookworm and malaria), causing blood loss and diets with very low iron content and low bioavailability (WHO, 2001; Beck *et al.*, 2014). It has been suggested that, the most successful dietary approaches for treating iron deficiency appear to be those that use multiple approaches to increase iron status. For example, increased intake of iron (particularly heme iron) and enhancers of iron absorption; decreased intake of iron absorption inhibitors, as well

as optimal timing of enhancers and inhibitors of iron absorption (Beck *et al.*, 2014). Maternal iron deficiency has also been implicated as a risk factor for preterm delivery, small-for-gestational-age and neonatal mortality. Among lactating women, iron deficiency has the same effects as on non-pregnant, non-lactating women of reproductive age: increased risk of iron deficiency anemia, reduced work and mental capacity, increased risk of postpartum depression and other emotional disorders, as well as reduced quality of mother-child interactions (Milman , 2011; Burke *et al.*, 2014).

Calcium-Iron Interaction

Calcium has been reported to be found to have a negative effect on iron absorption in humans (Lönnerdal, 2010). Over a range of “physiological” calcium intakes, iron absorption was inversely correlated to the calcium content of the meal (Hallberg *et al.*, 1991). Similar effects were observed for calcium salts (used for supplements) and milk/dairy products. An apparent solution to this nutritional problem would be to limit the calcium content of iron-containing meals so that iron absorption is optimized. It is well known that the same population groups that are vulnerable to iron deficiency; i. e., infants, children, and women of childbearing age, also have high requirements for calcium (Lönnerdal, 2010). Restricting the calcium content of one or, two of the major meals of the day creates considerable difficulties to achieve the recommended daily intakes of calcium. According to Lönnerdal, 2010, most studies on the effects of calcium on iron absorption show that an inhibition does occur however, the same basic methodology used by all these studies i.e., single meals, dual radioisotope labeling, and red blood cell iron incorporation, has some aspects that have been questioned and the possibility that it exaggerates the effect of dietary factors on iron absorption has been raised (Lönnerdal, 2010). Also, calcium given as carbonate or phosphate showed some inhibitory effect on iron absorption when added to meals with enhancers of iron absorption present (e. g., ascorbic acid) and that calcium citrate had no effect, while all three forms of calcium had a highly negative effect on iron absorption when they were added to a meal with inhibitors of iron absorption (e. g., phytate) present (Cook *et al.*, 1991). However, according to Lönnerdal in 2010, studies on humans in which Ca intake was substantially increased for long periods showed no changes in hematological

measures or indicators of iron status. Thus, the inhibitory effect may be of short duration and there also may be compensatory mechanisms. The interaction between Ca and Fe may be a luminal event, affecting Fe uptake through DMT1 (divalent metal transporter 1) at the apical membrane. However, it is also possible that inhibition occurs during Fe transfer into circulation (Lonnerdal, 2010).

I.2.3. Mineral deficiencies coupled to obesity and consequently cardiometabolic risk factors: Phenomenon of the Double Burden of Malnutrition (DBM)

Obesity is a paradoxical state of malnutrition that consists of excessive caloric intake and micronutrient deficiencies. This state can be partially explained by poor dietary choices and poor access to food rich in nutrients (Bhatti *et al.*, 2015). Also, micronutrient deficiencies have also been linked to a higher risk of overweight and obesity and other debilitating diseases, which can also have long-term consequences that include the risk of hypertension. Thus, micronutrient deficiency coupled with obesity may increase the cardiometabolic risk (Khadilkar *et al.*, 2012).

There is growing evidence that vitamin and mineral deficiencies are prevalent among overweight and obese individuals across a variety of populations in both developing and industrializing countries. Also, some studies have suggested the rates of obesity are increasing more rapidly in some regions of the world where micronutrient deficiencies are more prevalent (García *et al.*, 2009; Hwalla *et al.*, 2017). This pattern could suggest that the micronutrient deficiencies of individuals in these communities may be contributing to the increase in obesity rates (García *et al.*, 2009). This leads to the phenomenon of the double burden of malnutrition. This phenomenon is emerging within countries, communities, households and even at the individual level (Provo, 2013).

The double burden of malnutrition is defined by the World Health Organization (WHO) as the coexistence of undernutrition along with overweight/obesity or other nutrition-related noncommunicable diseases (NCDs). ‘Undernutrition’ encompasses stunting, wasting or thinness, as well as specific micronutrient deficiencies. ‘Overnutrition’ refers primarily to overweight or obesity. Overnutrition-related conditions or cardiometabolic

risk factors other than obesity include high blood pressure, hyperglycemia and diabetes and at-risk blood lipid profile. These conditions cluster as the metabolic syndrome (Delisle, 2018).

The dichotomy of undernutrition- and overnutrition-related conditions is no longer relevant. Nutrition-related NCDs such as diabetes and cardiovascular disease (CVD) are rapidly rising everywhere while undernutrition is still highly prevalent, particularly in low-income countries or regions. Urbanization as a major driver of the nutrition transition is occurring most rapidly in low-income countries, and this very rapid nutrition transition is largely responsible for the double burden now observed (Jones *et al.*, 2016b; Delisle, 2018). According to Delisle, the double burden may exist at the individual level. Various phenotypes of this double burden of malnutrition may be observed at the individual level, including the co-occurrence of obesity with stunting or micronutrient deficiencies and the combination of undernutrition (stunting, underweight or micronutrient deficiencies) with markers of CVD risk other than obesity. Also, according to this same author, the double nutritional burden may also take other forms in which undernutrition combines with nutrition-related cardiometabolic disease or risk markers, but in the absence of obesity (Delisle, 2018).

Evidence suggests that the nutrition transition in developing countries is occurring rapidly, which contributes to the double burden of malnutrition, i.e. the overlap of cardiometabolic risk factors and malnutrition within the same population group, the same household or the same individual (Zeba *et al.*, 2012a). This nutrition transition characterized by, the high consumption of western food such as fast foods, soft drinks and sweets, and less intake fruits and leafy vegetables in individuals (Khadilkar *et al.*, 2012) coupled to physical inactivity are associated with obesity and may also lead to micronutrient deficiencies. It has been reported that, despite an excess of dietary calorie intake, obese individuals have relatively high rates of micronutrient deficiencies (Via, 2012). As reported, Individuals who are obese or have other risk factors of cardiometabolic disease may be simultaneously undernourished, experiencing micronutrient deficiencies and associated disorders (e.g., anemia) despite consuming sufficient, or excess, dietary energy (Jones *et al.*, 2016a). Deficiencies of micronutrients

such as iron, calcium and magnesium have been shown to have potential negative effects on the causality of obesity and co-morbidities.

I.2.3.1. Magnesium deficiency and cardio metabolic risk factors

Magnesium is an essential electrolyte for living organisms and is the fourth most abundant mineral in the human body. Humans need to consume magnesium regularly to prevent magnesium deficiency. Based on magnesium's many functions within the human body, it plays an important role in prevention and treatment of many diseases. Low levels of magnesium have been associated with a number of chronic and inflammatory diseases, such as Alzheimer's disease, asthma, attention deficit hyperactivity disorder (ADHD), insulin resistance, type-2 diabetes mellitus, hypertension, cardiovascular disease (e.g., stroke), migraine headaches, and osteoporosis (Song *et al.*, 2005). Also, it has been pointed out that low Mg contributes to vascular calcification, accumulation of connective tissue in the vessel wall, altered lipid exchange between the vessel walls and blood, increased triglycerides, accumulation of oxalate in vessel walls, and reduced cholesterol transport by HDL (Schwalfenberg & Genui, 2017).

Magnesium is primarily found within the cell where it acts as a counter ion for the energy-rich ATP and nuclear acids. Magnesium is a cofactor in more than 300 enzyme systems that regulate diverse biochemical reactions in the body, including protein synthesis, muscle and nerve transmission, neuromuscular conduction, signal transduction, blood glucose control, and blood pressure regulation. Some magnesium dependent enzymes are Na⁺/K⁺-ATPase, hexokinase, creatine kinase, protein kinase, and cyclases (Gröber *et al.*, 2015). Currently, enzymatic databases list over 600 enzymes for which Mg²⁺ serves as cofactor and an additional 200 in which Mg²⁺ may act as activator (De Baaij *et al.*, 2015; Kostov, 2019). Magnesium is also necessary for structural function of proteins, nucleic acids or mitochondria. It is required for DNA and RNA synthesis, and for both aerobic and anaerobic energy production—oxidative phosphorylation and glycolysis—either indirectly as a part of magnesium-ATP complex, or directly as an enzyme activator. Magnesium also plays a key role in the active transport of calcium and potassium ions across cell membranes, a process that is

important for nerve impulse conduction, muscle contraction, vasomotor tone and normal heart rhythm (Gröber *et al.*, 2015).

Conditions that may lead to hypomagnesemia include alcoholism, poorly-controlled diabetes, malabsorption, endocrine causes (e.g., aldosteronism, hyperparathyroidism, hyperthyroidism), renal disease (e.g., chronic renal failure, dialysis) and medication use (Gröber *et al.*, 2015), chronic low dietary intake of magnesium may be the main reason for a total body Mg deficit (Schwalfenberg & Genui, 2017). In westernized populations, food refining leads to a reduction of the micronutrient density and thereby induces a marginal magnesium intake, resulting in a higher prevalence of Mg deficiency. In addition, a diet rich in animal foods and poor in vegetable foods induces acidosis and increased Magnesium urinary excretion (Rayssiguier *et al.*, 2010)

I.2.3.1.1. Implication of magnesium deficiency in metabolic syndrome

Cross-sectional evidence has shown that magnesium intake correlates significantly with features of the metabolic syndrome (or insulin resistance syndrome), including adiposity, hyperinsulinemia, insulin resistance, hypertriglyceridemia, and low HDL cholesterol and hypertension (Song *et al.*, 2005). Also, diabetes mellitus, both type-1 and type-2, are among the most common causes of magnesium deficiency (Palmer & Clegg, 2015; Ramadass *et al.*, 2015). The incidence of hypomagnesemia in patients with type 2 diabetes ranges widely, from 13.5%–47.7% (Palmer and Clegg, 2015; Gröber *et al.*, 2015). It has been pointed out that, the results of several clinical studies have shown that increased synthesis and release of proinflammatory cytokines may be the link between obesity and MetS. On the other hand, hypomagnesemia triggers low-grade chronic inflammation and Mg^{2+} deficit may be associated with the development of MetS. Researchers have found a link between Mg^{2+} levels, inflammation, and oxidative stress, as risk factors for the development of MetS. These findings support the hypothesis that MgD can play an important role in the pathophysiology of MetS and the actuation of the inflammatory reaction caused by the shortage of Mg^{2+} could be the link between MgD and MetS (Kostov, 2019).

I.2.3.1.2. Implication of magnesium deficiency in the onset of type 2 diabetes mellitus and Insulin Secretion

Magnesium deficiency involvement in the onset of type 2 diabetes mellitus is linked to the crucial role played by magnesium in glucose and insulin metabolism, mainly through its impact on tyrosine kinase activity of the insulin receptor, by transferring the phosphate from ATP to protein. Magnesium may also affect phosphorylase b kinase activity by releasing glucose-1-phosphate from glycogen. In addition, magnesium may directly affect glucose transporter protein activity 4 (GLUT4), and help to regulate glucose translocation into the cell (Song *et al.*, 2005; Ramadass *et al.*, 2015).

Although the exact mechanisms are not well understood, several mechanisms have been proposed concerning the involvement of magnesium deficiency in the development of type 2 diabetes and other metabolic effects (Song *et al.*, 2013).

Another mechanism of the involvement of MgD in the onset of T2DM is through the regulatory role played by Mg²⁺ in the insulin secretion from pancreatic beta cells. Secretion of insulin in response to increases in blood glucose levels occurs in two phases (Straub *et al.*, 2002). The first phase is rapid and occurs within the few minutes. This phase is followed by a second phase of insulin release, which is more prolonged. Observations show that the first phase of insulin secretion is lost in patients with T2DM (Nepton, 2013). In MgD, intracellular levels of ATP and MgATP decrease, which inhibits the closure and opening of KATP channels. This disturbs the coupling between the chemical signal (blood glucose) and the electrical stimulation of the beta-cells, resulting in a disturbance of the normal phases of insulin release. MgD may impair the functioning of the glucokinase, glucose-6-phosphate formation, and the accumulation of ATP in beta cells, which may impair closure of the KATP channels. This delays early and late plasma insulin responses to glucose. At physiological extracellular concentrations of Mg²⁺, intracellular levels of MgATP are sufficient for the normal opening of the KATP channels. In MgD, intracellular levels of MgATP decrease, which inhibits the opening of KATP channels. This induces a longer depolarization of the beta-cell plasma membrane followed by the release of more insulin. According to the author

of this statement, MgD can disrupt the normal functioning of beta cells and, thus, may trigger beta-cell dysfunction in T2DM (Kostov, 2019)

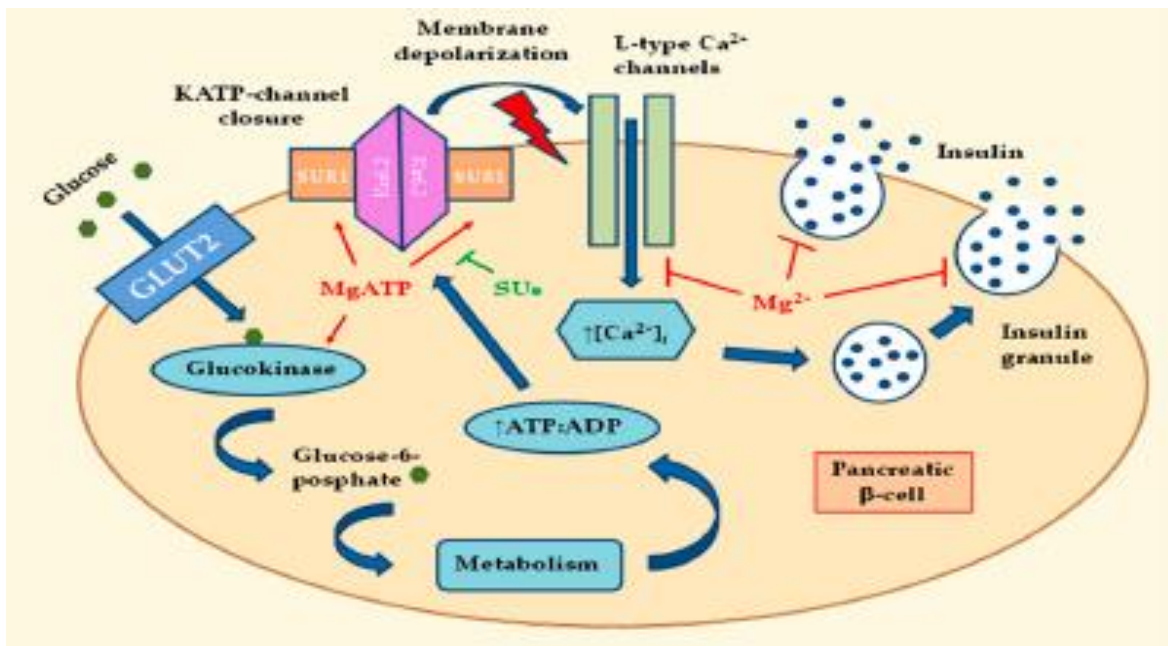


Figure 6 : Regulatory role of Mg²⁺ in insulin secretion from pancreatic beta cells.

The normal intracellular Mg²⁺ concentrations are of utmost importance for the optimal insulin secretion. The first step of beta-cell glucose metabolism is the conversion of glucose to glucose-6-phosphate by glucokinase, which subsequently results in a rise in intracellular ATP. MgD can directly influence the rate of GK activity because the enzyme's action depends on MgATP. Closure of the KATP channel depends on the binding of ATP to the Kir6.2 subunits. Opening of the KATP channel depends on the binding of MgATP to the SUR1 subunits. An important consequence of the closure of KATP channels is the depolarization of the beta-cell membrane, which stimulates Ca²⁺ influx through L-type Ca²⁺ channels and insulin release. In MgD, intracellular levels of ATP and MgATP decrease. This disturbs the coupling between the chemical signal (blood glucose) and the electrical stimulation of the beta cells, resulting in a disturbance of the normal phases of insulin release. SUs antagonize the binding of MgATP to the SUR1, which induces channel closure and potentiates insulin secretion. (Kostov, 2019)

Legend: GLUT2, glucose transporter type 2; KATP, ATP-sensitive K⁺ channel; SUR1, sulfonylurea receptor 1 subunit of KATP; Kir6.2, inwardly rectifying K⁺ channel subunit of KATP; SUs, sulfonylurea drugs; Mg²⁺, magnesium; MgATP, Mg²⁺-ATP complex; [Ca²⁺]_i, increased intracellular Ca²⁺ concentrations; ATP:ADP, increased ATP/ADP ratio.

1.2.3.1.3. Implication of magnesium deficiency in the onset of Hypertension

It has been reported by Gröber and collaborators that, substantial body of epidemiological and experimental researches are linking magnesium deficiency and cardiovascular diseases such as hypertension and atherosclerosis (Gröber *et al.*, 2015). Magnesium is involved in blood pressure regulation. Every modification of the endogenous magnesium status leads to changes in vascular tonus and, as a consequence, changes in arterial blood pressure (Kisters *et al.*, 2005). Magnesium deficiency increases angiotensin II-mediated aldosterone synthesis and the production of thromboxane and

vasoconstrictor prostaglandins (Barragán-Rodríguez *et al.*, 2008; Gröber *et al.*, 2015). Furthermore, alterations in the metabolism of calcium and magnesium have been implicated in the pathogenesis of primary hypertension (Gröber *et al.*, 2015).

I.2.3.1.4. Implication of magnesium deficiency in the onset of Dyslipidemia

It has been brought out that, an important characteristic of hyperlipidemia associated with magnesium deficiency is the accumulation of triglyceride rich lipoproteins and a decrease in the concentration of HDL (Sajjan and Shamsuddin, 2015). The plausible mechanism of the involvement of magnesium in lipid metabolism is linked to the fact that magnesium is an important co-factor for many rate limiting enzymes critical for lipid metabolism (Zhang *et al.*, 2014). It has been suggested that low magnesium may impair 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase inactivation via phosphorylation (Rosanoff and Seelig 2004). Whereas, sufficient levels of magnesium may decrease the activity of lecithin: cholesterol acyl transferase and HMG-CoA reductase, and increase lipoprotein lipase activity which will indirectly decrease the lipid levels (Zhang *et al.*, 2014). The HMG-CoA reductase is the rate limiting enzyme in cholesterol biosynthesis. Lipoprotein lipase is responsible for the conversion of triglycerides to HDL-C and thus leads to a decrease in hepatic synthesis and secretion of VLDL triglycerides (Duffoo, 2016). It has been pointed out that, epidemiologic evidence for the role of magnesium in improving blood lipid profiles remains controversial (Zhang *et al.*, 2014).

I.2.3.1.4. Implication of magnesium deficiency in the onset of Osteoporosis

In humans Mg deficiency contributes to osteoporosis. Low serum Mg is a co-contributing factor to osteopenia in adults with sickle cell anemia (Castiglioni *et al.*, 2013; Elshal *et la.*, 2012). Moreover, an association between serum Mg and bone density has been reported in pre and post menopausal women (Saito *et al.*, 2004; Song *et al.*, 2007). Mg intake was positively associated with bone mass density in surviving members of the Framingham study. It has been concluded from various results of various studies that, Mg supplementation is beneficial in osteoporotic women (Castiglioni *et al.*, 2013).

It has been found that, the mechanisms explaining the effects of Mg deficiency on the bone in humans are similar to what has been described in experimental models: (i) low Mg can directly affect the bone by altering the structure of apatite crystals and by acting on bone cells. Indeed, osteoporotic women with demonstrated Mg deficiency have larger and better organized crystals in trabecular bone than controls, while in women undergoing hormone replacement therapy bone Mg is increased and associates with low cristallinity index (Castiglioni *et al.*, 2013). (ii) Mg deficiency associates with the reduction of the levels of PTH, the induction of end-organ resistance to PTH and the decrease of vitamin D (Rude *et al.*, 2009; Castiglioni *et al.*, 2013). Interestingly, many osteoporotic post-menopausal women who are vitamin D deficient and have low PTH levels are also Mg deficient and Mg supplementation corrects these biochemical abnormalities (Sahota *et al.*, 2006; Castiglioni *et al.*, 2013). (iii) Mg deficiency associates with low grade inflammation (Rodriguez-Moran *et al.*, 2011; Song *et al.*, 2007; Castiglioni *et al.*, 2013) and inflammatory cytokines stimulate bone remodelling and osteopenia (Mazur *et al.*, 2007; Castiglioni *et al.*, 2013). (iv) Mg deficiency promotes endothelial dysfunction (Maier, 2012) and it is known that endothelial health is important for bone health (Warburton *et al.*, 2007; Castiglioni *et al.*, 2013).

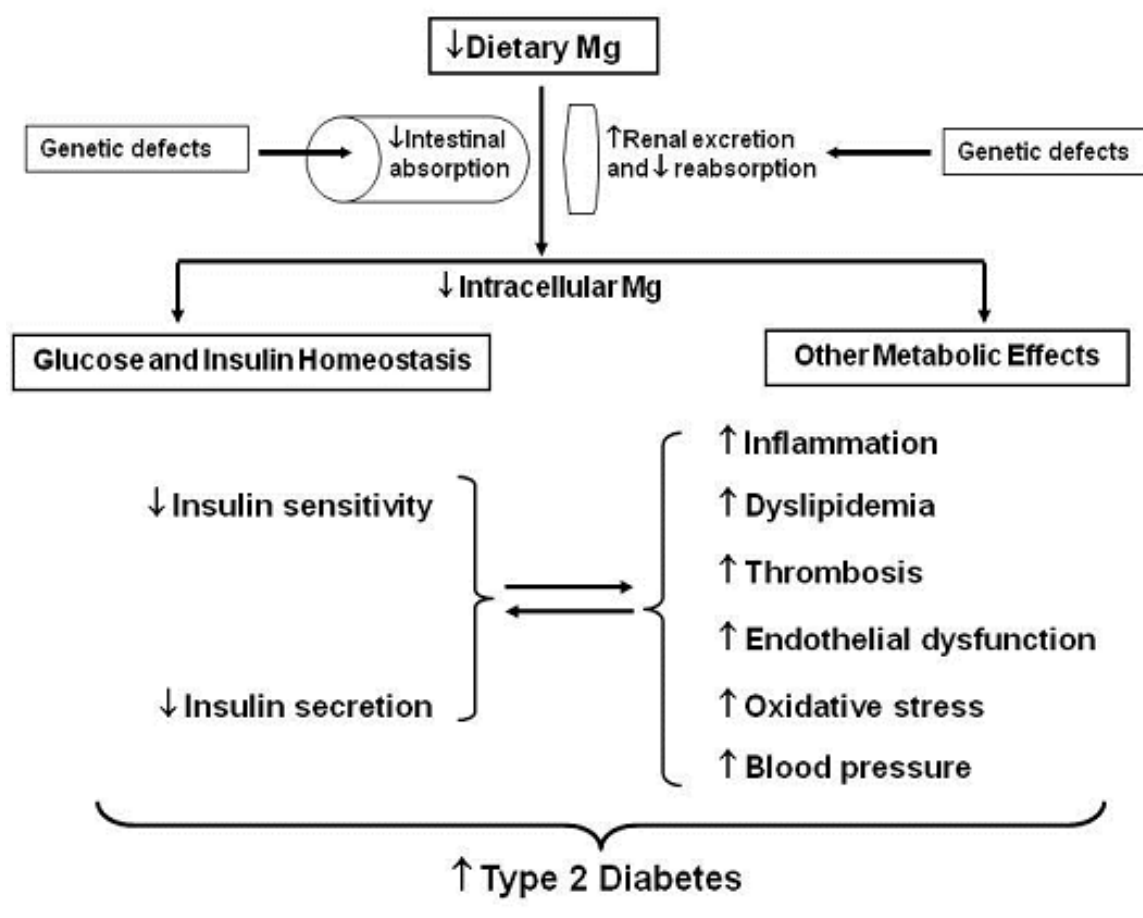


Figure 7 : Potential mechanisms underlying the relation between low magnesium intake, type 2 diabetes and other metabolic effects. Source: Song *et al.*, 2013

I.2.3.2. Calcium deficiency and cardio metabolic risk factors

Calcium is the body’s most abundant divalent cation. More than 99% of the body’s calcium is concentrated in the skeletal system, and approximately 1% is rapidly exchangeable with blood calcium while the remainder is more stable and exchanged only slowly. The small amount of calcium located outside the bone circulates in the serum, partly bound to protein and partly ionized. Ca acts as an intracellular “second messengers” impacting enzyme activity and release of many hormones such as insulin, aldosterone, vasopressin and rennin. Calcium is crucial for the maintenance of cell metabolic rate, nerve transmission, and muscle contraction (Edwards, 2005). In addition, it plays many essential tasks in the synthesis, release, and receptor responsiveness to neurotransmitters (Robinson *et al.*, 2010). Recommended adequate

intake for calcium for both men and women aged 19 to 50 years is 1000mg/day. Calcium is closely tied to magnesium and phosphorus regulation (Metheny, 2012).

Causes of hypocalcemia vary, but are known to include surgical hypoparathyroidism, acute pancreatitis, magnesium imbalances, hyperphosphatemia, alkalosis, malabsorption syndromes, infusion of citrate in blood products and a variety of drugs. However, hypocalcemia rarely results from decreased intake of calcium alone, as bone reabsorption can maintain normal levels for a prolonged period of time (Jones and Bartlett learning, 2010).

I.2.3.2.1. Implication of calcium deficiency in body fat accumulation

With regards to cardiometabolic risk factors, a lot of studies have assessed the association between low calcium intake and adiposity or obesity (Parikh and Yanovski, 2003; Beres *et al.*, 2009; García *et al.*, 2009). It has been reported that population studies have shown that low calcium intake is associated with greater fat mass while increased intake was negatively associated with body fat, BMI, and fat gain in adults. The inverse association between calcium intake and body weight was strongest when the source of calcium was dairy food (García *et al.*, 2009). Metabolically, the probable mechanism explaining the association between calcium intake and obesity is calcium's effect on calcium metabolism in adipocytes and thermogenesis. Low plasma calcium concentrations associated with low calcium intake can lead to a calcitriol (1, 25-dihydroxyvitamin D)-mediated increase in intracellular calcium ion ($[Ca^{2+}]$ ion) concentrations (Harvey-Berino *et al.*, 2005). Increased $[Ca^{2+}]$ ion concentrations, in turn, stimulate the expression and activity of fatty acid synthesis and inhibit lipolysis (Fig. 8) (Garcia *et al.*, 2009). Increased intake of calcium and dairy products inhibits lipogenesis and promotes lipolysis and lipid oxidation, thereby possibly inhibiting induced obesity (Zemel, 2004; Garcia *et al.*, 2009).

Also, in the review of Garcia and collaborators, it has been mentioned that clinical trials have reported that increasing the intake of calcium-rich foods in isocaloric diets reduces adiposity, which suggests that a pre-existing calcium deficiency increases the risk of obesity (García *et al.*, 2009). In a prospective study, it was found that, male and female participants with a higher dietary calcium intake had a “lower increase in waist

circumference” (Fumeron *et al.*, 2011). As possible mechanism, it has been stated that, the calcium levels of different dairy foods, in addition to their fat content, may have an impact on central adiposity (Hess *et al.*, 2016). Dietary calcium may contribute to the precipitation of longchain fatty acids, prevent their absorption in the intestine, and increase their excretion (Vavrusova & Skibsted 2014; Hess *et al.*, 2016). In addition, less fat absorption could contribute to weight control. The precipitation of long-chain fatty acids by calcium suggests a possible mechanism for dairy’s impact on central adiposity (Hess *et al.*, 2016).

I.2.3.2.2. Implication of calcium deficiency in blood pressure

Blood pressure is regulated by intracellular calcium in vascular smooth muscle cells, through vasoconstriction and variations of the vascular volume (Yim *et al.*, 2008; Cormick and Belizán, 2019). Low calcium intake seems to trigger both mechanisms, raising plasma parathyroid hormone (PTH) levels, that increase intracellular calcium directly or through calcitriol activation, and stimulating the renin–angiotensin–aldosterone signalling pathway that produces sodium and water reabsorption, thus increasing the vascular volume (Villa-Etchegoyen *et al.*, 2019; Heaney, 2006; Cormick and Belizán, 2019).

It has been hypothesized that low serum calcium may predispose women to pre-eclampsia for the following reasons: (1) by increasing parathyroid hormone release and thus increasing vascular smooth muscle intracellular calcium, which promotes vasodilatation; (2) by stimulating renin release, which increases angiotensin II levels; (3) by decreasing serum magnesium levels, inducing vasoconstriction in vascular smooth muscle; (4) by diminishing the effect of endothelial nitric oxide synthase, a calcium-dependent enzyme with vasodilatory action; and, finally (5) by reducing circulating prostacyclin, a calcium-dependent enzyme and a potent vasodilator (Richards *et al.*, 2014).

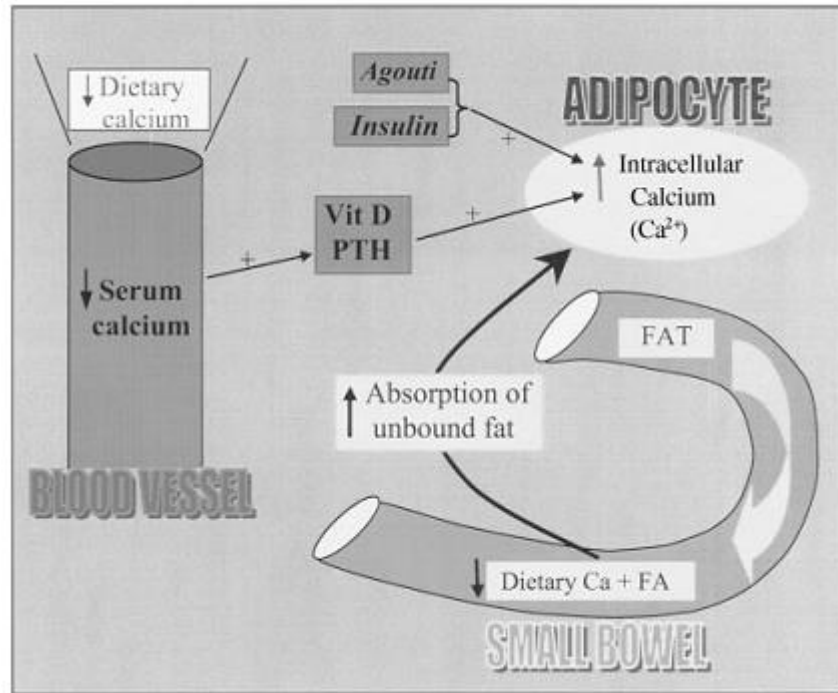


Figure 8 : Proposed mechanisms through which decreased dietary calcium may increase body weight.

Lower dietary calcium intakes (as found epidemiologically for obese subjects) can lead to increased concentrations of 1, 25(OH)₂ and PTH, which in turn may increase adipocyte [Ca²⁺] ions. These elevated intraadipocyte calcium concentrations might then increase the rate of lipogenesis and inhibit lipolysis, consequently leading to increased adiposity (Parikh and Yanovski, 2003).

I.2.3.2.3. Implication of calcium in dyslipidemia

It has been brought out that, a systematic review of calcium supplementation and lipid metabolism reported that calcium supplementation reduced LDL cholesterol and increased high-density lipoproteins (HDL) cholesterol. The authors explained that the possible mechanisms of these effects by the increase in dietary calcium include the suppression of calcitrophic hormones that reduce intracellular calcium in adipocytes, thus stimulating lipogenesis and lipid storage (Chen *et al.*, 2017; Cormick and Belizán, 2019). Also, Cormick and Belizán, stated that, besides, dietary calcium may decrease serum cholesterol by inhibiting cholesterol and saturated fatty acid absorption (Vinarova *et al.*, 2016; Cormick and Belizán, 2019).

I.2.3.2.4. Implication of calcium deficiency in Osteoporosis

Osteoporosis is globally the most common age-related skeletal disease, characterised by a progressive decline in bone mass and disruption of the bone micro-

architecture, resulting in an increased risk of fragility fractures. The IOF estimates that more than 200 million women worldwide suffer from osteoporosis. Numerous risk factors influence osteoporosis development, including oestrogen decline during menopause, immobilisation, old age and nutrition. In respect to nutrition, major osteoporosis risk factors are an insufficient calcium supply. Calcium is the main mineral present in bones, where it provides skeletal strength and serves as a reservoir for maintaining blood calcium levels in a physiological range (Fischer *et al.*, 2018). Calcium deficiency promotes bone loss through increased bone resorption in order to maintain the blood calcium concentration (Peacock, 2010). Calcium deficiency represent the main risk factors influencing osteoporosis development. Changes in serum calcium entail adaptations in bone remodelling, as in the case of increased bone resorption induced by low serum calcium. A low dietary calcium supply, which considerably reduces intestinal calcium absorption and increases PTH concentrations, stimulates bone turnover and excessive bone resorption to restore systemic calcium levels. These mechanisms favour bone loss and osteoporosis development (Fischer *et al.*, 2018).

I.2.3.3. Iron deficiency and cardio metabolic risk factors

Iron levels help to modulate the clinical manifestations of numerous systemic diseases. Iron is involved in binding and transporting oxygen and regulating cell growth and differentiation, as well as electron transport, DNA synthesis, and many important metabolic processes (Fernandez-Real *et al.*, 2015).

Iron is absorbed as Fe^{2+} in the proximal duodenum by the divalent metal transporter 1 (DMT1). It is transferred through the duodenal basolateral membrane facilitated by the iron exporter ferroportin (FPN), then undergoes oxidation by the membrane-bound copper containing ferroxidase hephaestin before being incorporated into transferrin for further transport into circulation. Most cells acquire iron via the uptake of transferrin-bound iron (Fe^{3+}) by the transferrin receptor (TfR1) (Aigner *et al.*, 2014).

25-amino acid peptide hormone hepcidin (hepatic bactericidal protein) maintains systemic iron homeostasis in a hormone-like negative feedback mechanism (Ganz, 2003). Hepcidin is secreted from hepatocytes in response to iron overload, inflammation, hypoxia or anemia. Hepcidin exerts its regulatory functions on iron

homeostasis via binding to FPN, thereby leading to FPN phosphorylation, degradation and consequently to blockage of cellular iron export which induces a decrease in serum iron (Nemeth *et al.*, 2004). Although in quantitative terms the liver is the main source of circulating hepcidin, macrophages, pancreatic islet cells and adipose tissue can also express hepcidin (Kulaksiz *et al.*, 2008; Aigner *et al.*, 2014).

Iron deficiency is considered to be the most common micronutrient deficiency and the major cause of anemia worldwide (Sharif *et al.*, 2011). The development of iron deficiency occurs in stages, beginning with the depletion of iron stores, followed by diminished iron transport, and finally the depletion of iron-containing proteins and enzymes, including hemoglobin, which results in iron deficiency anemia. It has been stated that in developing nations poor iron status occurs typically due to the lack of foods containing bioavailable iron (McClung & Karl, 2009).

It has been reported that a large number of studies have shown that iron metabolism plays a crucial role in the development of cardio metabolic disorders, such as MetS, type 2 diabetes, atherosclerosis and consequent cardiovascular diseases. According to the same author, previous epidemiological studies reported that the level of hepcidin, the key regulator of iron metabolism, was increased in children and adults with obesity and type 2 diabetes (Zhu *et al.*, 2016). A study conducted in adults with MetS demonstrated that higher hepcidin level was linked to the growing number of metabolic risk factors (Martinelli *et al.*, 2012). Zhu and collaborators observed that children with cardiometabolic risk factors had higher hepcidin level and lower levels of serum iron, transferrin and soluble transferrin receptor (sTfR) in comparison to healthy counterparts (Zhu *et al.*, 2016).

With regards to obesity, its relationship with iron deficiency has been investigated in many studies (Garcia-Valdes *et al.*, 2015; Cheng *et al.*, 2013). In adults, several analyses have demonstrated lower serum iron concentrations with higher BMI, particularly in women (Aigner *et al.*, 2014). Iron deficiency in obese individuals may be a result of low iron intake (e.g. due to an unbalanced diet), reduced iron absorption in the small intestine, and greater iron requirements caused by a larger blood volume. In addition, obesity is associated with a chronic low grade inflammation state. For this reason,

sequestration of iron through an inflammatory mediated mechanism can be one of the proposed causes of iron deficiency in obesity (Yanoff *et al.*, 2007; Menzie *et al.*, 2008). The interaction of iron homeostasis with obesity represents a Janus-faced clinical condition. On the one hand, obesity may promote iron deficiency by inhibition of dietary iron uptake from the duodenum. On the other hand, a condition termed “dysmetabolic iron overload syndrome (DIOS)” has become the most frequent differential diagnosis for elevated ferritin concentrations, affecting approximately one-third of subjects with nonalcoholic fatty liver disease (NAFLD) or metabolic syndrome (MetS). DIOS is characterized by increased serum ferritin concentrations with normal or mildly elevated transferrin saturation in subjects with various components of MetS or NAFLD (Aigner *et al.*, 2014).

The hepcidin-inflammation connection provides a succinct biological framework to explain the association of iron deficiency with obesity (Fig. 9). Hepcidin is an important regulator of iron homeostasis, inhibiting iron absorption at the enterocyte and sequestering iron at the macrophage, which could lead to decreased iron stores and hypoferrremia. Chronic inflammation caused by obesity, is associated with the expression and release of pro-inflammatory cytokines, including interleukin-6 (IL-6) and tumor necrosis factor- α (TNF- α). These pro-inflammatory cytokines may result in the release of hepcidin from the liver or adipose tissue. The potential role of hepcidin in the development of iron deficiency in the obese is supported by the discovery of elevated hepcidin levels in tissue from patients with severe obesity, and the positive correlation between adipocyte hepcidin expression and BMI (McClung & Karl, 2009).

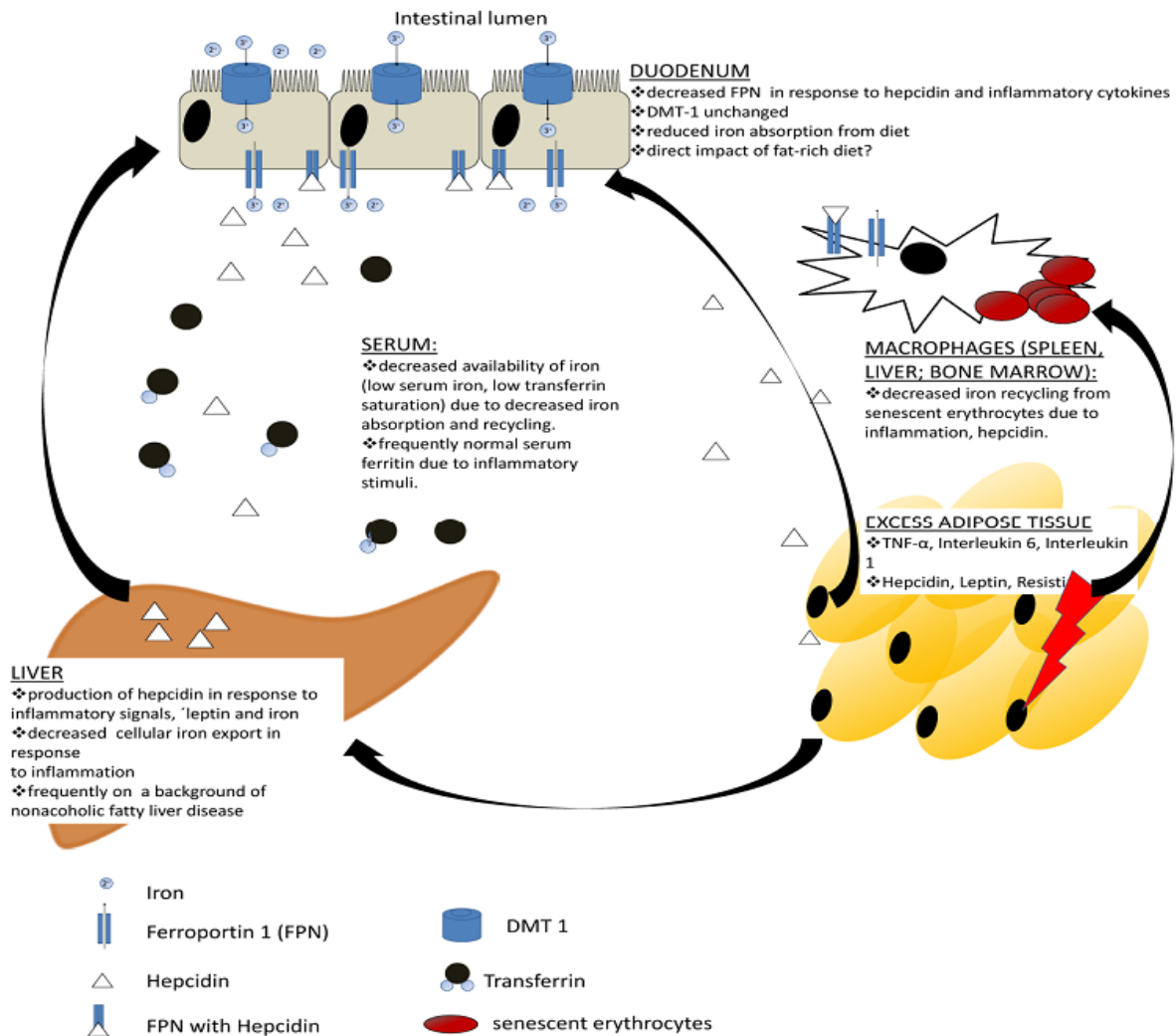


Figure 9 : Current understanding of molecular links between obesity and iron deficiency.

Obese adipose tissue is characterized by an increased production of several pro-inflammatory cytokines and adipokines as opposed to healthy lean adipose tissue. These may directly impact iron absorption from the enterocyte. Additionally, pro-inflammatory cytokines such as interleukin-1 and -6 represent potent inducers of hepcidin production in the liver, which may further impair iron absorption. Both cytokines and hepcidin lead to iron retention in spleen, liver or bone marrow macrophages, thereby lowering serum iron concentrations and iron availability for erythropoiesis. **SOURCE:** Aigner *et al.*, (2014)

Iron has also been observed to impact glucose metabolism. It has been reported that excess iron, once stored in the liver, interferes with glucose metabolism, causing hyperinsulinemia via both decreased insulin extraction and impaired insulin signaling (Fernandez-Real *et al.*, 2015). Hyperinsulinemic status, on the other hand, favors the intrahepatic deposition of iron. Insulin enhances the uptake of extracellular iron, inducing the redistribution of TfRs to the cell surface while downregulating hepcidin expression (Wang *et al.*, 2014; Fernandez-Real *et al.*, 2015).

CHAPTER II: MATERIAL AND METHODS

CHAPTER II: MATERIAL AND METHODS

II.1. Study duration, type and study period

The duration for this study was one year and it was cross sectional in design. During the one year, the study was divided into four phases: phase I or the preparatory phase which went from March 2013 to January 2014, phase II or the data collection phase which ran from January 2014 to August 2015 during the dry and rainy seasons, followed by phase III or the result reporting phase and phase IV or the data entry and processing phase.

II.2. Phases of the study

II.2.1. Phase I: Preparatory phase

This phase consisted mostly in seeking authorization from the local and administrative authorities and preparing the field activities.

II.2.1.1. Meeting the local and administrative authorities

Upon arrival in the different localities, applications containing the project of the study were deposited at the level of the Senior Divisional Officers (SDOs), parish priests and pastors of churches of each locality and the heads of health districts of each locality. Where the opportunity was given, an oral presentation of the project was done. Then, with the help of local guides, local chiefs were met and informed about the study and gave their oral consent when the study objectives were understood.

II.2.1.2. Sensitization of the population and choice of date of the health campaign

After the authorities in charged of the various study sites of this study understood and gave their consent for the study to be carried out, the LNNB fieldwork team with the help of the local authorities, sensibilised the population in churches, health centers, social gatherings like meeting places, maket place, schools, university and all the possible places where women of childbearing places could be found. After this sensibilisation phase, the administrative and local authorities choose the dates that where appropriate for the health campaigns in each study site.

II.2.1.3. Development of questionnaire

A questionnaire (*Appendix I*) was used to collect data and information from participants. The questionnaire consisted of a mixture of closed and open ended questions and it was divided into ten parts: A) Dietary habits; B) Alcoholic drink consumption; C) Tobacco consumption; D) Health history; E) Urbanisation; F) Socio-economic information; G) Information on Food security; H) Perception on physical activities; I) Perception on body aspects; J) Information on physical activity. This questionnaire was mainly developed from the WHO STEPWISE questionnaire for chronic diseases (WHO, 2009). Also, questions from previously validated questionnaires used in other studies (Ntandou, 2009) were included in this questionnaire. Some questions on dietary intakes and food diversity were modified from other studies to suit the sample of this study (Schaefer *et al.*, 2011). Other questions were worked out purposely for this study especially the questions on the semi quantitative food frequency questionnaire.

II.2.1.4. Training of Field workers

This training consisted in the recruitment of field workers in the different localities and the revision of data collection methods on dietary habits, socioeconomic parameters, socio-demographic data, lifestyle characteristics and physical activity level. Field workers were also trained on the standardization of the measurement of anthropometric parameters and the procedure of collection and transportation of blood samples. This was done by the Ph.D. research candidate (selection of research subjects, anthropometric measures and the administration of questionnaires) and by local collaborators. This training was done in many steps. There were working sessions between the researcher and the field workers on the comprehension and technique of administration of the questionnaire. Afterwards, the researcher and the field workers brought modifications to the questionnaire. Two sessions enabled the harmonization of the measurement of weight and height parameters between the Ph.D. candidate and the field workers. Then the whole team came together to plan and coordinate the field activities.

II.2.1.5. Pre-testing questionnaire

The pre-test questionnaire was completed by 20 volunteers chosen at random from the five regions of the study through an interview that allowed feedbacks. The time taken was recorded and comments from respondents led to changes, mainly in terms of the language, and structure of the questionnaire. Although the questionnaire had been validated in previous studies, it had to be tested again, since some changes had been made, to ensure its suitability for the study. Moreover, pre-testing the questionnaire could increase the validity and the reliability of the questionnaire itself (Marshall, 2005; Williams, 2003). The pre-test of the questionnaire enabled to appreciate the acceptability and the comprehension of the questionnaire.

II.2.2. Phase II: Data Collection Phase

This was the data collection phase proper. A questionnaire was conceived from the WHO STEPwise questionnaire (WHO, 2009) for chronic diseases and used as instrument for data collection. Data collection was carried out once for each individual. It consisted in the collection of data on the identity, the dietary habit, marital status, educational level, household size, source of revenue, profession, area or region of residence, health history, socioeconomic and socio-demographic level, food insecurity data and the level of physical activity (LPA) of the individual. The PhD research student, research assistants as well as well-trained health personnel administered the questionnaires to the participants. Data collection was done through a face to face interview; French, English and the local dialect were the languages used. The local dialect was used for individuals who neither spoke English nor French. In this case, the interviewer was a person who could easily speak the dialect and proceeded by translating orally the questions in the dialect for the participant to understand and then recording the answers either in English or French. Data were collected in data collection sites (health districts, health centers, churches, palace place) and this was done during the week, weekends and holidays.

After this, anthropometric parameters were taken by well trained personnel followed by the collection of blood samples by nurses.

Blood samples were collected only from participants who had indicated that they had fasted for 12 hours the previous night. These anthropometric parameters and blood samples were used for the evaluation of nutritional status and the diagnosis of metabolic diseases.

II.2.3. Phase IV: Data Entry and Processing

II.2.3.1. Data entry

Data were entered using excel Microsoft software. Data from each locality were entered each in different excel sheet and at the end, these data were fused to make one entire database.

II.2.3.2. Quality control of data

Quality control of completed questionnaires was undertaken by the Ph.D candidate. The same thing was done for the establishment of absolute frequencies, the median so as to identify aberrant data. Data for 30% of the participants were entered a second time and were compared to the initial data so as to verify whether there were any errors during the entry of data.

The final database was then cleaned and a code was attributed to each data after which the data were transferred to SPSS version 20.0 for statistical analysis.

II.2.4. Phase III: Reporting Results

This phase consisted in reporting the results from the evaluation of the nutritional status and the diagnosis of metabolic diseases. Each participant received her results individually by presenting the code which had been given to her on the data collection day. Using this code, her result sheet was found and given to her. Nutritional advice was then carried out and this was done by writing a serie of nutritional advice below the result section; this by taking into consideration the results of the participant we had in front of us before handling the result sheet to the patient. Serious cases were referred to health centers for treatment.

II.3. Inclusion and Exclusion Criteria

II.3.1. Inclusion criteria

Included in the study were women with the following baseline characteristics:

- aged 14 to 49 years old,
- of good health
- give their informed consent
- permanently live in the region or area under investigation

II.3.2. Exclusion criteria

Excluded from the study were women with the following baseline characteristics:

- pregnant women
- lactating women
- those that took dietary supplements like iron, calcium, magnesium...
- previously diagnosed hypertension or diabetes or those with a documented use of antihypertensive or antidiabetic medications
- physically handicapped and mentally ill

II.4. Study population

The study population was made up of women of childbearing age, aged 14-49 years randomly selected during mass health campaigns against cardiovascular risk factors and nutritional surveys

II.5. Sampling Procedure

A three-stage sampling technique was adopted for this study. In the first stage, regions were selected, in the second stage, cities, towns and villages were selected and in the third stage, women of childbearing age were targeted in various public gatherings in various study sites from which approbations were received. In addition to the region, towns and villages selected for this ongoing Nutrition and Health study, secondary data from other regions, towns and villages selected for a previous health campaign in 2013 on Metabolic Diseases by the Laboratory of Nutrition and Nutritional Biochemistry,

were exploited for this study. In the first stage of the sampling, 3 regions (West, Littoral and Far-North) included in the previous health campaign were chosen. In addition to these, the North West region was selected from the other seven regions of Cameroon making a total of four regions retained. The Northwest region was selected because together with the western region form the breadbasket of Cameroon.

In the second stage which consisted in choosing cities, towns, little towns and villages; the capital city Bafoussam of the western region was chosen and the political capital of the country (Yaounde) was also included. Four towns from the abovementioned regions which were randomly selected during the previous health campaign were also retained for this study and they were: Nkongsamba (littoral region), Maroua (far-north region), Dschang and Foumban (West Region). Three little towns: Mbengwi, Ndu, Wum were randomly selected from the North West region and one other town: Mbouda was selected from the west region. In addition, three villages selected during a previous campaign and proximate to three of the four little towns of the previous campaign were retained (Bafou, Bafolé, and Njimom). Two other villages were randomly selected during this study. They were Nyen which is proximate to Mbengwi in the North West region and Babadjou which is proximate to Mbouda in the west region.

In the third stage, in each area retained or randomly selected, five quarters were randomly selected making a total of 65 quarters. In each of these 65 quarters, announcements and posters stating the purpose, the exact date and time of the study were read and pasted in churches, in general assemblies of various groups, in secondary schools, high schools, universities and all other places where women of childbearing age could be found. At the beginning of data collection on the field, all women who voluntarily accepted to participate in the study were recruited during fieldwork. Later on, all women of childbearing age who met the inclusion criteria and had given their written consent were selected and included in the study population of this study. A written consent was obtained from the parent or guardian of women younger than 21 years. Figure 10 below summarises the sampling procedure.

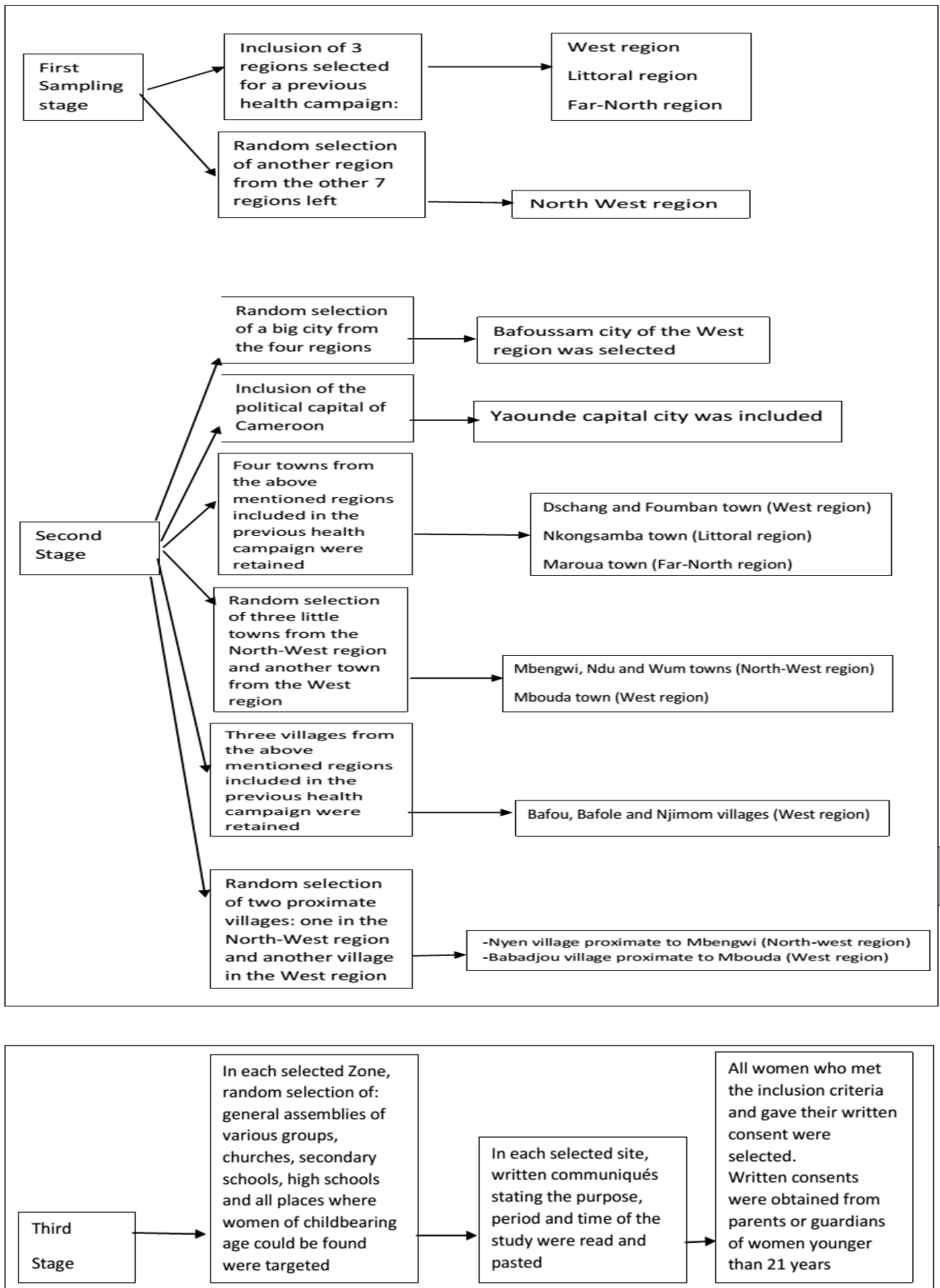


Figure 10: Sampling procedure

II.6. Location of Cameroon

Cameroon is located on the west coast of Central Africa and covers an area of 475,400 square kilometers (183,695 square miles). Its geographic coordinate is latitude 7.3697° N and longitude 12.3547° E. The topography of Cameroon is varied, ranging from tropical rain forests in the south to mountainous highlands in some western central regions, and semi-arid savannah in the far north. The population was estimated at 15,421,937 in July 2000 and is growing at an annual rate of 2.47 percent. Cameroon has ten regions with distinct regional culture, religion as well as ethnic differences. Cameroon has a diverse population comprising approximately 250 ethnic groups that form 5 regional/cultural groups. These are western highlanders (also called grass fielders), which include the Bamileke, Bamoun, and many smaller groups in the northwest; coastal tropical forest people, which include the Bassa, Douala, and many smaller groups in the southwest; southern tropical forest people, which include the Beti, Bulu, Fang, and Pygmies; the northern semi-arid regions and central highlands, which include the Fulani; and the Kirdi people of the northern desert and central highlands. In most areas, women are responsible for feeding their families. They grow staple food crops, while men clear the land and provide meat and oil as they grow the cash crops. Among the pastoral populations, men herd the livestock and women process dairy products (Njukwe *et al.*, 2014).



Map No. 4227 Rev.2 UNITED NATIONS
November 2015

Department of Field Support
Geospatial Information Section (formerly Cartographic Section)

Figure 11 : Location of Cameroon and location of the study areas on the map

-The Highlighted areas are the Regions which were randomly selected for this study except the Centre region which was chosen purposely for the capital city of the country

-The underlined and highlighted areas are the zones which were retained for this study in the various regions.

II.7. Description of study sites and study population of study sites

II.7.1. Description of study sites

1-) Center region (Yaounde city)

Yaounde is the capital of Cameroon and, with a population of approximately 2.5 million, the second largest city in the country after the port city Douala. It lies in the centre of the nation at an elevation of about 750 metres (2,500 feet) above sea level. Most of Yaoundé's economy is centered on the administrative structure of the civil service and the diplomatic services. The economy of Yaoundé is dependent on the administrative structure of the civil service and the diplomatic services. Owing to its status as the capital city of Cameroon, it is characterized by higher standards of living and relatively good security as compared to other parts of the country. The residents of Yaoundé engage in agricultural activities such as poultry and rearing of pigs (Wikipedia, 2018).

Plantain and melon are cultivated in association and in rotation with cassava and groundnut after fallow between 4 and 7 years. The main perennial crops are; cacao, palm oil, rubber and coffee, while the main food crops are cassava, groundnut, melon, plantain, cocoyam and in some extent yam, sweet potato and maize in large-scale. Cassava leave is consumed as vegetable and it constitute an important basis of nutritional balance (vitamins, mineral salts and proteins) in farming environment (Njukwe *et al.*, 2014).

2-) Western region (Bafoussam, Mbouda, Babadjou, Dschang, Foumban, Njimom, Bafou, Bafolé)

The relief constitute essentially of highlands between 1000 and 2300 masl but mountainous massifs and important craters do exist. The climate is of humid tropical type with two big seasons; an active relatively shorter dry season from November to February and the rainy season with high relative humidity. Some farming localities present densities of high population between 450 and 700 habitants/km² and majority of the population (above 80%) practice agriculture (Njukwe *et al.*, 2014). This is the breadbasket of Cameroon. Agriculture is greatly practiced in this region due to the fertile

agricultural lands and favorable climate that prevails. Food exists here in abundance; thus much is exported to other parts of the country. Markets in towns like Bafoussam are often flooded with fruits and vegetables that at times perish without ever getting a ready market. Onions, green beans, pepper, mangoes, avocados, tomatoes just to mention a few are the order of the day at the daily and weekly markets in the towns and villages of this region. Cash crops like coffee, cocoa and green beans are also heavily produced here; serving as a steady source of income to locals. The abundance in agricultural products partly suggests why this is the most densely populated province in the whole republic. It has a population density of about 199 inhabitants per square kilometer. The dominant ethnic groups here are the ‘bamilekes’ (Mphoweh and Futonge, 2009)

Bafoussam is the capital of the West Region of Cameroon, in the Bamboutos Mountains. It is the prefecture of the Mifi department, one of three arrondissements in the department. The city had a population of 239,287 inhabitants (at the 2005 Census). The city is the region's center of trade. People are farming coffee, tobacco and tea. A trading centre of the Bamileke peoples, it lies in a densely populated region where coffee, kola nuts, tobacco, tea, and cinchona (from which quinine is made) are grown and pigs and poultry are raised (Wikipedia, 2018).

Mbouda town is the chief place of the Bamboutos division. It runs on the central and south-west parts of this department and includes 4 divisions. It has a surface area of 437km² and it is bordered north by the Wabane commune (Region of south-west), south by the Bamougoum and Baleng villages, west by the Batcham village and east by the Galim and Babadjou villages (CVUC-UCCC, 2014).

Babadjou is a commune situated about 12km of Mbouda linking Mbouda to Bamenda. This community depends on the Bamboutos department in the region Western region of Cameroon. It is bordered in the north by Santa, in the east by Bamessingué, the west by the northwest region and the sud by the Balatchi group (a commune of Mbouda) (CVUC-UCCC, 2014)

Dschang town is located in the Menoua department in the West region. It spreads on a superficial of 262 Km² with 20 communities in its urban zone and 96 in its rural zone. It is bordered to the north by the Nkong-Zem commune, to the south by the Santchou

village, to the west by the Fongo-Tongo commune and the East by the Fokoué village (CVUC-UCCC, 2014).

Foumban is located in the West region of Cameroon in Noun department, the Foumban division. It is bordered in the East by Malantouen, west by the Koutaba commune, the north by the Njimom village and south by the Massagam village (CVUC-UCCC, 2014).

Bafou is a little municipality situated in the west of Cameroon, in the West region, Menoua division and Nkongni subdivision. Bafou is populated by the Bamileke people, situated at approximately 10 km from Dschang the closest city. The rural economy is sustain by agriculture, except some few government worker civil servant, the large part of the population depends on agriculture (Wikipedia, 2018).

3-) Far North region (Maroua)

This is the topmost extreme of Cameroon. It is in contact with the Lake Chad. Its population is over 2000 000 inhabitants and its headquarters is Maroua. This is the most populated region in Cameroon. It is largely occupied by the Bororo people also referred to as the Peuls. The dominant ethnic groups here are the ‘haoussas’. This area also has a vast expanse of rice fields in its plains. Other food crops are equally cultivated here, such as maize, millet and sorghum. Pastoral activities are highly practiced in this area, with the rearing of cattle, sheep and goats. The harsh nature of the weather during the dry season renders this activity very difficult. This has led to land degradation in several parts of this region (Mphoweh and Futonge, 2009).

4-) Littoral region (Nkongsamba, Ekangte)

The land consists of volcanic soils from Mount Cameroon and sediments of rock origin along the coast of the Atlantic Ocean. The climate is characterized by two seasons; a long season of rain from mid-March to mid-November and a dry season from mid-November to mid-March. The majority of the population lives in farming environment with the fundamental potential. Farmers, state cooperation and multinational companies are involved in the cultivation of coffee, cocoa, tea, palm oil, banana, rubber, for export and cassava, plantain, cocoyam and vegetables are cultivated for subsistence. Livestock

raising is of traditional type and concentrate on small ruminants and poultry (Njukwe *et al.*, 2014).

Nkongsamba is a city located in the Mounjo department, which is in the Littoral region. The city had a population of 104,050 inhabitants at the 2005 Census. The city is a centre of farming of oil palms, bananas and coffee, and is situated between two mountains, the Massif du Manengouba (2,396m) and Mount Nlonako. Mbo (Manenguba) is one of the languages used locally: in the district around, such languages as Kaa and Baneka are used, both reportedly somewhat like Mbo Wikipedia, 2018).

Ekangté is a nearby village of Nkongsamba in the Littoral region. Agriculture is the main activity in this locality.

5-) North-west region (Wum, Mbengwi, Ndu, Nyen)

The capital of this English speaking region is Bamenda. This is another region with high relief and mountainous landscape. It is also a region of lowland savannah, making it to be generally referred to as the grass fields. It also has an extensive flood plain at Ndop. This region has fertile soils and a favorable climate that account for massive production in the agricultural sector. Besides the west region, this is another breadbasket of the republic. Enormous quantities of food crops like maize, rice, beans, groundnut, vegetables, potatoes, cassava etc are produced here. Agro pastoral activities are also an important mainstay; mostly practiced by the ‘bororos’ on the hill slopes in places like Ndop, Sabga, Kumbo, Babanki and Kom. Over the years this has been a cause of conflict between farmers and grazers (Mphoweh and Futonge, 2009). Just like the west region, majority of the population (above 80%) practice agriculture and is also the main zone for the production of potato (Njukwe *et al.*, 2014).

Wum is a town and commune in Cameroon. It is the capital of Menchum division in the Northwest Province. Wum is the third biggest town in the North West Region of Cameroon. It lies on a plateau at an elevation of about 1100 m near the edge of the western highlands of Cameroon. It is 80 kilometres (50 mi) north of the regional capital Bamenda (by dirt road). It lies near Lake Wum, one of five small crater lakes within 15 km of the town in the hilly, volcanic landscape. Lake Nyos is 25 Km east to

it. In 2012 Wum's estimated population was 80,123 inhabitants (Wikipedia, 2018). It is bordered east by Fundong, North by Fungom subdivision, south by Bafut and west by the Mechum valley subdivision. Economic activities here involve agriculture involving the cultivation of crops like Maize, Cassava, Groundnuts, Beans, Yam, Potatoes, Sweet potatoes, Cabbage, Carrot, Okra (CVUC-UCCC, 2014)

Mbengwi municipality is the Divisional capital of Momo Division in the North West Region of Cameroon. The Municipality derives its name from its chief town Mbengwi. The Mbengwi is a Council area. This Council area is situated some 20 km to the west of Bamenda town and at an altitude ranging from 900m to 2000m above sea level. It is located on the western slopes of the Adamaoua between longitude 100 00' and 100 02' East, and between latitude 60 00' and 60 05' North. This Municipality lies in the transitional zone between the forest and grass land regions of western Cameroon. Mbengwi council has a surface area of 147,000 square kilometers and 22 municipal councilors. The main language spoken within the Mbengwi Council area is Meta. This is due to the fact that all the villages of the municipality have one common ancestry (CVUC-UCCC, 2014).

Ndu is a town and commune in Donga-Mantung Department of the Cameroonian region of Northwest Region. It lies at the northeast edge of the Bamenda Grassfields, on the eastern arc of the Ring Road. It is the highest elevation town in Cameroon. About 85,000 people live in Ndu commune. Most are Wimbum - the three clans which speak the Limbum language. Ndu commune contains the southeast part of Wimbum-land, including the villages of Talla, Ngarum, Taku, Ntundip, Luh, Ndu-town, Mbipgo, Njimkang, Njilah, Wowo, Sehn, Ntumbaw, Njirong, Ngulu, Nseh Macop, Sinna and Sop. Most Wimbum are farmers, raising maize, beans, Irish potatoes, yams, njama-njama, tomatoes, coffee, plantains, and rice. Most soils are rich and the rain is generally sufficient for good crops. At the south end of Ndu commune is the Ndu Tea Estate, the largest tea plantation in Cameroon. Some people raise cattle, horses, goats, sheep, and fowls. Ndu town is the administrative headquarters of the commune. The town includes a large market, hotels, schools, a hospital, a gendarmerie, and the Cameroon Baptist Theological Seminary (Wikipedia, 2018).

II.7.2. Areas and localities constituting the study sites

The following areas and localities made up our study sites:

- 1.) **Center region:** Djoungolo (Yaounde I), Melen (Yaounde III); Mimboman (Yaounde IV); Simbock, Biyem-Assi (Yaounde V)
- 2.) **Western region:**
 - a.) Bafoussam: Kamkop I (Bafoussam III)
 - b.) Mbouda: Saint-Anne Catholic Mission and Evangelic church of Cameroon, Mbouda Centre Parish.
 - c.) Babadjou: District hospital Babadjou
 - d.) Dschang: Foréké (Saint Paul's Apostle Parish)
 - e.) Foumban: Manka, Kuéka, Njiyouom
 - f.) Bafou and Bafole: Kekang II
 - g.) Njimom: Integrated Health Centre of Bafole
- 3.) **Far-North region:**
 - Maroua: a.) Kakataré (Maroua II) and
b.) Hardé (Pont-vert) (Maroua I)
- 4.) **Littoral region:**
 - a.) Nkongsamba: Ekangté Mbeng (Carrefour Lele)
 - b.) Ekangté: Ekangté Nko
- 5.) **North west region:**
 - a.) Wum: Wum District Hospital
 - b.) Mbengwi: Mbengwi District Hospital
 - c.) Ndu : Presbyterian Church Ndu
 - d.) Nyen : Chief Palace Nyen (Tab)

II.8. Calculation of sample size

The sample size was calculated using the formulas below (Magnani, 1997; UNICEF, 1995; FAO, 1990)

Step 1: Basic sample size calculation for random sampling

$$n = \frac{t^2 \times p(1-p)}{m^2}$$

Where:

n = required sample size

t = confidence level at 95% (standard value of 1.96)

p = estimated prevalence of malnutrition in the project area

m = margin of error at 5% (standard value of 0.05)

It has been estimated that 82% of women of childbearing age in Cameroon have low adjusted plasma zinc concentration (Engle-Stone, 2014).

$$n = 1.96^2 \times 0.82(1-0.82) / 0.05^2 = 226.81 = 227$$

Step 2: correcting the sample size for cluster sampling method

Since the survey was based on a cluster random sampling, the size of 227 is multiplied by a factor (D). This factor compensates for the effect of the sampling method. This factor is generally equals 2 in nutritional studies using a cluster sampling.

$$\text{Thus: } n \times D = 227 \times 2 = 454$$

Step 3: Sample size correction for unforeseen circumstances

5% of this calculated sample size has to be added, as unforeseen errors related to non-responses or registration errors should be taken into account.

$$n + 5\% = 454 \times 1.05 = 476.7 = 477$$

In total 480 women were to be recruited from the four regions and the capital city, but 608 women were recruited and included in the study to allow for precision.

II.9. Data on Nutritional Parameters

II.9.1. Dietary habits

a.) Food frequency for each food group

Dietary habits were assessed by collecting information on the frequency of consumption of various food groups using a food frequency questionnaire (FFQ). An inventory of all food items consumed through out the year in the various study areas was made in order to elaborate the FFQ. The FFQ therefore, included various food items (traditional diets; cereals imported and locally made, tubers, meat, fish, eggs, poultry; dairy products, fruits, vegetables; fats and oils; snacks, beverages; alcoholic drinks; sweet drinks; tea...) (Sharma, 2010). The questionnaire was designed to capture all foods/beverages typically consumed, including traditional foods not available in stores and those available seasonally in all the study areas. Data were collected on the number of days in the last 7 days a woman ate specific food items according to the following procedure:

1. The interviewed was asked about frequency of consumption (number of days in the last 7 days) of a food item over a recall period of the past 7 days.
2. The various food items were then grouped into eight standard food groups ('cereals and tubers group'; 'pulses (nuts and beans) group'; 'milk and dairy products group'; 'meat, fish and egg group'; 'vegetable group'; 'fruit group'; 'oil group'; 'sugar and derivative group') (WFP, 2008).
3. These informations were used to calculate the food frequency for each of the eight food groups consumed in the last seven days for each woman and for the study population. All the consumption frequencies of food items of the same group were summed, and the value of each group above 7 was recoded as 7.
4. Data on the frequency of consumption in a week of each food group were used to categorise each of the eight food group intake into three; *Low*: for food group intake 1-2 times per week; *Moderate*: for food group intake 3-4 times/week and *High*: for food group intake 5-7 times/week.

b.) Frequency of traditional diets in a week

Data on the frequency of intake of traditional diets in a week were also used to categorise traditional diets intake into three: *Low intake*: for 0-1 time per week; *moderate intake*: for intake 2-4 times per week and *high intake*: for 5-7 times per week.

c.) Food consumption score

With the information on the frequencies of intake per week of the eight food groups, the food consumption scores (FCS) were calculated based on method established by the World Food Programme (WFP) (WFP, 2008) as follows:

1. The value obtained for each food group was multiplied by its weight (weights are presented in the table below and they are based on nutrient density of the food groups) thus creating weighted food group scores.
2. The weighed food group scores were then summed, thus creating the Food Consumption Score (FCS).
3. Using the appropriate thresholds (see Table 5 below), the variable food consumption score were recoded from a continuous variable to a categorical variable for the Food Consumption Groups (FCGs) or profiles (WFP, 2008).

Table 4 : Weight for each food group to calculate the Food Consumption Score

Food items	Food groups	Weight
Corn, rice, millet, bread, plantains, cocoyams, cassava, sweet potatoes, irish potatoes...	Cereals and Tubers	2
Beans, groundnuts, nuts...	Pulses	3
Green leafy vegetables, green spices, other vegetables...	Vegetables	1
All fruits	Fruits	1
Cow meat, chicken, pork, eggs, fish, goat meat...	Meat and Fish	4
Milk and milk products	Milk	4
Biscuits, sweets, sweet drinks, sucres and other scury products	Sugar	0.5
Refined oil, red palm oil, butter, margarine...	Oil	0.5

Source: World Food Programme (WFP) (2008)

Table 5 : Thresholds for creating Food Consumption Groups (FCGs)

Food Consumption Score	Profile
0-21	Poor
21.5-35	Borderline
> 35	Acceptable

II.9.2. Assessment of Food security level: The household food insecurity access scale (HFIAS) (Coates *et al.*, 2007)

Household food insecurity was measured quantitatively using the qualitative tool Household Food Insecurity Access Scale (HFIAS). The tool was composed of nine questions that ask about modifications households made in their diet or food consumption patterns due to limited resources to acquire food. Three themes were covered by the tool: 1) experiencing anxiety and uncertainty about the household food supply; 2) altering quality of the diet; and 3) reducing quantity of food consumed. For this study, the questions were asked for a period of 30 days or four weeks. Based on the response to the nine questions and frequency of occurrence over the past 4 weeks, households were assigned a score that ranges from 0 to 27. A higher HFIAS score is indicative of poorer access to food and greater household food insecurity. The prevalence of food insecurity was evaluated by using the four food security categories: 1-) Food secure; 2-) mildly food insecure; 3-) moderately food insecure; 4-) severely food insecure. These categories were created sequentially for each individual based on the responses of the individual to the various HFIA questions. For convenience sake, the HFIA categories were grouped to form three groups; the first group was the food secure household group, the second group was the mildly food insecure households group; and the third group was formed by combining the moderately food insecure and the severely food insecure households.

II.9.3. Assessment of Socioeconomic status (SES) (CDHS, 2011; Ntandou, 2009)

The estimation of socioeconomic status (SES) score of the household was used as a proxy measure for household income. Household amenity scores were used to appraise SES of households. The information on ownership of some assets: bicycle, moto bike, car, radio, television, a mobile phone, refrigerator, taxi, ownership of house living in, plot of land, agricultural land, agricultural renting land, rentable house, presence of a remunerated house help, domestic animals, presence of electricity, house construction material (of floor, walls, roof), type of fuel use for cooking, source of drinking water, type of toilet was used. The sixteen items were dichotomous and coded '1' for the presence of the asset and '0' for the absence of the asset. Three answer items were attributed to the construction material of the floor of the house and were coded 1 for the less expensive asset, 2 of the intermediate and 3 for the most expensive; those of the walls were six and were coded 1 for the first and cheapest asset and 6 for the last and most expensive asset. The asset for the roof were four: the first and less expensive asset was coded 1 and the last and most expensive asset was coded 4; five assets were indicated for the types of fuel used for cooking: the first and cheapest fuel was coded 1 and the last and most expensive fuel coded 5. With regards to source of drinking water, there were three sources coded 1 to 3; with 1 indicating natural water source, 2: tap water and 3: mineral bottle water. Finally, the type of toilet also had three items, the first item coded 1 was for poor or no toilets and the code 3 was given to the last and modern toilet system. The amenity (or SES) score was the sum of these items for a maximum of 40. On the basis of tertiles, three SES levels were created and used: *low* (for total score between 0 to 9); *medium* (for total score between 10 to 19) and *high* (for total score between 20 to 31). No participant recorded a maximum score of 40.

II.10. Evaluation of Anthropometric measures

1.) Weight Measurement

A calibrated digital scale with maximum capacity of 150 kg (The Tanita™ BC-418 Segmental Body Composition Analyzer/Scale) was used to measure weight. The scale was placed on a flat, hard surface that allows participants to stand securely. The

participants was standing still in the middle of the scale's platform without touching anything and with the weight equally distributed on both feet. Participants were weighed with light clothing, shoes off and pockets emptied (Mushaphi, 2011). Participants who weighed more than 150 kg were asked for their estimated weight because the scales are inaccurate above this level. The average of two weights was recorded numerically on the record sheet to the nearest 0.1 kg

2.) Height Measurement

Height was measured with a stadiometer (portable height measuring 2m tape). The participants were standing without shoes, with heels together, arms to the sides, legs straight, shoulders relaxed and head in the Frankfort horizontal plane (looking straight ahead). Heels, buttocks, scapulae (shoulder blades) and back of the head were against the vertical surface of the stadiometer. The headboard was lowered to the highest point of the head with enough pressure to compress the hair. The height was taken twice and recorded to the nearest 0.1 cm (Mushaphi, 2011). When there was a doubt concerning the height value taken, this value was verified from the identical card of the participant.

3.) Calculation of the Body Mass Index (BMI)

The weight and the height were used to calculate BMI. The BMI was estimated using the Quetelet's index that is by dividing the weight (in kg) by the height (in m) squared (kg/m²) (Garrow and Webster, 1985).

$$\text{BMI (Kg/m}^2\text{)} = \frac{\text{Weight (Kg)}}{(\text{Height})^2 \text{ (m}^2\text{)}}$$

4.) Waist and Hip circumferences

A stretch-resistant tape that provides a constant 100 g of tension through the use of a special indicator buckle was used to measure the waist and hip circumferences. The subject stood with arms at the sides, feet positioned close together, and weight evenly distributed across the feet. Waist circumference measurement was made at the approximate midpoint between the lower margin of the last palpable rib and the top of the iliac crest and the hip circumference measurement was taken around the widest

portion of the buttocks. For both waist and hip, the tape was snug around the body, but not pulled so tight that it is constricting. The waist circumference was measured at the end of a normal expiration, when the lungs are at their functional residual capacity. The subject was advised to relax and take a few deep, natural breaths before the actual measurement was made, to minimize the inward pull of the abdominal contents during the waist measurement (WHO, 2008). Both measures were recorded to the nearest 0.1 cm. Waist and the hip circumferences were used to calculate the waist-hip ratio (WHR) defined as waist circumference divided by hip circumference. A raised waist-hip ratio was taken as 0.85 or more in women.

II.11. Evaluation of Nutritional Status

Nutritional status was evaluated using the Body Mass Index (BMI) calculated as shown above. The WHO BMI cut-offs (WHO, 2000a) were used to categorize individuals depending on their BMI value as either underweight, normal, overweight or obese as shown in the table below.

Table 6 : Body Mass Index range and category for nutritional status evaluation

BMI Range (Kg/m ²)	Category
<18.5	Underweight
18.5 to 24.9	Normal weight
25.0 to 29.9	Overweight
≥30	obese

II.12. Evaluation of Biological measures: Systolic and Diastolic Blood pressure

Systolic and diastolic blood pressures (BPs) were measured on the right arm of seated subjects, using an Automatic Blood Pressure Monitor with Heart Sense® (Samsung). The right arm was bare and was supported at heart level. Blood pressure

records were made three times on the upper right arm. The first measurement was taken after a 10 min rest in a sitting position and was followed by two subsequent measurements in the middle and at the end of the interview. The average of the three measurements was used to assess the presence or absence of hypertension/ high blood pressure. The Monitor also gave the value of the pulse.

II.13. Blood sample collection, processing and storage

Fasting blood samples were collected from 7: 30 a.m. to 10: 30 a.m. after 12-hours overnight fast. 5 ml of venous blood was collected in heparinated tubes by venipuncture in the hand from each participant. The tubes of blood were transported in an icebox at -4°C to the laboratory car where plasma was obtained through centrifugation of the blood at 3400 rpm for 10min. The supernatant constituting the plasma was collected into dry eppendorf tubes and the aliquots were stored at -20°C in a mini deep freezer fitted in the car; and then transported to the laboratory of nutrition and nutritional biochemistry (LNNB) and later used for the determination of the plasma levels of biochemical parameters. These biochemical parameters included: lipid profile biomarkers level (total cholesterol and triglycerides), Iron, calcium and magnesium concentrations as described below.

II.14. Determination of the biochemical parameters: plasma levels of lipid profile biomarkers and some minerals (Fe, Mg, Ca).

Biochemical biomarkers of interest were total cholesterol and triglyceride for the lipid profile; calcium, magnesium and iron for mineral profile.

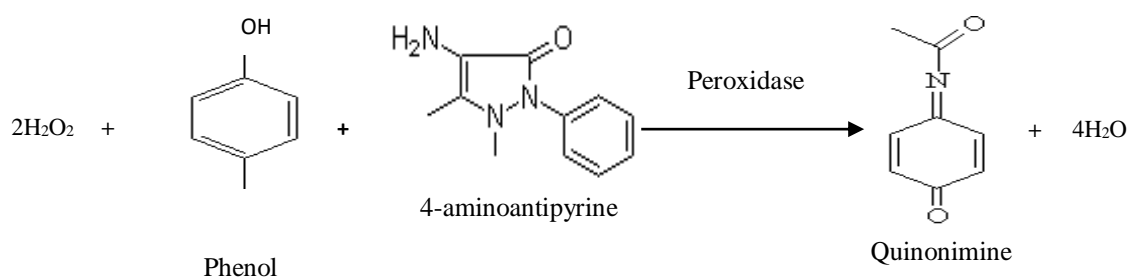
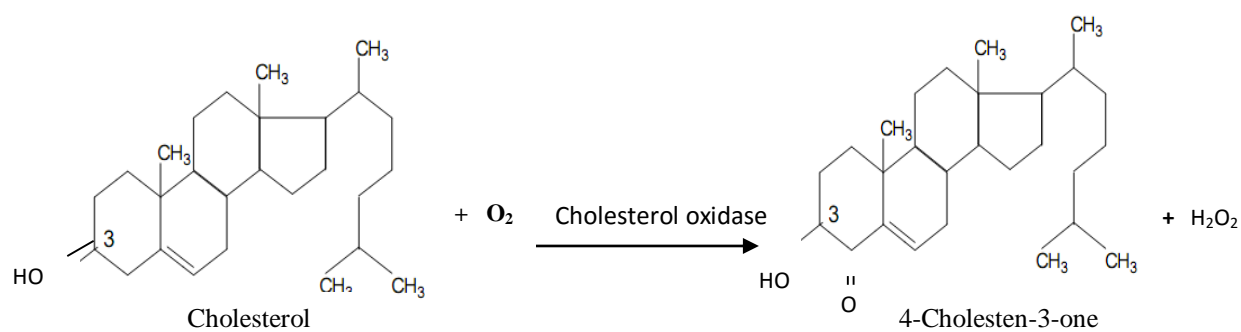
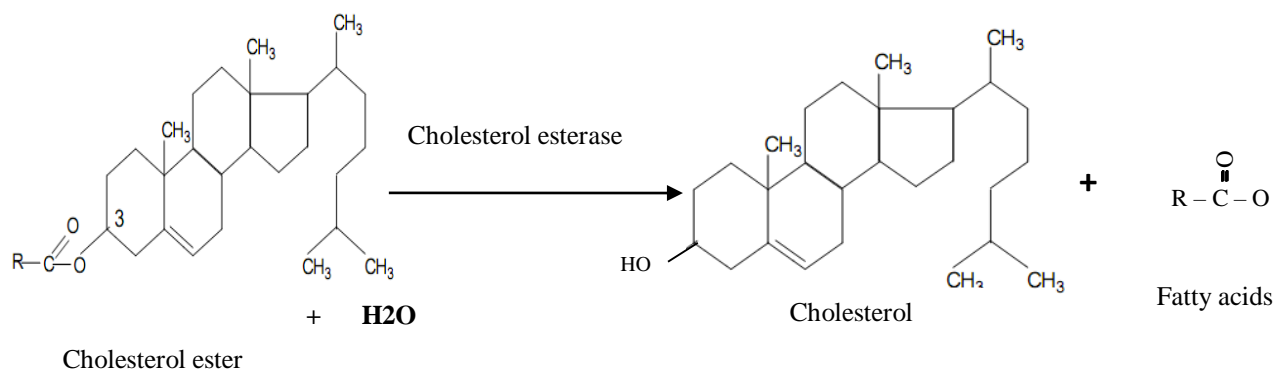
II.14.1. Lipid Profile parameters

II.14.1.1. Quantitative determination of cholesterol by the CHOD-PAP enzymatic colorimetric method (Sgmitalia kit)

➤ Principle (Naito, 1984)

The quantitative determination of cholesterol is based on the fact that cholesterol esterase catalyses the hydrolysis of cholesterol esters present in the sample to free cholesterol and fatty acids. The free cholesterol will be further oxidized into 4-

cholestenone and hydrogen peroxide by the cholesterol oxidase enzyme. Phenol and 4-aminophenazone will then combine with the hydrogen peroxide in the presence of peroxidase enzyme to give quinoneimine a red coloured product and water as described with the following equations.



The intensity of the colour formed is proportional to the cholesterol concentration in the sample.

➤ Different Reagents (Kits)

Reagent 1 (R1):

Phosphate buffer 50.0 mmol/l; Phenol 30.0mmol/l; Sodium Cholate 2.0mmoml/l

Reagent 2 (R2):

Cholesterol esterase $\geq 200\text{U/l}$; Cholesterol oxidase $\geq 160\text{U/l}$; Peroxidase $\geq 1600\text{U/l}$; 4-aminophenazone 0.9mmol/l ; no reactive stabilizers

Reagent 3 (R3):

Cholesterol standard 200mg/dl (5.17 mmol/l)

➤ Mixing Reagents

The contents of reagent 2 was dissolved with the corresponding volume of reagent 1 (buffer). The mixture was capped and mixed to dissolve for immediate use. The reagent was stable for 60 days at $+2$ to $+8^\circ\text{c}$.

➤ Assay procedure

After calibrating the instrument (spectrophotometer) with distilled water to zero, 1ml of WR was added to 10ul of sample, the standard was made of 1ml of WR with 10ml of standard solution and the blank contained just 1ml of WR. The solutions were mixed and incubated for 10 minutes at room temperature (25°C), and the absorbance (A) of the samples and standard were read against the blank at 505nm using a cuvette of 1cm light path. The colour was stable for at least 60 minutes. And the concentration of cholesterol in the sample was determined using the formular shown below:

$$\frac{\text{(A) sample}}{\text{(A) standard}} \times \text{standard concentration (5.2 mmol/l)} = \text{mg/dl cholesterol in the sample}$$

Conversion factor: $\text{mg/dL} \times 0.0259 = \text{mmol/l}$

Concentration of standard solution= 200mg/dl

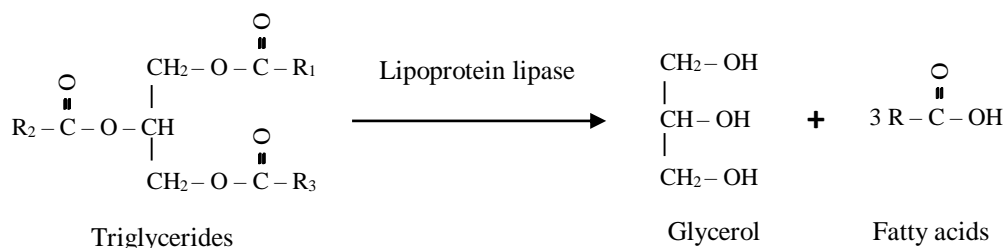
The bioassay of total cholestrol level in each sample was done twice for precision.

II.14.1.2. Quantitative determination of Triglycerides (TG) (Fortress kit)

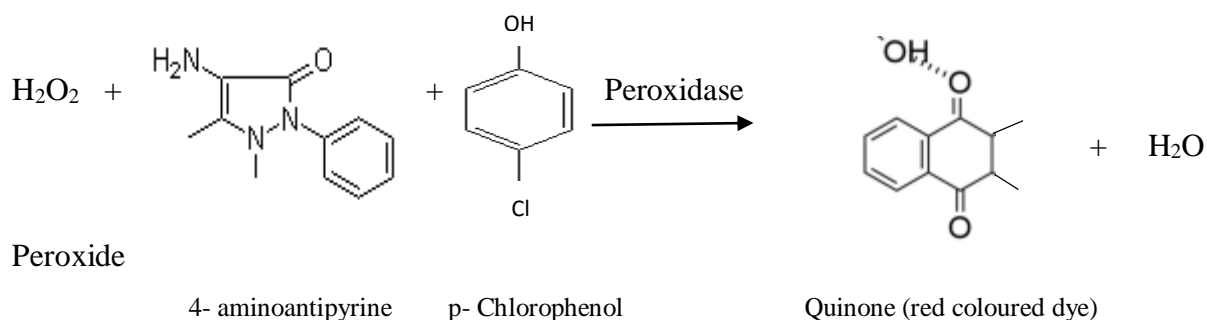
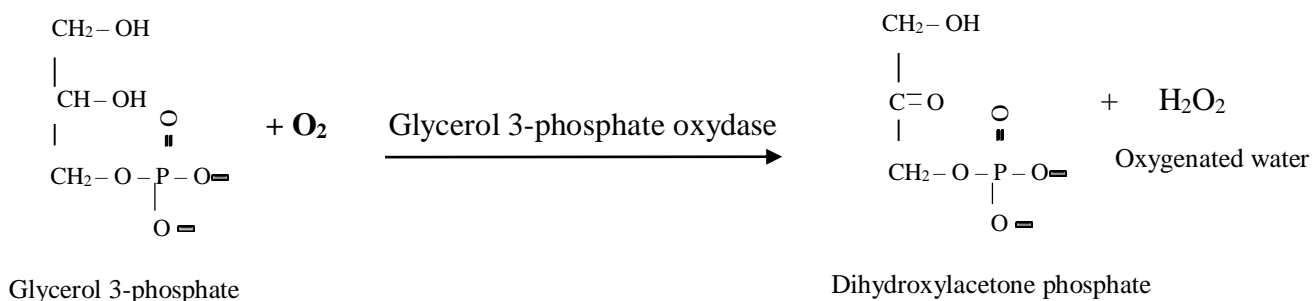
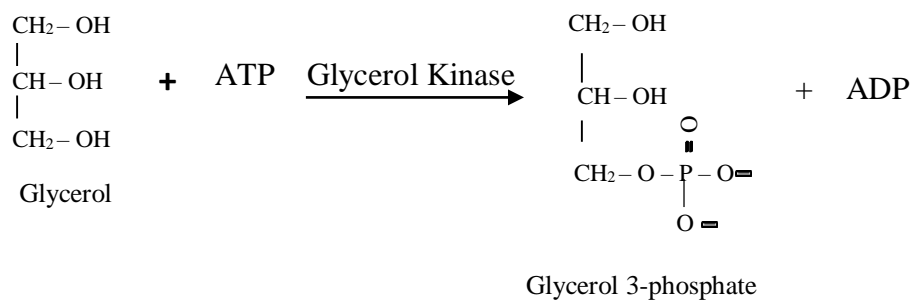
➤ Principle (Fossati and Principe, 1982)

Sample triglycerides incubated with lipoprotein lipase (LPL), liberate glycerol and free fatty acids. Glycerol is converted to glycerol- 3-phosphate (G3P) and adenosine -5-diphosphate (ADP) by glycerol kinase (GK) and ATP. Glycerol-3-phosphate (G3P) is

then converted by glycerol phosphate dehydrogenase (GPO) to dihydroxyacetone phosphate (DHAP) and hydrogen peroxide (H₂O₂). In the last reaction, hydrogen peroxide (H₂O₂) reacts with 4-aminophenazone (4-AP) and p-chlorophenol in the presence of peroxidase (POD) to give a red colored dye



R, R₁, R₂ and R₃ = Variable Radicals



The intensity of the color formed was proportional to the Triglycerides concentration in the sample.

➤ **Reagent (Kit) composition**

Reagent R (R1) Buffer:

Pipes Buffer pH 7.8 (50mmol/l); p-Chlorophenole (2mmol/l); Lipoprotein lipase (150000 U/l); Glycerolkinase (800U/l); Glycerol-3-P-Oxidase (4000 U/l); Peroxidase (440U/l); 4-Aminoantipyrine (0.7 mmol/l); ATP (0.3mmol/l); Mg 2+ (40mmol/l); Na-cholate 0.20 mmol/l; Potassium-Hexacyanoferrate (II) 1µmol/l.

Standard:

Triglycerides Concentration of 200mg/dl (2.28mmol/l)

➤ **Reagent stability**

The reagent (R1) was ready to use. The reagent was stable for 5 to 7 days at +2°C to +8°C or 3 months at -20°C

➤ **Assay procedure**

After calibrating the spectrophotometer with distilled water to zero, 1ml of WR was added to 10µl of sample, the standard was made of 1ml of WR with 10µl of standard solution and the blank contained just 1ml of WR. The solutions were mixed and incubated for 10 minutes at room temperature (37°C), and the absorbance (A) of the samples and standard were read against the blank at 505nm using a cuvette of 1cm light path. The colour was stable for at least 60 minutes. And the concentration of cholesterol in sample was calculated using the formular below:

$$\frac{(A)\text{Sample}}{(A)\text{Standard}} \times \text{Standard concentration} = \text{mg/dl Triglycerides in the sample}$$

Conversion factor; mg/dL x 0.0114 = mmol/l

Concentration of standard solution= 200mg/dl

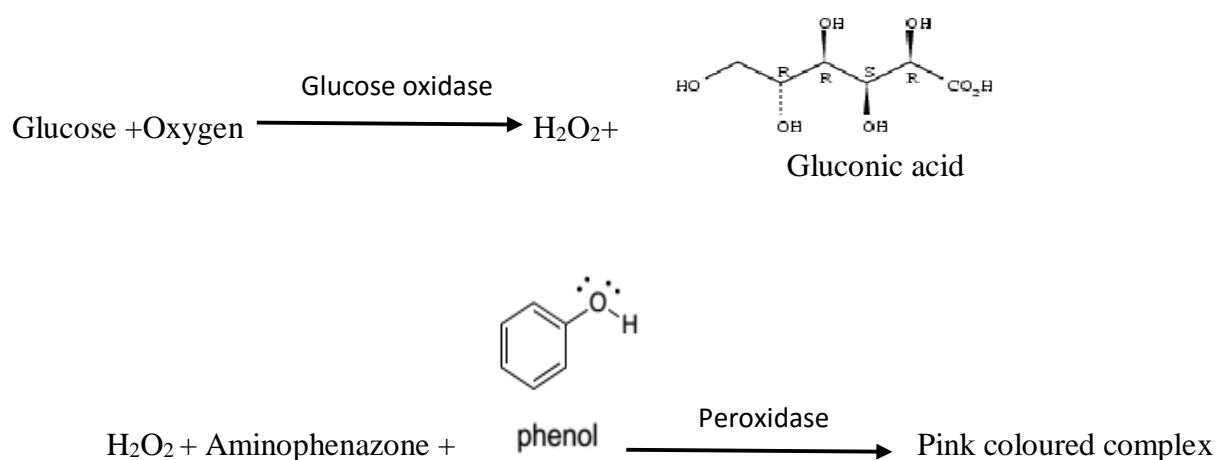
The bioassay of triglyceride level in each sample was done twice for precision.

II.14.2. Glucose concentration

II.14.2.1 Quantitative determination of Glucose using test-strips and a glucometer by the Glucose Oxidase-peroxidase method (Trinder, 1969)

Principle:

Glucose oxidase catalyses the oxidation of glucose to produce hydrogen peroxide and gluconic acid. The hydrogen peroxide, in the presence of enzyme peroxidase is broken down and the oxygen given off reacts with 4-aminophenazone and phenol to give a pink colour.



Fasting blood glucose was measured by this method immediately in the field using a glucometer (GlucoPlus™) and glucose test strips (GlucoPlusMD) directly at the participant's fingertip. The glucose test strips contained both enzymes: glucose oxidase and peroxidase on which a drop of blood was deposited and the quantity of glucose in the blood of the participant was determined following the above mentioned principle. The test strip with a drop of blood was inserted into the glucometer which gave the concentration (in mg/dl) of glucose in the blood of the participant.

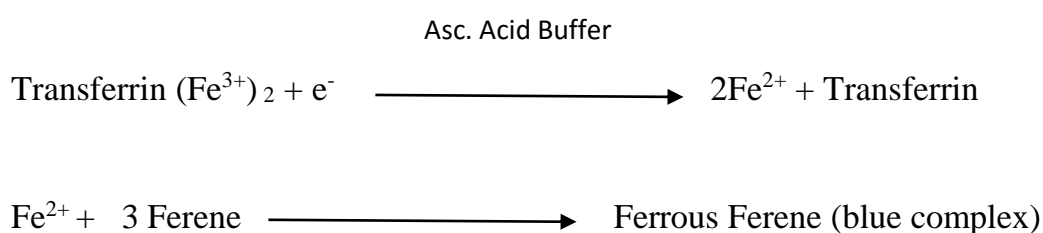
II.14.3. Bioassay of Minerals (Iron, Magnesium and Calcium)

The aliquots stored at -20°C were also used for the determination of the plasma levels of Iron, calcium and magnesium.

II.14.3.1. Bioassay of Iron by colorimetric quantitative determination or spectrophotometric method or compleximetric method (Tietz *et al.*, 1995; Itano, 1978)

Test Principle

Iron bound to transferrin is released in an acidic medium as ferric iron and is then reduced to ferrous iron in the presence of ascorbic acid. Ferrous iron forms a blue complex with Ferene 3-(2-pyridyl)-5, 6-difuryl-1, 2, 4-triazine-disulfonate). The absorbance at 595nm is directly proportional to the iron concentration.



Reagents (Kits)

Reagent 1 (R1):

Acetate Buffer, pH 4.9 (100mmol/l)

Reagent 2 (R2):

Ascorbic Acid (reductant) (99.7%)

Reagent 3 (R3):

Ferrozine (coloured) (40mmol/l)

Reagent 4 (R4):

Iron standard (100ug/dl)

Mixing Reagents (kits)

Reagents (kits) were ready to use.

To obtain the working reagent (WR), the contents of one tube R2 Reductant was dissolved in one bottle of R1 Buffer. Then the contents were capped and mixed gently to dissolve them.

Reagents and samples were first brought to room temperature and then in various cuvettes labelled WR blank, standard, sample blank and sample, the following were pipetted:

Table 7 : Manual Test procedure for plasma iron assay

	Working Reagent(WR) Blank	Standard	Sample Blank	Sample
WR (ml)	1.0	1.0	1.0	1.0
R3 (drops)	1	1	-	1
Distilled water (µl)	200	-	-	-
Standard (µl)	-	200	-	-
Sample (µl)	-	-	200	200

The mixture was then mixed and incubated for 5 minutes at 37°C or 10 minutes at room temperature. The absorbance (A) of standard and sample against WR blank were measured at 562nm. The bioassay of iron level in each sample was done twice for precision.

Calculation and expression of results

The concentration of iron in the sample was calculated as follows:

$$\frac{(A)\text{Sample}-(A)\text{ Sample Blank}}{(A)\text{ Standard}} \times \text{Standard concentration (100}\mu\text{g/dl)} = \mu\text{g/dl of Iron in sample}$$

$$\text{Conversion factor: } \mu\text{g/dl} \times 0.179 = \mu\text{mol/l}$$

II.14.3.2. Bioassay of Magnesium by colorimetric or spectrophotometric method Xylidyl Blue (Bohoun, 1962)

Test Principle

Magnesium reacts with Xylidyl Blue, in alkaline solution, giving a chelating crimson compound whose colour intensity is directly proportional to the magnesium concentration in the tested sample.

Reagents (Kits)

Reagent 1 (R1): Potassium carbonate 58.0 mmol/l; EGTA 0.04 mmol/l; Xylidyl Blue 0.0 mmol/l

Reagent 2 (R2): Magnesium standard (2.5 mg/dl)

Reagents (kits)

Reagents (kits) were liquid and ready for use

Reagents and samples were first brought to room temperature and then in various cuvettes labelled working reagent blank, standard and sample, the following were pipetted:

Table 8 : Manual Test procedure for plasma magnesium assay

	WR Blank	Standard/Calibrator	Sample
WR (R1)	2000 μ l	2000 μ l	2000 μ l
Distilled water	20 μ l		
Sample	-	-	20 μ l
Standard (R2)	-	20 μ l	-

The mixture was then mixed and incubated for 5 minutes at 37°C. The absorbance (A) of standard and sample against WR blank were measured at 520nm within 60minutes. The bioassay of magnesium level in each sample was done twice for precision.

Calculation and expression of results

The concentration of magnesium in sample was calculated as follows:

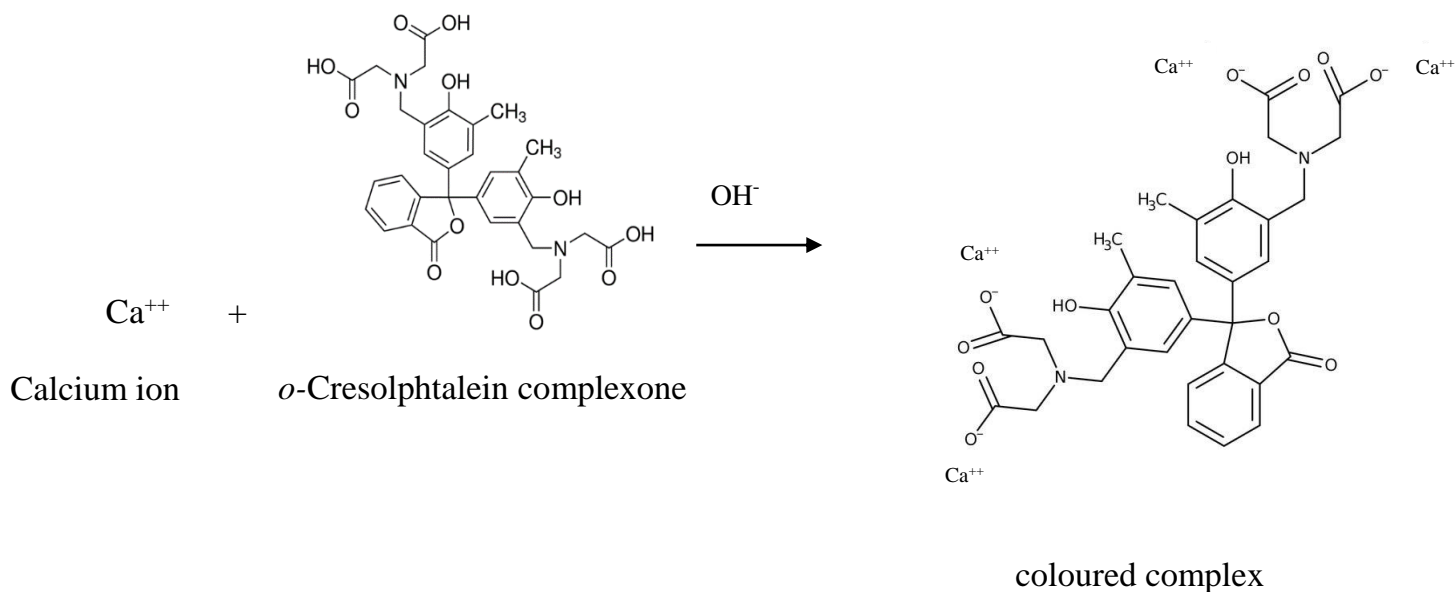
$$\frac{(A)\text{Sample}}{(A)\text{Standard}} \times \text{Standard concentration (2.5mg/dl)} = \text{mg/dl of Magnesium in the sample}$$

$$\text{Conversion factor: mg/dl} \times 0.41 = \text{mmol/l}$$

II.14.3.3. Quantitative determination of Calcium (Colorimetric method) (Farell, 1984)

Test principle

The measurement of calcium in the sample is based on formation of color complex between calcium and *o*-cresolphthalein in alkaline medium:



The intensity of the colour formed is proportional to the calcium concentration in the sample

Reagents (Kits)

Reagent 1 (R1): Buffer (Ethanalamine): 500mmol/l

Reagent 2 (R2): Chromogen: (*o*-cresolphthalein: 0.62 mmol/l; 8-Hydroxyquinolein: 69mmol/l) (8-Hydroxyquinolein is added to eliminate the interference by magnesium)

Reagent 3 (R3): Standard: standard aqueous primary: 10mg/dl

Reagents (kits)

Reagents (kits) were liquid and ready for use.

Reagents and samples were first brought to room temperature and then in various cuvettes labelled WR blank, standard and sample, the following were pipetted:

Table 9 : Manual Test procedure for plasma calcium assay

	WR Blank	Standard/Calibrator	Sample
R1 (ml)	1.0	1.0	1.0
R2 (ml)	1.0	1.0	1.0
Standard (R3) (µl)	-	20	-
Sample (µl)	-	-	20

The mixture was then mixed and incubated for 5 minutes at 37°C. The absorbance (A) of standard and sample against WR blank were measured at 570nm within 40minutes. The bioassay of calcium level in each sample was done twice for precision.

Calculation and expression of results

The concentration of calcium in sample was calculated as follows:

$$\frac{(A)\text{Sample}}{(A)\text{Standard}} \times \text{Standard concentration (10 mg/dl)} = \text{mg/dl of Calcium in the sample}$$

$$\text{Conversion factor: mg/dl} \times 0.25 = \text{mmol/l}$$

II.14.3.4. Reference values for plasma Calcium, Magnesium and Iron levels

-The reference values used in this study for plasma calcium level (Farell, 1984) were:

Normal level: plasma concentration between 8.5-10.5 mg/dL

Hypocalcemia: < 8.5 mg/dL

Hypercalcemia: > 10.5mg/dL

-The reference values used in this study for plasma magnesium level (Chernecky and Berger, 2013) were:

Normal level: plasma concentration between 1.7 - 2.2 mg/dL

Hypomagnesia: < 1.7 mg/dL

Hypermagnesia: > 2.2 mg/dL

-The reference values used in this study for plasma iron level (Thomas, 1998) were:

Normal level: plasma concentration between 30-150µg/dl

Iron deficiency: < 30µg/dl

Iron overload: > 150µg/dl

II.15. Evaluation of Metabolic syndrome

The metabolic syndrome was evaluated using International Diabetes Federation (IDF) criteria (Alberti *et al.*, 2006) which have been shown to be the most appropriate definition of metabolic syndrome for Cameroonian population (Mandob *et al.*, 2008). According to IDF criteria, central obesity (defined as waist circumference ≥ 80 cm in women) is a prerequisite in addition to two or more components. The International Diabetes Federation definition is shown in the table below:

Table 10 : Criteria for metabolic syndrome individual components (Alberti *et al.*, 2006)

Factors or Components	IDF Criteria
Raised triglycerides	≥ 150 mg/dl
Reduced HDL cholesterol	< 50 mg/dl for women
Raised blood pressure	Systolic BP ≥ 130 mmHg and/or diastolic BP ≥ 85 mmHg, or blood pressure lowering treatment
Raised fasting plasma glucose	FPG ≥ 100 mg/dl or previously diagnosed type 2 diabetes

II.16. Evaluation of Cardiometabolic risk factors

Biological cardiometabolic risk factors evaluated were five in total and included:

- 1.) Metabolic syndrome. The metabolic syndrome was evaluated using International Diabetes Federation (IDF) criteria (Alberti *et al.*, 2006) which have been shown to be an appropriate definition of metabolic syndrome for Cameroonian population. According to IDF criteria, waist circumference (>80 cm in women) is a prerequisite in addition to two or more of the following components: fasting triglyceride levels ≥ 150 mg/dl, fasting glucose levels ≥ 100 mg/dl, or hypertension (SBP ≥ 130 mmHg and/or DBP ≥ 85 mmHg, or blood pressure lowering treatment) in addition to fasting cholesterol levels ≥ 200 mg/dl.
- 2.) Obesity or overweight or central obesity;
- 3.) hypertension;
- 4.) hyperglycemia;
- 5.) dyslipidemia (hypertriglyceridemia and hypercholesterolemia).

IDF criteria were also used to evaluate the individual biological cardiometabolic risk factors (Alberti *et al.*, 2006).

II.17. Establishment of the phenotypes of the double burden of malnutrition and biological cardiometabolic risk factors at the individual level

These phenotypes were eight in number and were established by considering three criteria: the weight status, the presence of specific biological cardiometabolic risk factors and the mineral status at the individual level; except for phenotype I where the presence of at least one BCMRF was one of the criterion in addition to the mineral status. For phenotype VIII, only the weight status and the mineral status were considered to establish this phenotype. These phenotypes included:

Phenotype I: at least one mineral deficiency and one BCMRF

Phenotype II: overweight or obese+ at least one other BCMRF+ at least one mineral deficiency

Phenotype III: overweight or obese+ hypertension+ at least one mineral deficiency

Phenotype IV: overweight or obese+ hyperglycemia+ at least one mineral deficiency

Phenotype V: overweight or obese+ Hypertriglyceridemia + at least one mineral deficiency

Phenotyp VI: overweight or obese+ Hypercholesterolemia + at least one mineral deficiency

Phenotype VII: overweight or obese+ MetS + at least one mineral deficiency

Phenotype VIII: Underweight + at least one mineral deficiency

The use of the categorization of the 8 phenotypes of the DBM and cardio metabolic risk factors in this present study population was to show that in an individual, it was possible to observe the co-occurrence of obesity with micronutrient deficiencies and the combination of under nutrition with nutrition-related cardio metabolic risk markers other than obesity. Therefore categorising the phenotypes was to observed and understand all the possible combinations that could exist in this group of Cameroonian women in order to formulate different possible intervention strategies.

II.18. Evaluation of Physical activity level

Concerning physical activity levels, based on the Global Physical Activity Questionnaire (GPAQ) analysis guide developed by WHO, informations were recorded. The participants were interviewed on their usual physical activity on a typical day and during the week for transport, activity during leisure, work and housework as well as sitting. The subjects described their activities and the duration, during leisures (for example football, trekking, handball, jogging...) work (carrying heavy or light load on the head for long distances, standing up the whole day, walking the whole day, farm work...), active transport with regard to trekking, bicycle, motorbike or car. The main occupation of the participants was equally described with the aim of defining the corresponding level of physical energy expenditure. The average duration and the frequency per week for each activity were recorded. The level of intensity of the activities was expressed in metabolic equivalents (MET). Then the activities of each group was categorized according to intensity level according to the METs based on the compendium of physical activities (Ainsworth *et al.*, 2000): light (< 3.0 METs); moderate ($3.0 \leq \text{METs} \leq 6.0$ METs), and vigorous (> 6.0 METs) (Ntandou, 2009). For the main occupation, we considered vigorous work (e.g carrying heavy load on the head for long distances, brick layering, builder, farmer, truck pusher...), moderate work (e.g.: carrying light load on the head for short distances, gardening, carpentry, mechanician, teaching...) and light or sedentary work (e.g.: studying, house chores, computer work, office work, civil servant, waitress, buyer and seller etc). For transportation, we retained bicycling as vigorous, walking as moderate and motorbike, vehicle or car, as light activity. Three levels of energy expenditure were also considered for leisure activity: vigorous (football, basket-ball and intense physical exercises), moderate (dancing, gymnastics and moderate physical exercises), and light or sedentary (Television viewing, playing cards, sleeping...) (Ntandou *et al.*, 2008).

II.19. Ethical considerations

This study received the approval of the National Ethic Committee of Research for Human Health. The ethical clearance number is: N° 2014/08/488/CE/CNERSH/SP (appendix 3). The approval of the senior divisional officers and heads of health centres

of the different localities were also obtained. The verbal authorizations of chiefs of quarters and villages were obtained as well as those of the heads of families to carry out the study in their localities.

The participation of each individual was voluntary after she was explained the objectives of the study. Each participant signed in the presence of a local witness an informed consent form in which it was clearly explained that if at any time they wish to withdraw from the study without having to explain their reasons they may do so without any consequences (appendix 4). Illiterate participants gave their oral consent and affixed their thumb print as signature on the consent form in the presence of their literate witness. The participants were not remunerated for their participation.

Blood samples collected from participants was used for the bioassay of micronutrients levels. To ensure the confidentiality of the data, tubes containing blood were coded before their transfer to the laboratory for analysis.

Participants who were found to have poor nutritional status (poor dietary habits, low micronutrients levels and low weight status) after carry out all the analyses were given nutritional and dietetic advices and were referred to a hospital for better follow up and treatment. The results of the laboratory bioassays were given to the participants as soon as they were made available. Each participant received the visit of the nutritional biochemist who gave back the participant's results amongst which there was the weight, height, general nutritional status with respect to BMI, dietary habits and micronutrients level in blood. The nutritional biochemist of the research team explained the individual results to each participant and with the result form of the patients, directed those with a problem to a health centre. The files of the participants (questionnaires, forms, results of laboratory bioassays) were stored in LNNB of the University of Yaoundé I for a period of 5 years after the end of the study. The data collected were codified for statistical analysis and were used for the present thesis. The results of the analysis which were published in specialised or general scientific journals do not allow the identification of the participants. Parents or guidances of participants under 21 years of age were asked to sign the informed consent form for parents for their children to participate in the study.

II.20. Statistical Analysis

Data were analyzed using the IBM SPSS statistical software package version 20.0. Descriptive statistics including the frequency, mean, standard error of the mean, minimum and maximum were calculated for all variables. In addition to double data entry, these descriptive statistical tools were equally used to verify data entry errors especially in the verification of extreme values, missing values and corrections were done were required. Continuous variables were examined for adherence to a normal distribution and all mineral values, lipid profile biomarkers (total cholesterol and triglycerides) in plasma were normally distributed and hence no transformation was done.

Results were expressed as means and standard error of the mean for continuous variables, and as frequencies (%) for categorical variables. Prevalence is the proportion of individuals or a group of individuals in a population having a characteristic expressed as frequency. The prevalence was determined through the exploitation of cross tables followed by the Chi square test were necessary. Comparisons of parameters between groups were expressed as means plus or minus standard error of the mean. The Student's t-test for unpaired samples was used to detect differences in means between two groups of a continuous dependent variable; whereas, one-way analysis of variance (ANOVA) was used to detect differences in means between several groups (≥ 3 groups) in the presence of a continuous dependent variable. Multiple comparisons of group mean after the ANOVA test were performed using the least significant difference (LSD) post-hoc test.

Bivariate correlation analyses were performed to check for associations between variables. The Pearson's chi-square test was used to compare proportions between categorical variables. Pearson's correlation was used to check for the associations between two continuous variables of normal distribution, that is: the association between the frequency of intake of various food groups (exposures) with anthropometric, clinical and biochemical parameters (outcomes); associations between mineral concentrations; and associations between mineral concentrations and biological cardio metabolic risk factors. The use of Pearson's correlation test to assess the association between mineral

concentrations and biological cardio metabolic risk factors was to measure the strength of relationship between these continuous variables in order to find out a possible explanation of the probable high rates of biological cardio metabolic risk factors in this study population.

The Ca-Mg ratio has been found as a significant accurate marker of cardiovascular diseases mortality and all-cause mortality and was stated to be a more useful marker than Mg level alone in that study population (Sato *et al.*, 2018). Therefore, in this present study, tertiles of Ca-Mg ratio were created to assess if this marker could equally be an accurate predictor of individual biological cardio metabolic risk factors in this study population and, to assess the relationship between these tertile groups and individual biological cardio metabolic biomarkers and also, the prevalence of individual BCMRFs. The Ca-Mg ratio groups were obtained by dividing participants into three groups of the same number of participants. The high Ca-Mg ratio group had a Ca-Mg ratio of 8.99 ± 0.99 , the medium Ca-Mg ratio group had Ca-Mg ratio of 2.35 ± 0.04 and the low Ca-Mg ratio group had a Ca-Mg ratio of 0.46 ± 0.03 .

Multivariate Logistic regression model analyses were performed to assess the various predictors of nutritional deficiency markers (underweight, calcium deficiency, magnesium deficiency, and iron deficiency, and combined mineral deficiencies). Age adjusted logistic regression models were used to assess the predictors of individual biological cardio metabolic risk factors and the predictors of the eight phenotypes of the double burden of malnutrition and BCMRFs observed in this study.

Predictors of Nutritional deficiency markers which were assessed were the sociodemographic determinants such as the age groups, region of residence; and nutritional determinants such as the frequency of intake of the various food groups assessed in this study. In addition, for the nutritional markers, each of the mineral deficiencies were also assessed as potential determinants for other mineral deficiencies. Nutritional risk factors (FCS threshold, HFIAS threshold, moderate or low intake of vegetables and fruits, and excess intake of alcohol) of developing one or more nutritional deficiencies were equally assessed using logistic regression analyses. A magnesium deficient state (adjusted for calcium status, iron status and age), calcium deficient state

(adjusted for magnesium status, iron status and age), iron deficient state (adjusted for magnesium status, calcium status and age), low Ca-Mg ratio and high Ca-Mg ratio (adjusted for magnesium status, calcium status, iron status and age) were also assessed as predictors or determinants of individuals biological cardio metabolic risk factors. The choice of a variable as a confounder was based on the association the confounder had with the factor or characteristic studied. The level of statistical significance was $P < 0.05$.

CHAPTER III: RESULTS AND DISCUSSION

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III.1. RESULTS

III.1.1. Baseline characteristics of the study population

1152 women of childbearing age gave their consent after been explained the objectives of the study. 313 participants did not completely fill the questionnaires or did not have their anthropometric measures collected making a total of 839 with all these parameters. Among the 839 participants, in addition to the lipid profil, 608 had their blood mineral levels measured and were included in this study and the final analysis.

The sample population was therefore made up of 608 women of childbearing age of average 34.68 ± 0.39 years. Table 11 below presents the means \pm standard error of the means of the anthropometric, biochemical and demographic parameters of the study population.

Table 11 : Anthropometric, biochemical and demographic characteristics of the study population

Parameters	Mean	SEM	N
Age (years)	34.68	0.39	608
Weight (Kg)	74.15	0.68	608
Height (m)	1.59	0.00	608
WC (cm)	88.55	0.58	608
HC (cm)	105.57	0.56	608
WHR	1.00	0.00	608
FCS	31.03	0.86	608
HFIAS	5.03	0.35	608
Number of meals/day	2.75	0.02	608
Socioeconomic score	4.85	0.13	608

SEM: Standard error of mean; N: sample size; WC: Waist circumference; HC: Hip circumference; WHR: Waist to Hip ratio; FCS: Food Consumption score; HFIAS: Household Food Insecurity Access Scale

III.1.2. Distribution of participants by sociodemographic and nutritional characteristics and physical activity level

Table 12 presents the distribution of subjects of the study with respect to sociodemographic characteristics. Participants in the 31-40 years age group represented 34.4% of the study population. The North West region represented the highest number

of participants (36%) of the study population. 24.3 percent of the participants of the study had a primary educational level. Up to 38.3 percent of the women of the study were housewives and most of them (70.6 percent in the 95% CI) were married. Up to 38.0 percent of the participants of the study got their revenue from doing business. Most participants of the study (81.5 percent) were found in households with high socioeconomic status. Up to 39.3 percent of them had an acceptable food consumption score (FCS) and 48.8 percent of them were found in food secure households in the 95 percent confidence interval (CI); while most study participants (50.2 percent) had a low physical activity level.

Table 12 : Frequency of participants of the study by sociodemographic and nutritional characteristics and physical activity level

Characteristics	Frequency (n)	Percentage (%)	95% Confidence Interval	
			Lower limit	Upper limit
Age group				
<i>14-20 years</i>	63	10.4	8.1	12.8
<i>21-30 years</i>	141	23.2	19.7	26.8
<i>31-40 years</i>	209	34.4	30.3	38.1
<i>41-49 years</i>	195	32.1	28.1	36.0
Region/area of residence				
<i>Yaoundé</i>	72	11.8	9.2	14.6
<i>Littoral</i>	119	19.6	16.4	23.2
<i>West</i>	172	28.3	24.8	32.1
<i>North west</i>	219	36.0	31.9	39.8
<i>North</i>	26	4.3	2.6	5.9
Educational level				
<i>Illiterate</i>	121	19.9	16.6	23.0
<i>Literates</i>	89	14.6	11.7	17.4
<i>Primary</i>	148	24.3	20.9	27.8
<i>First cycle</i>	117	19.2	16.3	22.4
<i>Second cycle</i>	116	19.1	16.0	22.4
<i>University</i>	17	2.8	1.5	4.1
Profession				
<i>Student</i>	106	18.1	15.0	21.2
<i>Employed</i>	93	15.9	12.8	18.8
<i>Odd jobs/farmers</i>	110	18.8	15.7	21.9
<i>Business</i>	52	8.9	6.7	11.4
<i>Housewives</i>	247	38.3	34.2	42.2
Marital Status				
<i>Single</i>	74	12.2	9.5	14.6
<i>Married</i>	429	70.6	66.9	74.2
<i>Widow/Divorced</i>	105	17.3	14.3	20.4
Source of revenue				
<i>Permanent salary</i>	102	16.8	13.8	19.4
<i>Temporal salary</i>	44	7.2	5.3	9.5
<i>Business</i>	231	38.0	34.0	42.4
<i>Spouse</i>	137	22.5	19.3	26.0
<i>Help</i>	83	13.7	11.0	16.4
<i>Pension</i>	11	1.8	0.8	3.0
Socioeconomic status				
<i>Low</i>	14	2.8	1.6	4.3
<i>Medium</i>	80	15.8	12.3	18.9
<i>High</i>	413	81.5	78.1	85.0
FCS				
<i>Poor</i>	195	32.1	28.3	35.9
<i>Borderline</i>	174	28.6	25.0	32.4
<i>Acceptable</i>	239	39.3	35.2	43.6
HFIAS				
<i>Food secure</i>	161	48.8	43.0	54.8
<i>Mildly food insecure</i>	74	22.4	17.9	27.3
<i>Moderately & severely food insecure</i>	95	28.8	23.9	33.9
PAL				
<i>Low</i>	244	50.2	45.5	54.5
<i>Moderate</i>	221	45.5	41.2	50.0
<i>High</i>	21	4.3	2.7	6.4

n: frequency (Number of participants in that group); **FCS:** Food Consumption score; **HFIAS:** Household Food Insecurity Access

Scale; **PAL:** Physical Activity Level

Table 13 below presents the anthropometric, nutritional parameters and socioeconomic status of the study population with respect to age groups. The mean weight, WC, HC, BMI, Food consumption score (FCS), socioeconomic score (SES) were significantly different between the various age groups ($p < 0.05$)

Table 13 : Anthropometric, Nutritional and socioeconomic status parameters with respect to age groups

<i>Parameters</i>	<i>Age group (years)</i>	<i>Mean</i>	<i>SEM</i>	<i>P-value</i>
<i>Weight</i>	14-20	60.49	1.37	0.00
	21-30	70.26	1.23	
	31-40	76.01	1.08	
	41-49	79.37	1.31	
<i>Height</i>	14-20	1.94	0.03	0.479
	21-30	1.94	0.02	
	31-40	1.94	0.01	
	41-49	1.97	0.01	
<i>WC</i>	14-20	78.06	1.48	0.000
	21-30	83.84	1.77	
	31-40	89.15	0.82	
	41-49	94.70	1.04	
<i>HC</i>	14-20	96.21	1.44	0.000
	21-30	103.10	1.15	
	31-40	106.17	0.82	
	41-49	109.74	1.06	
<i>WHR</i>	14-20	1.00	0.00	0.910
	21-30	1.00	0.00	
	31-40	1.00	0.00	
	41-49	1.00	0.00	
<i>FCS</i>	14-20	38.48	2.85	0.018
	21-30	31.99	1.55	
	31-40	29.17	1.55	
	41-49	29.92	1.53	
<i>HFIAS</i>	14-20	6.83	1.00	0.137
	21-30	4.78	0.60	
	31-40	4.29	0.62	
	41-49	5.01	0.68	
<i>SES</i>	14-20	5.60	0.42	0.007
	21-30	5.36	0.28	
	31-40	4.34	0.21	
	41-49	4.79	0.23	

SEM: Standard error of mean; **WC:** Waist circumference; **HC:** Hip circumference; **WHR:** Waist to Hip ratio; **HFIAS:** Household Food Insecurity Access Scale; **FCS:** Food Consumption score; **SES:** Socioeconomic Status

III.1.3. Proportion of women with respect to Sociodemographic, nutritional and other parameters by age group

With respect to the area or region of origin, whatever the age group, most participants of the study were from the northwest region except for the 41-49 years age group which included most participants from the west region ($p < 0.05$). In all the age groups, a higher percentage of the participants of the study had a primary educational level except for the 14-20 years age group for which a higher proportion were just literates. With regards to the profession in the 14-20 years age group and in the 21-30 years age group, a higher percentage of the participants were students while in the 31-40 and 41-49 years age groups, a higher percentage were housewives ($p < 0.05$). Whatever the age groups a greater proportion of the study participants were married women. Also, a greater percentage of the participants of the study had an acceptable FCS in all the age groups ($p < 0.05$).

When the household food security level was considered, a higher percentage of the participants (6.7%) were found in moderately to severely food insecure households for the 14-20 years age group, while in the other age groups, most women were in food secure household. In the 14-20 and 41-49 years age groups, a significantly higher proportion of the participant had a moderate physical activity level (PAL) while in the 21-30 and 31-40 years age groups, a higher percentages had a low PAL.

Whatever the age group, women in high socioeconomic status (SES) households, were mostly represented and the difference between the various group was statistically significant ($p < 0.05$). Assessment of Traditional diets (TD) intake in a week by age group showed that in the 14-20 years age group a proportion of 5.8% had a low intake of TD while in the other groups a higher proportion of the study participants had a moderate intake in a week ($p > 0.05$).

Table 14 : Number of women with respect to sociodemographic, nutritional and other parameters by age group

Factors	14-20 years n (%)	21-30 years n (%)	31-40 years n (%)	41-49 years n (%)	X²	P-Value
Area of residence						
Yaoundé	6(1.0)	12(2.0)	30(4.9)	24(3.9)	54.755	0.000
Littoral	2(0.3)	24(3.9)	49(8.1)	44(7.2)		
West	10(1.6)	38(6.2)	58(9.5)	66(10.9)		
North west	45(7.4)	59(9.7)	60(9.9)	55(9.0)		
North	0(0.0)	8(1.3)	12(2.0)	6(1.0)		
Educational level						
Illiterate	13(2.1)	28(4.6)	47(7.7)	33(5.4)	23.415	0.076
Literates	15(2.5)	23(3.8)	20(3.3)	31(5.1)		
Primary	10(1.6)	31(5.1)	53(8.7)	54(8.9)		
First cycle	18(3.0)	26(4.3)	34(5.6)	39(6.4)		
Second cycle	6(1.0)	28(4.6)	47(7.7)	35(5.8)		
University	1(0.2)	5(0.8)	8(1.3)	3(0.5)		
Profession						
Student	46(7.9)	51(8.7)	8(1.4)	1(0.2)	248.687	0.000
Employed	0(0.0)	18(3.1)	44(7.5)	31(5.3)		
Odd jobs/farmers	7(1.2)	22(3.8)	43(7.4)	38(6.5)		
Business	1(0.2)	6(1.0)	24(4.1)	21(3.6)		
Housewives	5(0.9)	41(7.0)	86(14.7)	92(15.7)		
Marital status						
Single	14(2.3)	25(4.1)	23(3.8)	12(2.0)	33.56	0.000
Married	33(5.4)	87(14.3)	166(27.3)	143(23.5)		
Widow/Divorced	16(2.6)	29(4.8)	20(3.3)	40(6.6)		
FCS						
Poor	14(2.3)	34(5.6)	84(13.8)	63(10.4)	19.168	0.004
Borderline	14(2.3)	45(7.4)	52(8.6)	63(10.4)		
Acceptable	35(5.8)	62(10.2)	73(12.0)	69(11.3)		
HFIAS						
Food secure	20(6.1)	41(12.4)	52(15.8)	48(14.5)	8.478	0.205
Mildly food insecure	10(3.0)	25(7.6)	19(5.8)	20(6.1)		
Moderately and severely food insecure	22(6.7)	23(7.0)	22(6.7)	28(8.5)		
Number of meals per day						
≤ 2meals /day	12(2.0)	41(6.7)	50(8.2)	53(8.7)	3.398	0.758
3 meals/day	48(7.9)	95(15.6)	153(25.2)	135(22.2)		
≥ 4 meals/day	3(0.5)	5(0.8)	6 (1.0)	7(1.2)		
Physical Activity Level (PAL)						
Low	23(4.7)	55(11.3)	96(19.8)	70(14.4)	15.544	0.016
Moderate	24(4.9)	51(10.5)	70(14.4)	76(15.6)		
High	7(1.4)	2(0.4)	5 (1.0)	7(1.4)		
Socioeconomic status						
Low					13.373	0.037
Medium	0(0.0)	3(0.6)	7(1.4)	4(0.8)		
High	2(0.4)	15(3.0)	33(6.5)	30(5.9)		
	52(10.3)	104(20.5)	128(25.2)	129(25.4)		
Traditional diets intake						
Low	27(5.8)	45(9.7)	48(10.4)	56(12.1)	12.035	0.061
Moderate	23(5.0)	67(14.5)	102(22.0)	92(19.9)		
High	1(0.2)	0(0.0)	0(0.0)	2(0.4)		

n: Frequency of participants in that group; FCS: Food consumption score; HFIAS: Household Food Insecurity Access Scale; X²: Chi square value

III.1.4. Consumption frequency and mean consumption frequency of the various food groups in the study population

III.1.4.1. Consumption frequency of the various food groups in the study population

The table below portrays the frequency of intake of various food groups in a week. It reveals that most women (86.4%) of the study consumed cereals and tubers food groups 5 to 7 times or days in a week while 4.2% consumed this food group 1 to 2 times a week in the 95% confidence interval (CI). With regards to the pulse food group, up to 41.6% had a low intake (1 to 2 times in a week) while only 24.3% of the women had a high intake in a week. Most of the women (57 %) had a low intake of milk and dairy products food group in a week. Also, the meat, fish and egg group was consumed only 1 to 2 times in a week by up to 43.8 % of the women while only 27.3 % of them in the 95% CI had a high intake in a week. The vegetable food group and the fruits group were taken 5 to 7 times in a week by 33.1% and 9.8 % of the women respectively. The fats and oils group was eaten 5 to 7 times in a week by most women (56.9%) while the sugar group was taken only 1 to 2 times in a week by most women (57.2%) in the 95% CI.

Concerning the intake of traditional diets per week, most participants (61.3%) consumed traditional diets 2 to 4 times in a week and only 0.6% of them in the 95% CI (0.0-1.5) had a high intake in a week.

Table 15 : Frequency of consumption of the various food groups in the study population

<i>Food groups (N=608)</i>	Low intake (1-2 times/week) % (95%CI)	Moderate intake (3-4 times/week) % (95%CI)	High intake (5-7 times/week) % (95%CI)
<i>Cereals and tubers group</i>	4.2(2.4-5.9)	9.5(7.1-12.1)	86.4(83.4-89.3)
<i>Pulses group</i>	41.6(37.2-46.3)	34.1(30.0-38.9)	24.3(20.4-28.2)
<i>Milk and Dairy products group</i>	57.0(46.0-67.0)	27.0(18.0-36.0)	16.0(10.0-23.8)
<i>Meat, fish and eggs group</i>	43.8(38.3-49.3)	28.9(23.7-34.0)	27.3(22.4-32.5)
<i>Vegetables group</i>	38.5(34.0-42.9)	28.4(24.5-32.6)	33.1(28.7-37.3)
<i>Fruits group</i>	69.2(62.5-75.0)	21.0(15.7-25.9)	9.8(6.3-14.2)
<i>Fats and oils group</i>	32.3(27.1-37.8)	10.8(7.3-14.5)	56.9(51.4-62.8)
<i>Sugar group</i>	57.2(51.7-62.9)	22.1(17.8-26.7)	20.7(17.0-25.0)
	Low intake (0-1 times/week) % (95%CI)	Moderate intake (2-4 times/week) % (95%CI)	High intake (5-7 times/week) % (95%CI)
<i>Traditional diets</i>	38.0 (33.5-42.5)	61.3(56.6-65.7)	0.6 (0.0-1.5)

N: Sample Size of the study; CI: Confidence Interval

III.1.4.1. Mean Consumption frequency of the various food groups in the study population

In this study population, cereals and tubers food group were the most frequently consumed (6.29 ± 0.06), followed by fats and oils group; vegetables; pulses; meat, fish and eggs; then sugar group; milk and dairy products and finally fruits group (2.21 ± 0.11).

Table 16 : Mean frequency of consumption of various food groups in the study population

Food group	Mean	SEM
Cereals and tubers group	6.29	0.06
Pulses group	3.33	0.09
Milk and Dairy products group	2.77	0.18
Meat, fish and eggs group	3.32	0.12
Vegetables group	3.67	0.10
Fruits group	2.21	0.11
Fats and oils group	4.57	0.14
Sugar group	2.83	0.10

SEM: Standard Error of Mean

The table below presents the mean frequency intakes of various food groups by region of residence in the study population. In the Center region, it can be noted that most participants had a low mean intake in a week of milk and dairy products, meat, fish and egg, fruits and sugar food groups in a week.

In the littoral, most women had a low mean intake of milk and dairy products, meat, fish and egg, and fruits food groups in a week.

A low mean weekly intake of milk and dairy products, meat, fish and egg, fruits, vegetables and sugar food groups were observed in the western region.

Meanwhile in the NorthWest region, most subjects had a low mean weekly intake of milk and dairy products, fruits and sugar food groups while in the Far-North region it was observed that women in this region had a low weekly mean intake of pulses, milk and dairy products, meat, fish and egg, fruits and sugar food groups.

Table 17: Mean frequency of consumption of various food groups in a week in the study population by Region of Residence

Regions	Center Region	Littoral Region	West Region	NorthWest Region	Far-North Region
Food group					
Cereals and Tubers group	6.37±0.27	5.92±0.19	6.37±0.10	6.35±0.09	6.73±0.27
Pulses group	3.13±0.39	3.09±0.24	3.05±0.15	3.73±0.13	2.15±0.29
Milk and Dairy products	2.00±0.40	2.00±0.43	2.89±0.44	2.88±0.22	1.50±0.50
Meat, Fish and Egg group	2.86±0.80	1.81±0.22	2.95±0.19	3.93±0.17	1.75±0.47
Vegetables group	5.25±0.47	3.76±0.29	2.98±0.16	3.81±0.14	4.77±0.77
Fruits group	2.24±0.51	2.32±0.34	2.42±0.23	2.11±0.11	1.95±0.45
Fats and oil group	5.01±0.30	4.20±0.41	5.45±0.19	4.03±0.18	4.15±0.12
Sugar group	2.37±0.43	3.52±0.29	2.91±0.18	2.59±0.15	2.75±0.49

III.1.5. Nutritional status of women of childbearing age

III.1.5.1. Nutritional status of women of childbearing age in the study population

The overall mean calcium concentration (10.05±0.37) mg/dl and mean BMI (29.28±0.30) Kg/m² were significantly different (p<0.05) across all age groups, with the 41-49 years age group presenting the highest mean values. Whereas, the mean iron and magnesium concentrations, and mean Ca/Mg ratio were not significantly different across the various age groups.

Table 18: Mean plasma levels of Iron, Magnesium, calcium and BMI in the overall population and by age group

Parameters	Overall mean (n=608)	14-20 years (n=63)	21-30 years (n=141)	31-40 years (n=209)	41-49 years (n=195)	P-value
Iron conc. (µg/dl)n=504	174.40±8.31	142.09±28.74a	156.14±13.02a	179.89±14.13a	191.74±16.76a	0.256
Magnesium conc. (mg/dl)n=589	3.96±0.12	3.38±0.26a	4.00±0.22a	4.18±3.27a	3.96±0.11a	0.276
Calcium conc. (mg/dl)n=547	10.05±0.37	6.86±0.96a	9.66±0.71b,e	10.58±0.58c,e,f	10.84±0.75d,e,f	0.018
Ca-Mg ratio n=537	3.93±0.36	2.30±0.40a	3.62±0.54a	4.21±0.58a	4.39±0.87a	0.406
BMI (Kg/m²)	29.28±0.30	24.26±0.50a	27.92±0.66b,d	30.35±0.55c,e,g	30.66±0.48d,f,g	0.000

The prevalence of iron deficiency (ID) was 11.5%, that of Magnesium was 22.4% and Calcium was 48.3%. Only 2.1% of the women were underweight but up to 30.8% of them were overweight and 38.5% were obese among the 608 women included in this part of the study.

Table 19: Frequencies of various nutritional states in the study population

Parameters	Frequency	Percentage (%)	95% CI
Iron deficient(<30ug/dl)	58	11.5	9.1-14.3
Not Iron deficient	446	88.5	85.7-91.3
Magnesium deficiency(<1.7mg/dl)	132	22.4	18.9-26.0
Not Magnesium deficient	457	77.4	74.2-81.2
Calcium deficient (<8.5mg/dl)	264	48.3	43.9-52.5
Not Calcium deficient	283	51.7	47.7-55.8
Underweight(BMI<18.5Kg/m²)	13	2.1	1.2-3.5
Normal (18.5-24.9)	174	28.6	25.3-32.2
Overweight(BMI ≥ 25 kg/m²)	187	30.8	27.1-34.5
Obesity(BMI ≥ 30 kg/m²)	234	38.5	35.0-42.4

CI: Confidence Interval

III.1.5.2. Prevalence poor nutritional status of women of childbearing age with respect to age

The prevalence of iron and magnesium deficiencies were highest in the 31-40 years age group (3.8% and 7.6 respectively). Calcium deficiency was highest among women in the 41-49 years age group and this was 15.7% ($p<0.05$). Underweight was highest among women in the 21-30 years age group (0.8%) ($p<0.05$). The prevalence of overweight was highest in the 31-40 years age group (11.7%) while that of obesity was highest in the 41-49 years age group (16.1%) ($p<0.05$).

Table 20: Prevalence of poor nutritional status with respect to age in the study population

Parameters	14-20 years n (%)	21-30 years n (%)	31-40 years n (%)	41-49 years n (%)	P-value	Total
Iron deficiency	8(1.6)	14(2.8)	19(3.8)	17(3.4)	0.547	58(11.5)
Magnesium deficiency	18(3.1)	27(4.6)	45(7.6)	42(7.1)	0.524	132(22.4)
Calcium deficiency	37(6.8)	58(10.6)	83(15.2)	86(15.7)	0.020	264(48.3)
Underweight	2(0.3)	5(0.8)	2(0.3)	4(0.7)	0.557	13(2.1)
Overweight	12(2.0)	43(7.1)	71(11.7)	61(10.0)	0.000	187(30.8)
Obesity	6(1.0)	39(6.4)	91(15.0)	98(16.1)	0.000	234(38.5)

n: number of participants in the group; %: percentage

III.1.5.3. Prevalence of poor nutritional status in the study population by frequency of consumption of various food groups

The assessment of the prevalence of the various forms of malnutrition in association to the frequency of intake of various food groups in a week revealed that, a higher prevalence of Mg, Fe and Ca deficiencies, underweight, overweight and obesity were all associated to a high intake of cereals and tubers in a week ($p < 0.05$). A low intake of pluses (beans, nuts...) in a week whereas, was associated to a higher prevalence of Mg deficiency (30.5%), a significantly higher prevalence of Fe deficiency (51.1%), Ca deficiency (39.0%) and obesity (42.6%) ($p < 0.05$). A low intake of milk and dairy products in a week was significantly associated to higher rates of Mg deficiency (53.3%), Ca deficiency (57.1%) and overweight (58.3%) ($p < 0.05$).

A low intake of meat, fish and egg food group was associated to a higher level of Fe (41.4%) and Ca (42.3%) deficiencies although not statistically significant ($p > 0.05$) and a significantly higher rate of overweight (44.8%) ($p < 0.05$).

A higher prevalence of all the forms of malnutrition was associated to a low intake of vegetables in a week although not statistically significant, and a low intake of fruits in a week, with $p < 0.05$ between various intake categories for all forms of malnutrition except Fe deficiency.

A high intake of fats and oils (5 to 7 times) in a week was associated to a significantly higher prevalence of Mg deficiency (50.5%), Fe deficiency (70.0%), Ca deficiency (54.5%), overweight (54.5%) and obesity (63.2%) compared to the other

intake categories ($p < 0.05$). Whereas a low intake of sugar foods (1 to 2 times) in a week was instead associated to a significantly higher prevalence of these forms of malnutrition.

Table 21: Prevalence of poor nutritional status with respect to frequency of intake of various food groups

Food groups	Magnesium deficient	Iron deficient	Calcium deficient	Underweight	Overweight	Obesity
Cereals and tubers (1-2times/week) (3-4 times/week) (5-7 times/week)	P=0.000 5(4.0) 8(6.3) 113(89.7)	P=0.000 1(2.0) 2(4.1) 46(93.9)	P=0.000 12(5.4) 18(8.1) 192(86.5)	P=0.002 1(7.7) 0(0.0) 12(92.3)	P=0.000 4(2.6) 18(11.7) 132(85.7)	P=0.000 8(4.2) 15(7.9) 168(88.0)
Pulses (1-2times/week) (3-4 times/week) (5-7 times/week)	P=0.064 36(30.5) 51(43.2) 31(26.3)	P=0.031 23(51.1) 13(28.9) 9(20.0)	P=0.032 80(39.0) 74(36.1) 51(24.9)	P=0.039 8(66.7) 1(8.3) 3(25.0)	P=0.305 56(38.9) 47(32.6) 41(28.5)	P=0.001 72(42.6) 63(37.3) 34(20.1)
Milk and Dairy products (1-2times/week) (3-4 times/week) (5-7 times/week)	P=0.011 24(53.3) 13(28.9) 8(17.8)	P=0.174 7(58.3) 2(16.7) 3(25.0)	P=0.000 32(57.1) 16(28.6) 8(14.3)	P=/ 1(100.0) 0(0.0) 0(0.0)	P=0.005 14(58.3) 9(37.5) 1(4.2)	P=0.148 16(48.5) 10(30.3) 7(21.2)
Meat, fish and eggs (1-2times/week) (3-4 times/week) (5-7 times/week)	P=0.250 24(25.8) 32(34.4) 37(39.8)	P=0.639 12(41.4) 9(31.0) 8(27.6)	P=0.059 66(42.3) 45(28.8) 45(28.8)	P=0.097 1(11.1) 6(66.7) 2(22.2)	P=0.040 43(44.8) 30(31.2) 23(24.0)	P=0.132 46(41.8) 29(26.4) 35(31.8)
Vegetables (1-2times/week) (3-4 times/week) (5-7 times/week)	P=0.741 37(31.9) 43(37.1) 36(31.0)	P=0.126 21(47.7) 12(27.3) 11(25.0)	P=0.238 74(37.6) 55(27.9) 68(34.5)	P=0.670 4(40.0) 2(20.0) 4(40.0)	P=0.461 51(38.3) 40(30.1) 42(31.6)	P=0.077 63(40.4) 40(25.6) 53(34.0)
Fruits (1-2times/week) (3-4 times/week) (5-7 times/week)	P=0.000 59(67.0) 18(20.5) 11(12.5)	P=0.102 16(66.7) 8(33.3) 0(0.0)	P=0.000 88(73.3) 22(18.3) 10(8.3)	P=0.020 8(88.9) 0(0.0) 1(11.1)	P=0.000 45(68.2) 18(27.3) 3(4.5)	P=0.000 48(62.3) 17(22.1) 12(15.6)
Fats and oils (1-2times/week) (3-4 times/week) (5-7 times/week)	P=0.000 40(36.7) 14(12.8) 55(50.5)	P=0.000 6(20.0) 3(10.0) 21(70.0)	P=0.000 56(36.4) 14(9.1) 84(54.5)	P=0.102 5(71.4) 1(14.3) 1(14.3)	P=0.000 29(33.0) 11(12.5) 48(54.5)	P=0.000 24(25.3) 11(11.6) 60(63.2)
Sugar (1-2times/week) (3-4 times/week) (5-7 times/week)	P=0.000 50(54.3) 25(27.2) 17(18.5)	P=0.000 24(68.6) 3(8.6) 8(22.9)	P=0.000 95(61.7) 33(21.4) 26(16.9)	P=0.695 3(27.3) 5(45.5) 3(27.3)	P=0.001 50(49.5) 32(31.7) 19(18.8)	P=0.000 91(67.4) 22(16.3) 22(16.3)

1-2times/week: Low intake per week; 3-4 times/week: Medium intake per week; 5-7 times/week: High intake per week

III.1.6. Correlation between frequency of consumption of the various food groups with anthropometric parameters and nutrient concentration

Bivariate correlation analysis revealed that there was a negative but strong relation between BMI and frequency of intake of sugar food group in a week ($r = -0.108$, $p = 0.044$). Plasma magnesium (Mg) concentration was negatively but strongly associated to pulses food group ($r = -0.160$, $p = 0.001$) and meat, fish and egg food group intake in a week ($r = -0.190$, $p = 0.001$). Calcium concentration was also negatively but strongly

correlated to meat, fish and egg intake ($r=-0.124$, $p=0.040$) but positively and strongly correlated to the frequency of intake of traditional diet ($r=0.129$, $p=0.008$) in a week. Plasma iron (Fe) concentration was negatively but strongly associated to cereals and tubers intake in a week ($r=-0.109$, $p=0.026$), which means that plasma Fe concentration decreased as intake of this food group increased in a week. Ca-Mg ratio ($r=0.117$, $p=0.038$) was strongly and positively correlated to the frequency of intake of sugar in a week.

Table 22: Pearson's Correlation coefficient (rho (r)) between frequency of consumption of the various food groups with anthropometric parameters and nutrient concentration

Parameters	BMI Pearson's r (P-value)	Fe concentration Pearson's r (P- value)	Mg concentration Pearson's r (P- value)	Ca concentration Pearson's r (P-value)	Ca-Mg ratio Pearson's r (P-value)
Cereals and tubers frequency	0.017 (0.707)	-0.109 (0.026)*	-0.041 (0.369)	-0.010 (0.834)	0.013 (0.785)
Pulses and Beans frequency	-0.038 (0.419)	0.076 (0.139)	-0.160 (0.001)**	-0.077 (0.121)	0.018 (0.719)
Milk and Dairy products frequency	0.156 (0.120)	-0.048 (0.671)	-0.102 (0.330)	0.032 (0.768)	-0.026 (0.817)
Meat, fish and eggs frequency	0.038 (0.504)	-0.031 (0.622)	-0.190 (0.001)**	-0.124 (0.040)*	0.049 (0.432)
Vegetables frequency	0.002 (0.967)	0.023 (0.671)	-0.049 (0.319)	-0.097 (0.056)	-0.079 (0.124)
Fruits frequency	0.042 (0.528)	-0.021 (0.781)	-0.020 (0.767)	0.043 (0.549)	0.005 (0.950)
Fats and oils frequency	0.018 (0.352)	-0.119 (0.066)	0.097 (0.110)	0.100 (0.110)	0.032 (0.619)
Sugar frequency	-0.108 (0.044)*	0.048 (0.415)	0.018 (0.742)	0.107 (0.057)	0.117 (0.038)*
Traditional diets frequency	0.066 (0.159)	-0.001 (0.979)	0.091 (0.055)	0.129 (0.008)**	-0.013 (0.787)

Significance level: ** $p<0.01$, * $p<0.05$
0.000-0.009= very weak to no correlation
0.010-0.090= weak;
0.100-0.900= strong;
1.0=very strong correlation;
- Negative correlation; + positive correlation

III.1.7. Nutritional states in the study population by some sociodemographic, socioeconomic and nutritional parameters

III.1.7.1. Prevalence of poor weight states in the study population by Region of residence

The centre region regarded no underweight individual but had a high rate of obese and overweight participants ($p < 0.05$). In the littoral no woman was underweight but a high percentage (43.7%) was obese and 31.1% were overweight. The western region recorded the highest rate of obese women (45.3%) followed by over weights which were 33.1%. 1.7 % of the women in this region were underweight ($p < 0.05$).

In the Northwest region most participants were instead overweight (28.8%), followed by obese (28.3%) and 4.1% of women from this region were underweight ($p < 0.05$).

In the Far-North region, most were obese, followed by overweights then underweight women, this difference was statistically significant ($p < 0.05$).

Table 23: Abnormal Weight status with respect by Region of residence

Regions	Underweight (n) %	Overweight (n)%	Obese (n)%	P-value
Center Region	(0)0.0%	(22)30.6 %	(35)48.6%	0.001
Littoral Region	(0)0.0%	(37)31.1 %	(52)43.7 %	0.116
Western Region	(3)1.7%	(57)33.1%	(78) 45.3%	0.002
NorthWest region	(9) 4.1%	(63) 28.8%	(62)28.3%	0.000
Far-North region	(1)3.8%	(8) 30.8%	(7) 26.9%	0.752

n: number of participants in the group; %: percentage

III.1.7.2. Mineral status in the study population by Region of residence

With regards to mineral status, in the centre region, most women were Calcium deficient, none of them were Magnesium deficient and 10.3 % were Iron deficient compared to non Iron deficient women (89.7%) ($p < 0.05$).

In the Littoral region, most women were Calcium deficient (37.4 %) 3.6% were Iron deficient compared to 96.4 who were not and 2.5% were Magnesium deficient compared to 97.5% who were not Magnesium deficient ($p < 0.05$).

While in the Western region, most of them were Calcium deficient (32.1%), 23.4% were Magnesium deficient and 8.4% Iron deficient. Most women were Calcium deficient (74.3%).

In the Northwest region compared to non Calcium deficient women (25.7%) ($p < 0.05$). While 43.5% the women in this region were Magnesium deficient compared to 56.5% who were non deficient for this mineral ($p < 0.05$) and 19.3 % were Iron deficient.

In the Far-North region, 19.0 % were Iron deficient, no women were Magnesium deficient in this region and 16.7% were Calcium deficient. These results were significant compared to non-mineral deficient women ($p < 0.05$) for Iron and Calcium status.

Table 24: Mineral status with respect by Region of residence

Mineral Status Regions	Iron Status		Magnesium Status		Calcium Status	
	Iron deficient n (%)	Not Iron deficient n (%)	Magnesium deficient n (%)	Not Magnesium deficient n (%)	Calcium deficient n (%)	Not Calcium deficient n (%)
Center Region	6(10.3%)a	52(89.7%)b	0(0.0%)	72(100.0%)	28(40.6%)a	41(59.4%)a
Littoral Region	4(3.6%)a	106 (96.4%)a	3(2.5%)a	116(97.5%)b	40(37.4%)a	67(62.6%)b
Western Region	13 (8.4%)a	141 (91.6%)a	39(23.4%)a	128(76.6%)a	50 (32.1%)a	106 (67.9%)a
North West Region	31(19.3%)a	130(80.7%)a	90(43.5%)a	117 (56.5%)b	142(74.3%)a	49(25.7%)b
Far- North Region	4(19.0%)a	17(81.0%)b	0(0.0%)a	24 (25.0%)a	4(16.7%)a	20(83.3%)b

n: number of participants in the group; %: percentage

Different letters (a, b): significant difference between deficient and non deficient participants in that mineral status category

Same letters (a, a; b, b): no significant difference between deficient and non deficient participants in that mineral status category

III.1.7.3. Prevalence of poor nutritional status with respect to some socioeconomic and nutritional parameters

This study showed that Illiterates recorded the highest prevalence of Mg (25.8%) and Ca (22.3%) deficiencies and these were statistically significant ($p<0.05$). Fe deficiency was highest among those with a first cycle (27.6%). Overweight (26.2%) and obesity (27.4%) were highest among women with a primary education and these were statistically significant ($p<0.05$).

Housewives and married women recorded the highest prevalence of all the forms of malnutrition when the profession and the marital status were considered. The differences between the rates were statistically significant for almost all the forms malnutrition ($p<0.05$).

With respect to socioeconomic status, all the forms of malnutrition were most prevalent in high SES households ($p<0.001$), except for the prevalence of underweight which was inexistence in high SES households.

All the forms of malnutrition except iron deficiency (ID) were highest among participants of the study population with an acceptable food consumption score (FCS) and these differences were significant for magnesium and calcium deficiency rates ($p<0.001$). Participants of the study in food secure households presented the highest prevalence of all the forms of malnutrition. This difference was significant for Calcium, Magnesium, Iron deficiencies and obesity ($p<0.05$)

A moderate intake of traditional diets in a week were associated to higher prevalence of ID, Calcium deficiency, overweight and obesity ($p<0.001$). Magnesium deficiency was highest among those with a low intake ($p<0.001$).

Table 25: Prevalence of poor nutritional status with respect to subjects' socioeconomic and nutritional parameters

Parameters	Magnesium deficient	Iron deficient	Calcium deficient	Underweight	Overweight	Obesity
Educational level	P=0.000	P=0.127	P=0.000	P=0.846	P=0.000	P=0.000
<i>Illiterate</i>	34(25.8)	13(22.4)	59(22.3)	3(23.1)	35(18.7)	51(21.8)
<i>Literates</i>	23(17.4)	9(15.5)	45(17.0)	2(15.4)	26(13.9)	28(12.0)
<i>Primary</i>	31(23.5)	15(25.9)	57(21.6)	4(30.8)	49(26.2)	64(27.4)
<i>First cycle</i>	29(22.0)	16(27.6)	58(22.0)	4(30.8)	37(19.8)	36(15.4)
<i>Second cycle</i>	14(10.6)	5(8.6)	43(16.3)	0(0.0)	34(18.2)	49(20.9)
<i>University</i>	1(0.8)	0(0.0)	2(0.8)	0(0.0)	6(3.2)	6(2.6)
Profession	P=0.014	P=0.202	P=0.000	P=0.123	P=0.000	P=0.000
<i>Student</i>	17(13.0)	14(25.0)	53(20.9)	2(15.4)	25(13.9)	14(6.3)
<i>Employed</i>	30(22.9)	9(16.1)	43(17.0)	2(15.4)	37(20.6)	48(21.5)
<i>Odd jobs/farmers</i>	33(25.2)	8(14.3)	58(22.9)	2(15.4)	32(17.8)	37(16.6)
<i>Business</i>	16(12.2)	8(14.3)	15(5.9)	0(0.0)	17(9.4)	27(12.1)
<i>Housewives</i>	35(26.7)	17(30.4)	84(33.2)	7(53.8)	69(38.3)	97(43.5)
Marital Status	P=0.000	P=0.000	P=0.000	P=0.058	P=0.000	P=0.000
<i>Single</i>	9(6.8)	10(17.2)	21(8.0)	1(7.7)	29(15.5)	18(7.7)
<i>Married</i>	98(74.2)	36(62.1)	186(70.5)	8(61.5)	129(69.0)	180(76.9)
<i>Widow/Divorced</i>	25(18.9)	12(20.7)	57(21.6)	4(30.8)	29(15.5)	36(15.4)
SES	P=0.000	P=0.000	P=0.000	P=0.000	P=0.000	P=0.000
<i>Low</i>	1(0.9)	1(2.2)	3(1.4)	1(9.1)	5(3.2)	5(2.6)
<i>Medium</i>	2(1.8)	8(17.8)	21(9.8)	10(90.9)	21(13.5)	35(18.2)
<i>High</i>	106(97.2)	36(80.0)	190(88.8)	0(0.0)	130(83.3)	152(79.2)
FCS	P=0.000	P=0.799	P=0.000	P=0.116	P=0.313	P=0.311
<i>Poor</i>	13(9.8)	17(29.3)	74(28.0)	1(7.7)	59(31.6)	83(35.5)
<i>Borderline</i>	28(21.2)	21(36.2)	71(26.9)	5(38.5)	56(29.9)	67(28.6)
<i>Acceptable</i>	91(68.9)	20(34.5)	119(45.1)	7(53.8)	72(38.5)	84(35.9)
HFIAS	P=0.000	P=0.000	P=0.000	P=0.695	P=0.093	P=0.001
Food secure	75(58.1)	23(67.6)	100(54.6)	5(45.5)	42(43.8)	54(50.0)
Mildly food insecure	18(14.0)	7(20.6)	35(19.1)	3(27.3)	28(29.2)	25(23.1)
Moderately and severely food insecure	36(27.9)	4(11.8)	48(26.2)	3(27.3)	26(27.1)	29(26.9)
Traditional diets intake	P=0.000	P=0.000	P=0.000	P=0.132	P=0.000	P=0.000
<i>Low</i>	55(50.9)	17(39.5)	93(46.0)	8(72.7)	47(33.1)	59(34.3)
<i>Moderate</i>	52(48.1)	24(55.8)	108(53.5)	3(27.3)	95(66.9)	111(64.5)
<i>High</i>	1(0.9)	2(4.7)	1(0.5)	0(0.0)	0(0.0)	2(1.2)

SES: Socioeconomic status; FCS: Food Consumption Score; HFIAS: Household Food Insecurity Access Scale

III.1.8. Prevalence of Multiple mineral deficiencies

III.1.8.1. Prevalence of the coexistence of multiple mineral deficiencies

Most women in the study population had no mineral deficiency (45.4%) while up to 16.8% of them had two deficiencies and up to 1.6% of them had three deficiencies.

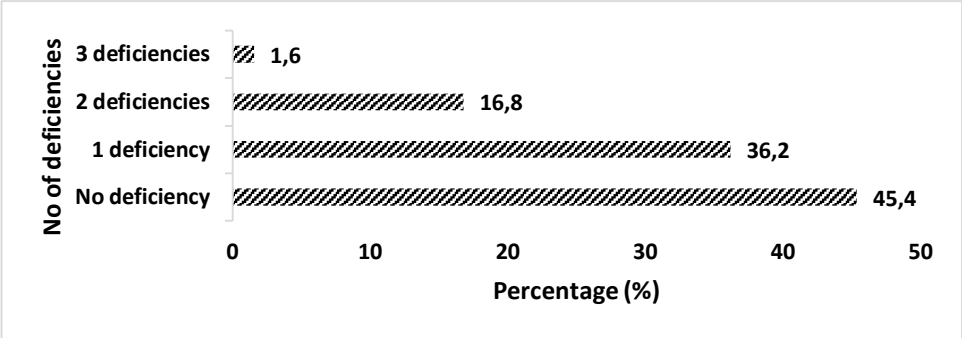


Figure 12: Coexistence of multiple mineral deficiencies in the study population

3 deficiencies: participants having all three mineral deficiencies; **2 deficiencies:** participants having two mineral deficiencies; **1 deficiency:** participants having one mineral deficiency; **No deficiency:** participants with no mineral deficiencies

III.1.8.1.1. Prevalence of the coexistence of multiple mineral deficiencies in the study population by area/region of residence

The figure below shows the presence of multiple mineral deficiencies among the study participants with respect to their area or region of residence. The western region recorded the highest percentage (14.5%) of participants with no mineral deficiencies followed by the littoral region. The northwest region recorded the highest percentages of participants with one (12.7%) and two (12.8%) mineral deficiencies followed by the western region. Individuals with three mineral deficiencies were found only in the northwest region and they represented 1.6 percent of the total population.

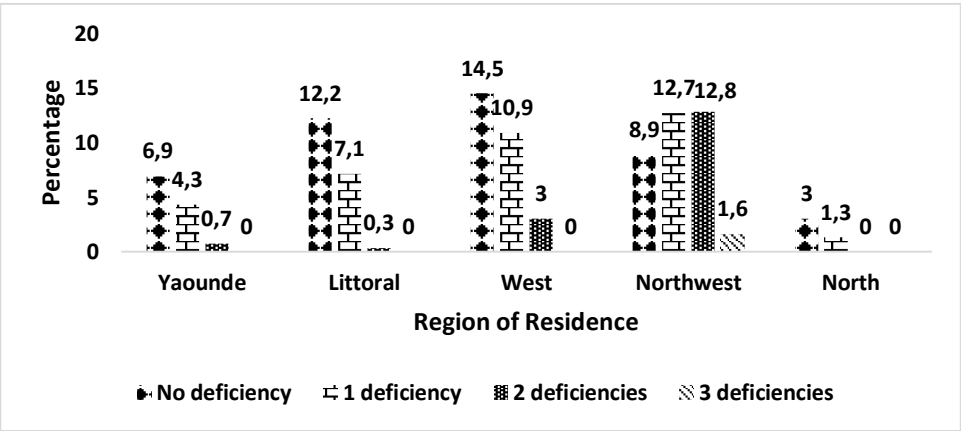


Figure 13: Coexistence of multiple mineral deficiencies in the study population by area/region of residence

3 deficiencies: participants having all three mineral deficiencies; **2 deficiencies:** participants having two mineral deficiencies; **1 deficiency:** participants having one mineral deficiency; **No deficiency:** participants with no mineral deficiencies

III.1.8.1.2. Prevalence of the coexistence of multiple mineral deficiencies in the study population with respect to age group

In the study population, the 31-40 years age group recorded the highest prevalence of women with no deficiencies (16.8%), the 41-49 years age group recorded the highest prevalence of those with one deficiency (12.0%). Also, the 41-49 years age group recorded the highest prevalence of those with 2 deficiencies (5.4%) and the highest prevalence of those with three deficiencies was among those in the 31-40 years age group.

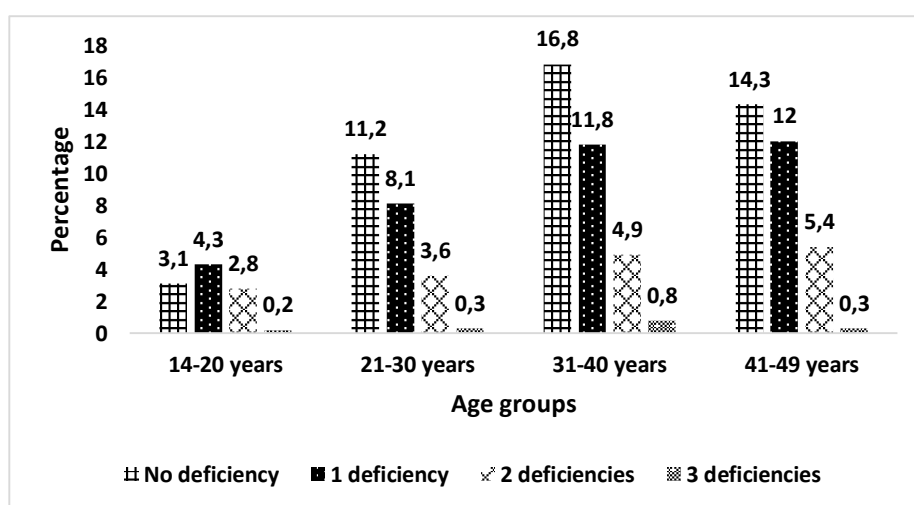


Figure 14: Coexistence of multiple mineral deficiencies in the study population with respect to age group

3 deficiencies: participants having all three mineral deficiencies; **2 deficiencies:** participants having two mineral deficiencies; **1 deficiency:** participants having one mineral deficiency; **No deficiency:** participants with no mineral deficiencies

III.1.8.2. Prevalence of specific combined mineral deficiencies in the study population

The table below presents the prevalence of combined mineral deficiencies in the study population. It was observed that, 15.1 percent of participants of the study population presented both Ca and Mg deficient status, 4.9 percent were both Ca and Fe deficient, 2.1 percent had both Mg and Fe deficiencies and 1.7 percent of the study population presented all three mineral (Ca, Mg and Fe) deficiencies in the 95 percent confidence interval.

Table 26: Prevalence of Ca/Mg, Mg/Fe, Ca/Fe and Ca/Mg/Fe deficiencies in the study population

	Mineral status	Frequency (%)	95% CI
Ca/Mg status	Ca/Mg deficient	90 (15.1)	(12.1-17.8)
	Not Ca/Mg deficient	506 (84.9)	(82.2-87.8)
Mg/Fe status	Mg/Fe deficient	13(2.1)	(1.2-3.3)
	Not Mg/Fe deficient	594(97.9)	(96.7-98.8)
Ca/Fe status	Ca/Fe deficient	29 (4.9)	(3.2-6.7)
	Not Ca/Fe deficient	567 (95.1)	(93.3-96.8)
Ca/Mg/Fe status	Ca/Mg/Fe deficient	10(1.7)	(0.7-2.8)
	Not Ca/Mg/Fe deficient	591(98.3)	(97.2-99.3)

Ca/Mg status: Combined Calcium and Magnesium status; **Mg/Fe status:** Combined Magnesium and Iron status; **Ca/Fe status:** Combined Calcium and Iron status; **Ca/Mg/Fe status:** Combined Calcium, Magnesium and Iron status; **Ca/Mg deficient:** Combined Calcium and Magnesium deficiencies; **Mg/Fe deficient:** Combined Magnesium and Iron deficiencies; **Ca/Fe deficient:** Combined Calcium and Iron deficiencies; **Ca/Mg/Fe deficient:** All three mineral deficiencies; %: percentage; CI: Confidence interval

III.1.8.2.1. Prevalence of specific combined mineral deficiencies in the study population by age groups

The 31-40 years age group presented the highest prevalence of those who were both Ca and Mg deficient (4.9 percent), both Mg and Fe deficient (1.2 percent) and also the highest prevalence of those with all three mineral deficiencies (0.8 percent) ($p>0.05$). The 31-40 years age group and the 21-30 years age group recorded the highest percentage of those with both Ca and Fe deficiencies (1.5 percent respectively) ($p>0.05$).

Table 27: Prevalence of Ca/Mg, Ca/Fe, Mg/Fe and Ca/Mg/Fe deficiencies in the study population by age groups

Mineral deficiencies	14-20 years	21-30 years	31-40 years	41-49 years	P-value
Ca/Mg deficiencies	16(2.7)	17(2.9)	29(4.9)	28(4.7)	0.066
Ca/Fe deficiencies	3(0.5)	9(1.5)	9(1.5)	8(1.3)	0.730
Mg/Fe deficiencies	1(0.2)	2(0.3)	7(1.2)	3(0.5)	0.518
Ca/Mg/Fe deficiencies	1(0.2)	2(0.3)	5(0.8)	2(0.3)	0.758

Ca/Mg deficiencies: Combined Calcium and Magnesium deficiencies; **Ca/Fe deficiencies:** Combined Calcium and Iron deficiencies; **Mg/Fe deficiencies:** Combined Magnesium and Iron deficiencies; **Ca/Mg/Fe deficiencies:** All three mineral deficiencies

III.1.8.2.2. Prevalence of specific combined mineral deficiencies in the study population by region of residence

The table below presents the prevalence of various combined mineral deficiencies in the study population by region of residence. It can be inferred that, the North-west region recorded the highest prevalence of those who were both Ca and Mg deficient (12.8%); Mg and Fe deficient (2.0%) and, Ca and Fe deficient (3.4%) ($p < 0.05$). Also, this region was the only area which recorded a prevalence of participants presenting all three mineral deficiencies and this prevalence was 1.7 percent ($p < 0.000$).

Table 28: Prevalence of Ca/Mg, Ca/Fe, Mg/Fe and Ca/Mg/Fe deficiencies in the study population by region of residence

Mineral deficiencies	Center	Littoral	West	North west	North	P-value
Ca/Mg deficiencies	0(0.0)	2(0.3)	12(2.0)	76(12.8)	0(0.0)	0.000
Ca/Fe deficiencies	4(0.7)	0(0.0)	5(0.8)	20(3.4)	0(0.0)	0.001
Mg/Fe deficiencies	0(0.0)	0(0.0)	1(0.2)	12(2.0)	0(0.0)	0.001
Ca/Mg/Fe deficiencies	0(0.0)	0(0.0)	0(0.0)	10(1.7)	0(0.0)	0.001

Ca/Mg deficiencies: Combined Calcium and Magnesium deficiencies; **Ca/Fe deficiencies:** Combined Calcium and Iron deficiencies; **Mg/Fe deficiencies:** Combined Magnesium and Iron deficiencies; **Ca/Mg/Fe deficiencies:** All three mineral deficiencies

The mean plasma Ca-Mg ratio was significantly higher among participants of the study population with a Magnesium deficient state compared to those who were not Magnesium deficient while with respect to Calcium status, this ratio was higher among those who were not Calcium deficient compared to those who were deficient for this mineral ($p < 0.001$). Participants who were not Ca/Mg deficient had a higher mean Ca-Mg ratio compared to those who were ($p > 0.05$) in the 95 % confidence interval (CI).

Table 29: Mean plasma Ca-Mg ratio with respect to Mg, Ca and Ca/Mg status

	Mineral status	Mean±SEM	95% CI
Magnesium status	Magnesium deficient (n=118)	8.03±1.57a	(4.91-11.14)
	Not Mg deficient (n=419)	2.78±0.11b	(2.56-3.00)
Calcium status	Ca deficient (n=257)	1.62±0.23a	(1.15-2.09)
	Not Ca deficient (n=280)	6.05±0.64b	(4.78-7.32)
Ca/Mg status	Ca/Mg deficient (n=90)	2.66±0.65a	(1.36-3.96)
	Not Ca/Mg deficient (n=447)	4.19±0.42a	(3.36-5.02)

Ca/Mg status: Combined Calcium and Magnesium statuses; **SEM:** Standard Error of Mean; **CI:** Confident Interval;

In the same line, values with different letters (a and b) are significantly different at $p < 0.001$ and those with same letters (a and a) are not significantly different at $p > 0.05$.

III.1.8.3. Correlation between plasma mineral concentrations in the study population

Calcium plasma level was strong and positively correlated to Mg plasma level and Ca-Mg ratio in the plasma ($p < 0.01$). The same was observed for Mg with regards to Ca plasma level but this mineral was strongly but negatively correlated to plasma Ca-Mg ratio ($p < 0.01$). Whereas, Fe plasma concentration was not correlated to any of the other minerals levels.

Table 30: Pearson's Correlation (rho (r)) between plasma mineral concentrations

Mineral concentration	Calcium concentration	Magnesium concentration
Calcium concentration	1	0.214**
Magnesium concentration	0.214**	1
Iron concentration	0.090	0.030
Ca-Mg ratio	0.301**	-0.247**

* $p < 0,05$; ** $p < 0,01$ for binary correlation

III.1.8.4. Nutritional Predictors of single and combined specific mineral deficiencies

Binary logistical regression analysis revealed that, a low intake compared to a high intake of animal protein sources like fish, meat and eggs was a predictor of Mg deficiency (odd ratio=3.836, $p=0.000$) and combined Ca and Mg deficiencies (odd ratio=2.270, $p=0.022$) among the study participants. Participants living in the center region were 6.453 times more likely of iron deficiency ($p < 0.05$), compared to those living in the north region of Cameroon. Also, all age groups were predictors of Ca deficiency and combined Ca and Mg deficiencies compared to the 14- 20 years age groups.

Table 31: Some sociodemographic parameters and Food consumption frequencies as predictors of Ca, Mg, Fe, Ca/Mg, Ca/Fe and Ca/Mg/Fe deficiencies in the study population

Parameters	Magnesium deficient OR(CI)p-val	Iron deficient OR(CI)p-val	Calcium deficient OR(CI)p-val	Ca/Mg deficiencies OR(CI)p-val	Ca/Fe deficiencies OR(CI)p-val	Mg/Fe deficiencies OR(CI)p-val	Ca/Mg/Fe deficiencies OR(CI)p-val
Age groups	1	1	1	1	1	1	1
14-20 years	1.674(0.837-3.349)0.145	1.778(0.690-4.580)0.233	2.350(1.223-4.518) 0.010	2.283(1.160-5.314)0.019	0.737(0.192-2.824)0.656	1.121(0.100-12.594)0.926	1.169(0.104-13.148)0.899
21-30 years	1.460(0.768-2.776)0.248	1.813(0.735-4.468)0.196	2.557(1.372-4.766) 0.003	2.113(1.060-4.211)0.033	1.164(0.305-4.442)0.824	0.463(0.056-3.837)0.476	0.688(0.079-6.006)0.735
31-40 years	1.485(0.777-2.839)0.232	1.869(0.748-4.674)0.181	1.925(1.026-3.611)0.041	2.030(1.014-4.065)0.046	1.217(0.312-4.741)0.777	1.032(0.105-10.104)0.978	1.619(0.144-18.169)0.696
41-49 years							
Region of residence	1	1	1	1	1	1	1
North	/	2.138(0.535-8.551)0.282	0.312(0.096-1.019)0.054	/	/	/	/
Center	/	6.453(1.463-28.470) 0.014	0.350(0.111-1.102)0.073	/	/	/	/
Littoral	/	2.656(0.769-9.169)0.122	0.454(0.147-1.406)0.171	/	/	/	/
West		1.026(0.317-3.320)0.966	0.072(0.023-0.224) 0.000				
North west							
Cereals and tubers	1	1	1	1	1	1	1
(5-7 times/week)	1.170(0.419-3.268)0.764	2.329(0.302-17.981)0.417	0.552(0.213-1.431)0.221	0.748(0.261-2.140)0.588	0.921(0.115-7.354)0.938	0.468(0.056-3.903)0.468	0.344(0.40-2.974)0.332
(1-2 times/week)	1.737(0.787-3.836)0.172	2.620(0.610-11.250)0.195	1.209(0.632-2.309)0.566	1.731(0.660-4.536)0.265	1.146(0.258-5.091)0.858	1.195(0.150-9.523)0.866	0.873(0.106-7.167)0.899
(3-4 times/week)							
Pulses	1	1	1	1	1	1	1
(5-7 times/week)	1.622(0.932-2.822)0.087	0.751(0.331-1.705)0.494	1.100(0.672-1.800)0.704	1.616(0.859-3.039)0.137	1.074(0.378-3.056)0.893	1.780(0.433-7.313)0.424	2.393(0.524-10.933)0.260
(1-2 times/week)	0.797(0.467-1.363)0.407	1.076(0.438-2.643)0.873	0.878(0.527-1.465)0.619	0.870(0.474-1.595)0.653	1.401(0.437-4.488)0.570	1.383(0.336-5.700)0.654	1.871(0.408-8.572)0.420
(3-4 times/week)							
Milk and dairy products	1	1	1	1	1	1	1
(5-7 times/week)	1.429(0.453-4.501)0.542	1.629(0.356-7.456)0.530	0.792(0.238-2.631)0.703	0.448(0.116-1.728)0.243	1.461(0.195-10.959)0.712	/	/
(1-2 times/week)	1.055(0.293-3.803)0.935	3.150(0.452-21.947)0.247	0.583(0.146-2.323)0.445	0.397(0.090-1.755)0.223	0.981(0.108-8.933)0.986	/	/
(3-4 times/week)							
Meat, fish and eggs	1	1	1	1	1	1	1
(5-7 times/week)	3.836(2.056-7.158)0.000	1.130(0.437-2.924)0.801	1.461(0.812-2.628)0.206	2.270(1.126-4.573)0.022	1.393(0.413-4.693)0.593	0.391(0.042-3.619)0.408	0.531(0.053-5.298)0.590
(1-2 times/week)	1.371(0.735-2.558)0.321	1.055(0.383-2.905)0.918	1.071(0.554-2.074)0.838	1.232(0.610-2.489)0.561	1.546(0.391-6.110)0.534	0.553(0.049-6.293)0.633	1.115(0.068-18.332)0.939
(3-4 times/week)							
Vegetables	1	1	1	1	1	1	1
(5-7 times/week)	1.187(0.699-2.016)0.526	0.581(0.267-1.264)0.171	1.062(0.664-1.699)0.800	1.344(0.750-2.406)0.321	0.377(0.118-1.203)0.099	0.155(0.019-1.283)0.084	0.182(0.022-1.536)0.117
(1-2 times/week)	0.628(0.368-1.072)0.088	0.835(0.351-1.984)0.683	1.005(0.603-1.673)0.986	0.953(0.525-1.728)0.873	0.560(0.154-2.041)0.560	0.200(0.022-1.816)0.153	0.271(0.028-2.6467)0.261
(3-4 times/week)							
Fruits	1	1	1	1	1	1	1
(5-7 times/week)	1.475(0.600-3.622)0.397	/	0.696(0.258-1.879)0.475	1.224(0.460-3.253)0.686	/	/	/
(1-2 times/week)	1.500(0.537-4.188)0.439	/	1.080(0.354-3.289)0.893	1.594(0.513-4.956)0.420	/	/	/
(3-4 times/week)							

Ca/Mg deficiencies: Combined Calcium and Magnesium deficiencies; **Ca/Fe deficiencies:** Combined Calcium and Iron deficiencies; **Mg/Fe deficiencies:** Combined Magnesium and Iron deficiencies; **Ca/Mg/Fe deficiencies:** All three mineral deficiencies; **1-2 times/week:** Low intake per week; **3-4 times/week:** Medium intake per week; **5-7 times/week:** High intake per week; **OR:** Odds ratio; **CI:** Confidence Interval; **p-val:** p-value

In table 32 below, a Ca deficient status was a very weak predictor of Mg deficiency compared to a non deficient Ca status

Table 32: Calcium deficiency as predictors of other mineral deficiencies adjusted for frequent intake of meat, fish and eggs

Parameters	Not Calcium deficient	Odds ratio (95% CI)P
Magnesium deficiency	1	0.345(0.192-0.620)0.000
Iron deficiency	1	0.436(0.163-1.164)0.098

CI: Confidence Interval; P: p-value

In table 33 below, a Mg deficient status was a very weak predictor of Ca deficiency compared to a non Mg deficient status

Table 33: Magnesium deficiency as predictors of other mineral deficiencies adjusted for frequent intake of meat, fish and eggs

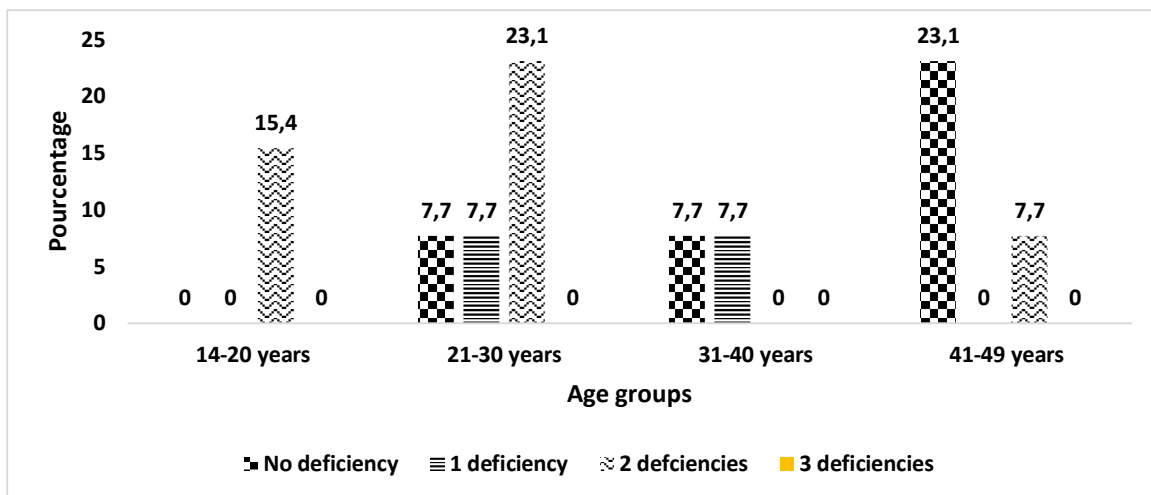
Parameters	Not Magnesium deficient	Odds ratio (95% CI)
Calcium deficiency	1	0.345(0.192-0.620)0.000
Iron deficiency	1	0.796(0.408-1.552)0.503

CI: Confidence Interval; P: p-value

III.1.8.5. Prevalence of multiple mineral deficiencies with respect to abnormal weight status and age group

III.1.8.5.1. Prevalence of multiple mineral deficiencies in underweight participants with respect to age group

Among the underweights, the 41-49 years age group recorded the highest prevalence of those with no deficiencies (23.1%), while the 21-30 years and the 31-40 years age groups recorded the highest prevalence of those with one deficiency, 7.7% respectively. The prevalence of two deficiencies was highest among underweights in the 21-30 years age group (23.1%) while no underweights in any of the age groups recorded three mineral deficiencies.

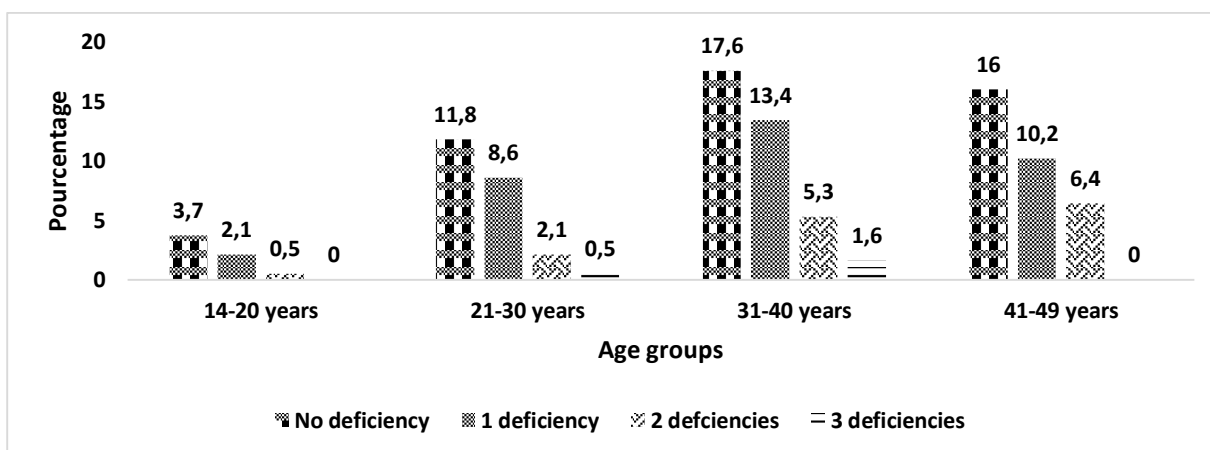


3 deficiencies: participants having all three mineral deficiencies; **2 deficiencies:** participants having two mineral deficiencies; **1 deficiency:** participants having one mineral deficiency; **No deficiency:** participants with no mineral deficiencies

Figure 15: Coexistence of multiple mineral deficiencies in underweight participants with respect to age group

III.1.8.5.2. Prevalence of multiple mineral deficiencies in overweight participants with respect to age group

Among overweight participants, the 31-40 years age group recorded the highest prevalence of those with no mineral deficiencies, those with one mineral deficiency and those with three mineral deficiencies (17.6%, 13.4 and 1.6% respectively). The 41-49 year age group recorded the highest prevalence of overweight women with two deficiencies (6.4%).

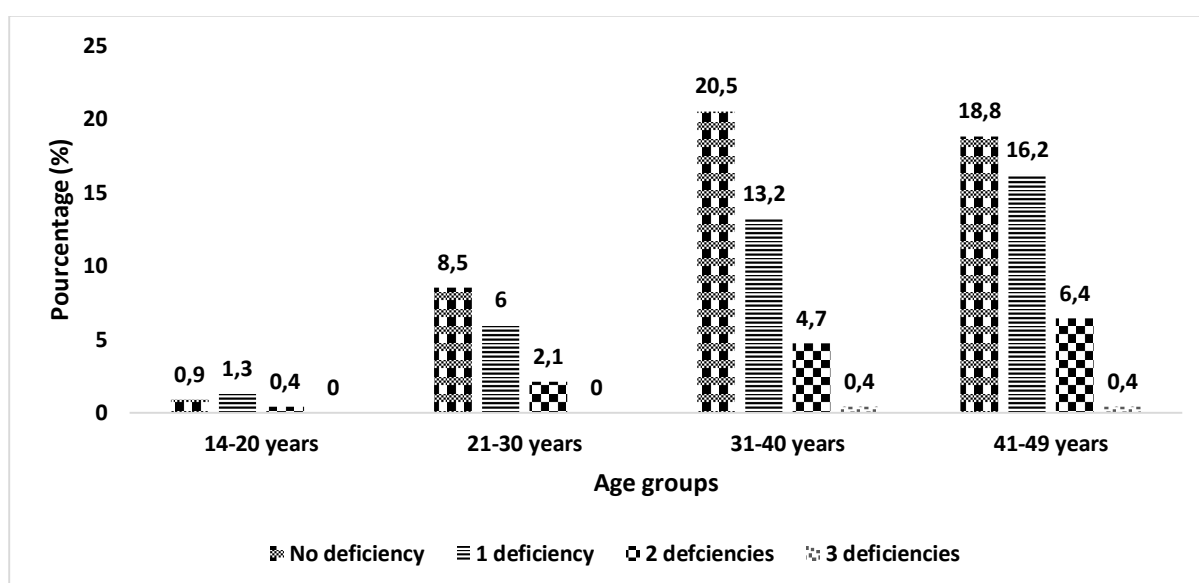


3 deficiencies: participants having all three mineral deficiencies; **2 deficiencies:** participants having two mineral deficiencies; **1 deficiency:** participants having one mineral deficiency; **No deficiency:** participants with no mineral deficiencies

Figure 16: Coexistence of multiple mineral deficiencies in overweight participants with respect to age group

III.1.8.5.3. Prevalence of multiple mineral deficiencies in obese participants with respect to age group

Among obese participants, the 31-40 years age group recorded the highest percentage of women with no mineral deficiencies (20.5%), while the 41-49 years age group recorded the highest prevalence of obese women with one mineral deficiency and obese women with two mineral deficiencies (16.2% and 6.4% respectively). While the 31-40 years and the 41-49 years age group recorded the highest prevalence of obese women with three mineral deficiencies (0.4 % respectively).



3 deficiencies: participants having all three mineral deficiencies; **2 deficiencies:** participants having two mineral deficiencies; **1 deficiency:** participants having one mineral deficiency; **No deficiency:** participants with no mineral deficiencies

Figure 17: Coexistence of multiple mineral deficiencies in obese participants with respect to age group

III.1.9. Prevalence of specific mineral deficiencies with respect to abnormal weight status

III.1.9.1. Prevalence of specific mineral deficiencies and underweight at the individual level

Up to 15.4% of the study population was underweight and iron deficient, 38.5% was underweight and magnesium deficient, and 53.8% was underweight and calcium deficient in the 95% CI.

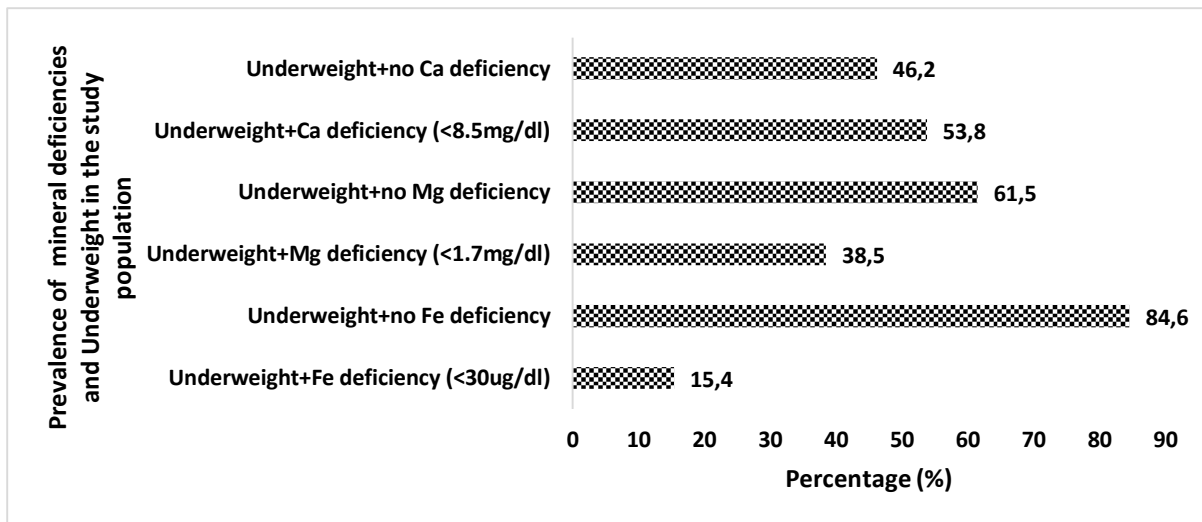


Figure 18: Prevalence of the coexistence of specific mineral deficiencies and underweight at the individual level in the study population

III.1.9.2. Prevalence of specific mineral deficiencies and overweight at the individual level

The assessment of specific mineral deficiencies among overweight participants of the study population, revealed that, 8.6% of overweight were iron deficient, 21.9% of them were magnesium deficient and 39.6% of overweight participants were calcium deficient in the 95% confident interval.

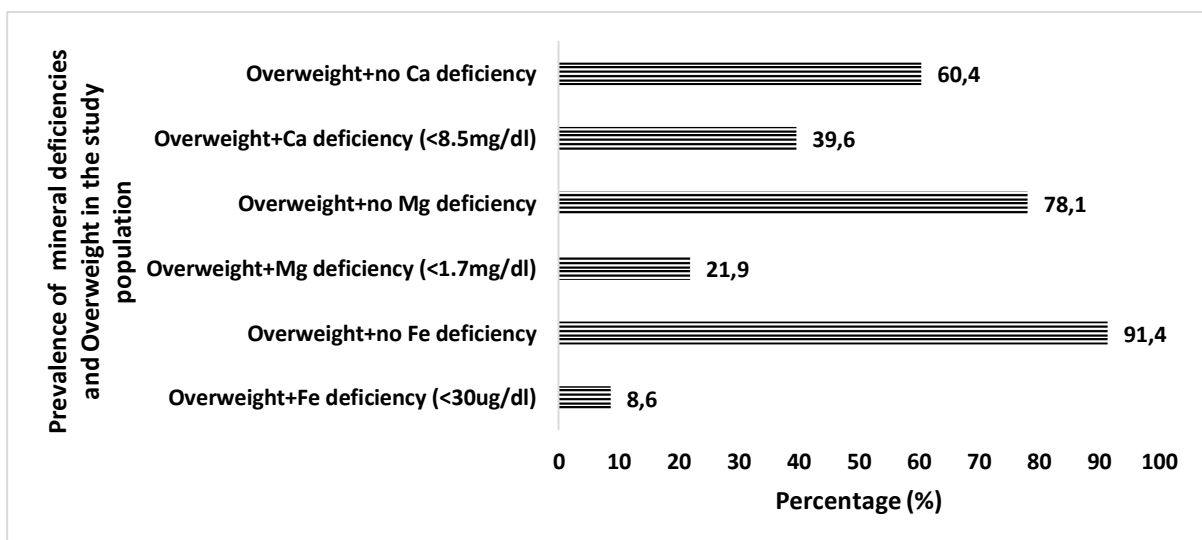


Figure 19: Prevalence of the coexistence of specific mineral deficiencies and overweight at the individual in the study population

III.1.9.3. Prevalence of specific mineral deficiencies and obesity at the individual level

Among obese participants of the study population, it was observed that 7.3% of them were iron deficient, 20.5% were magnesium deficient and 38.9% were calcium deficient in the 95% CI.

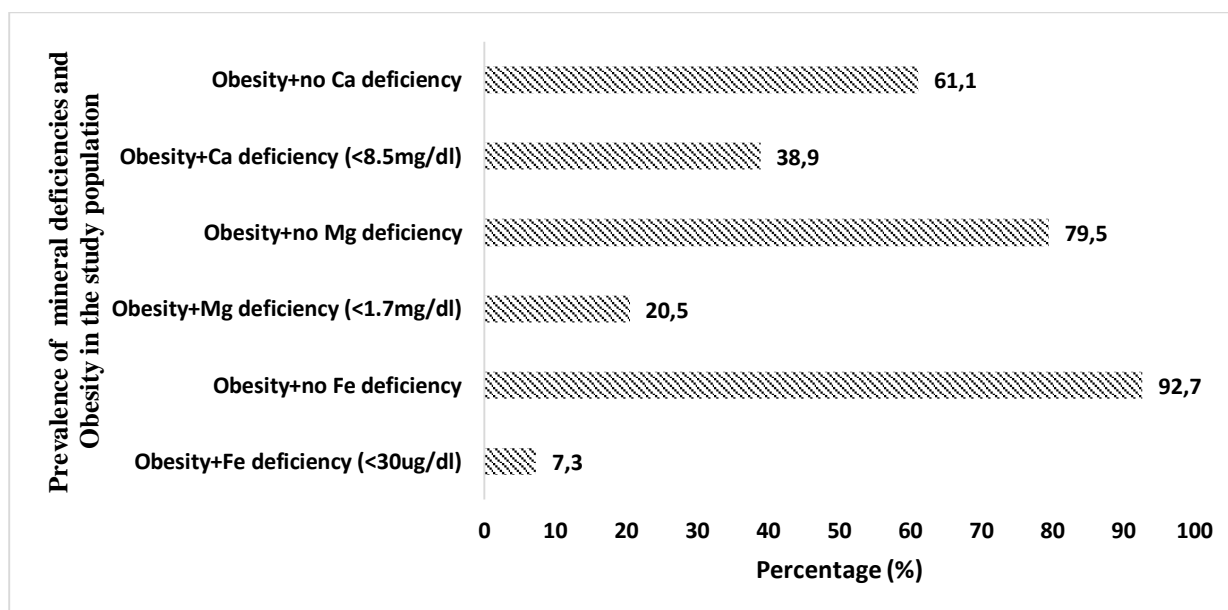


Figure 20: Prevalence of the coexistence of specific mineral deficiencies and obesity at the individual level in the study population

III.1.10. Risk factors of Nutritional deficiencies

III.1.10.1. Risk factors of developing one or more nutritional deficiencies

Logistical regression analysis revealed that, a borderline food consumption score (OR= 1.586; p=0.028), an acceptable FCS (OR= 2.733; p=0.000), was a risk factor of developing at least one or more nutritional deficiencies among these women of childbearing age in the 95% CI. A mildly food insecure household (OR=0.326; p=0.000) and the rare intake of vegetables (OR: 0.447; p=0.000) were less likely to be predictors of developing one or more nutritional deficiencies.

Table 34: Nutritional risk factors of developing one or more nutritional deficiencies (underweight, iron, calcium or magnesium deficiencies)

Risk factors	Odd ratio 95% CI	P-Value
FCS threshold		
Poor[‡]		
Borderline	1.586 (1.050-2.393)	0.028
Acceptable	2.733 (1.848-4.042)	0.000
HFIAS threshold		
Food secure[‡]		
Mildly food insecure	0.326 (0.178-0.594)	0.000
Moderately and severely food insecure	0.565 (0.315-1.012)	0.055
Vegetable intake		
High intake[‡]		
Moderate intake	0.756 (0.498-1.149)	0.190
Rare intake	0.447 (0.308-0.649)	0.000
Fruit intake		
High intake[‡]		
Moderate intake	1.012 (0.430-2.383)	0.979
Rare intake	0.529 (0.253-1.109)	0.092
Alcohol consumption		
Never[‡]		
<Occasional	1.176(0.799-1.731)	0.411
Moderate	0.881(0.548-1.416)	0.600
High	0.861(0.455-1.629)	0.645

[‡]J=Reference Category; FCS: Food Consumption Score; HFIAS: Household Food Insecurity Access Scale; **High intake:** 5-7

times per week; **Medium intake:** 3-4 times per week; **Low intake:** 1-2 times per week; **CI:** Confidence Interval

III.1.10.2. Stepwise covariates Logistical Regression analysis of Nutritional risk factors for developing one or more nutritional deficiencies

The step by step analysis of nutritional risk factors showed that the household food security level was the factor that mostly determined the development of one or more nutritional deficiencies in these women. Women who were in mildly food insecure households were less likely (OR=0.326) of having one or more nutritional deficiencies (p=0.000) in the 95% CI. Also, those in moderately and severely food insecure households were less likely of having one or more nutritional deficiencies (p=0.044) in the 95% CI compared to those in food secure households (Table 20, STEP 6).

Table 35: Stepwise covariates Logistical Regression analysis of Nutritional risk factors for developing one or more nutritional deficiencies (underweight, iron, calcium or magnesium deficiencies)

Risk factors	Odd ratio 95% CI	P-Value
STEP 1 FCS threshold		
Poor ^f	1.0	
Borderline	1.075 (0.347-3.327)	0.901
Acceptable	0.947 (0.293-3.058)	0.927
HDDS threshold		
Not acceptable ^f	1.0	
Acceptable	0.726 (0.245-2.151)	0.564
HFIAS threshold		
Food secure ^f	1.0	
Mildly food insecure	0.324 (0.175-599)	0.000
Moderately and severely food insecure	0.555 (0.305-1.011)	0.054
Vegetable intake		
High intake ^f	1.0	
Moderate intake	0.907(0.492-1.673)	0.756
Rare intake	0.455(0.217-0.954)	0.037
Fruit intake		
High intake ^f	1.0	
Moderate intake	0.924(0.379-2.255)	0.862
Rare intake	1.372(0.598-3.147)	0.455
Alcohol consumption		
Never ^f	1.0	
Occasional	0.930 (0.526-1.644)	0.802
Moderate	0.933 (0.443-1.967)	0.856
High	1.310 (0.412-4.161)	0.647
STEP 2 FCS threshold		
Poor ^f	1.0	
Borderline	1.045 (0.341-3.197)	0.939
Acceptable	0.921 (0.289-2.938)	0.890
HDDS threshold		
Not acceptable ^f	1.0	
Acceptable	0.739 (0.250-2.181)	0.583
HFIAS threshold		
Food secure ^f	1.0	
Mildly food insecure	0.327 (0.177-0.603)	0.000
Moderately and severely food insecure	0.555 (0.305-1.008)	0.053
Vegetable intake		
High intake ^f	1.0	
Moderate intake	0.912 (0.496-1.678)	0.768
Rare intake	0.458 (0.219-0.954)	0.037
Fruit intake		
High intake ^f	1.0	
Moderate intake	0.920 (0.377-2.245)	0.854
Rare intake	1.372 (0.599-3.142)	0.455
STEP 3 HDDS threshold		
Not acceptable ^f	1.0	
Acceptable	0.707(0.293-1.705)	0.440

HFIAS threshold		
Food secure ^J	1.0	
Mildly food insecure	0.328 (0.178-0.605)	0.000
Moderately and severely food insecure	0.557 (0.306-1.011)	0.054
Vegetable intake		
High intake ^J	1.0	
Moderate intake	0.933 (0.512-1.697)	0.820
Rare intake	0.471 (0.231-0.959)	0.038
Fruit intake		
High intake ^J	1.0	
Moderate intake	0.933 (0.383-2.270)	0.879
Rare intake	1.406 (0.620-3.189)	0.414
STEP 4 HFIAS threshold		
Food secure ^J	1.0	
Mildly food insecure	0.333 (0.181-0.612)	0.000
Moderately and severely food secure	0.547 (0.302-0.992)	0.047
Vegetable intake		
High intake ^J	1.0	
Moderate intake	0.951(0.524-1.727)	0.869
Rare intake	0.507(0.256-1.006)	0.052
Fruit intake		
High intake ^J	1.0	
Moderate intake	0.930(0.382-2.262)	0.872
Rare intake	1.435 (0.634-3.251)	0.386
STEP5 HFIAS threshold		
Food secure ^J	1.0	
Mildly food insecure	0.334 (0.182-0.611)	0.000
Moderately and severely food insecure	0.568 (0.315-1.024)	0.060
Vegetable intake		
High intake ^J	1.0	
Moderate intake	1.001 (0.556-1.800)	0.998
Rare intake	0.548 (0.281-1.073)	0.079
STEP6 HFIAS threshold		
Food secure ^J	1.0	
Mildly food insecure	0.326 (0.178-0.594)	0.000
Moderately and severely food insecure	0.547 (0.305-0.983)	0.044

J=Reference Category; FCS: Food Consumption Score; **HDDS:** Household dietary diversity score; **HFIAS:** Household Food

Insecurity Access Scale; **High intake:** 5-7 times per week; **Medium intake:** 3-4 times per week; **Low intake:** 1-2 times per week;

CI: Confidence Interval

III.1.11. Influence of mineral status on biological cardiometabolic risk factors

III.1.11.1. Anthropometric, Biochemical and clinical characteristics of the study population

The table below presents the mean of the biochemical, anthropometric and clinical characteristics of the general population.

Table 36: Anthropometric, Biochemical and Clinical characteristics of the population

Parameters	Mean	SEM	N
WC (cm)	88.55	0.58	608
HC (cm)	105.57	0.56	608
Total cholesterol concentration (mg/dl)	143.03	2.67	608
Triglyceride concentration (mg/dl)	111.03	3.09	608
Glucose concentration(mg/dl)	101.85	1.62	608
SBP (mmHg)	126.32	0.77	608
DBP (mmHg)	83.71	0.63	608
Cardiac frequency	73.31	0.92	608

WC: Waist Circumference; HC: Hip circumference; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; SEM: Standard Error of Mean

III.1.11.2. Anthropometric, Biochemical and clinical characteristics of the study population with respect to age group

The table below presents the mean of the biochemical, anthropometric and clinical characteristics of the general population with respect to the age groups. The mean WC, HC, triglyceride concentration, SBP and the DBP were significantly different between the various age groups ($p < 0.05$).

Table 37: Anthropometric, Biochemical and Clinical characteristics of the population with respect to age group

Parameters	14-20 years (N=63)	21-30 years (N=141)	31-40 years (N=209)	41-49 years (N=195)	P-value
WC(cm)	78.06±1.48a	83.84±1.17b,a	89.15±0.823c,b,a	94.70±1.04d,c,b	0.000
HC (cm)	96.21±1.44a	103.10±1.15b,a	106.17±0.82c,b,a	109.74±1.06d,c,b	0.000
TC concentration (mg/dl)	146.94±10.53a	147.25±5.47a	137.79±4.03a	144.33±4.86a	0.534
TG concentration (mg/dl)	104.40±9.80a	95.23±5.25a,b	113.26±5.63a,c,e	122.19±5.56a,d,e	0.012
Glucose concentration (mg/dl)	97.49±3.88a	106.46±3.69a	99.16±2.86a	102.82±2.72a	0.299
SBP (mmHg)	117.87±2.16a	122.42±1.37a,b	125.75±1.18b,c,d	132.49±1.53c,d,e	0.000
DBP (mmHg)	79.82±1.70a	81.24±1.23a,b	83.90±1.11a,b,e	86.54±1.16b,d,e	0.003
Cardiac Frequency	70.22±3.40a	74.49±2.10a	73.53±1.41a	73.22±1.58a	0.672

TC: Total Cholesterol; TG: Triglyceride; WC= Waist Circumference; HC= Hip circumference; SBP=Systolic Blood Pressure; DBP=Diastolic Blood Pressure; Results are expressed in terms of mean values ± SEM (standard error of the mean). In the same line, values with different letters (a, b, c, d and e) are significantly different at $p < 0.05$.

III.1.11.3. Prevalence of biological cardiometabolic risk factors

The table below presents the prevalence of BCMR and individual BCMRF in the study population. Up to 92.4% of the women had one or more BCMRF. The prevalence

of abdominal obesity was 70.4%. Up to 29.1 % of the women were hypertensive according to the IFD criteria, 43.1% of them were hyperglycemic, hypertriglyceridemia was found in 21.7 % of them and hypercholesterolemia in 16.1% of the women. Up to 42.1% of the study population had MetS in the 95% CI.

Table 38: Prevalence of biological cardiometabolic risk (BCMR) and Individual biological cardiometabolic risk factors in the study population

BCMRF and Individual risk factors	Frequency	Percentage (%)	95% CI
BCMR			
Absence	46	7.6	5.6-9.7
Presence	562	92.4	90.3-94.4
Abdominal obesity (WC>80cm)	428	70.4	66.5-73.6
Overweight (BMI 25.0 – 29.9 Kg/m²)	187	30.8	26.8-34.4
Obese (BMI ≥ 30 Kg/m²)	234	38.5	34.4-42.7
High Blood pressure (SBP≥130; DBP≥85)mmHg	177	29.1	25.5-32.6
Hyperglycemia (Fasting glucose level≥100mg/dL)	262	43.1	38.8-47.0
Hypertriglyceridemia (plasma TG≥ 150 mg/dL)	132	21.7	18.5-24.8
Hypercholesterolemia (plasma TC≥ 200 mg/dL)	98	16.1	13.3-19.2
Metabolic syndrome (MetS) (WC>80cm+2 or more components)	256	42.1	38.3-46.1

WC= Waist Circumference; BMI: Body Mass Index; SBP=Systolic Blood Pressure; DBP=Diastolic Blood Pressure; TC: Total Cholesterol; TG: Triglyceride; CI: Confidence Interval; MetS: Metabolic Syndrome

III.1.11.4. Prevalence of biological cardiometabolic risk factors by age group

Participants of the study population in the 31-40 years age group recorded the highest prevalence (32.6%) of those with BCMR compared to the other age groups and this was statistically different (p=0.000). The highest prevalence of abdominal obesity (27.8%), obesity (16.1%), high blood pressure (13.3%), hyperglycemia (15.1%), hypertriglyceridemia (8.9%), hypercholesterolemia (5.3%) and MetS (18.8%) were observed among participant in the 41-49 years age group and this was statistically significant (p<0.05) for all parameters except for hypercholesterolemia (p>0.05).

Table 39: Prevalence of BCMR and Individual BCMRF by age group

Parameters	14-20 years n (%)	21-30 years n (%)	31-40 years n (%)	41-49 years n (%)	P-value
BCMR					
Presence	51(8.4)	126(20.7)	198(32.6)	187(30.8)	0.000
Absence	12(2.0)	15(2.5)	11(1.8)	8(1.3)	
Abdominal obesity (WC>80cm)	21(3.5)	78(12.8)	160(26.3)	169(27.8)	0.000
Overweight (BMI 25.0 – 29.9 Kg/m²)	12(2.0)	43(7.1)	71(11.7)	61(10.0)	0.165
Obese (BMI ≥ 30 Kg/m²)	6(1.0)	39(6.4)	91(15.0)	98(16.1)	0.000
High Blood pressure (SBP≥130; DBP≥85)mmHg	9(1.5)	30(4.9)	58(9.5)	81(13.3)	0.000
Hyperglycemia (Fasting glucose level≥100mg/dL)	18(3.0)	66(10.9)	86(14.1)	92(15.1)	0.049
Hypertriglyceridemia (plasma TG≥ 150 mg/dL)	12(2.0)	21(3.5)	45(7.4)	54(8.9)	0.042
Hypercholesterolemia (plasma TC≥ 200 mg/dL)	11(1.8)	26(4.3)	29(4.8)	32(5.3)	0.697
Metabolic syndrome (MetS) (WC>80cm+2 or more components)	10(1.6)	39(6.4)	93(15.3)	114(18.8)	0.000

BCMRF: Biological cardiometabolic risk factors; **WC=** Waist Circumference; **BMI:** Body Mass Index; **SBP=**Systolic Blood Pressure; **DBP=**Diastolic Blood Pressure; **TC:** Total Cholesterol; **TG:** Triglyceride; **CI:** Confidence Interval; **MetS:** Metabolic Syndrome

III.1.11.5. Prevalence of biological cardiometabolic risk factors with respect to mineral status and Ca-Mg ratio groups

Among participants who were deficient in one of the minerals, higher rates of all the BCMRF was associated to participants of the study who were Ca deficient. The rates of hyperglycemia, hypercholesterolemia and MetS were slightly higher in the low Ca-Mg ratio group ($p>0.05$) while the rates of overweight/obesity and hypertriglyceridemia were slightly higher in the medium Ca-Mg ratio group compared to the other groups ($p>0.05$); and abdominal obesity and high blood pressure rates were higher in the high Ca-Mg ratio group compared to the other groups ($p>0.05$).

Table 40: Frequencies of biological cardio metabolic risk with respect to mineral status and mean Ca-Mg ratio group

	Magnesium status		Iron status		Calcium status		Mean Ca-Mg ratio groups		
	N (%)		N (%)		N (%)		N (%)		
BCMRF	Mg deficient	Not Mg deficient	Fe deficient	Not Fe deficient	Ca deficient	Not Ca deficient	0.46±0.03 (N= 179)	2.35±0.04 (N= 179)	8.99 ±0.99 (N= 179)
Overweight/obesity (BMI≥25Kg/m²)	89(21.9)a	317(78.1)b	33(9.5)a	314(90.5)b	165(43.9)a	211(56.1)b	108(20.1)a	134(25.0)a	125(23.3)a
Abdominal obesity (WC>80cm)	95(16.1)a	318(54.0)b	33(6.5)a	325(64.5)b	171(31.3)a	209(38.2)b	116(21.6)a	127(23.6)b	129(24.0)c
High Blood pressure (SBP≥130;DBP≥85)mmHg	52(8.8)a	208(35.3)b	24(4.8)a	195(38.7)b	114(20.8)b	123(22.5)b	48(8.9)a	55(10.2)a	56(10.4)a
Hyperglycemia (Fasting glucose level≥100mg/dL)	62(10.5)a	189(32.1)b	27(5.4)a	184(36.5)b	111(20.3)a	117(21.4)a	76(14.2)a	72(13.4)a	75(14.0)a
Hypertriglyceridemia (plasma TG≥ 150 mg/dL)	20(3.4)a	105(17.8)b	10(2.0)a	100(19.8)b	60(11.0)b	60(11.0)b	35(6.5)a	44(8.2)a	38(7.1)a
Hypercholesterolemia (plasma TC≥ 200 mg/dL)	32(5.4)a	61(10.4)b	10 (2.0)a	69(13.7)a	51(9.3)a	38(6.9)b	33(6.1)a	22(4.1)a	31(5.8)a
Metabolic syndrome (MetS) (WC>80cm+2 or more components)	54(9.2)a	193(32.8)b	20(4.0)a	190(37.7)b	109(19.9)a	118(21.6)a	77(14.3)a	73(13.6)a	73(13.6)a

BCMRF: Biological cardiometabolic risk factors; WC= Waist Circumference; BMI: Body Mass Index; SBP=Systolic Blood Pressure; DBP=Diastolic Blood Pressure; TC: Total Cholesterol; TG: Triglyceride; CI: Confidence Interval; MetS: Metabolic Syndrome

-In the same line, values with different letters (a, b and c) are significantly different at p<0.05.

III.1.11.6. Influence of mineral status on biological cardio metabolic biomarkers

Only the mean high systolic blood pressure of those who were iron deficient was significantly higher than that of those who were not iron deficient (p=0.021).

Table 41: Influence of iron status on biological cardio metabolic biomarkers

Parameters	Iron deficient	Not Iron deficient	P-value
BMI (≥25Kg/m²)	31.50±0.92	32.43±0.37	0.438
Central obesity (>80 cm)	94.50±1.94	94.97±0.70	0.838
SBP (≥130 mmHg)	153.16±5.56	144.35±1.08	0.021
DBP(≥ 85 mmHg)	99.58±3.02	97.37±0.89	0.427
Elevated glycaemia (≥100 mg/dl)	127.86±9.02	128.92±3.17	0.906
Elevated triglyceridemia (≥ 150 mg/dl)	233.28±25.37	213.33±6.05	0.338
Elevated cholesterol (≥ 200mg/dl)	249.46±20.57	252.13±5.60	0.873

BMI: Body Mass Index; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure;

III.1.11.6.1. Influence of Magnesium status on biological cardio metabolic biomarkers

There were no significant differences between the mean BCMRF of those who were magnesium deficient and that of those who were not.

Table 42: Influence of Magnesium status on biological cardio metabolic biomarkers

Parameters	Magnesium deficient (n=89)	Not Magnesium deficient (n=406)	P-value
BMI ($\geq 25\text{Kg/m}^2$)	32.04 \pm 0.66	32.32 \pm 0.37	0.721
Central obesity (>80 cm)	94.90 \pm 1.40	94.65 \pm 0.66	0.862
SBP (≥ 130 mmHg)	142.07 \pm 2.16	145.82 \pm 1.14	0.151
DBP (≥ 85 mmHg)	94.82 \pm 1.28	97.82 \pm 0.88	0.114
Elevated glycaemia (≥ 100 mg/dl)	135.09 \pm 5.62	125.46 \pm 2.94	0.114
Elevated triglyceridemia (≥ 150 mg/dl)	252.91 \pm 19.49	216.84 \pm 7.15	0.053
Elevated cholesterol (≥ 200mg/dl)	258.44 \pm 10.62	253.31 \pm 6.70	0.671

BMI: Body Mass Index; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure;

III.1.11.6.2. Influence of Calcium status on biological cardio metabolic biomarkers

There were no significant differences between the mean BCMRF of those who were calcium deficient and that of those who were not.

Table 43: Influence of Calcium status on biological cardio metabolic biomarkers

Parameters	Calcium deficient (n=165)	Not Calcium deficient (n=211)	P-value
BMI ($\geq 25\text{Kg/m}^2$)	32.18 \pm 0.50	32.62 \pm 0.48	0.533
Central obesity (>80 cm)	94.05 \pm 0.78	95.60 \pm 0.95	0.223
SBP (≥ 130 mmHg)	144.61 \pm 1.43	146.12 \pm 1.47	0.473
DBP (≥ 85 mmHg)	96.38 \pm 0.94	98.34 \pm 1.29	0.227
Elevated glycaemia (≥ 100 mg/dl)	131.33 \pm 4.31	123.93 \pm 3.60	0.188
Elevated triglyceridemia (≥ 150 mg/dl)	224.33 \pm 9.80	212.80 \pm 7.62	0.355
Elevated cholesterol (≥ 200mg/dl)	250.16 \pm 7.21	255.80 \pm 8.77	0.618

BMI: Body Mass Index; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure;

III.1.11.6.3. Influence of Calcium-to-Magnesium ratio groups on biological cardio metabolic biomarkers

There were no significant differences in the means of the biological cardiometabolic risk factors between the various Ca-Mg ratio groups ($p > 0.05$) except for hyperglycemia whose mean was highest in the medium Ca-Mg ratio group compared to other groups ($p < 0.05$).

Table 44: Influence of Calcium-to-Magnesium ratio groups on biological cardio metabolic biomarkers

Parameters	Low Ca-Mg ratio group (n=179)	Medium Ca-Mg ratio group (n=179)	High Ca-Mg ratio group (n=179)	P-value
BMI ($\geq 25\text{Kg/m}^2$)	32.56 \pm 0.63a	31.93 \pm 0.61a	32.85 \pm 0.58a	0.536
Central obesity (>80 cm)	95.46 \pm 1.22a	93.02 \pm 0.81a	96.26 \pm 1.24a	0.095
SBP (≥ 130 mmHg)	145.16 \pm 1.90a	145.73 \pm 1.87a	145.66 \pm 1.73a	0.973
DBP (≥ 85 mmHg)	95.97 \pm 0.96a	99.02 \pm 1.44a	98.21 \pm 1.66a	0.271
Elevated glycaemia (≥ 100 mg/dl)	124.68 \pm 4.68a	136.14 \pm 6.09b	119.65 \pm 2.84c	0.043
Elevated triglyceridemia (≥ 150 mg/dl)	228.83 \pm 12.29a	231.00 \pm 12.17a	197.63 \pm 6.37a	0.055
Elevated cholesterol (≥ 200mg/dl)	248.33 \pm 7.77a	254.27 \pm 7.42a	258.87 \pm 12.50a	0.730

BMI: Body Mass Index; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure;

III.1.11.7. Associations between mineral concentrations and biological cardio metabolic biomarkers

Plasma iron concentration was strongly and positively correlated only to a waist circumference $\geq 80\text{cm}$ ($r= 0.108$, $p=0.041$)

Calcium concentration was strongly and significantly associated to high BMI ($\text{BMI} \geq 25\text{kg/m}^2$) ($r= 0.104$, $p=0.044$) but strongly and negatively associated to an elevated plasma triglyceride concentration ($r= -0.220$, $p=0.016$).

There was a strong negative and significant association only between an elevated plasma triglyceride concentration and magnesium concentration ($r=-0.192$, $p=0.032$)

Plasma Ca-Mg ratio was strongly and significantly correlated to a waist circumference ≥ 80 cm ($r=0.113$, $p=0.029$)

Table 45: Pearson's Correlation (r) between mineral concentrations and BCMRFs

BCMRFs	Fe conc	Ca Conc	Mg Conc	Ca-Mg ratio	Frequency (N)
BMI($\geq 25\text{kg/m}^2$)	0.091	0.104*	0.042	0.074	376
Hyperglycemia ($\geq 100\text{mg/dl}$)	-0.045	-0.105	-0.081	-0.061	228
SBP($\geq 140\text{mmHg}$)	-0.055	0.092	0.064	-0.008	206
DBP($\geq 90\text{mmHg}$)	0.000	0.098	0.051	-0.001	237
Hypertriglyceridemia (TG$\geq 150\text{mg/dl}$)	0.057	-0.220*	-0.192*	-0.007	120
Hypercholesterolemia (TC$\geq 200\text{mg/dl}$)	-0.059	0.039	0.121	-0.006	89
WC($\geq 80\text{cm}$)	0.108*	0.054	0.042	0.113*	380

BMI: Body Mass Index; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; TG: Triglycerides; TC: Total Cholesterol; WC: Waist circumference; Fe Conc: Iron concentration; Ca Conc: Calcium concentration; Mg Conc: Magnesium Concentration; Ca-Mg ratio: Calcium-to-Magnesium ratio

Significance level: * $p<0.05$, ** $p<0.01$

0.000-0.009= very weak to no correlation

0.010-0.090= weak;

0.100-0.900= strong;

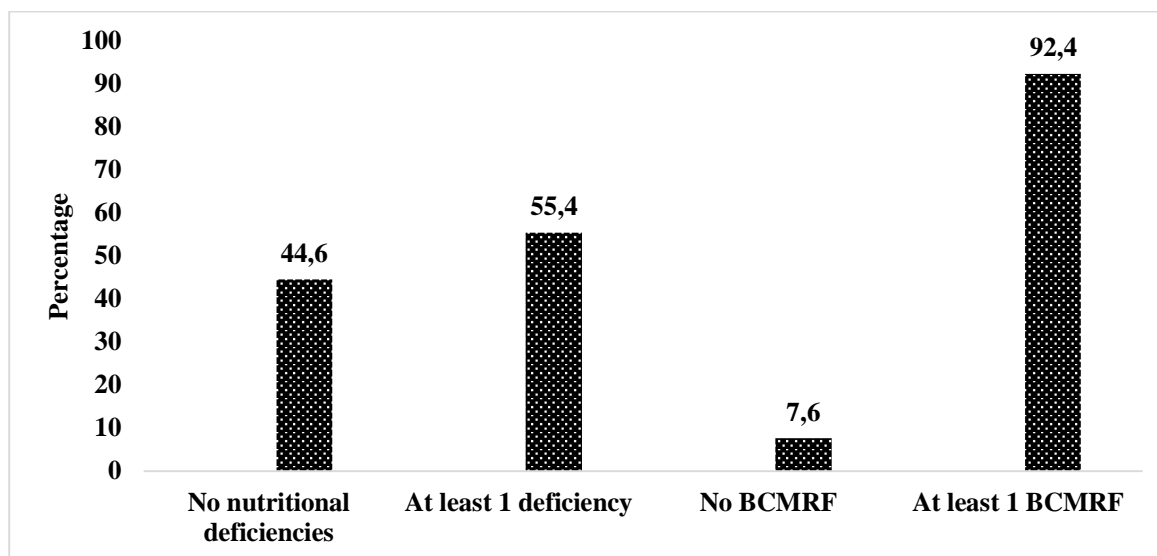
1.0=very strong correlation;

- Negative correlation; + positive correlation

III.1.12. Prevalence of Nutritional deficiencies and biological cardio metabolic risk factors

III.1.12.1. Prevalence of at least 1 nutritional deficiency and at least 1 biological cardiometabolic risk factors (BCMRF)

There were up to 92.4% of women in the study with at least one biological cardiometabolic risk factor and 55.4% of them with at least one nutritional deficiency.

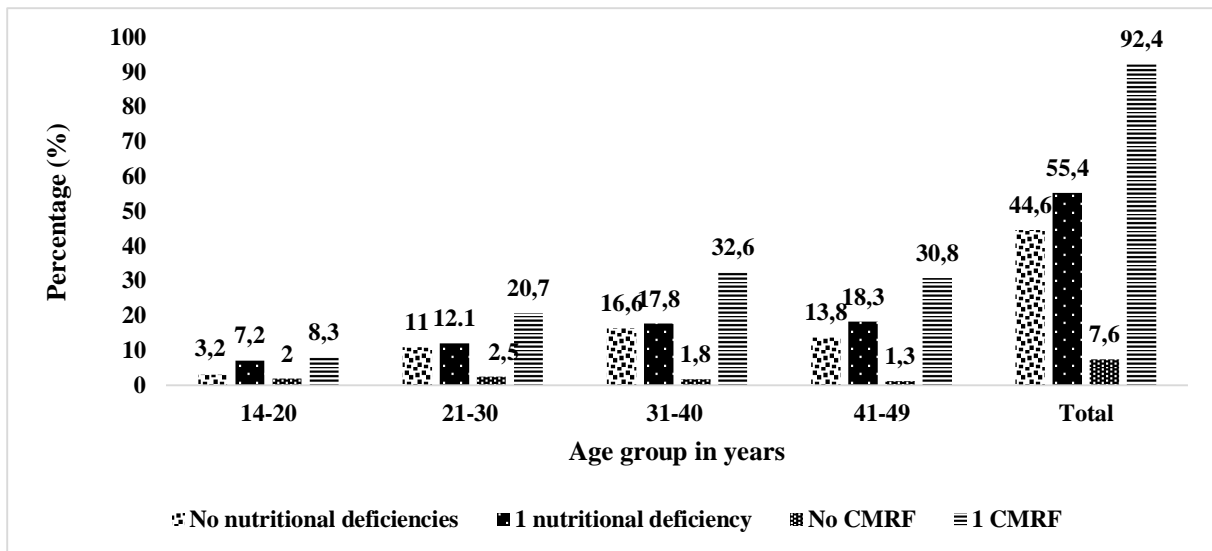


Nutritional deficiencies: (Underweight, calcium, magnesium, iron deficiencies); BCMRF: Biological Cardiometabolic risk factors

Figure 21: Prevalence of at least 1 nutritional deficiency and at least 1 biological cardiometabolic risk factors (CMRF)

III.1.11.2. Prevalence of at least 1 nutritional deficiency and at least 1 cardiometabolic risk factors (CMRF) according to age groups

55.4% of the participants had at least one nutritional deficiency and 92.4% of them had at least one CMRF. The highest percentage of at least one nutritional deficiency was observed among women in the 41-49 years age group (18.3%) while the highest percentage of at least one CMRF was in the 31-40 years age group (32.6%) (Figure 22).



CMRF: Cardiometabolic risk factors; Nutritional deficiencies include one of these: underweight or calcium or magnesium or iron deficiencies (N=608)

Figure 22: Prevalence of at least 1 nutritional deficiency and at least 1 cardiometabolic risk factors (CMRF) according to age groups

III.1.12.3. Prevalence of the coexistence of nutritional deficiencies and BCMRF

Up to 52.0% of women of childbearing age of the study population had at least one nutritional deficiency and at least one biological cardiometabolic risk factor. 35.4% of them were overweight or obese with at least one mineral deficiency. While 36.5% had central obesity with at least one mineral deficiency. Among those with at least one mineral deficiency, 14.6% had a high blood pressure, 23.2% were hyperglycemic, 11.2% had a high plasma triglyceride concentration and 9.4% had a high plasma cholesterol level. Metabolic syndrome among these women was 22.2%. The prevalence of the double burden of malnutrition was 36.7% in the 95% (32.9-40.5) confidence interval.

Table 46: Prevalence of the coexistence of Nutritional deficiencies and BCMRF at the individual level in the study population

Phenotypes	Frequency	Percentage	95% CI
Nutritional deficiencies + BCMRF	316	52.0	47.9-55.9
Overweight/obese+ mineral deficiencies	215	35.4	31.4-39.3
Central obesity+ mineral deficiencies	222	36.5	32.9-40.3
HTN+ mineral deficiencies	89	14.6	12.0-17.9
Hyperglycemia+ mineral deficiencies	141	23.2	20.1-26.6
Hypertriglyceridemia+ mineral deficiencies	68	11.2	8.9-13.7
Hypercholesterolemia+ mineral deficiencies	57	9.4	7.1-12.0
Metabolic syndrome+ mineral deficiencies	135	22.2	18.9-25.3
Double burden of malnutrition	223	36.7	32.9-40.5

Nutritional deficiencies: (Underweight, calcium, magnesium, iron deficiencies)

Double burden: (Underweight+mineral deficiency (n=8) and Overweight/obese+mineral deficiency (n=215))

HTN: Hypertension; BCMRF: Biological cardiometabolic risk factors

III.1.12.3.1. Prevalence of the coexistence of nutritional deficiencies and BCMRF by age group

Women in the 40-49 years age group recorded the highest prevalence of the various phenotypes of nutritional deficiencies and BCMRF followed by those in the 31-40 years age group except for the hyperglycemia and at least one mineral deficiency group which was highest among women in the 31-40 years age group followed by those in the 41-49 years age group. The prevalence of the double burden of malnutrition increased with age and it was 14.1% in the 41-49 years age group and this was statistically significant from the other age groups ($p=0.001$).

Table 47: Prevalence of the coexistence of Nutritional deficiencies and BCMRF at the individual level with respect to age group

Phenotypes	14-20 years N (%)	21-30 years N (%)	31-40 years N (%)	41-49 years N (%)	Total N (%)	P-value
Nutritional deficiencies + CMRF	6.2(38)	10.9 (66)	17.1 (104)	17.8 (108)	52.0 (316)	0.205
Overweight/obese+ mineral deficiencies	1.5(9)	6.6(40)	13.3(81)	14.0(85)	35.4(215)	0.000
Central obesity+ mineral deficiencies	1.8(11)	6.2(38)	13.0(79)	15.5(94)	36.5(222)	0.000
HTN+ mineral deficiencies	1.0(6)	1.5(9)	4.4(27)	7.7(47)	14.6(89)	0.000
Hyperglycemia+ mineral deficiencies	1.8(11)	5.8(35)	8.1(49)	7.6(46)	23.2(141)	0.706
Hypertriglyceridemia+ mineral deficiencies	1.2(7)	2.1(13)	3.8(23)	4.1(25)	11.2(68)	0.782
Hypercholesterolemia+ mineral deficiencies	1.3(8)	2.6(16)	2.5(15)	3.0(18)	9.4(57)	0.447
Metabolic syndrome+ mineral deficiencies	1.0(6)	3.1(19)	7.4(45)	10.7(65)	22.2(135)	0.000
Double burden of malnutrition	1.8(11)	7.2(44)	13.5(82)	14.1(86)	223(36.7)	0.001

Nutritional deficiencies: (Underweight, calcium, magnesium, iron)

Double burden: (Underweight+mineral deficiency (n=8) and Overweight/obese+mineral deficiency (n=215))

%; Percentage of BCMRF with respect to micronutrient status

III.1.12.4. Specific mineral deficient state and Specific Ca-Mg ratio groups as predictors of individual BCMRF

III.1.12.4.1. Magnesium deficient state as predictors of individual BCMRF

Multivariate logistical regression analysis revealed that Magnesium deficiency was a predictor of hypercholesterolemia (OR: 1.962, p=0.024) when adjusted for iron status, calcium status and age

Table 48: Odds of developing individual biological cardio metabolic risk factors with respect to Magnesium deficiency adjusted for Fe, Ca status and age

BCMRF	Not Mg deficient	Adjusted Odd Ratio	P-value
Overweight/obese	1.0	1.309(0.752-2.276)	0.341
Abdominal obesity	1.0	1.526 (0.845-2.754)	0.161
High Blood pressure (SBP≥130;DBP≥85)mmHg	1.0	0.727 (0.422-1.254)	0.252
Hyperglycemia (Fasting glucose level≥100mg/dL)	1.0	0.984 (0.605-1.601)	0.948
Hypertriglyceridemia (plasma TG≥ 150 mg/dL)	1.0	0.423 (0.216-0.828)	0.012
Hypercholesterolemia (plasma TC≥ 200 mg/dL)	1.0	1.962 (1.093 -3.521)	0.024
Metabolic syndrome (MetS) (WC>80cm+2 or more components)	1.0	0.933 (0.564-1.546)	0.789

BCMRF: Biological Cardiometabolic Risk factors; **SBP:** Systolic Blood Pressure; **DBP:** Diastolic Blood Pressure; **TG:** Tryglycerides; **TC:** Total Cholesterol; **MetS:** Metabolic syndrome; **WC:** Waist circumference

III.1.12.4.2. Calcium deficient state as predictors of individual BCMRF

Multivariate analysis revealed that calcium deficiency was not a predictor of developing any of the BCMRF compared to normal calcium status when adjusted for iron status, magnesium status and age.

Table 49: Odds of developing individual biological cardio metabolic risk factors with respect to Calcium deficiency adjusted for Fe, Mg status and age

BCMRF	Not Ca deficient	Adjusted Odd Ratio	P-value
Overweight/obese	1.0	0.633(0.405-0.989)	0.045
Abdominal obesity	1.0	0.652 (0.408-1.043)	0.074
High Blood pressure (SBP≥130;DBP≥85)mmHg	1.0	0.899 (0.584-1.383)	0.628
Hyperglycemia (Fasting glucose level≥100mg/dL)	1.0	1.047 (0.703-1.558)	0.822
Hypertriglyceridemia (plasma TG≥ 150 mg/dL)	1.0	1.278 (0.800-2.042)	0.304
Hypercholesterolemia (plasma TC≥ 200 mg/dL)	1.0	1.413 (0.824 -2.422)	0.209
Metabolic syndrome (MetS) (WC>80cm+2 or more components)	1.0	1.060 (0.703-1.599)	0.782

BCMRF: Biological Cardiometabolic Risk factors; **SBP:** Systolic Blood Pressure; **DBP:** Diastolic Blood Pressure; **TG:** Tryglycerides; **TC:** Total Cholesterol; **MetS:** Metabolic syndrome; **WC:** Waist circumference

III.1.12.4.3. Iron deficient state as predictors of individual BCMRF

Iron deficiency was not a predictor of developing any of the BCMRF compared to normal iron status when adjusted for calcium status, magnesium status and age

Table 50: Odds of developing individual biological cardio metabolic risk factors with respect to Iron deficiency adjusted for Mg, Ca status and age

BCMRF	Not Fe deficient	Adjusted Odd Ratio	P-value
Overweight/obese	1.0	0.552(0.280-1.085)	0.085
Abdominal obesity	1.0	0.516 (0.255-1.045)	0.066
High Blood pressure (SBP\geq130;DBP\geq85)mmHg	1.0	0.887 (0.435-1.806)	0.740
Hyperglycemia (Fasting glucose level\geq100mg/dL)	1.0	1.419 (0.754-2.669)	0.278
Hypertriglyceridemia (plasma TG\geq 150 mg/dL)	1.0	0.903 (0.413-1.974)	0.798
Hypercholesterolemia (plasma TC\geq 200 mg/dL)	1.0	0.743 (0.297 -1.858)	0.525
Metabolic syndrome (MetS) (WC$>$80cm+2 or more components)	1.0	0.733 (0.369-1.458)	0.376

BCMRF: Biological Cardiometabolic Risk factors; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; TG: Tryglycerides; TC: Total Cholesterol; MetS: Metabolic syndrome; WC: Waist circumference

III.1.12.4.4. Low Ca-Mg ratio as predictors of individual BCMRF

Low Ca-Mg ratio group was not a predictor of any of the biological cardiometabolic risk factors after adjusting for age, calcium and magnesium status.

Table 51: Odds of developing individual biological cardio metabolic risk factors with respect to low group and adjusted for age, calcium and magnesium status

BCMRF	Medium Ca-Mg group	Adjusted Odd Ratio	P-value
Overweight/obese	1.0	1.230 (0.626-2.415)	0.548
Abdominal obesity	1.0	1.291 (0.643-2.592)	0.472
High Blood pressure (SBP\geq130;DBP\geq85)mmHg	1.0	1.133 (0.531-2.416)	0.747
Hyperglycemia (Fasting glucose level\geq100mg/dL)	1.0	1.071 (0.593-1.937)	0.820
Hypertriglyceridemia (plasma TG\geq 150 mg/dL)	1.0	0.643 (0.315-1.314)	0.226
Hypercholesterolemia (plasma TC\geq 200 mg/dL)	1.0	0.732 (0.336-1.592)	0.431
Metabolic syndrome (MetS) (WC$>$80cm+2 or more components)	1.0	1.465 (0.788-2.725)	0.228

BCMRF: Biological Cardiometabolic Risk factors; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; TG: Tryglycerides; TC: Total Cholesterol; MetS: Metabolic syndrome; WC: Waist circumference; Ca-Mg: Calcium-to-Magnesium ratio

III.1.12.4.5. High Ca-Mg ratio as predictors of individual BCMRF

A high Ca-Mg ratio group was not a predictor of any of the biological cardiometabolic risk factors after adjusting for age, calcium and magnesium status.

Table 52: Odds of developing individual biological cardio metabolic risk factors with respect to high Ca-Mg ratio group and adjusted for age, Ca and Mg status

BCMRF	Medium Ca-Mg ratio group	Adjusted Odd Ratio	P-value
Overweight/obese	1.0	0.813(0.414-1.597)	0.548
Abdominal obesity	1.0	0.775 (0.386-1.555)	0.472
High Blood pressure (SBP≥130;DBP≥85)mmHg	1.0	1.133 (0.591-2.170)	0.707
Hyperglycemia (Fasting glucose level≥100mg/dL)	1.0	0.933 (0.516-1.688)	0.820
Hypertriglyceridemia (plasma TG≥ 150 mg/dL)	1.0	1.555 (0.761-3.176)	0.226
Hypercholesterolemia (plasma TC≥ 200 mg/dL)	1.0	1.366 (0.628-2.972)	0.431
Metabolic syndrome (MetS) (WC>80cm+2 or more components)	1.0	0.683 (0.367-1.269)	0.228

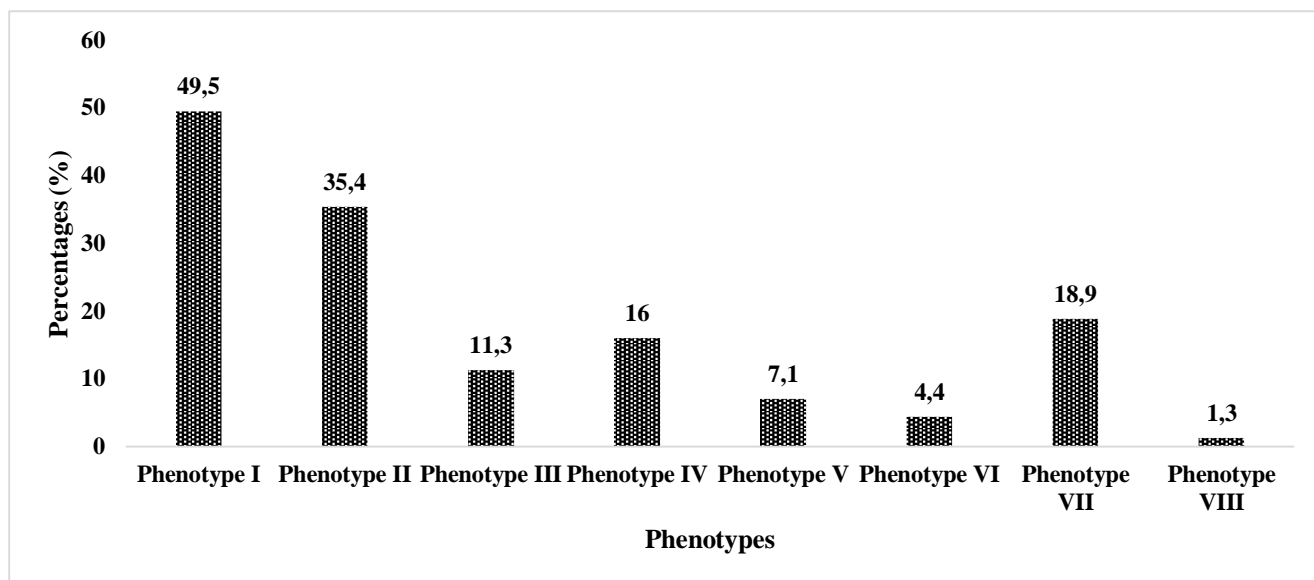
BCMRF: Biological Cardiometabolic Risk factors; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; TG: Tryglycerides; TC: Total Cholesterol; MetS: Metabolic syndrome; WC: Waist circumference; Ca-Mg: Calcium-to-Magnesium ratio

III.1.13. Coexistence of the double burden and biological cardiometabolic risk factors in the study population

III.1.13.1. Phenotypes of the double burden of malnutrition and BCMRF at the individual level in the study population

Considering the phenotypes of the double burden of malnutrition in association to BCMRF, 49.5 % of the participants had at least 1 mineral deficiency and at least 1 BCMRF (*Phenotype I*), 35.4% of them were overweight or obese and had at least 1 mineral deficiency in addition to one other CMRF (*Phenotype II*). 11.3 % were overweight or obese, had hypertension and at least 1 mineral deficiency (*Phenotype III*). The prevalence of *phenotype IV* (being overweight or obese and hyperglycemic and having at least one mineral deficiency) was 16%. Those with *phenotype V* were 7.1% (overweight or obese, having a high serum triglyceride level and at least one mineral deficiency). 4.4% were overweight or obese with an elevated total cholesterol level and at least 1 mineral deficiency (*Phenotype VI*), participants with *Phenotype VII*

(overweight or obese with MetS and at least 1 mineral deficiency) were 18.9% and lastly, 1.3% of participants were underweight with at least one mineral deficiency (*Phenotype VIII*).



Phenotype I: at least one mineral deficiency and one CMRF; **Phenotype II:** overweight or obese+ at least one other CMRF+ at least one mineral deficiency; **Phenotype III:** overweight or obese+ hypertension+ at least one mineral deficiency; **Phenotype IV:** overweight or obese+ hyperglycemia+ at least one mineral deficiency; **Phenotype V:** overweight or obese+ Hypertriglyceridemia + at least one mineral deficiency; **Phenotype VI:** overweight or obese+ Hypercholesterolemia + at least one mineral deficiency; **Phenotype VII:** overweight or obese+ MetS + at least one mineral deficiency, **Phenotype VIII:** Underweight + at least one mineral deficiency

Figure 23: Phenotypes of the double burden of malnutrition and BCMRF in the study population at the individual level

III.1.13.2. Phenotypes of the double burden of malnutrition and BCMRF at the individual level in the study population by age group

The prevalence of Phenotype I (16.9%), Phenotype II (14.0%), Phenotype III (6.4%), Phenotype V (3.1%), Phenotype VI (2.0%) and Phenotype VII (9.2%) were highest in the 41-49 years age group; and there was a significant difference between this age group and the other age groups for phenotype II, phenotype III and VII. Phenotype IV was highest in the 31-40 years age group (6.2%) and this was statistically different from the other age groups (14-20 age group: 0.5%; 21-30 age group: 3.1%; 41-49 age group: 6.1%) ($p < 0.05$). The prevalence of individual presenting characteristics of phenotype VIII was highest in the 21-30 years age group (0.7%).

Table 53: Phenotypes of the double burden of malnutrition and BCMRF in the study population by age group

Phenotypes	14-20 years N (%)	21-30 years N (%)	31-40 years N (%)	41-49 years N (%)	Total N (%)	P-value
Phenotype I	33(5.4)	64(10.5)	101(16.6)	103(16.9)	301(49.5)	0.544
Phenotype II	9(1.5)	40(6.6)	81(13.3)	85(14.0)	215(35.4)	0.000
Phenotype III	1(0.2)	6(1.0)	23(3.8)	39(6.4)	69(11.3)	0.000
Phenotype IV	3(0.5)	19(3.1)	38(6.2)	37(6.1)	97(16.0)	0.035
Phenotype V	2(0.3)	5(0.8)	17(2.8)	19(3.1)	43(7.1)	0.086
Phenotype VI	0(0.0)	7(1.2)	8(1.3)	12(2.0)	27(4.4)	0.208
Phenotype VII	1(0.2)	17(2.8)	41(6.7)	56(9.2)	115 (18.9)	0.000
Phenotype VIII	2(0.3)	4(0.7)	1(0.2)	1(0.2)	8(1.3)	0.099

Phenotype I: at least one mineral deficiency and one CMRF; **Phenotype II:** overweight or obese+ at least one other CMRF+ at least one mineral deficiency; **Phenotype III:** overweight or obese+ hypertension+ at least one mineral deficiency; **Phenotype IV:** overweight or obese+ hyperglycemia+ at least one mineral deficiency; **Phenotype V:** overweight or obese+ Hypertriglyceridemia + at least one mineral deficiency; **Phenotype VI:** overweight or obese+ Hypercholesterolemia + at least one mineral deficiency; **Phenotype VII:** overweight or obese+ MetS + at least one mineral deficiency, **Phenotype VIII:** Underweight + at least one mineral deficiency

III.1.13.3. Coexistence of the double burden of malnutrition and biological cardiometabolic risk factors in the study with respect to sociodemographic and nutritional parameters

III.1.13.3.1. Coexistence of the double burden of malnutrition and biological cardiometabolic risk factors in the study with respect to sociodemographic parameters

III.1.13.3.1.1. Coexistence of the double burden of malnutrition and biological cardiometabolic risk factors in the study with respect to region of resident

The prevalence of the various phenotypes of the DBM & BCMRFs at the individual level was highest in the Northwest region then Western followed by Littoral region then Center region and finally Far-North region. These prevalences were statistically significant for phenotypes I, II, VI, VII and VIII.

Table 54: Prevalence of the phenotypes of the double burden of malnutrition and BCMRF in the study population by Region of resident

Regions Phenotypes	Center Region	Littoral Region	Western Region	North West Region	North Region	p-value
Phenotype I	28(4.8%)	38(6.2%)	80(13.2%)	147(24.2%)	7(1.2%)	0.000
Phenotype II	23(3.8%)	34(5.6%)	65(10.7%)	90(14.8%)	3(0.5%)	0.012
Phenotype III	5(0.8%)	13(2.1%)	23(3.8%)	26(4.3%)	2(0.3%)	0.642
Phenotype IV	7(1.2%)	10(1.6%)	31(5.1%)	49(8.1%)	0(0.0%)	0.001
Phenotype V	3(0.5%)	7(1.2%)	9(1.5%)	21(3.5%)	3(0.5%)	0.291
Phenotype VI	1(0.2%)	3(0.5%)	2(0.3%)	21(3.5%)	0(0.0%)	0.000
Phenotype VII	7(1.2%)	21(3.5%)	32(5.3%)	53(8.7%)	2(0.3%)	0.037
Phenotype VIII	0(0.0%)	0(0.0%)	1(0.2%)	7(1.2%)	0(0.0%)	0.048

Phenotype I: at least one mineral deficiency and one CMRF; **Phenotype II:** overweight or obese+ at least one other CMRF+ at least one mineral deficiency; **Phenotype III:** overweight or obese+ hypertension+ at least one mineral deficiency; **Phenotype IV:** overweight or obese+ hyperglycemia+ at least one mineral deficiency; **Phenotype V:** overweight or obese+ Hypertriglyceridemia + at least one mineral deficiency; **Phenotype VI:** overweight or obese+ Hypercholesterolemia + at least one mineral deficiency; **Phenotype VII:** overweight or obese+ MetS + at least one mineral deficiency, **Phenotype VIII:** Underweight + at least one mineral deficiency

III.1.13.3.1.2. Coexistence of the double burden of malnutrition and biological cardiometabolic risk factors in the study with respect to educational level

Phenotype I (11.7%), II (9.2%), IV (3.9%), V (2.1%) and VII (4.3%) were highest among women with a primary educational level followed by those with no educational level (Phenotype I (11.5%), II (7.6%), IV (3.6%) V (1.3%, the same percentage was observed for literates) and VII (4.1%)) then those with a first cycle of education although with regards to phenotype IV women who were literate (3.1%) had the highest prevalence after those with no education. Phenotype III was highest among illiterates (3.0%). While phenotype VI was mostly prevalent among first cycle of education (1.2%). Phenotype VIII whereas was highest among women with primary (0.5%) and those with first cycle education (0.5%).

Table 55: Prevalence of the phenotypes of the double burden of malnutrition and BCMRF in the study population by educational level

Phenotypes	Illiterate N (%)	Literate N (%)	Primary N (%)	1 st cycle N (%)	2 nd cycle N (%)	University N (%)	Total N (%)	P-value
Phenotype I	69(11.3)	52(8.6)	71(11.7)	62(10.2)	44(7.2)	3(0.5)	301(49.5)	0.002
Phenotype II	46(7.6)	33(5.4)	56(9.2)	43(7.1)	35(5.8)	2(0.3)	215(35.4)	0.261
Phenotype III	18(3.0)	9(1.5)	17(2.8)	14(2.3)	10(1.6)	1(0.2)	69(11.3)	0.694
Phenotype IV	22(3.6)	19(3.1)	24(3.9)	18(3.0)	14(2.3)	0(0.0)	97(16.0)	0.225
Phenotype V	8(1.3)	8(1.3)	13(2.1)	7(1.2)	7(1.2)	0(0.0)	43(7.1)	0.717
Phenotype VI	4(0.7)	6(1.0)	6(1.0)	7(1.2)	4(0.7)	0(0.0)	27(4.4)	0.662
Phenotype VII	25(4.1)	19 (3.1)	26(4.3)	22(3.6)	22(3.6)	1(0.2)	115(18.9)	0.755
Phenotype VIII	2(0.3)	0(0.0)	3(0.5)	3(0.5)	0(0.0)	0(0.0)	8(1.3)	0.410

Phenotype I: at least one mineral deficiency and one CMRF; **Phenotype II:** overweight or obese+ at least one other CMRF+ at least one mineral deficiency; **Phenotype III:** overweight or obese+ hypertension+ at least one mineral deficiency; **Phenotype IV:** overweight or obese+ hyperglycemia+ at least one mineral deficiency; **Phenotype V:** overweight or obese+ Hypertriglyceridemia + at least one mineral deficiency; **Phenotype VI:** overweight or obese+ Hypercholesterolemia + at least one mineral deficiency; **Phenotype VII:** overweight or obese+ MetS + at least one mineral deficiency, **Phenotype VIII:** Underweight + at least one mineral deficiency

III.1.13.3.1.3. Coexistence of the double burden of malnutrition and biological cardiometabolic risk factors in the study with respect to marital status

Married women recorded the highest percentage of all the phenotypes when marital status was concerned.

Table 56: Prevalence of the phenotypes of the double burden of malnutrition and BCMRF in the study population with respect to marital status

Phenotypes	Single N (%)	Married N (%)	Widow/ divorced N (%)	Total N (%)	P-value
Phenotype I	25(4.1)	215(35.4)	61(10.0)	301(49.5)	0.005
Phenotype II	18(3.0)	154(25.3)	43(7.1)	215(35.4)	0.066
Phenotype III	2(0.3)	51(8.4)	16(2.6)	69(11.3)	0.027
Phenotype IV	6(1.0)	77(12.7)	14(2.3)	97(16.0)	0.074
Phenotype V	3(0.5)	31(5.1)	9(1.5)	43(7.1)	0.497
Phenotype VI	0(0.0)	20(3.3)	7(1.2)	27(4.4)	0.095
Phenotype VII	4(0.7)	88(14.5)	23(3.8)	115(18.9)	0.006
Phenotype VIII	0(0.0)	6(1.0)	2(0.3)	8(1.3)	0.525

Phenotype I: at least one mineral deficiency and one CMRF; **Phenotype II:** overweight or obese+ at least one other CMRF+ at least one mineral deficiency; **Phenotype III:** overweight or obese+ hypertension+ at least one mineral deficiency; **Phenotype IV:** overweight or obese+ hyperglycemia+ at least one mineral deficiency; **Phenotype V:** overweight or obese+ Hypertriglyceridemia + at least one mineral deficiency; **Phenotype VI:** overweight or obese+ Hypercholesterolemia + at least one mineral deficiency; **Phenotype VII:** overweight or obese+ MetS + at least one mineral deficiency, **Phenotype VIII:** Underweight + at least one mineral deficiency

III.1.13.3.2. Coexistence of the double burden of malnutrition and biological cardiometabolic risk factors in the study with respect to nutritional parameters

III.1.13.3.2.1. Coexistence of the double burden of malnutrition and biological cardiometabolic risk factors in the study with respect to Food Consumption score (FCS) threshold

Participants with an acceptable FCS recorded the highest prevalence of all the phenotypes (Phenotype I: 23.7%; Phenotype II: 16.4%; Phenotype III: 5.3%; Phenotype IV: 8.9%; Phenotype V: 3.6%; Phenotype VI: 3.5%; VII: 10.0% and VIII: 0.7%) compared to those with a poor and borderline FCS. The differences were statistically significant ($p < 0.05$) between all the groups of the various phenotypes of the DBM & BCMRFs except for phenotypes V and VIII ($p > 0.05$).

Table 57: Prevalence of the phenotypes of the double burden of malnutrition and BCMRF in the study population with respect to FCS

Phenotypes	Poor N (%)	Borderline N (%)	Acceptable N (%)	Total N (%)	P-value
Phenotype I	75(12.3)	82(13.5)	144(23.7)	301(49.5)	0.000
Phenotype II	59(9.7)	56(9.2)	100(16.4)	215(35.4)	0.025
Phenotype III	26(4.3)	11(1.8)	32(5.3)	69(11.3)	0.047
Phenotype IV	21(3.5)	22(3.6)	54(8.9)	97(16.0)	0.001
Phenotype V	9(1.5)	12(2.0)	22(3.6)	43(7.1)	0.178
Phenotype VI	3(0.5)	3(0.5)	21(3.5)	27(4.4)	0.000
Phenotype VII	31(5.1)	23(3.8)	61(10.0)	115(18.9)	0.003
Phenotype VIII	1(0.2)	3(0.5)	4(0.7)	8(1.3)	0.490

Phenotype I: at least one mineral deficiency and one CMRF; **Phenotype II:** overweight or obese+ at least one other CMRF+ at least one mineral deficiency; **Phenotype III:** overweight or obese+ hypertension+ at least one mineral deficiency; **Phenotype IV:** overweight or obese+ hyperglycemia+ at least one mineral deficiency; **Phenotype V:** overweight or obese+ Hypertriglyceridemia + at least one mineral deficiency; **Phenotype VI:** overweight or obese+ Hypercholesterolemia + at least one mineral deficiency; **Phenotype VII:** overweight or obese+ MetS + at least one mineral deficiency, **Phenotype VIII:** Underweight + at least one mineral deficiency

III.1.13.3.2.2. Coexistence of the double burden of malnutrition and biological cardiometabolic risk factors in the study with respect to household food insecurity access score (HFIAS) threshold

Whatever the phenotype, participants in food secure households were those with the highest prevalence (Phenotype I: 27 %; Phenotype II: 18.8 %; Phenotype III: 5.7 %; Phenotype IV: 9.8 %; Phenotype V: 4.7 %; Phenotype VI: 2.4%; VII: 10.9 % and VIII:

0.7%). The differences between the various household food security groups was significant only for phenotypes I and IV ($p < 0.05$).

Table 58: Prevalence of the phenotypes of the double burden of malnutrition and BCMRF in the study population with respect to HFIAS threshold

Phenotypes	Food secure N (%)	Mildly food insecure N (%)	Moderate and Severely food insecure N (%)	Total N (%)	P-value
Phenotype I	164 (27)	53 (8.7)	84 (13.8)	301(49.5)	0.006
Phenotype II	114 (18.8)	42 (6.9)	59 (9.7)	215(35.4)	0.408
Phenotype III	35 (5.7)	14 (2.3)	20 (3.3)	69 (11.3)	0.945
Phenotype IV	59 (9.8)	12 (2)	26 (4.2)	97(16.0)	0.029
Phenotype V	28 (4.7)	3(0.5)	12 (1.9)	43(7.1)	0.113
Phenotype VI	15 (2.4)	4 (0.7)	8 (1.3)	27(4.4)	0.592
Phenotype VII	66(10.9)	20 (3.2)	29 (4.8)	115(18.9)	0.237
Phenotype VIII	4(0.7)	2(0.3)	2(0.3)	8(1.3)	0.918

Phenotype I: at least one mineral deficiency and one CMRF; **Phenotype II:** overweight or obese+ at least one other CMRF+ at least one mineral deficiency; **Phenotype III:** overweight or obese+ hypertension+ at least one mineral deficiency; **Phenotype IV:** overweight or obese+ hyperglycemia+ at least one mineral deficiency; **Phenotype V:** overweight or obese+ Hypertriglyceridemia + at least one mineral deficiency; **Phenotype VI:** overweight or obese+ Hypercholesterolemia + at least one mineral deficiency; **Phenotype VII:** overweight or obese+ MetS + at least one mineral deficiency, **Phenotype VIII:** Underweight + at least one mineral deficiency

III.1.13.3.2.3. Coexistence of the double burden of malnutrition and biological cardiometabolic risk factors in the study with respect to fruits intake

The highest prevalence of all the phenotypes (Phenotype I: 38.0%; Phenotype II: 26.5%; Phenotype III: 8.2%; Phenotype IV: 12.5%; Phenotype V: 5.6%; Phenotype VI: 2.3%; Phenotype VII: 13.7% and Phenotype VIII: 1.2%) were recorded among participants who had a low intake in a week. The differences were statistically significant for all the categories of fruit consumption for all the phenotypes except for phenotypes III, IV, V and VIII.

Table 59: Prevalence of the phenotypes of the double burden of malnutrition and BCMRF in the study population with respect to intake fruits

Phenotypes	Low intake (1-2 times /week) N (%)	Moderate intake (3-4 times /week) N (%)	High intake (5-7 times/week) N (%)	Total N (%)	P-Value
Phenotype I	231(38.0)	48(7.9)	22(3.6)	301 (49.5)	0.022
Phenotype II	161(26.5)	36(5.9)	18(3.0)	215(35.4)	0.011
Phenotype III	50(8.2)	14(2.3)	5(0.8)	69 (11.3)	0.140
Phenotype IV	76(12.5)	13(2.1)	8(1.3)	97(16.0)	0.458
Phenotype V	34(5.6)	5(0.8)	4(0.7)	43 (7.1)	0.532
Phenotype VI	14(2.3)	9(1.5)	4(0.7)	27(4.4)	0.000
Phenotype VII	83(13.7)	21(3.5)	11(1.8)	115(18.9)	0.018
Phenotype VIII	7(1.2)	1(0.2)	0(0.0)	8(1.3)	0.779

Phenotype I: at least one mineral deficiency and one CMRF; **Phenotype II:** overweight or obese+ at least one other CMRF+ at least one mineral deficiency; **Phenotype III:** overweight or obese+ hypertension+ at least one mineral deficiency; **Phenotype IV:** overweight or obese+ hyperglycemia+ at least one mineral deficiency; **Phenotype V:** overweight or obese+ Hypertriglyceridemia + at least one mineral deficiency; **Phenotype VI:** overweight or obese+ Hypercholesterolemia + at least one mineral deficiency; **Phenotype VII:** overweight or obese+ MetS + at least one mineral deficiency, **Phenotype VIII:** Underweight + at least one mineral deficiency

III.1.13.3.2.4. Coexistence of the double burden of malnutrition and biological cardiometabolic risk factors in the study with respect to vegetables intake

Phenotypes I (24.2%) ($p < 0.05$), II (16.4%) ($p > 0.05$), III (4.6%) ($p > 0.05$), IV (8.4%) ($p < 0.05$), V (3.8%) ($p > 0.05$), VI (3.3%) ($p < 0.05$), VII (9.2%) ($p > 0.05$) and VIII (0.7%) ($p > 0.05$) were mostly observed among participants with a high consumption of vegetables.

Table 60: Prevalence of the phenotypes of the double burden of malnutrition and BCMRF in the study population with respect to intake of vegetables

Phenotypes	Low intake (1-2 times/week) N (%)	Moderate (3-4 times/week) N (%)	High (5-7 times/week) N (%)	Total N (%)	P-value
Phenotype I	81(13.3)	73(12.0)	147(24.2)	301(49.5)	0.001
Phenotype II	61(10.0)	54(8.9)	100(16.4)	215(35.4)	0.092
Phenotype III	20(3.3)	21(3.5)	28(4.6)	69(11.3)	0.312
Phenotype IV	22(3.6)	24(3.9)	51(8.4)	97(16.0)	0.028
Phenotype V	9(1.5)	11(1.8)	23(3.8)	43(7.1)	0.155
Phenotype VI	3(0.5)	4(0.7)	20(3.3)	27(4.4)	0.003
Phenotype VII	29(4.8)	30(4.9)	56(9.2)	115(18.9)	0.085
Phenotype VIII	2(0.3)	2(0.3)	4(0.7)	8(1.3)	0.857

Phenotype I: at least one mineral deficiency and one CMRF; **Phenotype II:** overweight or obese+ at least one other CMRF+ at least one mineral deficiency; **Phenotype III:** overweight or obese+ hypertension+ at least one mineral deficiency; **Phenotype IV:** overweight or obese+ hyperglycemia+ at least one mineral deficiency; **Phenotype V:** overweight or obese+ Hypertriglyceridemia + at least one mineral deficiency; **Phenotype VI:** overweight or obese+ Hypercholesterolemia + at least one mineral deficiency; **Phenotype VII:** overweight or obese+ MetS + at least one mineral deficiency, **Phenotype VIII:** Underweight + at least one mineral deficiency

III.1.13.3.3. Coexistence of the double burden of malnutrition and biological cardiometabolic risk factors in the study with socioeconomic status score

The table below presents the frequencies of the various phenotypes of the DBM & BCMRFs with respect to socioeconomic status. It can be noted that whatever the phenotype, women with a high SES recorded the highest percentages.

Table 61: Prevalence of the phenotypes of the double burden of malnutrition and BCMRF in the study population with respect to SES score

Phenotypes	Low SES N (%)	Medium SES N (%)	High SES N (%)	Total N (%)	P-value
Phenotype I	2(0.4)	23(4.5)	218(43.0)	243(47.9)	0.000
Phenotype II	2(0.4)	17(3.4)	156(30.8)	175(34.5)	0.005
Phenotype III	0(0.0)	11(2.2)	45(8.9)	56(11.0)	0.310
Phenotype IV	1(0.2)	3(0.6)	72(14.2)	76(15.0)	0.005
Phenotype V	0(0.0)	6(1.2)	29(5.7)	35(6.9)	0.579
Phenotype VI	0(0.0)	3(0.6)	21(4.1)	24(4.7)	0.613
Phenotype VII	0(0.0)	11(2.2)	84(16.6)	95(18.7)	0.073
Phenotype VIII	0(0.0)	0(0.0)	6(1.2)	6(1.2)	0.501

Phenotype I: at least one mineral deficiency and one CMRF; **Phenotype II:** overweight or obese+ at least one other CMRF+ at least one mineral deficiency; **Phenotype III:** overweight or obese+ hypertension+ at least one mineral deficiency; **Phenotype IV:** overweight or obese+ hyperglycemia+ at least one mineral deficiency; **Phenotype V:** overweight or obese+ Hypertriglyceridemia + at least one mineral deficiency; **Phenotype VI:** overweight or obese+ Hypercholesterolemia + at least one mineral deficiency; **Phenotype VII:** overweight or obese+ MetS + at least one mineral deficiency, **Phenotype VIII:** Underweight + at least one mineral deficiency

III.1.13.4. Predictors of the various phenotypes of the double burden of malnutrition and BCMRF at the individual level

Regression analysis revealed that predictors of phenotype I were a secondary educational level (OR: 2.171, p=0.003) and a university level (OR: 6.192, p=0.006), a rare intake of fruits in a week (OR: 2.079, p=0.048), a low intake of vegetables (OR: 2.042, p=0.000), being in a mildly food insecure household (OR: 2.500, p=0.002). Women in households with a high SES were less likely of developing the characteristics of this phenotype. Also, married women widows and divorced women were less likely of developing the characteristics of this phenotype.

Predictors of phenotype II were: a university level, a low intake of fruits and a low intake of vegetables in a week.

A borderline FCS (OR: 2.280, p=0.028) was a predictor of phenotype III.

Risk factors of phenotype VI were, a low intake of vegetables and being in a mildly food insecure household.

A low intake of vegetable was a predictor of phenotype V (OR=2.329, p=0.038) after adjusting for age.

Table 62: Predictors of the Phenotypes (I, II, III, IV, V) of the double burden and BCMRF at the individual level adjusted for age

Phenotypes	Phenotype I		Phenotype II		Phenotype III		Phenotype IV		Phenotype V	
Risk factors	OR (95% CI)	P	OR (95% CI)	P	OR (95% CI)	P	OR (95% CI)	P	OR (95% CI)	P
Age group										
14-20 ^J	1.0		1.0		1.0		1.0		1.0	
21-30	1.323(0.730-2.400)	0.356	0.421 (0.190-0.932)	0.033	0.363 (0.043-3.079)	0.353	0.321 (0.091-1.128)	0.076	0.891 (0.168-4.725)	0.893
31-40	1.176(0.669-2.067)	0.573	0.263 (0.123-0.562)	0.001	0.130 (0.017-0.986)	0.048	0.225 (0.067-0.756)	0.016	0.370 (0.083-1.648)	0.192
41-49	0.983(4.956-14.452)	0.952	0.216 (0.101-0.461)	0.000	0.065 (0.009-0.480)	0.065	0.214 (0.063-0.719)	0.013	0.304 (0.069-1.342)	0.116
Educational Level										
Illiterate ^J	1.0		1.0		1.0		1.0		1.0	
Literate	0.944 (0.542-1.644)	0.839	0.966 (0.539-1.731)	0.906	1.579(0.653-3.815)	0.310	0.748(0.372-1.507)	0.417	0.672(0.239-1.893)	0.452
Primary	1.439 (0.888-2.333)	0.140	1.085(0.655-1.799)	0.751	1.574(0.757-3.273)	0.225	1.211(0.637-2.301)	0.560	0.781(0.311-1.965)	0.600
First cycle	1.177(0.706-1.963)	0.532	1.015(0.592-1.740)	0.840	1.328(0.611-2.883)	0.474	1.167(0.585-2.330)	0.660	1.091(0.379-3.136)	0.872
Second cycle	2.171(1.291-3.652)	0.003	1.530(0.883-2.651)	0.129	2.055(0.890-4.747)	0.092	1.721(0.830-3.569)	0.145	1.151(0.402-3.301)	0.793
University	6.192(1.691-22.673)	0.006	4.787(1.036-22.111)	0.045	2.651(0.322-21.821)	0.365	/	/	/	/
Marital status										
Single ^J	1.0		1.0		1.0		1.0		1.0	
Married	0.508(0.303-0.852)	0.010	0.704(0.393-1.261)	0.238	0.289(0.068-1.233)	0.094	0.468(0.194-1.131)	0.092	0.680(0.199-2.318)	0.537
Widow/divorced	0.368(0.198-0.683)	0.002	0.500(0.254-0.985)	0.045	0.194(0.042-0.892)	0.035	0.610(0.220-1.691)	0.342	0.507(0.130-1.975)	0.328
SES										
Low ^J	1.0		1.0		1.0		1.0		1.0	
Medium	0.423 (0.087-2.047)	0.285	0.622 (0.126-3.075)	0.560	/	/	1.984 (0.191-20.619)	0.566	/	/
High	0.146 (0.032-0.666)	0.013	0.225 (0.049-1.029)	0.054	/	/	0.316(0.040-2.461)	0.217	/	/
FCS										
Poor ^J	1.0		1.0		1.0		1.0		1.0	
Borderline	0.701 (0.463-1.061)	0.093	0.870 (0.554-1.366)	0.546	2.280 (1.082-4.881)	0.030	0.795 (0.418-1.513)	0.485	0.612 (0.249-1.500)	0.283
Acceptable	0.412 (0.280-0.608)	0.000	0.509 (0.336-0.770)	0.001	0.839 (0.472-1.491)	0.549	0.359 (0.206 -0.625)	0.000	0.412 (0.184-0.926)	0.032
Fruit intake										
High intake ^J	1.0		1.0		1.0		1.0		1.0	
Moderate intake	1.260(0.549-2.894)	0.585	1.347(0.589-3.081)	0.480	0.686(0.217-2.171)	0.521	1.573(0.577-4.284)	0.376	1.905(0.473-7.673)	0.365
Low intake	2.079(1.007-4.295)	0.048	2.431(1.183-4.995)	0.016	1.534(0.551-4.273)	0.413	1.721(0.744-3.982)	0.205	1.802(0.594-5.463)	0.298
Vegetables intake										
High intake ^J	1.0		1.0		1.0		1.0		1.0	
Moderate intake	1.241(0.823-1.871)	0.304	1.080(0.702-1.662)	0.725	0.723(0.387-1.350)	0.309	1.266(0.737-2.173)	0.393	1.200(0.563-2.554)	0.637
Low intake	2.042(1.408-2.962)	0.000	1.657(1.111-2.472)	0.013	1.232(0.663-2.288)	0.509	2.241(1.301-3.862)	0.004	2.329(1.047-5.184)	0.038
Food security level										
Food secure ^J	1.0		1.0		1.0		1.0		1.0	
Mildly food insecure	2.500(1.414-4.419)	0.002	1.353(0.751-2.437)	0.315	1.021(0.430-2.421)	0.963	2.648(1.204-5.821)	0.015	3.990(0.892-17.841)	0.070
Moderately/severely food insecure	1.525(0.891-2.611)	0.123	1.064(0.616-1.838)	0.823	0.881(0.406-1.910)	0.748	1.331(0.711-2.489)	0.371	1.365(0.538-3.465)	0.513
Traditional diets										
High intake ^J	1.0		1.0		1.0		1.0		1.0	
Moderate intake	1.951(0.173-22.026)	0.589	0.971(0.074-12.703)	0.982	3.489(0.242-50.239)	0.358	/	/	/	/
Low intake	1.438(0.127-16.3065)	0.769	0.892(0.068-11.731)	0.931	3.776(0.258-55.307)	0.332	/	/	/	/

J = Reference category for comparison

Phenotype I: at least one mineral deficiency and one CMRF; **Phenotype II:** overweight or obese+ at least one other CMRF+ at least one mineral deficiency; **Phenotype III:** overweight or obese+ hypertension+ at least one mineral deficiency; **Phenotype IV:** overweight or obese+ hyperglycemia+ at least one mineral deficiency; **Phenotype V:** overweight or obese+ Hypertriglyceridemia + at least one mineral deficiency; **Phenotype VI:** overweight or obese+ Hypercholesterolemia + at least one mineral deficiency; **Phenotype VII:** overweight or obese+ MetS + at least one mineral deficiency, **Phenotype VIII:** Underweight + at least one mineral deficiency

Predictors of Phenotype VI included: a low intake of fruits in a week (OR: 4.696, p=0.010) and a moderate (OR: 3.088, p=0.044) and a low intake of vegetables in a week (OR: 5.982, p=0.004).

Predictors of phenotype VII included a low intake of fruits and a low intake of vegetables in a week.

None of the factors studied were predictors of phenotype VIII after adjusting for age.

Table 63: Predictors of the Phenotypes (VI, VII, VIII) of the double burden and BCMRF adjusted for age at the individual level

Phenotype	Phenotype VI		Phenotype VII		Phenotype VIII	
	OR (95% CI)	P	OR (95% CI)	P	OR (95% CI)	P
Age group						
14-20 ^J	1.0		1.0		1.0	
21-30	/		0.118 (0.015-0.905)	0.040	1.123 (0.200-6.296)	0.895
31-40	/		0.066 (0.009-0.491)	0.008	6.820 (0.608-76.491)	0.120
41-49	/		0.040 (0.005-0.296)	0.002	6.361 (0.5672-71.362)	0.134
Educational Level						
Illiterate ^J	1.0		1.0		1.0	
Literate	0.460 (0.124-1.701)	0.244	0.907 (0.451-1.824)	0.784	/	
Primary	0.870 (0.239-3.172)	0.833	1.383 (0.739-2.586)	0.311	0.712 (0.115-4.419)	0.715
First cycle	0.523 (0.148-1.852)	0.315	1.111 (0.574-2.149)	0.755	0.682 (0.110-4.246)	0.682
Second cycle	1.024 (0.249-4.205)	0.974	1.213 (0.631-2.333)	0.562	/	
University	/		4.185 (0.522-33.566)	0.178	/	
Marital status						
Single ^J	1.0		1.0		1.0	
Married	/		0.288 (0.101-0.822)	0.020	/	
Widow/divorced	/		0.237 (0.076-0.732)	0.012	/	
SES						
Low ^J	1.0		1.0		1.0	
Medium	/		/		/	
High	/		/		/	
FCS						
Poor ^J	1.0		1.0		1.0	
Borderline	0.911 (0.180-4.599)	0.910	1.215 (0.669-2.204)	0.523	0.348 (0.035-3.429)	0.366
Acceptable	0.147 (0.043-0.504)	0.002	0.448 (0.272-0.738)	0.002	0.407 (0.044 -3.753)	0.428
Fruit intake						
High intake	1.0		1.0		1.0	
Moderate intake	0.993 (0.278-3.549)	0.993	1.247 (0.500-3.112)	0.636	/	
Low intake	4.696 (1.438-15.339)	0.010	2.503 (1.135-5.520)	0.023	/	
Vegetables intake						
High intake ^J	1.0		1.0		1.0	
Moderate intake	3.088 (1.031-9.251)	0.044	1.101 (0.658-1.842)	0.714	1.033 (0.184-5.802)	0.970
Low intake	5.982 (1.745-20.505)	0.004	1.892 (1.143-3.132)	0.013	1.341 (0.239-7.508)	0.739
Food security level						
Food secure ^J	1.0		1.0		1.0	
Mildly food insecure	1.881 (0.509-6.945)	0.343	1.589 (0.788-3.207)	0.196	0.820 (0.131-5.114)	0.831
Moderately/severely food insecure	0.899 (0.336-2.400)	0.831	1.283 (0.680-2.420)	0.441	1.052 (0.167-6.609)	0.957
HHDS						
Not acceptable ^J	1.0		1.0		1.0	
Acceptable	0.279 (0.095-0.822)	0.021	0.605 (0.385-0.951)	0.029	0.322 (0.039-2.679)	0.294
Traditional diet						
High ^J	1.0		1.0		1.0	
Moderate	11.856 (0.659-213.355)	0.094	2.224 (0.148-33.392)	0.563	/	
Low	15.301 (0.867-270.163)	0.063	1.814 (0.120-27.453)	0.668	/	

J = Reference category for comparison; **Phenotype VI**: overweight or obese+ Hypercholesterolemia + at least one mineral deficiency; **Phenotype VII**: overweight or obese+ MetS + at least one mineral deficiency, **Phenotype VIII**: Underweight + at least one mineral deficiency

III.2. DISCUSSION

III.2.1. Baseline characteristics and Prevalence of poor nutritional states

The main aim of this study was to assess the prevalence of malnutrition still a great public health issue in Cameroon, among women of childbearing age. Baseline characteristics of the study population showed that, the mean age was 34.68 ± 0.39 years. A higher percentage (34.4%) of the participants were in 31-40 years age group and up to 36.0 percent of the total population were from the North West region. 24.3 percent of the women had a primary educational level and 19.9 percent were illiterates. Up to 38.3 percent of the study population were housewives and most of the participants (70.6 percent) were married. Business was the professional activity which served as source of revenue for up to 38.0 percent of the participants. In addition, up to 81.5 percent of the study population were found in households with high socioeconomic status. Evaluation of food consumption score (FCS) revealed that, 39.3 percent of the study population had an acceptable FCS while 32.1 percent had a poor score. 48.8 percent of them were found in food secure households. Most of the women (86.4% and 56.9% respectively) had a high intake of ‘cereals and tubers’ and ‘Fats and oils’ food groups in a week. Whereas only 16.0 percent and 9.8 percent of them had a high intake of ‘milk and dairy products’ and ‘fruits’ food groups in a week respectively in the 95 percent confidence interval.

Assessment of the prevalence of malnutrition revealed that all the forms studied were present in the study population with calcium deficiency been the most prevalent form followed by obesity, overweight then magnesium deficiency, iron deficiency and underweight. Calcium deficiency and obesity all increased with age and their prevalence were highest in the 41-49 years age group; the other forms were highest in the 31-40 years age group. Malnutrition can take several forms including hunger, undernutrition, overnutrition and micronutrient deficiencies (Fanzo, 2012) and the present study shows the high rates of almost all these forms. The prevalence of underweight (2.1%) was lower than that reported among women aged 15-49 years in Cameroon by WHO (WHO, 2017). This estimate of underweight was also lower than those of other African nations namely: Togo, Rwanda, Benin, Chad, DR of Congo equally reported by WHO in 2017 among women of childbearing age. In this report, Cameroon has been cited among the

countries in WHO African regions with the lowest rates of underweight (WHO, 2017). Equally, the present observed rate of underweight was lower than the rate (6 %) of thin Cameroonian women observed in the 2018 Cameroon DHS (NIS & ICF, 2020). The low rate of underweight among women in Cameroon could be as a result of increased food production and availability although the eating habits are still very unhealthy among most Cameroonians in general. The observed rates of overweight (30.8%) and obesity (38.5%) were higher than the proportions observed in the 2004, 2011, 2018 DHSs which were 29.40% in 2004 as compared to 32.17% in 2011 and 37% in 2018 for obesity (DHS, 2004; DHS, INS and ORC Marco, 2011; NIS & ICF, 2020), making an increase of 2.77% between 2004 and 2011 (Yongsi and Ngwa, 2015) and an increase of 4.83 % between 2011 and 2018. These percentages show that the prevalence of underweight among reproductive women in Cameroon is on the decrease while that of overweight and obesity is on the rise.

Low plasma calcium (Ca) level was observed as the most prevalent (48.3%) mineral deficiency among the study population followed by magnesium (Mg) (22.4%) and then iron (Fe) (11.5%). Therefore, the prevalence of Ca deficiency was twice that of Mg and four times that of Fe, confirming the hypothesis that, interventions such as direct fortification and supplementations strategies (Tanya *et al.*, 2011), are improving iron status and leading to the reduction of Fe deficiency in Cameroon. For example, the prevalence of iron deficiency anemia often used as a proxy indicator for iron deficiency was 45 percent in 2004 (DHS and NIS, 2004) and 39.7 percent in 2011 as reported by WHO in 2017 (WHO, 2017) among women of childbearing in Cameroon, which are higher than the present rate of iron deficiency (11.5%) showing that iron deficiency and related conditions are decreasing. In the recent DHS, the prevalence of anaemia among women has been reported not to have changed since 2011 (NIS & ICF, 2020). Iron deficiency anemia has been reported to increase at puberty and menstruating. One menstruating woman out of five has depleted iron stores. Also, it has been reported in a review that iron indices such as hemoglobin, ferritin, and % transferrin saturation have been observed to be lower in menstruating women than women in their luteal phase (Miller, 2016). Infections such as malaria and hookworms, which cause blood loss and

diets with very low iron content and low bioavailability might also be causes of iron deficiency in this study population.

The assessment of Ca and Mg deficiencies are neglected and their prevalence are high in various regions of the country especially among women. To our knowledge, in Cameroon, no published data are available on the prevalence of calcium and magnesium deficiencies among women of childbearing age. However, a study on the dietary mineral supplies in Africa found that the risk of dietary calcium deficiency is estimated at 11-25 percentage and those for magnesium and iron deficiencies are estimated at 0-10 percentage respectively in Cameroon due to inadequate intakes. While 108.9 million i.e. 36 percent of people in West Africa have been estimated to have dietary calcium deficiency; 0.4 million i.e. 0.1 percent have been estimated to have dietary magnesium deficiency and 0.9 million i.e. 0.3 percent for iron deficiency (Joy *et al.*, 2014). The observed prevalence of Ca was higher than that observed by Saeedian *et al.* among women of childbearing age in Iran which were 3.2% but that of Mg was slightly lower than the rate of magnesium deficiency they observed which was 22.6% (Saeedian *et al.*, 2015). The high prevalence (above the threshold of public health significance of 15 percent (WHO, 2010)) of Ca and Mg deficiencies in the population can be explained by the low intake of food sources of these minerals. For example, in the present study population, it was observed that, only 16.0% of participants had a frequent intake of milk and dairy products, 27.3% of them a high intake of meat, fish and eggs, 33.1% had a frequent intake of vegetables and only 9.8% a frequent intake of fruits in a week; all of which are potential sources of these minerals. But it has been pointed out that decrease calcium intake alone rarely results in calcium deficiency and other causes may include low magnesium imbalance, malabsorption, a variety of drugs, acute pancreatitis etc. (Metheny, 2012). Hypomagnesium whereas has been linked to reduced intake caused by poor nutrition, from reduced absorption and increased gastrointestinal loss, such as in chronic diarrhoea, malabsorption or bowel resection/bypass, poor condition or disease (Swaminathan, 2003; Jahnen-Dechent and Ketteler, 2012). Deficiencies might also be triggered by increased magnesium excretion in some medical conditions such as diabetes mellitus, renal tubular disorders, hypercalcaemia, hyperthyroidism or

aldosteronism or in the course of excessive lactation or use of diuretics etc (Jahnen-Dechent and Ketteler, 2012).

III.2.2. Determinants of poor nutritional status

III.2.2.1. Sociodemographic Determinants of poor nutritional status

The evaluation of the sociodemographic determinants of poor nutritional status revealed that, the northwest region showed the highest prevalence of all the forms of malnutrition except that of obesity which was highest in the western region, the latter showing the next highest rates after the northwest region. Mg deficient individuals were absent in Yaoundé and the north region. In the 2004 DHS, the northwest and western regions were also among the regions with the highest prevalence of maternal overweight (DHS, 2004). The fact that these regions recorded the highest prevalence of all the forms of malnutrition could be linked to their dietary habits. The northwest and the west regions are zones of Cameroon that have humid and wet climates, influencing natural food resources and dietary habits. Tubers are available all year round in these regions, bean species are the main sources of plant protein (Nkengfack *et al.*, 2011). Tubers and fufu made from some tubers are often eaten in these region which provide the bulk of energy in the diets. In addition, many of the traditional soups and dishes of these regions, contain large amounts of palm oil, the basic fat used in the Cameroonian kitchen (Mennen *et al.*, 2000). Tubers and palm oil which are energy dense but poor in nutrients like Ca and Mg are usually eaten with small amounts of vegetables which provide little amounts of micronutrients. Furthermore, the beneficial effects of micronutrients found in these vegetable may be hindered by the large quantities of refined palm oil and sometimes groundnuts with which these vegetables are prepared (Ntentie *et al.*, 2014). The soup of a traditional diet like “Achu and yellow soup”, frequently eaten in both regions has been shown to be very low in Ca and Mg; and to contain no Fe in 100g edible portion compared to groundnut soup, eru, okro pod soup (Sharma *et al.*, 2007) but has high energy content contributed by cocoyam (achu). Therefore, the energy dense and low micronutrients content of most diets coupled to the lack of dietary diversification in these regions might be reasons for the high prevalence of all the forms of malnutrition. Also, high rates of overweight and

obesity might equally be attributed to low levels of physical activity observed among most women of the study. In the Center region and Littoral, no underweight women were observed instead, most women were overweight and obese in both regions. The low physical activity observed in this study population in general might account for these high rates of overnutrition in both regions. Also, the fact that in the Center region the study was carried out in an urban area (Yaounde) and most study participant were from the semi-urban area (Nkongsamba) in the Littoral region might also explain this high rates of overweight and obesity observed in these regions. These urban and semi-urban areas might favour the onset of overweight and obesity among women residing there; since it has been observed by Ntentie and collaborators that overweight and obesity rates were highest in these urbanization areas (Ntentie *et al.*, 2014). Mg deficient participants were not observed in the Center region but a low rate was observed in the Littoral region. The results observed in the Center region with respect to Mg status could be due to the high mean frequency intake of vegetables and moderate mean frequency intake of pulses; all of which are food sources of magnesium observed in the center region compared to other regions in this study. Vegetables especially green leafy vegetable have a high content of Magnesium since this element forms the center of the green pigment chlorophyll of these group of vegetables making them major sources of Mg (Grober *et al.*, 2015). The same observation was made in the Far-North region with regards to Mg status.

Higher rates of magnesium and calcium deficiencies were associated to participants of the study with no educational level; higher rates of overweight and obesity were associated to a primary educational level while that of iron deficiency to a first cycle of education. Housewives and married women recorded the highest numbers of all the forms of malnutrition with calcium deficiency been the most prevalent mineral deficiency among them. The high prevalence of Ca and Mg deficiencies among those with no educational level is consistent with the results observed in a study in Nepal whereby more respondents who did not attend school at all had poor nutritional status (Acharya *et al.*, 2017). The high level of overweight and obesity among women with a primary educational is consistent with a finding made in Cameroon which showed that women aged 15-49 years in Cameroon with a primary educational level were 1.47 times

more likely to be overweight (Yongsi and Ngwa, 2015). The fact that a higher prevalence of all the forms of malnutrition were associated to no or low educational levels (illiteracy, primary educational level and secondary level) may be due to the fact that this group are less empowered to access economic resources and therefore less likely to get access to quality and diversified foods, rich in micronutrients, low in calories and saturated fatty acids that enhance better micronutrient status and prevent weight gain. Although as pointed out by Fezeu *et al.* being educated in Cameroon is not necessarily associated with a better remuneration in the job market (Fezeu *et al.*, 2005). A study carried out in the North west region of Cameroon among university students, stated limited knowledge as possible reason for unhealthy dietary patterns and high obesity rates among this population group (Niba *et al.*, 2017); this could equally account for the high rates of overweight/obesity and other forms of malnutrition observed among women with no educational level and those with a low level in this same region in the present study and in the entire study population equally. The result that, a high level of Ca deficiency was observed among housewives and married women in this study, may be due to the lack of good dietary habits. For example, the infrequent or lack of consumption of Ca rich foods such as milk and dairy products has been observed among most Cameroonian women and in the African population in general (Prentice *et al.*, 2009). This observation is supported by the finding from our study whereby, only 16% of the study population frequently took milk and dairy products in a week; a finding which needs nutritional intervention.

III.2.2.2. Nutritional determinants of poor nutritional states

Mg deficiency was highest among those with a low intake of traditional diets (TDs) ($p < 0.001$). The fact that a high SES, an acceptable FCS and been in a food secure household were all associated to higher rates of almost all the forms of malnutrition studied may be explained by the ongoing nutritional transition which Cameroon is experiencing in all its localities especially the semi-urban areas as observed by Ntentie and collaborators (Ntentie *et al.*, 2014). Despite the fact that in the present study most households were food secure and had a high SES, and the fact that most participants had an acceptable FCS, most participants of the study in general have lifestyles that reflect

those of western societies; characterized by physical inactivity, low consumption of fruits and vegetables, and infrequent intake of traditional diets mostly replaced by energy dense processed foods low in micronutrients. These may explain the observed association between higher rates of various forms of malnutrition studied and a high SES, an acceptable FCS and a food secure household and equally the low intake of TD in a week.

The frequent intake of cereals and tubers was associated to higher rates of all the forms of malnutrition while low intakes of pulses; milk and dairy products; vegetables; fruits and sugar food groups in a week were all associated to higher rates of all forms of malnutrition. Also, a low weekly intake of meat, fish and eggs, was associated to higher levels of iron and calcium deficiencies. In all these cases, the number of calcium deficient individuals were highest. Optimum intake of food sources of various micronutrients are known to prevent deficiencies. All forms of undernutrition, occur due to insufficient intake or even sufficient intakes combined with impaired absorption due to infection, disease, or inflammation (Bailey *et al.*, 2015). Equally, the fact that most women of the study consumed nutrient dense food groups (pulses; meat, fish and egg; milk and dairy products) only 1-2 times a week might explain the above observed high rates of all forms of malnutrition. These three food groups have the highest weights according to the World Food Programme (WFP, 2008) and therefore when eaten in sufficient quantities contribute substantially to the FCS of an individual and to good nutritional status. It should be noted that the determination of food group weights is based on an interpretation of nutrient density. Nutrient density is a term used to subjectively describe a food groups's quality in terms of caloric density, macro- and micronutrient content, and actual quantities typically eaten. Although subjective, this weighting attempts to give greater importance to foods such as meat and fish, usually considered to have greater nutrient density and lesser importance to foods such as sugar. The highest weight was attached to foods with relatively high energy, good quality protein, and a wide range of micronutrients that can be easily absorbed (Wiesmann *et al.*, 2009). These various mechanisms may explain the association between higher rates of underweight and mineral deficiencies with low intakes of nutrient dense food groups. However, the higher levels of all forms of malnutrition in association to frequent intake

of cereals and tubers in this study, may be related to the high energy and low micronutrients content of food items of this group. In addition, the high level of anti-nutrients such as phytates in cereals and tubers (Joy *et al.*, 2014) frequently eaten in this study population, might also lead to a high prevalence of mineral deficiencies especially Magnesium and Calcium.

Fe level was negatively correlated to cereal and tubers intake. Meanwhile, Magnesium plasma level decreased as the frequency of intake of pulses and beans; meat, fish and egg food groups increased in a week. Also, Ca plasma level was negatively correlated to meat, fish and egg intake. This negative association between the frequency of intake of mineral food sources and plasma level of the various minerals studied may be attributed to the presence of anti-nutrients in the food source or the diets containing the mineral food sources. Anti-nutrients have been shown to inhibit the intestinal absorption of some key minerals like Ca, Fe, Mg and Zn there by reducing their dietary bioavailability (Hurrell and Egli, 2010; Joy *et al.*, 2014). For example anti-nutrients such as phytic acid salts, referred to collectively as phytate are the compounds which have received the most attention (Joy *et al.*, 2014). Phytate is a potent inhibitor of Fe, Mg, Ca and Zn absorption in the human intestine (Hurrell and Egli, 2010; Miller *et al.*, 2007; Joy *et al.*, 2014). It has been reported that, 68% of total dietary phytate supply in Africa is from ‘Cereals’, with 17% from ‘Pulses and Beans’ and 7% from ‘Roots and Tubers’ (Joy *et al.*, 2014). Also, with particular reference to calcium, not only is dairy product intake minimal in many African countries, but typical diets also contain high amounts of phytates, oxalates and tannins that are likely to reduce the absorption of calcium (Prentice *et al.*, 2009). These anti-nutrients may therefore be contributory factors to the lack of association or negative association between food mineral sources intake and the plasma concentrations of various minerals studied here. Nutrient-nutrient interaction may also lead to these negative associations observed (Joy *et al.*, 2014). For example, possible deficiency in vitamin D may also result in this negative association, since it is known that a deficiency in this vitamin reduces intestinal absorption of calcium by up to 50% (Fong and Khan, 2012), although the level of this vitamin was not evaluated in our study. With respect to magnesium level, high calcium intakes have been reported to caused lower absorption and great excretion of magnesium leading to

Mg deficiency (Rosanoff *et al.*, 2016) although we did not measure the dietary intakes of these minerals.

A low intake compared to a high intake of animal protein sources like fish, meat and eggs was a predictor of Mg deficiency and combined Ca and Mg deficiencies among the study participants. It has been reported that fish and meat have an intermediate magnesium concentration (Altura, 1994; Gröber *et al.*, 2015). Therefore, the low consumption of these food sources with intermediate Mg levels might be taken as earlier stated with high quantities of foods such as cereals with very high levels of anti-nutrients such as phytate, oxalates and tubers with intermediate levels (Joy *et al.*, 2014). This might be the reason why a low intake of these animal protein food sources was a predictor of not only Mg deficiency but also that of combined Ca and Mg deficiencies.

Ca plasma concentration was strongly associated to the frequent intake of traditional diets in a week. It has been reported by Ponka and collaborators that, some traditional dishes in Cameroon have substantial amounts of minerals and other micronutrients. For example, Koki and “Tenue militaire” two traditional dishes frequently consumed among the participants in this study population, have been reported by these authors to contain 38.9 mg of calcium /100 g and 24.4mg of calcium /100g of edible portion respectively (Ponka *et al.*, 2016). So, the high mineral content, especially high calcium content of most traditional dishes frequently consumed by the women of the study, might enhance this positive association observed between the frequent intake of traditional diets and plasma calcium level. As suggested, the consumption of traditional dishes rich in key nutrients should be encouraged (Ponka *et al.*, 2016).

The frequent intake of sugar containing foods like beverages has been shown to be negatively associated with bone mineral density (Wyshak and Frisch, 1994; McGartland *et al.*, 2003). Also, it has been pointed out that too much simple sugar can decrease bone mineral density (Gazella, 2009). These mechanisms might lead to an increase calcium plasma level leading to a high Ca-Mg ratio. These can explain the observed positive correlation between frequent intake of sugar and sugar derivatives and the increase plasma Ca-Mg ratio.

III.2.3. Prevalence of multiple mineral deficiencies

The evaluation of multiple mineral deficiencies revealed that 16.8 percent of the participants of the study population had 2 mineral deficiencies and 1.6 percent had 3 deficiencies with the northwest region recording the highest prevalence and the far-north region recording no cases of two or three deficiencies. The prevalence of multiple deficiencies increased with age. 15.1 percent presented a Ca and Mg deficiency, 4.9 percent a Ca and Fe deficiency, 2.1 percent a Mg and Fe deficiency and 1.7 percent had all deficiencies (Ca, Mg and Fe) with the northwest region still presenting the highest prevalence of all these specific combined deficiencies followed by the west region. This presence of two or three mineral deficiencies in the study population could be the result of inadequate intakes of food sources of these minerals as noted in this study. For example, up to 57.0 percent of the participants of the study had an infrequent intake of calcium food sources like milk and dairy products in a week and only 16.0 percent a frequent intake in the study population. Also, the lack of dietary diversification and the monotony of meals among the general Cameroonian population might result in this observed prevalence of multiple mineral deficiencies at the individual level. Another cause could be the lack of nutritional knowledge which could be associated to the low educational level of most participants of the study. It was observed in this study that 24.1 percent of the subjects had a primary educational level and 19.9 percent were illiterate. Also, as earlier stated, a higher prevalence of individual mineral deficiencies (Ca deficiency and Mg deficiency) was associated to participants of the study with no educational level. Therefore this low educational level might portray the lack of a knowledge on the choice of healthy foods, rich in micronutrients that enhance good health. Studies have shown that nutritional knowledge in Cameroon is lacking (Nkengfack *et al.*, 2011; Niba *et al.*, 2017). The increase prevalence of specific mineral deficiencies with age indicate that intervention strategies should be age specific. Even though most countries recommend a range of age- and sex-specific nutrient intake values associated with minimum risk of insufficient intake and without risk of adverse effects for the majority of the population (Neufeld and Cameron, 2012), interventions strategies aiming at reducing multiple micronutrient deficiencies should be very age specific even within specific population groups like women of childbearing age.

Equally, intervention strategies should be in addition to age and sex specific, region specific with emphasis laid on the characteristics and cultures of each region. For example, the North West followed by the western region recorded almost the highest prevalence for all concurrent specific mineral deficiencies. This might be due to the representative sample population of the participants from this region to the study population size compared to the other regions. Notwithstanding this, the findings from this study suggest that, intervention strategies should also take into consideration the area or region of residence of the targeted population. Staple foods like root vegetables observed to be important sources of Calcium in Western Africa (Joy *et al.*, 2014), have also been found to be frequently consumed in the North West region of the country. Sweet potatoes, cassava used in making ‘garri’ a foodstuff greatly consumed in this region, irish potatoes, cocoyam could be bio fortified with calcium and magnesium and anti-nutrients like phytate in these various root vegetables that hinder the intestinal absorption of these mineral reduced in other to ameliorate the status of these minerals in this region. This could be coupled to the nutritional education of the people of the region on the benefits of the frequent intake of fruits and vegetables which they themselves cultivate so as to prevent multiple mineral deficiencies such as calcium, magnesium and iron at the individual level.

The 21-30, 31-40, and the 41-49 years age groups were all predictors of Ca deficiency and combined Ca and Mg deficiencies among the study participants of the study population. This finding can be supported by the fact that as pointed out, metabolism is altered due to aging (Hwalla *et al.*, 2017). Thereby slowing down or completely hindering the absorption of these minerals and leading to increase likeliness of mineral deficiencies with age.

III.2.4. Mineral-Mineral interaction

III.2.4.1. Magnesium Hypothesis of high plasma Ca-Mg ratio in Magnesium deficient individuals

Mean plasma Ca-Mg ratio was significantly higher among participants who were Mg deficient and those who were not Ca deficient compared to others ($p < 0.001$). Also, plasma Ca-Mg ratio was strongly and positively correlated to Ca level and negatively

correlated to Mg level in the plasma. In addition, a low intake of ‘meat, egg, fish’ food group was a predictor of Mg deficiency and combined Ca and Mg deficiencies in this study.

Mean glycemia was highest in the medium Ca-Mg ratio group compared to other groups ($p < 0.05$). Neither a low nor a high Ca-Mg ratio group were predictors of individual cardiometabolic risk factors after adjusting for age, calcium and magnesium status.

Plasma Ca-Mg ratio was strongly and significantly correlated to a waist circumference ≥ 80 cm ($r = 0.113$, $p = 0.029$)

A higher mean plasma Ca-Mg ratio among Mg deficient participants was confirmed by the observation that, plasma Ca-Mg ratio increased, as Mg level decreased and as Ca level increased in the plasma, as shown by bivariate correlation analysis. It has been observed that low or inadequate intakes of food sources of Magnesium might lead to dietary magnesium deficiency (Rosanoff *et al.*, 2012; Joy *et al.*, 2014). Similar observations were made in this study whereby, low intakes of the ‘meat, eggs, fish’ food groups were predictors of not just Mg deficiency but equally predictors of combined Ca and Mg deficiencies in an individual. In addition, other causes of Mg deficiency might include excessive excretion of this mineral due to poor health condition (Jahnen-Dechent and Ketteler, 2012). Therefore, a Mg deficient state, might result in calcium leaching from bones into the bloodstream consequently leading to the observed high plasma Ca-Mg ratio. It has been pointed out that Ca interacts and naturally antagonizes Mg in the absorption from the intestinal tract into the bloodstream (Huang *et al.*, 2014). Mg deficiency impacts on the bone indirectly by affecting the homeostasis of the two master regulators of calcium homeostasis, i.e., parathyroid hormone (PTH) and 1,25(OH)₂-vitamin D thus leading to hypocalcemia (Castiglioni *et al.*, 2013). Magnesium deficiency has been postulated to lead to elevated levels of parathyroid hormone which cause calcium to leach from bones. Insufficient parathyroid hormone levels result in excessive urinary calcium loss by reducing bone remodeling and low intestinal absorption of calcium (Cooper and Gittoes, 2008). Also, Mg deficiency could hinder the body’s production of vitamin D, further weakening bones. Bone

demineralization in all cases, can result in elevated plasma Ca concentration existing with low plasma Mg concentration in a Mg deficient individual and consequently a high plasma Ca-Mg ratio. This high Ca-Mg ratio has been found to be associated to higher rates of individual cardiometabolic risk factors such as abdominal obesity and high blood pressure in the present study, although it was not a risk factor for these CMRF parameters. Very few studies have assessed plasma or serum Ca-Mg ratio. A study reported that an elevated serum calcium to magnesium ratio was associated with an increased risk of high-grade prostate cancer adjusted for serum calcium and magnesium (Dai *et al.*, 2011). Equally, a study in 2018 has concluded that a high Ca-Mg ratio was significantly associated with all-cause and cardiovascular mortality (Sato *et al.*, 2018). As postulated, it is possible that the cellular calcium activation phenomenon is part of the pathology of a dietary magnesium deficit caused by low dietary magnesium, which can be exacerbated by a high dietary calcium-to-magnesium ratio, and this inappropriate calcium activation at the cellular level can lead to type 2 diabetes mellitus, cardiovascular diseases, or other manifestations of magnesium deficiency (Rosanoff *et al.*, 2012) like osteoporosis. Huang and collaborators also hypothesized that, inadequate Mg intakes may cause a decrease in the extracellular Mg, leading to the influx of Ca into the cells, which may ultimately contribute to the development of CVD and metabolic disorder (Huang *et al.*, 2014). A study showed that, abundant levels of calcium-regulating hormones such as PTH and calcitriol caused not only a decrease in bone mineral content but also increased intracellular calcium in vascular smooth muscle. Increased calcium ion concentration produces contraction and therefore vasoconstriction leading to high blood pressure (Martinez, 1998). High levels of calcitriol can stimulate influx of Ca⁺⁺ ions in adipocytes which might promote lipogenesis and prevents lipolysis by increasing fatty acid synthase activity and expression inhibiting hormone-sensitive lipase thereby resulting in increase overall adiposity (Torres and Sanjuliani, 2012). In addition, a strong physiological/cellular link between a rising intracellular ratio of calcium to magnesium and aspects of metabolic syndrome, including hypertension, hyperinsulinemia, insulin resistance, and left ventricular cardiac hypertrophy have been proven (Resnick, 1992). This evidence may explain the observation that, the rates of abdominal obesity and high blood pressure were

highest among the high Ca-Mg ratio group compared to the other groups in the present study. Also, it has been reported that, the high intracellular calcium induced by magnesium deficiency may induce both insulin resistance and hypertension (DiNicolantonio *et al.*, 2018).

Furthermore, very high Ca intakes can result in negative Mg balance if Mg intake is low. The Ca balance is positive with high Ca-Mg dietary ratios, but in such a circumstance, the Ca deposition can be in the soft tissues such as the arteries and kidneys, as well as in bones. High Mg intakes do not interfere with Ca retention and improve Ca retention unless intake is very low. This is implemented by the favorable effect Mg has on the hormones that control Ca absorption and its metabolism (Seelig, 2009).

III.2.5. Influence of multiple mineral deficiencies and specific mineral deficiencies on the prevalence of underweight and overnutrition and vice versa; and predictors of nutritional deficiencies and overweight/obesity

In this study, the prevalence of the double burden of malnutrition was studied at the individual level. It was observed that among underweight participants, up to 46.2% had two mineral deficiencies with those in the 21-30 years age group having the highest number of those with this nutritional double burden of malnutrition. 15.4 percent (2 women) were underweight and iron deficient, 38.5 percent (5 women) were underweight and magnesium deficient and 53.8 percent (7 women) were underweight with calcium deficiency. In Cameroon, no study has reported the prevalence of underweight in association to micronutrient deficiencies at the individual level. The Demographic and Health survey of 2004 assessed the prevalence of underweight and anemia at the population level among women of childbearing age and not at the individual level. According to this survey, seven percent of mothers of children under the age of five in Cameroon were underweight, and 45 percent of them were anemic (DHS, 2004). Most studies in Cameroon have reported the double burden mostly at the population level. For example, low plasma zinc level has been reported in Cameroon in 2014 among women of childbearing in association to child stunting. In this study, it was reported that 33% of children aged 12–59 months were stunted and 82% of women of childbearing age had low adjusted plasma zinc concentration (PZC); stunting and PZC

together with dietary zinc intake were the three indicators of zinc status which were used to measure the risk of zinc deficiency at the population level (Engle-Stone *et al.*, 2014). A study carried out in Vietnam on micronutrient deficiencies (zinc, vitamin B12, iron, folate, vitamin A) among reproductive age women found that the double burden of malnutrition in terms of overweight and underweight also existed at the population level (Lailou *et al.*, 2012). Also, a study in Ghana on the double burden of malnutrition in terms of overweight/obesity and underweight found that Overweight and obesity are becoming a common phenomenon among Ghanaian women while underweight still remains a problem (Doku & Neupane, 2015). Equally a very recent review article on the double burden of malnutrition among women of reproductive age in 55 low-and middle-income countries concluded that, although the prevalence of underweight declined, this declined has been superseded by the dramatic increase of overweight; and that none of the 55 LMICs is likely to eradicate malnutrition in women by 2030, Cameroon included (Hasan *et al.*, 2021).

Among women who were overweight, up to 34.2% of them exhibited one mineral deficiency, with those in the 31-40 years age group exhibiting the highest prevalence. 14.4 percent of overweight had two mineral deficiencies, with the 41-49 years age group showing the highest prevalence of this double burden. 8.6 percent of overweight women were also iron deficient, 21.9 percent were overweight and magnesium deficient and 60.4 percent overweight and calcium deficient. Women in the 41-49 years age group recorded the highest prevalence of those who were obese and had one mineral deficiency and those who were obese with two mineral deficiencies. Among those with this weight status (obese), 7.3 percent were iron deficient, 20.5 percent were magnesium deficient and 38.9 percent were calcium deficient in the 95% CI. These results portray the fact that in Cameroon among women of childbearing age, the double burden of malnutrition is prevalent at the individual level in terms of adiposity and mineral deficiencies. Some studies have also observed this phenomenon of the double burden of malnutrition in terms of adiposity or related conditions and nutrient deficiencies or related conditions among women of childbearing age in other developing countries (Zeba *et al.*, 2012a; Jones *et al.*, 2016a). It has been pointed out that individuals who are obese or have other risk factors of cardiometabolic disease may be simultaneously undernourished,

experiencing micronutrient deficiencies and associated disorders (e.g., anemia) despite consuming sufficient, or excess, dietary energy (Garcia *et al.*, 2009). The fact that in the present study there existed obese women who were iron deficient although the prevalence of this kind of DBM was lower than the prevalence of obesity in association to no iron deficiency is consistent with other studies which found that the prevalence of iron deficiency was high in obese women (Cepeda-Lopez *et al.*, 2011) and that iron level was significantly lower in obese women (Stankowiak-Kulpa *et al.*, 2017). The observed iron deficiency among overweight/obese participants could be explained by various mechanisms. Firstly, to the fact that overweight and obesity may be associated with poor dietary Fe intake. Secondly, women with obesity absorb less Fe (Mujica-Coopman *et al.*, 2015). Studies have shown that higher BMIs are associated with lower fractional Fe absorption in childbearing age women, independent of Fe status (Zimmermann *et al.*, 2008). Obesity may promote iron deficiency by inhibition of dietary iron uptake from the duodenum (Aigner *et al.*, 2014). Thirdly, the pro-inflammatory state present in most obese can impair Fe status by decreasing Fe bioavailability and affecting Fe status biomarkers (Mujica-Coopman *et al.*, 2015). A likely explanation is that chronic adiposity-related inflammation increases circulating hepcidin, thereby decreasing intestinal Fe absorption or increasing reticuloendothelial Fe sequestration (Zeba *et al.*, 2012b). There are clear associations between mineral deficiencies and obesity in various populations, and there is evidence to suggest that such deficiencies can affect leptin and insulin metabolism. Mineral deficiency in obesity may not be due to only inadequate intakes, but also due to changed metabolism and excretion (Astrup & Bugel, 2010). In this study, up to 38.9 percent of obese women were calcium deficient. Scientific literature has suggested that low calcium intake could act as a contributory factor in the rise in obesity (Beres *et al.*, 2009); which could be one possible explanation to this observed double burden (calcium deficiency among obese women) at the individual level.

Single covariate logistical regression analysis revealed, a borderline food consumption score (FCS) and an acceptable FCS were predictors of nutritional deficiencies in the study population. Also, an acceptable food consumption score was a risk factor of developing one mineral deficiency and being overweight or obese. As observed earlier

in this study, although most women of the study had an acceptable FCS and a borderline score, their diets in a week were frequent in cereals and tubers, fats and oils, vegetables food groups which made up the FCS. ‘Cereals and tubers’ and ‘fats and oils’ food groups are known to be poor in minerals but dense in carbohydrates or energy. Also, vegetables contain a high content of micronutrients, but the way these vegetable food groups are cooked affect their nutrient content level. This may explain why women with these acceptable and borderline scores were more likely of nutritional deficiencies and those with an acceptable score most likely to have the double burden in terms of mineral deficiency and overweight/obesity. Multiple covariates logistical regression analysis revealed that those in mildly food insecure households and those in moderately and severely food insecure households were less likely of having at least one nutritional deficiency than those in food secure households. Therefore, women in food secure households were at a greater risk of nutritional deficiencies than those in food insecure homes. One explanation to this could be that a wide array of relatively cheap foods but poor in micronutrients may be easily accessible to this group of women (food secure). Also, although food may be available in food secure households, inadequate consumption of certain foods can be exacerbated by other medical conditions like HIV infection, which increases resting energy expenditure and also impairs the metabolic functions in absorption, storage, and utilization of nutrients (Katona and Katona-Apte, 2008; Ivers and Cullen, 2011).

III.2.6. Prevalence of biological cardiometabolic risk factors (BCMRFs) with respect to mineral states and correlation between mineral concentrations and cardio metabolic biomarkers

The study of the prevalence of BCMRFs revealed that a very high percentage of women of the study (92.4%) had one or more BCMRFs. A higher prevalence of all these BCMRFs was not associated to a magnesium deficient status nor iron deficient status. Whereas a higher prevalence of hypercholesterolemia was associated to a calcium deficient status compared to that associated to a non deficient status ($p < 0.05$). Risk factor assessment revealed that magnesium deficiency was a predictor of hypercholesterolemia. Also, it was observed that magnesium plasma level

was negatively correlated to high triglyceride (TG) level but not to other biomarkers. This observed result is similar to other studies which have reported a positive effect of magnesium on lipid metabolism (Kirsten *et al.*, 1988; Duffoo, 2016). Also, a positive correlation between serum Ca-Mg ratio and serum cholesterol has been reported (Rasic-Milutinovic *et al.*, 2012). The plausible mechanism of the involvement of magnesium in lipid metabolism is linked to the fact that magnesium is an important co-factor for many rate limiting enzymes critical for lipid metabolism (Zhang *et al.*, 2014). It has been suggested that low magnesium may impair 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase inactivation via phosphorylation (Rosanoff and Seelig 2004). Whereas, sufficient levels of magnesium may decrease the activity of lecithin: cholesterol acyl transferase and HMG-CoA, and increase lipoprotein lipase activity which will indirectly decrease the lipid levels (Zhang *et al.*, 2014). The HMG-CoA reductase is the rate limiting enzyme in cholesterol biosynthesis. Lipoprotein lipase is responsible for the conversion of triglycerides to HDL-C and thus leads to a decrease in hepatic synthesis and secretion of VLDL triglycerides (Duffoo, 2016). It has been pointed out that, epidemiologic evidence for the role of magnesium in improving blood lipid profiles remains controversial (Zhang *et al.*, 2014).

In addition, it was observed that, calcium concentration was positively associated to higher BMI while it was negatively associated to high triglyceride levels and no relationships existed with the other cardiometabolic biomarkers. Iron concentration whereas was only positively associated to elevated waist circumference. Most studies have instead found an inverse association between high calcium intake and adiposity (Parikh and Yanovski; 2003; Beres *et al.*, 2009). One possible mechanism as to how calcium leads to adiposity is that lower dietary calcium intakes can lead to increased concentrations of 1,25-dihydroxyvitamin D (1,25(OH)₂) and parathyroid hormone (PTH), which in turn may increase adipocyte intracellular calcium ions concentration. These elevated intra-adipocyte calcium concentrations might then increase the rate of lipogenesis and inhibit lipolysis, consequently leading to increased adiposity (Parikh and Yanovski; 2003). The lack of an inverse association between calcium concentration and higher BMI in our study may be due to the lack of adjustment for confounding factors such as physical activity level. In adults, several analyses demonstrated lower

serum iron concentrations with higher adiposity (BMI), particularly in women (Aigner *et al.*, 2014). This is contrary to the observation made in the present study whereby plasma iron concentration was positively correlated with higher adiposity (central obesity). One possible explanation of the involvement of increasing iron concentration in higher central obesity could be the “dysmetabolic iron overload syndrome” (DIOS) which has been observed to affect one-third of subjects with nonalcoholic fatty liver disease (NAFLD) or metabolic syndrome (MetS). DIOS is characterized by increased serum ferritin concentrations with normal or mildly elevated transferrin saturation in subjects with various components of MetS or NAFLD (Aigner *et al.*, 2014).

III.2.7. Influence of the double burden (overweight/obesity and one or more mineral deficiencies) on the prevalence of individual biological cardiometabolic risk factors

III.2.7.1. Prevalence of the phenotypes of the double burden of malnutrition and cardiometabolic risk factors at the individual level in the study population

The assessment of the prevalence of nutritional deficiencies and biological cardiometabolic risk factors (BCMRF) revealed that 92.4% of the women had at least one risk factor and 55.4% of them had at least one nutritional deficiency. Women in the 41-49 years age group recorded the highest prevalence of those with at least one nutritional deficiency and those in the 31-40 years age group recorded the highest prevalence of those with at least one BCMRF. The overall prevalence of the double burden of malnutrition in this study population was 36.7%. Also, at least one mineral deficiency and at least one CMRF (Phenotype I) existed at the individual level. In addition, 35.4% of the participants had the double burden (overweight/obese and one mineral deficiency) of malnutrition in addition to a CMRF (Phenotype II). The overall prevalence of the double burden which was 36.7% in our study was higher than that found in women in Burkina Faso which was 30.4% (Zeba *et al.*, 2012a). In this same study in Burkina Faso, it was found that the double burden of malnutrition within individual adults is widespread in Ouagadougou, and this was accentuated in women; it also showed that 5.2% of individuals (men and women) had one or two micronutrient

deficiencies combined with overweight/obesity and at least one other CMRF (Zeba *et al.*, 2012a). This prevalence was lower than that observed in our study which was 35.4%. Among individuals who presented a double burden (at least one mineral deficiency and overweight/obese), 11.3% had a high blood pressure, 16% were hyperglycemic, 7.1% had hypertriglyceridemia, 4.4% had a high plasma cholesterol level and 18.9% had MetS whereas 1.3% of the participants were underweight with at least one mineral deficiency. Obesity is highly correlated with a constellation of disorders including dyslipidemia, insulin resistance, and hypertension, which are hallmarks of the metabolic syndrome (MS). Also, micronutrient deficiencies are linked to a higher risk of overweight and obesity and other debilitating diseases; according Khadilkar and collaborators, Micronutrient deficiencies coupled with obesity may increase the cardiometabolic risk (Khadilkar *et al.*, 2012).

The prevalence of hypertension in the study among participants with the double burden (at least one mineral deficiency and overweight/obese) was 11.3 %. This was lower than the prevalence of hypertension in a study carried out by Azantsa and collaborators in the city of Yaounde Cameroon which was 20.4% for systolic blood pressure (SBP) but higher than 8.6% for diastolic blood pressure (DBP) among women (Azantsa *et al.*, 2010); and lower than the high blood pressure rates observed in another study carried out in the city of Douala Cameroon which was 17.2% in the urban areas of the city and 5.8% in the peri-urban areas among women (Tachang *et al.*, 2013). In addition this observed level was lower than that found in the Burkina Faso study which was in 21.9% (both gender included) (Zeba *et al.*, 2012a). Among the three minerals evaluated, magnesium and calcium deficiencies have been shown to contribute directly to hypertension while the level of iron deficiency observed in this population may indirectly contribute to hypertension. Iron deficiency and anemia may impair mitochondrial and cellular energy homeostasis and further increase inactivity and fatigue of obese participants further aggravating weight gain (Aigner *et al.*, 2014); weight gain in turn aggravating hypertension. Magnesium deficiency whose prevalence in the study population was 22.4% as earlier stated, may be a contributing factor to high blood pressure. Magnesium is involved in blood pressure regulation. It has been pointed out that previous findings have indicated that Mg deficiency might affect blood pressure,

thus leading to hypertension (Rosique-Esteban *et al.*, 2018). As earlier stated, every modification of the endogenous magnesium status leads to changes in vascular tonus and, as a consequence, changes in arterial blood pressure. Magnesium deficiency increases angiotensin II-mediated aldosterone synthesis and the production of thromboxane and vasoconstrictor prostaglandins thereby elevating blood pressure (Gröber *et al.*, 2015). Calcium deficiency which was 48.3% in the study population, on the other hand may also be a contributing factor to the observed high blood pressure in the study population. Calcium deficiency could be caused by vitamin D deficiency or insufficient parathyroid hormone levels or resistance of these hormones or by inadequate dietary intake (Fong and Khan, 2012). Insufficient parathyroid hormone levels resulting in excessive urinary calcium loss by reduced bone remodeling and low intestinal absorption of calcium and would lead to hypocalcemia (Cooper and Gittoes, 2008). Therefore, disturbances in calcium metabolism include increased urinary calcium excretion and abundance of calcium-regulating hormones such as parathyroid hormone and calcitriol. These hormones cause decreases in bone mineral content and increase intracellular calcium in vascular smooth muscle. Increased calcium ion concentration produces contraction and therefore vasoconstriction leading to high blood pressure (Martinez, 1998). Magnesium deficiency associated to overweight/obesity might also be involved in the onset of type 2 diabetes as observed in this present study. Magnesium deficiency caused by low dietary intake or low intestinal absorption due to genetic defects has been shown to result in low intracellular levels of magnesium. These have been proposed to affect glucose and insulin homeostasis through decreasing tyrosine kinase activity at insulin receptors (Suarez *et al.*, 1995) and to increase intracellular calcium levels (Barbagallo *et al.*, 2003); leading to an impairment in insulin signaling thereby reducing insulin sensitivity and secretion. These in association with other metabolic effects (increased dyslipidemia, inflammation, oxidative stress, endothelial dysfunction, blood pressure) also influenced by low intracellular magnesium levels lead to type 2 diabetes (T2DM) (Song *et al.*, 2013). Therefore, sufficient levels of Magnesium may reduce the risk of T2DM by improving insulin sensitivity and secretion. (Villegas *et al.*, 2009). Also, it has been reported that data from observational studies suggest a beneficial role of Mg intake in T2DM prevention, whereas results from

interventional studies have shown beneficial effects on HOMA-IR and fasting glucose (Rosique-Esteban *et al.*, 2018). With regard to calcium deficiency in association to overweight/obesity observed in this study and its possible implication in hyperglycemia or type 2 diabetes, it has been suggested that calcium plays a role in the development of T2DM because of inverse associations observed between calcium intake and body weight (Villegas *et al.*, 2009). Calcium is essential for insulin-mediated intracellular processes in insulin responsive tissues such as skeletal muscle and adipose tissue (Ojuka, 2004), suggesting a potential mechanism to explain associations between calcium insufficiency and the risk of T2DM (Zemel, 1998).

Phenotype V and Phenotype VI were observed in 7.1% of participants and 4.4% of participants respectively. While phenotype VII (overweight/obesity with MetS and at least 1 mineral deficiency) was observed in 18.9% of the participants. This prevalence of metabolic syndrome was higher than that observed among women in the littoral region by Tachang and collaborators which was 3.7% among urban dwellers and 1.4% among peri-urban dwellers (Tachang *et al.*, 2013); and differed from that observed by Ntentie and collaborators in Cameroon which were 13% in urban area, 20.7% in less urbanized areas and 9.9% in rural areas among women (Ntentie *et al.*, 2014). Hyperlipidemia and/or MetS observed in overweight/obese individuals are common features usually associated with obesity. It has been shown that magnesium deficiency combined with the high-fructose diet, which appears to be a causative factor in the metabolic syndrome, induces insulin resistance, hypertension, dyslipidemia, endothelial activation, and prothrombotic changes (Rayssiguier *et al.*, 2006). Cross-sectional evidence has shown that magnesium intake correlates significantly with features of the metabolic syndrome (or insulin resistance syndrome), including adiposity, hyperinsulinemia, insulin resistance, hypertriglyceridemia, and low HDL cholesterol and hypertension (Song *et al.*, 2005). Also, it has been pointed out that studies have found low serum magnesium levels to be associated with elevated risk of MetS (Zhang *et al.*, 2014). In addition, in a review article, the authors pointed out that, findings from studies on Mg and MetS show an inverse association with the prevalence of MetS (Rosique-Esteban *et al.*, 2018). According to the latter authors, data appeared to support

the hypothesis that higher magnesium intake, predominantly from diet, may be beneficial for cardiometabolic health (Zhang, *et al.*, 2014).

III.2.7.2. Determinants and predictors of the phenotypes of the double burden and BCMRF in the study population

The prevalence of all the phenotypes except that of phenotype IV were highest in the 41-49 years age group although whatever the age group, this parameter (age group) was not a risk factor for developing phenotype I ($p>0.05$). This shows that, the double burden of malnutrition and BCMRF increased with age in the study population. This is in accordance with the findings of Tachang *et al.* who observed that for all the three definitions of Metabolic Syndrome used, the prevalence estimates increased with age (Tachang *et al.*, 2013). Ntentie and collaborators also observed the same trend with regards to MetS in the three areas of study (Ntentie *et al.*, 2014). Also another study carried out in Cameroon found out that, the rate of obesity among women of reproductive age increased with age and the rate was highest 35-49 age group (Yongsi and Ngwa, 2015). This trend was also shown in a review article on the nutritional status of Nigerian women of childbearing where overweight and obesity were found to increase with age and the prevalence was highest in the 40-49 years age group (Lindsay *et al.*, 2012).

The prevalence of the various phenotypes of the DBM & BCMRFs at the individual level was highest in the Northwest region then Western region. These regions recorded the highest rates of mineral deficiencies and overweight and obesity. It is therefore not surprising that the highest rates of these various phenotypes of the DBM & BCMRFs were highest in the Northwest then the western region. Urbanisation as a major driver of nutrition transition might be responsible for this observed phenotypes of DBM & BCMRFs in these regions (Delisle, 2018). Also, the low mean frequency intake of vegetables and fruits observed in both regions and the overall low physical activity observed in this general population of women of childbearing age might explain the high presence of this phenomenon observed in both regions.

Phenotypes I, II, IV, V and VII were mostly found among those with a primary education. This is consistent with a finding by Yongsi and Ngwa who observed that

women aged 15-49 years in Cameroon with a primary educational level were 1.47 times more likely to be overweight (Yongsi & Ngwa, 2015). Women with no education had the highest prevalence of Phenotypes I, II and VII after those with a primary level but phenotype III was highest among illiterates. The fact that women with no educational level were among those who recorded a high prevalence of most phenotypes is consistent with a study in India which found that women with no education were more likely to have multiple CMRF compared to those with some education (Gupta *et al.*, 2015). The ongoing nutritional transition observed in almost all areas of Cameroon (Fezeu *et al.*, 2007; Assah *et al.*, 2011; Ntentie *et al.*, 2014) might explain why participants of the study with a primary education and those with no education showed the highest levels of 5 out of the 8 phenotypes studied. A general westernization of lifestyle habits of urban population (Fezeu *et al.*, 2007; Assah *et al.*, 2011) but also of less urbanized populations (Ntentie *et al.*, 2014) has been observed in Cameroon. Also, women with a secondary educational level and those with a higher educational level were 2.171 times and 6.192 times respectively more likely of developing phenotype I (at least one mineral deficiency and one CMRF) in the 95% CI. Women with a higher or university education were also most likely of developing characteristics of phenotype II. This is consistent with the study by Yongsi and collaborator in Cameroon which noticed that the educational level influenced women's weight. Specifically, it was observed that a higher educational level presented the highest risk for women of developing obesity a CMRF (Yongsi and Ngwa, 2015). A high educational level is usually associated with a high socioeconomic status. For example, the level of obesity in developing countries has been observed to be greater in the higher socioeconomic status segments of society. Evidence of this exist in Cameroon in a study carried out by Fezeu and collaborators (Fezeu *et al.*, 2005). In our study it was also found that a high SES, was associated with a high prevalence of obesity. This can be a possible explanation to what has been observed above, that is, the fact that the few women with a high educational level were likely of having characteristics of phenotypes I and II. Although in the present study, it was observed that, most women irrespective of the educational level were found in households with high socioeconomic status and women in these households were less likely of developing characteristics of phenotype I. Also,

with the ongoing nutritional transition and the adoption of western lifestyle which Cameroon is experiencing, irrespective of the socioeconomic status, the intake of poor quality diets (mostly rich in sugar and sweetened beverages, processed foods, edible oils, salt and animal-source foods) that lack micronutrients may be enhanced.

Regression analyses revealed that married women were 0.508 times less likely to develop characteristics of phenotype I and widows or divorced women were 0.368 times less likely to develop characteristics of this phenotype compared to single women. This is contrary to the findings of Yongsu and Ngwa, who observed that married women are more likely to gain weight (Yongsu and Ngwa, 2015) and therefore more likely of being exposed to other CMRF. The fact that we did not control potential confounders like physical activity level, food consumption, and socioeconomic status in risk factor analysis might also account for the above observation.

Participants with an acceptable FCS presented the highest prevalence of all the phenotypes established in this study. Regression analysis also showed that women with an acceptable FCS were 0.412 times ($p=0.000$) less likely of developing characteristics of phenotype I compared to those with a poor score. This could be partly due to the fact that in this study, a poor FCS, portrays mostly a dietary habit rich in carbohydrates sources like starch (tubers and cereals) but low in proteins sources (meat, fish, milk) and other micronutrient rich foods such as fruits. The Cameroonian eating habits have been characterized as low in fruits and vegetables consumption but high in the consumption of foods rich in starch and sugar (Kyobutungi, 2008). Also, the nutrition transition characterized by urbanization and westernization of lifestyles has also been observed in Cameroon (Ntentie *et al.*, 2014; Fezeu *et al.*, 2007; Assah *et al.*, 2011); and as in many African countries, this transition leads to an increase consumption of nutrient-poor foods (high in fat and sugar and low in many important nutrients, such as dietary fibre, calcium, folate, and vitamin A, D, and E) and increased rates of overweight or obese (Hopping *et al.*, 2010). These could also explain the fact that the prevalence of overweight/ obesity were not only high in the study population but occurred with one or more mineral deficiencies and were associated with CMRF. It was also observed that, a borderline FCS was a predictor of group III.

Whatever the phenotype, participants in food secure households were those with the highest prevalence. The explanation here might be that, most women in the study population were in food secure households (48.8%). This food security state may expose them to obesity and other CMRF like hypertension, hyperglycemia, hypertriglyceridemia, hypercholesterolemia and MetS. Similar findings were made in Malaysia where it was observed that food secure women had the highest prevalence of MetS (29.6%) compared to other groups. Also, it was reported that women with food insecurity had lower risk of abdominal obesity, elevated plasma glucose, cholesterol, LDL-cholesterol and MetS as compared to women with food security whose risk was high (Shariff *et al.*, 2014). According to this author, for food secure households, despite the access to a variety of food choices, the increasing food prices could also force them to avoid relatively expensive foods (i.e. fruits, vegetables, fish and lean meats rich in micronutrients) (Shariff *et al.*, 2014; Drewnowski, 2009). Similar observations have been made in Cameroon (Nkengfack *et al.*, 2011). Avoidance of such quality but expensive foods enables such households to maintain food variety and higher energy intake at a lower cost favouring the association of food security, micronutrient deficiencies, obesity and other BCMRFs. This study also revealed that women in mildly food insecure households were 2.500 times more likely of developing characteristics of group I ($p=0.002$) and 2.648 times of developing characteristics of group VI ($p=0.015$) in the 95% CI after adjusting for age. This result is similar to two studies conducted in developed countries which have consistently shown that mild but not severe food insecurity was associated with obesity among women (Seligman *et al.*, 2010; Nord, 2007). Low food security has also been related to higher BMI for Asian women. A possible mediator of the association between food insecurity and obesity according to these authors is dietary behaviour, where food-insecure individuals may reduce the quality and/or quantity of foods consumed (Leung *et al.*, 2012). Also, it has been stated that budgetary constraints may result in increased purchasing of low-cost, non-nutrient rich and energy-dense foods, and decreased consumption of healthy foods, such as fruit and vegetable (Vedovato *et al.*, 2016) leading to subsequent chronic diseases together with the deficiencies of certain micronutrients.

Participants of the study population with a low fruit intake per week recorded the highest prevalence of all the phenotypes of the DBM and BCMRFs. Whereas, with regards to vegetable intake, almost all the phenotypes were mostly prevalent among participants with a high intake. Also, Binary linear regression analysis showed that, participants with a low intake of fruits and those with a low intake of vegetables were 2.079 ($p=0.048$) and 2.042 ($p=0.000$) times respectively more likely of developing characteristics of phenotype I. A low intake of fruits and vegetables were also predictors of phenotypes II, VI, and VII. In addition, a low intake of vegetables in a week was also a predictor of IV and V. In this study, it was observed that up to 81.1% of women had a low intake of fruits per week which might explain the fact that most women of this group recorded the highest percentages of all the phenotypes of the DBM and BCMRFs. de Almeida Ventura *et al.* also observed an inadequate intake of fruits among women of a study in Brazil. They also found a significant association between high concentrations of LDL-cholesterol and lower fruit intake (de Almeida *et al.*, 2014). A study in Canada also pointed out that infrequent consumption of fruit and vegetables explain the low intake of nutrients like calcium, folate, vitamin A and Vitamin D (Folchetti *et al.*, 2014). Also, Folchetti *et al.* found a beneficial role of fruits and vegetables, as well as antioxidant vitamins, in the pathophysiological process of cardiometabolic diseases (Folchetti *et al.*, 2014). Ntentie *et al.* observed in a study carried out in Cameroon that the less urbanized and the rural areas had the highest frequency of low fruit intake (Ntentie *et al.*, 2014). It is well established that low fruits and vegetables intakes especially in women of childbearing age is associated with micronutrient deficiencies (Bartley *et al.*, 2005). In addition, higher contents of dietary fiber and other bioactive phytochemicals in fruits and vegetables especially polyphenolic compounds, with potential anti-oxidative, antiinflammatory, hypo-lipidemic and vascular protective properties, make them excellent dietary choices for cardiovascular health (Zern and Fernandez, 2005; Mirmiran *et al.*, 2015). Women with a high vegetable intake had the highest prevalence of all the phenotypes of the DBM and BCMRFs. This could be supported by the fact that green vegetable soups which are mostly eaten in Cameroon, are generally cooked with larger amounts of oil added to important quantities of protein sources (meat or fish, groundnut) which result in high fat green vegetable sauce; in which the high amount of fats may

probably interfere with the known beneficial effect of fibers on the health prevention and management of nutritional related diseases (Ntentie *et al.*, 2014). The fact that the low consumption of vegetables was a risk factor of developing characteristics of phenotypes I, II, IV, V, VI, and VII might be associated to the low fiber and micronutrient intakes usually linked to low intake of vegetables (Folchetti *et al.*, 2014). It has been stated that generally, diets rich in fruits and vegetables are associated with higher intakes of fiber. High-fiber diets are more satiating, reduce the absorption of other nutrients including lipid and glucose, improve insulin sensitivity and stimulate fat oxidation (Du *et al.*, 2010; Mirmiran *et al.*, 2015). In addition, decreased dietary glycemic index, reduced total fat intake, increased intake of nutrients including magnesium and non-nutrient bioactive compounds may contribute to the FV intake and cardiovascular associations. Different colors of FV represent various contents of phytochemicals, which may explain the associations of certain subgroup of FV intakes with cardiometabolic risk factors. The health benefits of FV have been proposed to be attributed to the additive and synergistic effects of these bioactive compounds (phytochemicals) (Liu, 2003; Mirmiran *et al.*, 2015). Therefore, the association between mineral deficiency or deficiencies and overweight/obesity in an individual, might result in the onset of individual biological cardiometabolic risk factors.

**CONCLUSION, RECOMMENDATIONS
AND PERSPECTIVES**

CONCLUSION

The evaluation of the nutritional characteristics among this group of Cameroonian Women revealed that in general, they had unhealthy dietary habits frequent in Carbohydrate rich foods and fats and oils but infrequent in proteins, and vitamins and minerals rich foods and equally, most of these participants had a high socioeconomic status. The assessment of the prevalence of malnutrition revealed that all the forms of malnutrition studied were present in the study population with calcium deficiency being the most prevalent form. The prevalence of all these forms increased with age. Nutritional determinants of these forms of malnutrition included a frequent intake (i.e. 5-7 times) in a week of 'cereals and tubers' food group; and a low intake (i.e. 1-2 times) in a week of 'pulses', 'milk and dairy products', 'meat, fish and eggs', and 'vegetables' and 'fruits' food groups. All the forms of malnutrition were highest in the northwest region followed by the western region. A low educational level, a high socioeconomic status, an acceptable food consumption score and being in a food secure household, were equally determinants of almost all these forms of malnutrition.

The assessment of the prevalence of multiple mineral deficiencies revealed that the northwest region was the only region which recorded a percentage (1.6 percent) of participants of the study population with all three mineral deficiencies. Specific combined mineral deficiencies such as Ca/Mg deficiencies, Ca/Fe deficiencies, Mg/Fe deficiencies and Ca/Mg/Fe deficiencies existed in the study population and their rates were highest in the 31-40 years age group and in the northwest region. The study of mineral-mineral interaction showed that, a magnesium deficient state was associated to a high Ca-Mg ratio; this result was supported by the finding that Mg concentration was negatively and strongly correlated to Ca-Mg ratio whereas, Ca concentration was strongly and positively correlated to Ca-Mg ratio. All age groups were predictors of calcium deficiency and combined Ca and Mg deficiencies in an individual. A low intake (i.e. 1-2 times) of 'meat, fish and eggs' food group in a week was a predictor of Mg and combined Ca and Mg deficiencies. Ca deficiency was the most prevalent mineral deficiency among those who were underweight, overweight or obese.

Higher rates of all BCMRFs were observed to be associated to a Ca deficient state compared to a Mg and Fe deficient state. The overall prevalence of the double burden of malnutrition in this study population was 36.7%. Various phenotypes of the double burden of malnutrition and BCMRFs existed at the individual level and the most prevalent phenotypes were groups I and II. The most outstanding predictors were mild food insecurity for phenotypes I and IV, and a low intake (i.e. 1-2 times in a week) of fruits for phenotypes I, VI and VII and a low intake (i.e. 1-2 times in a week) of vegetables for phenotypes I, II, IV, V, VI and VII. Therefore, the occurrence of mineral deficiency or deficiencies and excess weight (overweight/obesity) in an individual, might result in the onset of individual biological cardiometabolic risk factors.

RECOMMENDATIONS, LIMITATIONS OF STUDY AND PERSPECTIVES

Recommendations

- 1.) Research on bio-fortification of staple foods such as cassava, sweet potatoes, cocoyam, and rice in Cameroon; especially the bio-fortification of staple foods specific to each region of the country with Ca and Mg should be part of interventional strategies directed at alleviating dietary Ca and Mg deficiencies.
- 2.) The government should subsidise Calcium and Magnesium rich foods such as fish, meat, milk, beans and dairy products so as to improve the status of these minerals not only among all women of childbearing age especially those of low socioeconomic status in these regions most notably in the Northwest region where most women suffered from these mineral deficiencies but in the entire population too.
- 3.) Research directed towards improving micronutrient status of women of childbearing age in Cameroon in particular and the population in general should focus on methods of reducing anti-nutrients in cereals and tubers which make up a great proportion of most meals in Cameroon. Especially in the Center, Far-North, Northwest and Western regions where the frequency of consumption of these food groups are very high.
- 4.) Dietary diversification strategies such as increasing the production and consumption of micronutrient-dense foods; incorporating enhancers of micronutrient absorption in household diets should be encouraged in households and in community through nutritional education by nutritional experts. For example, the consumption of calcium rich foods should be taken with food sources of enhancers of Ca absorption such as: fruits and vegetables, vitamin D food sources such as oily fish, egg... especially in the Western region where the vegetable food group mean frequency consumption was observed to be low.
- 5.) Furthermore, nutritional education aiming at discouraging the consumption of calorie dense foods low in micronutrients, refined foods, sweetened beverages, excessive alcoholic beer intake and sedentary lifestyle but that enhance the

consumption of traditional diets rich in micronutrients, vegetables and fruits should be prioritized in order to reduce the prevalence of obesity and related consequences.

- 6.) Also, where possible, nutritional experts in Cameroon should personalize diets in order to prevent if not, manage both sides of malnutrition and their consequences among women of childbearing age in all these regions.
- 7.) Calcium and magnesium rich vegetables like okro, green leafy vegetables, keleng keleng, huckleberry, pumpkin leaves, cassava leaves and bitter leaves; and pulses like beans and groundnuts should be eaten as main vegetable food sources and pulses food sources respectively of calcium and magnesium to prevent deficiencies from these minerals especially in the Northwest and Western regions with the highest rates of these mineral deficiencies and moderate and low mean frequency consumption of vegetable food group respectively.
- 8.) Multisectorial approach should be used as a gateway in fighting both sides of malnutrition especially among women of childbearing age in these regions; mostly among women in the Northwest and West regions which presented the highest rates of overweight/obesity and all mineral deficiencies and equally highest rates of various phenotypes of the double burden of malnutrition and biological cardiometabolic risk factors at the individual level.

Limitations of the Study

This study has some limitations that should be mentioned.

- 1.) Self-reported data on which dietary assessment was based tend to over- or underestimate actual food consumption.
- 2.) Bioassay of mineral concentrations in samples was done using ready to use reagents following standard procedures described by reagent producers which are not quite appropriate methods for the evaluation of mineral concentrations. A more appropriate method would be to use flame atomic absorption spectroscopy.

Perspectives

- 1.) Assess Vitamin D status in the study population in order to better understand calcium and magnesium status
- 2.) Carry out this study among the same population group while taking into consideration the physical activity level of women in each of these regions in order to understand the exact influence of this lifestyle parameter on the Nutritional status of this study population.
- 3.) Carry out this study in the other remaining regions of the country and among the other segments of the Cameroonian population in order to have a bigger picture of the status of these minerals in Cameroon
- 4.) Carry out dietary habit assessment using the 24 hours recall method and food consumption tables to better grasp the influence of food habits and dietary intakes on mineral status of women of childbearing age and the general population.
- 5.) Study gene-mineral interaction in order to understand the influence of this interaction on the onset of cardiometabolic risk factors in this study population.
- 6.) Study Osteoporosis in this study population which might be another consequence of the high rates of calcium deficiency and high rates of obesity observed in this study.

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APPENDIXES

APPENDIXES

APPENDIX 1: QUESTIONNAIRES

NUTRITION AND HEALTH STUDY

CODE : _____	Tel. _____	Age : _____	Sex : _____
Participant's Name:			
Place of birth:		Ethnic :	
Region of Origin :			
Division of Origin :			
Profession :		Religion :	
Hormonal Status:			

PARAMETERS OF INTEREST :

Weight (kg): _____ Height (m) : _____
 BMI (kg/m²): _____ % Body fat: _____
 Waist circumference (cm): _____ Hip circumference (cm): _____
 SBP /DBP mm Hg _____
 FC..... GLYCAEMIA:mg/dL

A DIETARY HABITS

1 How many times do you usually eat per day ?

_____ Number of times / day

2 At which time/moment of the day do you eat ? _____

3 Do you cook your meals yourself ? Always /Often;

Occasionally /Sometimes ;

Rarely /Never

3a Who cooks your meals ? _____

4 Do you sometimes eat out of the house ?

Yes 4a, 4b 1 No 5 0

4a When you eat out of the house, where do you generally eat?

(several answers are possible)

VIP Restaurant; Restaurant ; mama put

Road ; Work/ canteen of the company

Cafeteria

4b For which reason(s) do you eat out of the house? (several answers are possible)

Work place far away from the house ; For pleasure

Nothing to eat in the house ; Other reasons (precise)

5 During the last 7 days, did you eat out of the house? No

Yes

5a How many days in the week? _____ Nber of days/week

How many times these days _____ Nber of times /day

6 During the last 7 days, did you buy cooked food in the street?

No Yes

6a How many days in the week? _____ Nber of days/week

How many times these days? _____ Nber of times /day

7 During the last 7 days, did you eat in a restaurant?

No Yes

7a 6a How many days in the week? _____ Nber of days/week

How many times these days? _____ Nber of times /day

8 During the last 7 days, did you eat in a mama put?

No Yes

8a How many days in the week? _____ Nber of days/week

How many times these days? _____ Nber of times /day

9 During the last 7 days, did you eat in a canteen of a company?

No Yes

9a How many days in the week? How many times these days?

_____ Nber of days/week _____ Nber of times /day

10 During the last 7 days, did you eat in a cafeteria

No Yes

Question 11,12 &13:	Yes	No	Nber days /Week	Nber of times/ day
11 During the last 7days, did you eat:				
a) Aerated beverage(Coca, Fanta,				
b) Bonbons				
c) Chocolate (paste, butter, powder...)				
d)Fried (plantain, irish potatoes,sweet potatoes)				
e) Hamburger				
h) Popcorn				
j) Pizza				
k) Ice cream				
l) Cake (cream, with chocolate)				
12- During the last 7days, did you eat:				
a) Pasta products (Macaroni, spaghetti, etc.)				
b) Pork-butcheries (jambon, sausages,)				
c) Canned fish (Sardines, pilchard, ...)				
d) Conserved vegetables (peas, ...)				
e) Cheese				
f) salade				
g) Shawarma				
h) Biscuits (salty, sweetened)				
13a- During the last 7days, did you eat:				
Makara (Table tennis) (Banana+ Cassava) or Accra (Koki Bean)				
a) Koki and complements				
b) rice pap or corn pap				
c) fufu millet; rice or sorghum or corn fufu				
d) plantain banana and sauce				
e) Bobolo and sauce				
f) Cassava fufu and sauce				
g) tubers and sauces (tomato or groundnut)				
h) Beans				
i)Rice +sauce				
j) eru+ water futu				
k)okok + Complement				
l) ndole+ Complement				
m) Tomato soup				
n) Groundnut soup				
o) Vegetables				
p) fruits				
q) Achu and yellow soup				
r)Fufu corn and djamadjama				
s) Porridge cocoyam/plaintain +bitter leaves				
t) Egusi pudding or Egusi soup				
u) Porridge yam or Hot pot (irish)				
v) Pound Beans + banana or Irish or plantain				
w)Corn cake				
Kawacoco bible				
Ekwang				
y) Fufu corn and Yellow soup (ou Okru)				
z) Fufu corn and Nkui				
Porridge banana or Porridge plaintain				
Kawacoco				
Koki corn				
Corn chaff				
Eggs, Fish, Meat, Milk, Dairy products (Encircle all possibilities)				
Refined oil (Precise) :				
Palm oil				
Yellow or sweet yam				
Others (Specify)				
1-				
2-				
13b What are your traditional dishes?				
13c How many times do you eat them per week ?				

- Tous les jours ; Plusieurs fois par semaine
 À peu près une fois par semaine ; Tous les mois
 Rarement/jamais

15a Do you paste your bread with butter or chocolate
 No Yes

15b If yes at what frequency do you do it?

- Every day ; Most days/week ; About once per week ; Every month ; Rarely/Never

16 Do you consider that you eat too much sugar for your health?
 Yes No undecided

17 Do you consider that you eat too much fat and oils for your health? Yes No undecided

18 Do you consider that you eat too much salt for your health?
 Yes No Undecided

19 Do you consider that you eat too much for your health?
 Yes No Undecided

20 Do you consider that by changing your way of eating you can ameliorate your health? Yes No Undecided

21 Compared to other people of your age, you would say that your usual food/diet is.....

- Very good Good Fairly good
 Bad Undecided

22 Compared to other people of your age you would say that your health is in general...

- Very good Good Fairly good Bad
 Undecided

23 Do you receive informations on diet ? Yes No

23a If yes, by which means do you receive information on diet (several answers are possible)

- Radio Television Newspapers, review or brochures
 Books or courses Health professionals (doctors, dietetists, nurses)
 Others (precise) _____

23b Would you like to receive informations on diets/foods?

- Yes No

23c By which means would you like to receive information on diets / foods (several answers are possible) :

- Radio Television Newspapers, review or brochures
 Books or courses Health professionals (doctors, dietetists, nurses) Others (precise) _____

24 Do you currently follow a particular diet for your health?
 No Yes

24a For which reason do you follow this diet? Specify _____

25 In your household, are there any foods which are prohibited to you? No Yes

25a If Yes, Which? _____

25b Why are these foods prohibited?

D) HEALTH HISTORY

30a Do you know your status with respect to these diseases?

Diseases	Yes	No
a. Cardiovascular Diseases		
b. Hypertension		
c. High Cholesterol		
d. Diabetes		

30b In your family (paternal or maternal side), did someone suffer or suffers of one of the following diseases:

Diseases	Yes	No
a. Cardiovascular Diseases		
b. Hypertension		
c. High Cholesterol		
d. Diabetes		

30c Do you take any dietary supplements like: a.) iron: Yes

- No; b.) calcium: Yes No; c.) magnesium: Yes No; d.) others: Yes No

B-ALCOHOLIC DRINK CONSUMPTION

26 Do you consume alcoholic drinks? Yes ; No

26a If yes, describe your alcohol consumption during the last 12 months

Type of drink consumed	yes	No	Nber of times /week	Nber of times /month	Usual Quantity
Traditional local Beer (e.g .bilibili, chā, Mbouh, Raffia....)					
Ordinary or imported beer					
Traditional local wine					
Wine and imported aperitifs					
Locally distilled drinks (<i>fofo, arky, ...</i>) (<i>Roi, Fighter, Lion d'or, Officier, Nikita, Bravo, Zed, Kitoko, King Arthur ...</i>)					
Imported distilled drinks (<i>whisky, Vat 69, J&B...</i>)					
Usual Quantity					
Maximum consumed quantity in one intake last month					

C) TOBACCO CONSUMPTION

27 Do you actually smoke ? Yes No

28 Are you a former smoker? Yes No

28a If you smoke at what frequency?

- Everyday occasionally

28b What do you smoke ?

Type of tobacco consumed	Yes	No	36c. Smokes everyday
			Number per day
Cigarette			
Cigare			
Pipe			
Tobacco powder			

For occasional smokers, How many sticks when this happens?

28c At what age did you start smoking? _____ years

29a If you are a former smoker at what frequency did you ?

- Everyday occasionally

Specify the type of tobacco and the number of sticks per/day

29b At what age did you start smoking? _____ Years

29c At what age did you stop smoking? _____ years

E) URBANISATION

31a Where were you born? Specify the place of birth:

- Rural area ; Secondary city /little city ; Big city

31b How long have you lived in your place of birth? Specify:

32 For how long have you continuously lived in the current region? _____ Years

33a Previously, where did you live?

- Does not apply (has always lived in Mbengwi since birth)

Big city ; Other secondary city; In the village /Rural area ;
 Abroad: (Specify) _____

33b For how long have you lived in the current place? _____
 _____ Years

34 For which reason(s) do you now live here?

Specify _____

35 Since the age of 5 years, how long (in years) have you already spent in one of the following localities:

Big city(Chief town of Region) :	Secondary city (Chief town of Division) :	Rural area :	Abroad (urban) :	Abroad (rural) :

F) SOCIO-ECONOMIC INFORMATIONS

36 What is your current matrimonial status? Married or lives with a partner widow or widower, divorced, separated single / Fiancé, lives alone

37 In which type of household do you live? Monogamous polygamous

38 What is the gender of the head of the family? Male Female

39 What is your highest level of education?

- No schooling or illiterate
 Literate ; Primary
 First cycle secondary education (1st cycle, Form 1 to 5)
 Second cycle and Professional school
 University

40 What is your main source of revenue?

- Salary (permanent employment)
 Salary (daily or temporal)
 Business ; Help, assistance ; Partner
 Other _____

41 Have you worked or exerted a business or any activity that generated money during the last 12 months?

- No Yes

41a If yes, For how long have you worked during this period? _____
 _____ Number of months

41b What was your main job? Give all possible details _____

42 What is the main source of revenue of your partner? _____

43 What is the total number of people in your household (Including you)?

_____ Number of women of child bearing age 15 to 49 years;
 _____ Number of women aged above 49 years; _____ Number of men aged above 15 years; _____ Number of adolescents (12 ≤ years ≤ 15) ; _____ Number of children (< 12 year)

43a How many pregnancies have you carried to the end _____
 _____ Number of full-term pregnancies (Women)

44 Do you or someone in your household possesses?

Goods possessed	Yes	No	If yes, total number in the household
Bicycle			
Moto bike			
Car			
Radio			
Television (TV)			
A mobile phone			
Refrigerator			
Land			
House on rentage			
taxi			

45 Do you or someone in your household possesses :

Rearing	Yes	No	If yes, specify the type of rearing (ex. chicken, sheep, Pigs, goats, Cows, Rabbits, rats)
domestic			
Buisness			
Land/ Farming land			
Land/ Renting farming land			

46 Does your household possess a house help? (house help, servant) Yes No

47 What is your status in the house in which you live? Owner ; Tenant; Free housing

House of function ; Other (Specify) _____

48 Record of the material of the floor of the house

Ground, sand, stone ; cement

Tile, marble, ceramic, jeflex, carpet

Others (Specify) _____

49 Record of the material of the wall of the house

Palms, cartons, oilcloth ; Wood, bamboos

Sheet metal; Mud brick house Semi hard / baked ground

cement brick ; Others (Specify) _____

50 Record the material of the roof of the house Straw;

Sheet metal; Pave (concrete)

Tile ; Others (Specify) _____

51 Do you have electricity? Yes No

51a If no, what is the main source of energy used as light in your house?

Petrol Gas Other _____

51b Do you have a complementary source of energy

(power generating unit, solar panel.) No Yes

52 In your household, what is the principal source of energy that you use in your kitchen: Firewood; Charcoal; Petrol; Gas;

Electricity; Other _____

53 What is the principal source of provision of drinking water in your household?

Bottle water 1 ; Running water (tap) 2

Drilling equipped with manual pump (Public Fountain) 3

Spring 4 ; Well 5

Surface water (river, sea, lake) 6

Tank 7 : Rain water 8

Others _____ 66

54 Which kind of toilets do members of your household generally use ?

Modern toilet water system (toilets)

Improved ventilated pit latrines (VIP)

Pit Latrines with flag stone

Pit latrines without flagstone/ open pit

Truck/bin

Composting toilets

No installations (bush, beach, etc.)

Others, Specify:

I do not know/ No response

55 Does your household have possesses all the following

equipment (should be in a good condition)?

Sofa ; Table ; Armchair ; Cupboard ; Wardrobe ;

Sewing machine ; Wrist watch ; Clock ; Cable ; Telephone

line ; Cutlass ; Axe ; Gas cooker ; Refrigerator ; Freezer

G) INFORMATION ON FOOD SECURITY

56 In the past four weeks, did you worry that your household would not have enough food? No Yes

56a If yes, How often did this happen? Rarely (once or twice in the past four weeks); Sometimes (three to ten times in the past four weeks); Often (more than ten times in the past four weeks)

57 In the past four weeks, were you or any household member not able to eat the kinds of foods you preferred because of a lack of resources? No Yes

57a If yes, How often did this happen? Rarely (once or twice in the past four weeks); Sometimes (three to ten times in the past four weeks); Often (more than ten times in the past four weeks)

58 In the past four weeks, did you or any household member have to eat a limited variety of foods due to a lack of resources? No Yes

58a If yes, How often did this happen? Rarely (once or twice in the past four weeks); Sometimes (three to ten times in the past four weeks); Often (more than ten times in the past four weeks)

59 In the past four weeks, did you or any household member have to eat some foods that you really did not want to eat because of a lack of resources to obtain other types of food? No Yes

59a If yes, How often did this happen? Rarely (once or twice in the past four weeks); Sometimes (three to ten times in the past four weeks); Often (more than ten times in the past four weeks)

60 In the past four weeks, did you or any household member have to eat a smaller meal than you felt you needed because there was not enough food? No Yes

60a If yes, How often did this happen? Rarely (once or twice in the past four weeks); Sometimes (three to ten times in the past four weeks); Often (more than ten times in the past four weeks)

61 In the past four weeks, did you or any other household member have to eat fewer meals in a day because there was not enough food? No Yes

61a If yes, How often did this happen? Rarely (once or twice in the past four weeks); Sometimes (three to ten times in the past four weeks); Often (more than ten times in the past four weeks)

62 In the past four weeks, was there ever no food to eat of any kind in your household because of lack of resources to get food? No Yes

62a If yes, How often did this happen? Rarely (once or twice in the past four weeks); Sometimes (three to ten times in the past four weeks); Often (more than ten times in the past four weeks)

63 In the past four weeks, did you or any household member go to sleep at night hungry because there was not enough food? No Yes

63a If yes, How often did this happen? Rarely (once or twice in the past four weeks); Sometimes (three to ten times in the past four weeks); Often (more than ten times in the past four weeks)

64 In the past four weeks, did you or any household member go a whole day and night without eating anything because there was not enough food? No Yes

64a If yes, How often did this happen? Rarely (once or twice in the past four weeks); Sometimes (three to ten times in the past four weeks); Often (more than ten times in the past four weeks)

H) PERCEPTIONS ON PHYSICAL ACTIVITIES

65 Compared to other people of your age, you would say that you do: Less Physical activity as much physical activity More physical activity Undecided

66 What is your main means of transport in the day: walking bicycle motorbike car

67 Main attitude adopted when exercising your job/work:

Sited; Standing; Walking;

Carrying heavy loads Carrying light loads

I) PERCEPTIONS ON BODY ASPECTS

68 Among the following figures/outlines, which one mostly resembles the subject (the investigator encircles the appropriate figure)

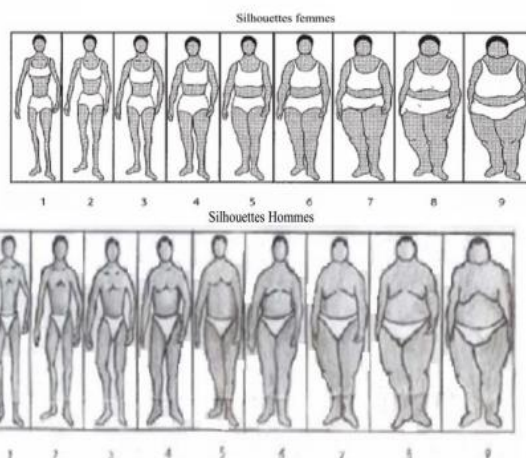
1 2 3 4 5 6 7 8 9

69 Among the following figures/outlines, which one resembles you the most (encircle the appropriate figure according to the gender of the subject)?

1 2 3 4 5 6 7 8 9 NSP / NR

70 Among the following figures/outlines, to which would you like to resemble (encircle the appropriate figure according to the gender of the subject)? 1 2 3 4 5 6 7 8 9 NSP / NR

71 For what reason(s) would you like to resemble this figure/outline? Specify : _____



J) INFORMATION ON PHYSICAL ACTIVITY

1) Activity during work

a) Which intensity of physical activity does your work requires? Light ; Moderate ; Average ; Intense ; Very intense

b-) Does your work involve an activity of vigorous-intensity that causes large increases in breathing or heart rate like [carrying or lifting heavy loads, digging or construction work] for at least 10 minutes continuously? Yes No

c-) In a typical week, on how many days do you do activities of vigorous intensity as part of your work? Number of day.....

d How much time do you spend doing activities of vigorous-intensity at work on a typical day? Hours..... Minute.....

e-) Does your work involve an activity of moderate-intensity, that causes small increases in breathing or heart rate such as brisk

walking [or carrying light loads] for at least 10 minutes continuously? Yes No

f-) In a typical week, on how many days do you do activities of moderate intensity as part of your work? Number of days

g-) How much time do you spend doing activities of moderate-intensity at work on a typical day? Hours.....
Minute.....

2-) Travel to and from places

a-) Do you walk or use a bicycle (pedal cycle) for at least 10 minutes continuously to get to and from places? Yes No

b-) In a typical week, on how many days do you walk or bicycle for at least 10 minutes continuously to get to and from places? Number of days

c-) How much time do you spend walking or bicycling for travel on a typical day? Hours..... . Minute.....

3-) Recreational Activities

a-) Do you do any vigorous-intensity sports, fitness or recreational (leisure) activities that cause large increases in breathing or heart rate like [running or football or handball] for at least 10 minutes continuously? Yes No

b-) In a typical week, on how many days do you do vigorous intensity sports, fitness or recreational (leisure) activities? Number of days

c-) How much time do you spend doing vigorous-intensity sports, fitness or recreational activities on a typical day? Hours..... Minute.....

d-) Do you do any moderate-intensity sports, fitness or recreational (leisure) activities that cause a small increase in breathing or heart rate such as brisk/rapid walking, cycling for at least 10 minutes continuously? Yes No

e-) In a typical week, on how many days do you do moderate intensity sports, fitness or recreational (leisure) activities? Number of days

f-) How much time do you spend doing moderate-intensity sports, fitness or recreational (leisure) activities on a typical day? hours..... Minute.....

4-) Sedentary behaviour

a-) How much time do you usually spend sitting or reclining/lying on a typical day doing nothing? Hours..... Minute.....

APPENDIX 2: PATIENT RESULT FORM



Cameroon Nutritional Sciences Society (C.N.S.S.)

Société Camerounaise des Sciences de la Nutrition (S.C.S.N.)

P.O. Box 8418, Yaounde, Cameroon Tel. 75791138 / 77671375



HEALTH CAMPAIGN ON METABOLIC DISEASES

Code # :	Sex	Age:
Name :		
ANTHROPOMERIC MEASURES AND BLOOD PRESSURE		
Weight (kg):	Height (m) :	
BMI ¹ (kg/m ²)	Waist circumference (cm) :	
% Body fat:	Hip circumference (cm) :	
Systolic Blood Pressure (mm Hg)	/	pool
Diastolic Blood Pressure (mm Hg)	/	
BIOCHEMICAL MARKERS		
Biochemical parameters	Obtained values	Reference Values
Fasting blood glucose level (mg / dL)		(< 110mg / dL)
Total cholesterol (mg / dL)		(< 200 mg / dL)
LDL Cholesterol (mg / dL)		(< 150 mg / dL)
HDL Cholesterol (mg / dL)		(> 45 mg / dL)
Triglyceride (mg / dL)		(< 150 mg / dL)

CONCLUSIONS:

- Weight status: **Normal weight ; Overweight ; Obese ;** Surplus weight of about.....kg
- Blood pressure: **Normal ; High ;** Hypertension **Yes no**
- Glycaemia : **Normal, High :** Diabetes **Yes no**
- Total Cholesterolemia **Normal, High** Total Hypercholesterolemia **Yes no**
- LDL Cholesterolemia **Normal, High** HDL Cholesterol **Normal, Low**
- Triglyceridemia **Normal, High** Hypertriglyceridemia **Yes no**

RECOMMANDATIONS:

- Loss some weight,
- Control regularly your blood pressure and or your blood sugar level,
- Reduce salt in your meals,
- Reduce the quantity of oil in your meals and avoid too fatty foods,
- Increase your frequency of consumption of fruits and vegetables,
- Increase your physical activity level and do a lot of walking,
- Reduce your frequency of consumption of eggs (2 eggs/week),
- Reduce if possible or stop tobacco and alcohol consumption,
- If you are already under antihypertensor and/or antidiabetic treatments respect the regular intake of these drugs,
- Return to the consumption of basic traditional meals rich in fibers and antioxidants,
- Needs to meet a medical doctor,
- Keep the same rhythm and good lifestyle.

APPENDIX 3: ETHICAL CLEARANCE

COMITE NATIONAL D'ETHIQUE DE LA RECHERCHE POUR LA SANTE HUMAINE

Arrêté N° 0977/A/MINSANTE/SESP/SG/DROS/ du 18 avril 2012 portant création, organisation et fonctionnement des comités d'éthique de la recherche pour la santé humaine au sein des structures relevant du Ministère en charge de la santé publique

N° 2014/08/488/CE/CNERSH/SP

Yaoundé, le 26 août 2014

Cnethique_minsante@yahoo.fr -

CLAIRANCE ETHIQUE

Le Comité National d'Ethique de la Recherche pour la Santé Humaine (CNERSH), en sa session ordinaire du 05 août 2014, a examiné le projet de recherche intitulé «**Nutrition and health study**» soumis par le **Docteur NGONDI Judith Laure**, Investigateur Principal, Faculté des Sciences/ Université de Yaoundé 1.

Le projet est d'un grand intérêt scientifique et social. Cette étude permettra d'évaluer l'impact des habitudes alimentaires et nutritionnelles, notamment sur l'apparition des maladies du cœur. La procédure de l'étude est bien documentée et claire. Les risques liés au prélèvement sanguin seront minimisés par un personnel qualifié. La notice d'information et le formulaire de consentement éclairé, en français et en anglais, sont bien élaborés et simples à comprendre. Les mesures prises pour garantir la confidentialité des données collectées sont présentes dans le document. Les CVs des Investigateurs les décrivent comme des personnes compétentes, capables de mener à bien cette étude. Pour toutes ces raisons, le Comité National d'Ethique approuve pour une durée d'un an, la mise en œuvre de la présente version du protocole.

Les Investigateurs sont responsables du respect scrupuleux du protocole approuvé et ne devraient y apporter aucun amendement aussi mineur soit-il, sans avis favorable du CNERSH. Les investigateurs sont appelés à collaborer pour toute descente du CNERSH pour le suivi de la mise en œuvre du protocole approuvé. Le rapport final du projet devra être soumis au CNERSH et aux autorités sanitaires du Cameroun.

La présente clairance peut être retirée en cas de non respect de la réglementation en vigueur et des recommandations susmentionnées.

En foi de quoi, la présente clairance éthique est délivrée pour servir et valoir ce que de droit.

Ampliations

- MINSANTE

LE PRESIDENT

Dr. Lazare KAPTUE

N.B : cette clairance éthique ne vous dispense pas de l'autorisation administrative de recherche (AAR), exigée pour mener cette étude sur le territoire camerounais. Cette dernière vous sera délivrée par le Ministère de la Santé Publique.



APPENDIX 4: PROJECT TITLE: NUTRITION AND HEALTH STUDY

INFORMATION AND CONSENT FORMS

INFORMATION FORM

Good day Sir, Madame, Miss

This information sheet will give you all the information about the project. In fact, cardiovascular risk factors such as obesity, type 2 diabetes and hypertension have become a big public health problem recently; and their number is rising slowly around the world and particularly in Cameroon. Substances called nutrients contained in the foods we eat can help prevent from heart diseases. But with urbanization and increasing wealth most people are now leaving their traditional foods which contain most of these nutrients and are eating foods which are poor in good nutrients but very rich in poor fats, poor oils, sugars and bad substances such as cholesterol all of which are responsible of heart diseases, stroke and some cancers. Many research have shown that eating fruits and vegetables low in fats and calories but rich in many nutrients may reduce the risk of heart diseases, stroke, cancers, obesity, diabetes, hypertension and even poor nutrition. In other to find new ways of preventing and managing these diseases so as to reduce their occurrence, our research team design this study that aimed at investigating the impact of nutritional transition on heart and metabolic (cardio-metabolic) risk factors in Cameroon.

Aim: This project will help:

- To evaluate the influence of changing feeding habits on some parameters like: blood glucose level, lipid profile, blood pressure, body weight and substances like micronutrients levels in blood; which when are affected can lead to diabetes, dyslipidaemia, hypertension, obesity and even under nutrition.
- To understand the relationship between nutritional changes and cardiovascular risk factors in Cameroon
- To put in place strategies of managing cardio-metabolic risk factors linked to nutritional change.

Objective

This project seeks to evaluate the impact of nutritional transition or change on the occurrence of cardio metabolic risk factors in Cameroon.

Procedure

- Information about your identity (age, sex, marital status) as well as anthropometric measurements such as your weight, height, waist and hip circumference; family history of heart diseases and stroke; and life style (feeding habits and physical activity) will be collected using a questionnaire.
- All information given to us, including the results of your examinations will be treated with care in total secret. No information will be given out or published without your consent.
- Blood sample collection: a little quantity of your blood will be collected from you after you have fasted for 10 hours the night before. During this exercise, you may feel tired after your blood has been collected but you need to be patient. There will also be a finger-prick. This quantity of your blood will be used to evaluate fasting blood sugar level (glycaemia), biochemical markers of interest such as total cholesterol, triglycerides, HDL cholesterol and LDL cholesterol. In addition to evaluating all these parameters, the levels of some important substances called micronutrients will also be evaluated in the little quantity of blood of women of childbearing age (14 to 49 years).
- The collection of blood sample will be painful but the risk will be made little since only specialized medical personnel will be allowed to collect blood samples.
- Your blood pressure and total body fat mass will also be measured by simple measurements.
- Participating in this study will be advantageous because if your examinations show us that you have any of the above mentioned diseases or that you are at the point of developing them, we will inform you, give you dietetic advices (since knowing that you are predisposed to cardio metabolic diseases you will as much as possible reduce the consumption of high fat diets, tobacco and alcohol. Also if you are poorly fed or at risk of being poorly fed, nutritional advice will be given to you so that you can restore your nutritional status. This will protect you against cardiovascular diseases and improve your health status) and we will refer you to a hospital for treatment and better follow up.

You are free to choose whether to participate or not. If at any time you wish to stop the interview without having to explain your reasons you may do so. For this reason, it is important that the information you give us be correct and as truthful as possible.

- The field work will be done during a two year period and the samples will be kept during the entire study program (five years) after which they will be destroyed.
- This project has been approved by the National Ethical Committee
- Upon your demand, you would be informed on the project's global results.

CONTACTS

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- B) Pr. OBEN Julius Enyong, Professor**, University of Yaoundé I, Nutritional Biochemistry, Tel: (00 237) 77 74 50 87
- C) M'BOBDA MOMDJO Christelle**, PhD Student, University of Yaoundé 1, Laboratory of Food Sciences and Metabolism. Tel: (00 237) 70 47 69 56.
- D) TCHUENTE TONOU Boris**, Masters Student, University of Yaounde 1, Laboratory of Food Sciences and Metabolism. Tel: (00 237) 77 93 08 56
- E) NGUEDJO WANDJI Maxwell**, Masters Student, University of Yaounde 1, Laboratory of Food Sciences and Metabolism. Tel: (00 237) 97 17 63 77
- F) NATIONAL ETHIC COMMITTEE OF RESEARCH FOR HUMAN HEALTH**
Tel: (00 237) 22 76 21 14

CONSENT FORM OF PARENT

I,

Parent of.....

After the aim, objectives, advantages and disadvantage of the study on nutrition and health have been well explained to me, I understand

- a. What it involves
- b. That refusal to participate in this study will not affect my child's treatment or care in any way
- c. That my child or I, may withdraw at any time and this decision will not affect us adversely in any manner
- d. That the global research results can be sent back to me on my request

I therefore agree that my child participates in this study by

- Answering the questions yes no
- having his/her body measurements taken yes no
- allowing the collection of his/her blood sample yes no

Full name of participant (Child) _____

Full name of Mother or caregiver of child _____

Date _____

Postal address _____

Signature or thumb print _____

Date __/__/____ (DD/MM/YYYY)

CONSENT FORM OF PARTICIPANT

The aim, objectives, advantages and disadvantages of the study nutrition and health have been well explained to me and I understand

- a. What it involves
- b. That if I refuse to participate my treatment or care will not be affected in any way
- c. That I may withdraw at any time and it will not affect me badly in any way
- d. That the global research results can be sent back to me if I ask

I therefore agree to participate in this study by

- Answering the questions yes no
- The collection of the measurement of my body yes no
- The collection of my blood sample yes no
- To get the results obtained at the end of the study yes no

Full name of participant _____

Date _____

Postal address _____

Signature or thumb print _____

Date __/__/____ (DD/MM/YYYY)

APPENDIX 5: ARTICLE PUBLISHED

I-Article Published

M'bobda, C.M., Ngondi, J.L., Ntentie, F.R., Tchuenta, B.R.T., Nguedjo, M.W., Azantsa, B.G.K. and Oben, J.E. (2020) Assessment of Dietary Habits and Nutritional Status of Women of Childbearing Age in Cameroon: A Cross Sectional Study. *Open Journal of Epidemiology*, 10, 369-392. <https://doi.org/10.4236/ojepi.2020.104030>

Assessment of Dietary Habits and Nutritional Status of Women of Childbearing Age in Cameroon: A Cross Sectional Study

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Open Access

Abstract

Background: Malnutrition due to poor feeding habits, is still a serious public health issue in Cameroon. The objective of this study was to assess the dietary patterns and nutritional status of women of childbearing age in various geographical settings in Cameroon so as to propose intervention strategies. **Methods:** In a cross sectional study conducted from January 2014 to August 2015, women aged 14 - 49 years were randomly selected from the capital city Yaounde and four regions of Cameroon. Anthropometric measures were collected for nutritional status assessment. Data on diet habits and socio-demographic parameters were collected. **Results:** 608 women of reproductive age with average age 34.68 ± 0.39 years made up the study. Dietary patterns assessment revealed that, most study participants had a frequent intake of carbohydrate rich foods and fats and oils rich foods; but an infrequent intake of protein rich foods and vitamin and minerals rich foods in a week. Nutritional status assessment revealed that, 2.1% were underweight, 28.6% had a normal weight, 30.8% overweight, and 38.5% obese. The grass field regions presented the highest prevalence of underweight, overweight and obesity. Higher malnutrition levels were linked to low intakes of pulses and beans; milk and dairy products; vegetables and fruits food groups. **Conclusion:** Women of the study population had an unhealthy dietary pattern and a poor nutritional status. Therefore, strategies such as nutritional education are warranted and should be age and region specific, so as to target specific groups of women and ensure adequate nutritional status and health.

Keywords

Diet Pattern, Nutritional Status, Childbearing Age, Women, Cameroon

1. Introduction

Maternal nutrition status prior to conception is believed to affect embryonic and fetal growth [1]; and pre-pregnancy weight is a common indicator of a woman's nutritional status. Under nutrition in women contributes to 20% of maternal deaths [2]. Pre-pregnancy under nutrition, has been stated to lead to an increased risk of fetal loss, preterm birth, anemia, infections, fetal growth restriction (FGR), birth defects, low birth weight (BW), brain damage, admission to neonatal intensive care unit, and a longer duration of hospital stay, signs of the metabolic syndrome accompanied the catch-up in body weight and central adiposity [3]. A child's future nutrition status is affected before conception and is greatly dependent on the mother's nutrition status prior to, and during pregnancy [4]. Globally, maternal under nutrition and its consequences is estimated to account for 3.1 million child deaths annually [5]. In Africa, the lowest rates of underweight among women are found in Benin, Cameroon, Ghana, Lesotho, Rwanda, Swaziland and Togo [6]. Studies have found associations between dietary patterns and health outcomes and biomarkers, including the body mass index (BMI) [7]. Cohort studies have suggested that healthy dietary patterns up to 3 years before pregnancy resulted in a healthy pregnancy and pregnancy outcomes. In these studies, healthy dietary patterns are characterized by high intake of fruits, vegetables, legumes, nuts, and fish, and low intake of red and processed meat [5]. The need for maintaining optimal food habits throughout a woman's lifetime is essential to optimize her health and that of her offspring [8]. Women of reproductive age have the same dietary requirements as the general population [9]. In order to meet dietary guidelines, women of childbearing age should restrict simple carbohydrates while complex carbohydrates in the form of starches, legumes, seeds and bread should be limited to reasonable quantities. Useful protein sources can be meat, fish, cheese and dairy products (source of calcium), supplemented with small amounts of butter and vegetable fats [10]. Women of reproductive age, especially those who are planning a pregnancy, should be counseled to consume a well-balanced diet including fruits and vegetables, calcium rich foods, and protein-containing foods daily and increase their consumption of iron-rich or iron-fortified foods in conjunction with vitamin C-rich foods to enhance iron absorption [10]. In Cameroon, to our knowledge, few or no studies have assessed the dietary patterns of women of childbearing. Studies have been published on the dietary intakes of adolescent girls [11] [12] and women of childbearing age [13] in Cameroon. Therefore, with the increasing prevalence of obesity among women of childbearing age in Cameroon [14], it was therefore important to carry out this study which aimed at assessing the

preconception dietary consumption patterns, nutritional status and determinants of dietary patterns and nutritional status of childbearing age women in Cameroon. This is in order to provide adequate dietary advices in preconception specific to these groups of Cameroonian women, so as to prevent malnutrition; and adverse malnutrition pre-pregnancy and pregnancy outcomes.

2. Materials and Methods

2.1. Study Design and Setting Population

In a cross-sectional study conducted from January 2014 to August 2015, women of childbearing age randomly selected during mass health campaigns on nutritional and cardio-metabolic risk factors surveys residing in the selected areas made up the study population. The selected areas include: the political capital of the country (Yaounde), the Littoral, the Western, North-West and Far-North Regions of the country. From the western region, Bafoussam the capital city, Mbouda town-the chief place of the Bamboutos division; Babadjou, a village situated about 12 km of Mbouda; Dschang town located in the Menoua department; Fouban located in the Noun department; Bafou village situated in the Menoua division made up the study sites from this region. The far-north region was equally included, and from this region, its headquarters Maroua served as study area. The littoral region another region which made up the study setting had as study sites Nkongsamba a city located in the Mungo department and Ekangte a nearby village of Nkongsamba made up the study area. The last region for this study was the northwest region. The study sites from this region included Wum, Mbengwi, Ndu towns and Nyen village [15].

2.2. Inclusion and Exclusion Criteria

Eligible participants were Cameroonian-born women of childbearing age, aged 14 - 49 years who lived in the study areas for at least 6 months and who gave their informed consent. Pregnant or lactating women, those taking dietary supplements as well as physically and mentally disabled women were excluded from the study.

2.3. Sampling Procedure

The sample size was calculated based on the formula for basic sample size calculation for random sampling [16] [17]. The 95% confidence level, 5% margin of error and 82% prevalence of low adjusted plasma zinc concentration among women of childbearing age in Cameroon [13] were used. 480 subjects were required for the minimum sample size, but 608 women were included in the study to allow for precision. A multiple-stage sampling which was divided into three stages was adopted for the survey. In the first stage, regions were selected, in the second stage, cities, towns and villages were selected and in the third stage, women of childbearing age were targeted in various public gatherings in various study sites from which approbations were received. In addition to the region,

towns and villages randomly selected for this study, secondary data from other regions, towns and villages selected were used. In the first stage of the sampling, 3 regions (West, Littoral and Far-North) included in the previous health campaign were chosen. In addition to these, the North West region was randomly selected from the other seven regions of Cameroon using simple random sampling method (balloting) making a total of four regions retained. In the second stage consisting in randomly choosing cities, towns, little towns and villages; a big city (Bafoussam) was chosen at random from the 4 regions above and the political capital of the country (Yaounde) was also included. Four towns from the abovementioned regions which were randomly selected during the previous health campaign were also retained for this study and they were: Nkongsamba (littoral region), Maroua (far-north region), Dschang and Foumban (West Region). Three little towns: Mbengwi, Ndu, Wum were randomly selected from the North West region and one other town: Mbouda was selected from the west region. In addition, three villages selected during a previous campaign and proximate to three of the four little towns of the previous campaign were retained (Bafou, Bafolé, and Njimom). Two other villages were randomly selected during this study. They were Nyen which is proximate to Mbengwi in the North West region and Babadjou which is proximate to Mbouda in the west region. In the third stage, in each area retained or randomly selected, five quarters were randomly selected making a total of 65 quarters. In each of these 65 quarters, announcements and posters stating the purpose, the exact period and time of the study were read and pasted in churches, in general assemblies of various groups, in secondary schools, high schools, universities and all other places where women of childbearing age could be found. All women of childbearing age who met the inclusion criteria and gave their written consent were selected. A written consent was obtained from the parent or guardian of women younger than 21 years.

2.4. Research Instruments: Questionnaires for Data Gathering

Well-structured questionnaires were administered by well-trained health personnel. A questionnaire was conceived from the WHO STEPWISE questionnaire [18] to collect data on the identity (age, gender), the area or region of residence, the marital status, profession and educational level. Data collection was done through a face to face interview in data collections sites (health districts, health centers, churches, palace place ...). French, English and the local dialect were the languages used.

2.5. Dietary Habits

Food frequency for each food group

Dietary habits were assessed by collecting information on the frequency of consumption of various food groups using a food frequency questionnaire (FFQ). The FFQ included various food items consumed throughout the year

(traditional diets; imported cereals and locally made cereals, tubers, meat, fish, eggs, poultry; dairy products, fruits, vegetables; fats and oils; snacks, beverages; alcoholic drinks; sweet drinks; tea ...) [19]. The questionnaire was designed to capture all foods/beverages typically consumed, including traditional foods not available in stores and those available seasonally. Data were collected on the number of days in the last 7 days a participant ate specific food items. The various foods were then grouped into eight food items (cereals and tubers group; pulses and beans group; milk and dairy products group; meat, fish and egg group; vegetable group; fruit group; oil group; sugar and derivative group) [20]. These information were used to calculate the food frequency for each of the eight food items consumed in the last seven days for each participant and for the study population. All the consumption frequencies of food items of the same group were summed, and the value of each group above 7 was recorded as 7. Data on the frequency of consumption in a week of each food group were used to categorize each of the eight food group intake into three; *Low*: for food group intake 1 - 2 times per week; *Moderate*: for food group intake 3 - 4 times/week and *High*: for food group intake 5 - 7 times/week. The eight food groups obtained were further grouped into three food groups depending on their specific nutrient densities. These included carbohydrate and proteins rich foods, vitamins and minerals rich foods, fats and oils, and sugars rich foods.

Frequency of traditional diets in a week

Data on the frequency of intake of traditional diets in a week were also used to categorize traditional diets intake into three: *Low intake*: for 0 - 1 time per week; *moderate intake*: for intake 2 - 4 times per week and *high intake*: for 5 - 7 times per week

2.6. Anthropometric Parameters

The weight was recorded to the nearest 0.1 kg using an electronic balance (The Tanita™ BC-418 Segmental Body Composition Analyzer/Scale) with participants wearing light clothing. Height was measured with a Harpended™ stadiometer to the nearest 0.1 cm. Body Mass Index (BMI) was calculated by the formula $BMI = \text{Weight (kg)}/\text{Height}^2 \text{ (m)}$ and expressed as kg/m^2 . BMI was categorized as follows [21]: underweight: $BMI < 18.5 \text{ kg}/\text{m}^2$; normal weight: $BMI = 18.5 - 24.9 \text{ kg}/\text{m}^2$; overweight: $BMI = 25.0 - 29.9 \text{ kg}/\text{m}^2$; obese: $BMI \geq 30.0 \text{ kg}/\text{m}^2$.

2.7. Ethical Consideration

This study was approved by the National Ethic Committee of Research for Human Health of Cameroon (N° 2014/08/488/CE/CNERSH/SP). Authorizations were obtained from local and administrative authorities of each area of the capital city and four regions. Written consent was obtained from each woman who agreed to participate. Also, the parent or the guardian of each adolescent girl gave his/her consent for the child to participate in the study. Consents were obtained after the objectives, the minimal risks and benefits of the study were well

explained. To ensure the confidentiality of the collected data, codes were attributed to each participant and no name was revealed. All participants received their results individually and nutritional advices were given to each subject with respect to her nutritional state upon completion of the study measurements.

2.8. Statistical Analysis

Data were analyzed using the IBM SPSS statistical software package version 20.0. Results are expressed as means with standard error of the mean for continuous variables, or as percentages for categorical variables. Categorical variables were compared by the Chi square test and continuous variables compared by one way analysis of variance (ANOVA) followed by post hoc LSD. Regression analyses were performed to evaluate risk. The level of statistical significance was $P < 0.05$.

3. Results

3.1. Characteristics of the Study Population

Overall 1152 women of childbearing age were recruited for the study among which, 996 gave their consent after been explained the objectives of the study. 157 participants did not completely fill the questionnaires due to lack of patience or lack of interest in the study during the study course or lack of time, making a total of 839 participants who completely filled the questionnaires. Of the 839 participants, 231 of them had missing anthropometric measures and other important parameters, making a total of 608 with all the parameters required for this study and were therefore included in this study and the final analysis. The average age of the sample population was 34.68 ± 0.39 years. An analysis of socio-demographic data revealed that participants in the 31 - 40 years age group represented 34.4% of the study population. The North West region represented the highest number of participants (36%) of the study population. 24.3 percent of the participants of the study had a primary educational level. Up to 38.3 percent of the women of the study were housewives and most of them (70.6% in the 95% CI) were married (**Table 1**).

3.2. Dietary Patterns of the Population

Concerning the consumption of foods rich in carbohydrate, proteins, vitamins and minerals, fats and oils and sugars in a week, most participants (86.4%) of the study consumed cereals and tubers food groups 5 to 7 times in a week while 4.2% consumed this food group 1 to 2 times a week in the 95% confidence interval (CI). With regards to protein rich foods such as the pulse food group, up to 41.6% had a low intake (1 to 2 times in a week) while only 24.3% of the women had a high intake in a week. While, most of the women (57%) had a low intake of milk and dairy products food group in a week. Also, the meat, fish and egg group was consumed only 1 to 2 times in a week by up to 43.8% of the women while only 27.3% of them in the 95% CI had a high intake in a week. With regards to vitamins and minerals rich foods, the vegetable food group and

Table 1. Distribution of study population by socio-demographic parameters.

Characteristics	Frequency (N)	Percentage (%)	95% Confidence Interval	
			Lower	Upper
Age group				
14 - 20 years	63	10.4	8.1	12.8
21 - 30 years	141	23.2	19.7	26.8
31 - 40 years	209	34.4	30.3	38.1
41 - 49 years	195	32.1	28.1	36.0
Area of residence				
Yaoundé	72	11.8	9.2	14.6
Littoral	119	19.6	16.4	23.2
West	172	28.3	24.8	32.1
North west	219	36.0	31.9	39.8
Far North	26	4.3	2.6	5.9
Educational level				
Illiterate	121	19.9	16.6	23.0
Literates	89	14.6	11.7	17.4
Primary	148	24.3	20.9	27.8
First cycle	117	19.2	16.3	22.4
Second cycle	116	19.1	16.0	22.4
University	17	2.8	1.5	4.1
Profession				
Student	106	18.1	15.0	21.2
Employed	93	15.9	12.8	18.8
Odd jobs/farmers	110	18.8	15.7	21.9
Business	52	8.9	6.7	11.4
Housewives	247	38.3	34.2	42.2
Marital Status				
Single	74	12.2	9.5	14.6
Married	429	70.6	66.9	74.2
Widow/Divorced	105	17.3	14.3	20.4

the fruits group were taken 5 to 7 times in a week by 33.1% and 9.8% of the participants respectively (**Table 2**). The fats and oils group was eaten 5 to 7 times in a week by most women (56.9%) while sugar rich foods (sugar food group) was taken only 1 to 2 times in a week by most women (57.2%) in the 95% CI (**Table 2**).

3.3. Consumption Frequency of Various Food Groups by Age Group

The frequency of consumption of various food groups by age group is shown in **Table 3**. It can be seen that, a high intake of carbohydrate rich food was mostly observe among the 31 - 40 years age group (28.7%) followed by the 41 - 49 years age group (26.9%), although there was no significant difference ($p > 0.05$). A

moderate intake was mostly observed equally among the 31 - 40 years age group. A low intake of carbohydrate rich foods was mostly observed among the 41 - 49 years age group (1.6%) ($p > 0.05$). A high intake (5 - 7 times in a week) of protein rich foods was mostly observed among participants of the 31 - 40 years age group. 7.9% of the participants in this age group had a high intake of for pulses, 8.0% of them had a high intake of milk and dairy products ($p < 0.05$) and 8.8% of them had a high intake of meat, fish and egg food group ($p > 0.05$). Whereas, a low intake of this food category was mostly observed among the 41-49 years age group (19% for milk and dairy products ($p < 0.05$) and 14% for meat, fish and egg food group ($p > 0.05$)). The study of the frequency of consumption of vitamins and minerals rich foods revealed that, the 31 - 40 years age group (12.6% for vegetable intake and 21.0% for fruits intake) presented the highest percentage of participants with a low intake of this food category followed by the 41 - 49 years age group (12.4% for vegetable intake and 17.4% for fruits intake). With regards to the intake of fats and oils food group, the 31 - 40 years age group showed the highest percentage of low intake (9.7%) although there was no significant difference compared to other age group ($p > 0.05$). The 31 - 40 year age group recorded the highest percentage (7.5%) of a high intake of sugar food items compared to the other age groups ($p < 0.05$).

Table 2. Frequency of consumption of Carbohydrate, proteins, minerals and vitamins, lipid and sugar rich foods in the study population.

Food groups (N = 608)	Low intake (1 - 2 times/week) % (95%CI)	Moderate intake (3 - 4 times/week) % (95%CI)	High intake (5 - 7 times/week) % (95%CI)
Carbohydrate and proteins rich foods			
Cereals and tubers (Carbohydrate rich foods)	4.2 (2.4 - 5.9)	9.5 (7.1 - 12.1)	86.4 (83.4 - 89.3)
Pulses (Protein rich foods)	41.6 (37.2 - 46.3)	34.1 (30.0 - 38.9)	24.3 (20.4 - 28.2)
Milk and Dairy products (Protein rich foods)	57.0 (46.0 - 67.0)	27.0 (18.0 - 36.0)	16.0 (10.0 - 23.8)
Meat, fish and eggs (Protein rich foods)	43.8 (38.3 - 49.3)	28.9 (23.7 - 34.0)	27.3 (22.4 - 32.5)
Vitamins and Minerals rich foods			
Vegetables	38.5 (34.0 - 42.9)	28.4 (24.5 - 32.6)	33.1 (28.7 - 37.3)
Fruits	69.2 (62.5 - 75.0)	21.0 (15.7 - 25.9)	9.8 (6.3 - 14.2)
Lipids and sugars rich foods			
Fats and oils (lipids rich foods)	32.3 (27.1 - 37.8)	10.8 (7.3 - 14.5)	56.9 (51.4 - 62.8)
Sugar (sugar rich foods)	57.2 (51.7 - 62.9)	22.1 (17.8 - 26.7)	20.7 (17.0 - 25.0)

%: percentage; CI: Confidence Interval.

Table 3. Frequency of consumption of Carbohydrate, proteins, minerals and vitamins, lipid and sugar rich foods in the study population by age group.

Food groups	Age groups 14 - 20 years	21 - 30 years	31 - 40 years	41 - 49 years	P-Value
Carbohydrate rich foods					
Cereals and tubers					
(1 - 2 times/week)	5 (1.0)	3 (0.6)	5 (1.0)	8 (1.6)	0.441
(3 - 4 times/week)	4 (0.8)	14 (2.8)	17 (3.4)	13 (2.6)	
(5 - 7 times/week)	48 (9.5)	108 (21.3)	145 (28.7)	136 (26.9)	
Proteins rich foods					
Pulses					
(1 - 2 times/week)	22 (4.8)	49 (10.7)	64 (14.0)	55 (12.0)	0.851
(3 - 4 times/week)	15 (3.3)	36 (7.9)	48 (10.5)	57 (12.5)	
(5 - 7 times/week)	14 (3.1)	26 (5.7)	36 (7.9)	35 (7.7)	
Milk and Dairy products					
(1 - 2 times/week)	11 (11.0)	17 (17.0)	10 (10.0)	19 (19.0)	0.046
(3 - 4 times/week)	4 (4.0)	7 (7.0)	5 (5.0)	11 (11.0)	
(5 - 7 times/week)	5 (5.0)	1 (1.0)	8 (8.0)	2 (2.0)	
Meat, fish and eggs					
(1 - 2 times/week)	13 (4.2)	42 (13.6)	37 (12.0)	43 (14.0)	0.153
(3 - 4 times/week)	8 (2.6)	23 (7.5)	27 (8.8)	31 (10.1)	
(5 - 7 times/week)	17 (5.5)	17 (5.5)	27 (8.8)	23 (7.5)	
Vitamins and Minerals rich foods					
Vegetables					
(1 - 2 times/week)	15 (3.5)	43 (10.0)	54 (12.6)	53 (12.4)	0.926
(3 - 4 times/week)	16 (3.7)	32 (7.5)	37 (8.6)	37 (8.6)	
(5 - 7 times/week)	15 (3.5)	33 (7.7)	50 (11.7)	44 (10.3)	
Fruits					
(1 - 2 times/week)	26 (11.6)	43 (19.2)	47 (21.0)	39 (17.4)	0.453
(3 - 4 times/week)	4 (1.8)	10 (4.5)	18 (8.0)	15 (6.7)	
(5 - 7 times/week)	2 (0.9)	5 (2.2)	6 (2.7)	9 (4.0)	
Fats and oils and sugars rich foods					
Fats and oils					
(1 - 2 times/week)	13 (4.5)	25 (8.7)	28 (9.7)	27 (9.4)	0.895
(3 - 4 times/week)	2 (0.7)	10 (3.5)	9 (3.1)	10 (3.5)	
(5 - 7 times/week)	26 (9.0)	41 (14.2)	48 (16.7)	49 (17.0)	
Sugar					
(1 - 2 times/week)	18 (5.2)	56 (16.1)	55 (15.8)	70 (20.1)	0.003
(3 - 4 times/week)	7 (2.0)	25 (7.2)	26 (7.5)	19 (5.5)	
(5 - 7 times/week)	17 (4.9)	18 (5.2)	26 (7.5)	11 (3.5)	

3.4. Nutritional Status of the Study Population

In the population, only 2.1% of the women were underweight but up to 30.8% of them were overweight and 38.5% were obese in the 95% confident interval (Table 4). The prevalence of underweight was highest among women in the 21 - 30 years age group (0.8%) followed by those in the 41 - 49 years age group (0.7%) ($p > 0.05$). While that of overweight was highest in the 31 - 40 years age group (11.7%) and this was significantly different across all the age groups ($p < 0.05$). Study participants in the 41 - 49 years age group recorded the highest

prevalence obesity (16.1%). These differences were significant across all the age groups ($p < 0.05$) (**Table 4**).

3.5. Influence of Socio-Demographic Factors on the Nutritional Status of the Study Population

The assessment of nutritional status with respect to some socio demographic factors revealed that, with respect to the region/area of residence, the prevalence of underweight, overweight and obesity were highest in the northwest region and these were: 69.2% for underweight, 33.7% for overweight and 26.5% for obesity ($p < 0.001$). The western region recorded the next highest prevalence of all the forms of malnutrition and the differences in the rates were statistically significant ($p < 0.001$). Overweight (26.2%) and obesity (27.4%) were highest among study participants with a primary education and these were statistically significant ($p < 0.001$). Housewives and married women recorded the highest prevalence of underweight, overweight and obesity when the profession and the marital status were considered. The differences between the rates were statistically significant ($p < 0.001$). All these results are shown in **Table 5**.

3.6. Influence of Dietary Patterns on the Nutritional Status of the Study Population

Nutritional status assessment in association to the frequency of intake of various food categories in a week as shown in **Table 6** revealed that, a higher prevalence of underweight ($p < 0.05$), overweight and obesity ($p < 0.001$) were all associated to a high intake of cereals and tubers in a week. A low intake of pluses (beans, nuts ...) in a week whereas, was associated to a higher prevalence of obesity (42.6%) ($p < 0.05$) (**Table 6**).

A low intake of protein rich foods such as milk and dairy products in a week was significantly associated to a higher rate of overweight (58.3%) ($p < 0.05$). A low intake of meat, fish and egg food group was associated to a significantly higher rate of overweight (44.8%) ($p < 0.05$). A higher prevalence of underweight, overweight and obesity was linked to a low intake of vegetables in a week although not statistically significant (**Table 6**). It was also observed that, a high intake of fats and oils (5 to 7 times) in a week was associated to a significantly higher prevalence of overweight (54.5%) and obesity (63.2%) compared to the other intake categories ($p < 0.001$). Whereas a low intake of sugar foods (1 to 2 times) in a week was instead associated to a significantly higher prevalence of these forms of malnutrition (**Table 6**).

3.7. Risk Factors Influencing Abnormal Weight Status among Women of Childbearing Age in Cameroon

Socio-demographic risk factor assessment as shown in **Table 7** revealed that women in the 31 - 40 years age group were 2.187 times at risk of becoming overweight in the 95% confident interval. Also, all the age groups were risk factors of developing obesity $p < 0.05$ in the 95% CI. Based on the region or area of

residence, participants living the North West Region were 0.417 times less likely of becoming obese compared to those living in Yaounde $p < 0.05$ in the 95% CI. Risk assessment based on marital status showed that married women were 2.249 times more likely of becoming obese compared to single women, $p < 0.05$ in 95% CI. The educational level was not a risk factor of a poor nutritional status.

Table 4. Nutritional status of women of childbearing age in the overall population and by age group.

Age groups Weight Status	Overall % (95% CI)	14 - 20 Years	21 - 30 years	31 - 40 years	41 - 49 years	P- value
Underweight , n = 13 (BMI < 18.5 kg/m ²)	2.1 (1.2 - 3.5)	2 (0.3)	5 (0.8)	2 (0.3)	4 (0.7)	0.557
Normal , n = 174 (BMI = 18.5 - 24.9 kg/m ²)	28.6 (25.2 - 31.7)	43 (7.1)	54 (8.9)	45 (7.4)	32 (5.3)	0.001
Overweight , n = 187 (BMI ≥ 25 kg/m ²)	30.8 (27.1 - 34.5)	12 (2.0)	43 (7.1)	71 (11.7)	61 (10.0)	0.001
Obesity , n = 234 (BMI ≥ 30 kg/m ²)	38.5 (35.0 - 42.4)	6 (1.0)	39 (6.4)	91 (15.0)	98 (16.1)	0.001

CI: Confidence Interval; BMI: Body Mass Index; P-value Significant at $p < 0.05$.

Table 5. Nutritional status with respect to socio-demographic parameters of the subjects.

Parameters	Underweight Frequency (%)	Normal Frequency (%)	Overweight Frequency (%)	Obesity Frequency (%)
Area of residence	P = 0.018	P < 0.001	P < 0.001	P < 0.001
Yaoundé	0 (0.0)	15 (2.5)	22 (11.8)	35 (15.0)
Littoral	0 (0.0)	30 (4.9)	37 (19.8)	52 (22.2)
West	3 (23.1)	34 (5.6)	57 (30.5)	78 (33.3)
North west	9 (69.2)	85 (14.0)	63 (33.7)	62 (26.5)
North	1 (7.7)	10 (1.6)	8 (4.3)	7 (3.0)
Educational level	P = 0.846	P = 0.093	P < 0.001	P < 0.001
Illiterate	3 (23.1)	32 (5.3)	35 (18.7)	51 (21.8)
Literates	2 (15.4)	33 (5.4)	26 (13.9)	28 (12.0)
Primary	4 (30.8)	31 (5.1)	49 (26.2)	64 (27.4)
First cycle	4 (30.8)	40 (6.6)	37 (19.8)	36 (15.4)
Second cycle	0 (0.0)	33 (5.4)	34 (18.2)	49 (20.9)
University	0 (0.0)	5 (0.8)	6 (3.2)	6 (2.6)
Profession	P = 0.123	P < 0.001	P < 0.001	P < 0.001
Student	2 (15.4)	65 (11.1)	25 (13.9)	14 (6.3)
Employed	2 (15.4)	6 (1.0)	37 (20.6)	48 (21.5)
Odd jobs/farmers	2 (15.4)	39 (6.7)	32 (17.8)	37 (16.6)
Business	0 (0.0)	8 (1.4)	17 (9.4)	27 (12.1)
Housewives	7 (53.8)	51 (8.7)	69 (38.3)	97 (43.5)
Marital Status	P = 0.058	P = 0.105	P < 0.001	P < 0.001
Single	1 (7.7)	26 (4.3)	29 (15.5)	18 (7.7)
Married	8 (61.5)	112 (18.4)	129 (69.0)	180 (76.9)
Widow/Divorced	4 (30.8)	36 (5.9)	29 (15.5)	36 (15.4)

Table 6. Nutritional status with respect to frequency of intake of various food groups among women of childbearing age.

Food groups	Underweight Frequency (%)	Normal Frequency (%)	Overweight Frequency (%)	Obesity Frequency (%)
Carbohydrates rich foods				
Cereals and tubers	P = 0.002	P = 0.614	P < 0.001	P < 0.001
(1 - 2 times/week)	1 (7.7)	8 (1.6)	4 (2.6)	8 (4.2)
(3 - 4 times/week)	0 (0.0)	15 (3.0)	18 (11.7)	15 (7.9)
(5 - 7 times/week)	12 (92.3)	125 (24.7)	132 (85.7)	168 (88.0)
Proteins rich foods				
Pulses	P = 0.039	P = 0.971	P = 0.305	P = 0.001
(1 - 2 times/week)	8 (66.7)	54 (11.8)	56 (38.9)	72 (42.6)
(3 - 4 times/week)	1 (8.3)	45 (9.8)	47 (32.6)	63 (37.3)
(5 - 7 times/week)	3 (25.0)	33 (7.2)	41 (28.5)	34 (20.1)
Milk and Dairy products	P = /	P = 0.409	P = 0.005	P = 0.148
(1 - 2 times/week)	1 (100.0)	22 (22.0)	14 (58.3)	16 (48.5)
(3 - 4 times/week)	0 (0.0)	8 (8.0)	9 (37.5)	10 (30.3)
(5 - 7 times/week)	0 (0.0)	8 (8.0)	1 (4.2)	7 (21.2)
Meat, fish and eggs	P = 0.097	P = 0.555	P = 0.040	P = 0.132
(1 - 2 times/week)	1 (11.1)	45 (14.6)	43 (44.8)	46 (41.8)
(3 - 4 times/week)	6 (66.7)	24 (7.8)	30 (31.2)	29 (26.4)
(5 - 7 times/week)	2 (22.2)	24 (7.8)	23 (24.0)	35 (31.8)
Vitamins and mineral rich foods				
Vegetables	P = 0.670	P = 0.735	P = 0.461	P = 0.077
(1 - 2 times/week)	4 (40.0)	47 (11.0)	51 (38.3)	63 (40.4)
(3 - 4 times/week)	2 (20.0)	40 (9.3)	40 (30.1)	40 (25.6)
(5 - 7 times/week)	4 (40.0)	43 (10.0)	42 (31.6)	53 (34.0)
Fruits	P = 0.020	P = 0.428	P < 0.001	P < 0.001
(1 - 2 times/week)	8 (88.9)	54 (24.1)	45 (68.2)	48 (62.3)
(3 - 4 times/week)	0 (0.0)	12 (5.4)	18 (27.3)	17 (22.1)
(5 - 7 times/week)	1 (11.1)	6 (2.7)	3 (4.5)	12 (15.6)
Fats and oils and sugar rich foods				
Fats and oils	P = 0.102	P = 0.475	P < 0.001	P < 0.001
(1 - 2 times/week)	5 (71.4)	35 (12.2)	29 (33.0)	24 (25.3)
(3 - 4 times/week)	1 (14.3)	8 (2.8)	11 (12.5)	11 (11.6)
(5 - 7 times/week)	1 (14.3)	55 (19.1)	48 (54.5)	60 (63.2)
Sugar	P = 0.695	P = 0.092	P = 0.001	P < 0.001
(1 - 2 times/week)	3 (27.3)	55 (15.8)	50 (49.5)	91 (67.4)
(3 - 4 times/week)	5 (45.5)	18 (5.2)	32 (31.7)	22 (16.3)
(5 - 7 times/week)	3 (27.3)	28 (8.0)	19 (18.8)	22 (16.3)

Table 7. Socio-demographic risk factors of abnormal weight status (BMI) among women of Childbearing age in Cameroon.

Risk factors	Underweight OR (CI 95%) p-value	Overweight OR (CI 95%) p-value	Obesity OR (CI 95%) p-value
Age group (Years)			
14 - 20	1.0	1.0	1.0
21 - 30	0.893 (0.168 - 4.725) p = 0.893	1.865 (0.904 - 3.846) p = 0.092	3.632 (1.450 - 9.102) p = 0.006
31 - 40	3.393 (0.468 - 4.594) p = 0.227	2.187 (1.096 - 4.364) p = 0.026	7.326 (3.025 - 17.743) p < 0.001
41 - 49	1.566 (0.280 - 8.758) p = 0.610	1.935 (0.963 - 3.888) p = 0.064	9.568 (3.954 - 23.298) p < 0.001
Area of residence			
Yaoundé	1.0	1.0	1.0
Littoral	/	1.025 (0.544 - 1.933) p = 0.938	0.820 (0.456 - 1.476) p = 0.509
West	/	1.126 (0.622 - 2.039) p = 0.694	0.877 (0.506 - 1.522) p = 0.641
North west	/	0.918 (0.514 - 1.640) p = 0.772	0.417 (0.241 - 0.722) p = 0.002
Far-North	/	1.010 (0.382 - 2.670) p = 0.984	0.389 (0.146 - 1.040) p = 0.060
Educational Level			
University	1.0	1.0	1.0
Illiterate	/	0.746 (0.256 - 2.174) p = 0.591	1.336 (0.464 - 3.848) p = 0.592
Literate	/	0.757 (0.253 - 2.261) p = 0.618	0.842 (0.283 - 2.505) p = 0.757
Primary	/	0.907 (0.317 - 2.598) p = 0.856	1.397 (0.491 - 3.978) p = 0.531
First cycle	/	0.848 (0.291 - 2.468) p = 0.762	0.815 (0.280 - 2.374) p = 0.707
Second cycle	/	0.760 (0.260 - 2.221) p = 0.616	1.341 (0.464 - 3.873) p = 0.588
Marital status			
Single	/	1.0	1.0
Married	/	0.667 (0.401 - 1.111) p = 0.120	2.249 (1.279 - 3.955) p = 0.005
Widow/divorced	/	0.592 (0.314 - 1.115) p = 0.105	1.623 (0.833 - 3.161) p = 0.154

OR: Odd Ratio; CI: Confidence Interval; significant at $p < 0.05$.

4. Discussion

The present work was undertaken to provide information on the dietary habits and nutritional status of women of childbearing age in Cameroon. The assessment of the dietary patterns of women of childbearing age of the study revealed that in a week, most women (more than 50%) consumed cereals and tubers, and fats and oils food groups while a few of them had a high intake of vegetables, fruits, milk and dairy products, meat, fish and eggs and also pulses food groups in a week, suggesting an overall unhealthy food habit among most women of the study. Also, in general, in all the age groups, cereals and tubers food group, and fats and oils group were mostly consumed 5 to 7 times in a week. With regards to pulses intake, in all the age groups, beans and nuts food group were mostly eaten 1 to 2 times in a week. The same observation was made for protein rich foods groups such as milk and dairy products food group, meat, fish and egg food group; and vitamins and minerals rich food groups such as vegetables group and fruit group; and sugar food group intake in a week. Overall, in all these five food categories or groups mentioned above, more than 40% of the participants reported a low intake in a week. Nation-wide data on dietary intake and patterns are not available in Cameroon for women of childbearing age. Dietary patterns are population specific, since they are influenced by sociocultural factors and food availability [22]. The observation made in this study has earlier been stated whereby, Cameroonian eating habits is mainly characterized by low

fruits and vegetables consumption, added to the higher consumption of foods rich in starch and sugar [23]. As earlier shown, in the present study, dietary habits are mostly characterized by cereals and tubers like rice, maize, cassava, corn fufu ... showing that carbohydrate rich food serve as the major source of energy among the majority of women of the study. Similar findings were found in a study in Nepal among women of childbearing age [24]. The main reason for the lack of a balanced diet in the food habits of women of the study population could be that there is a considerable lack of nutritional knowledge regarding healthy dietary patterns and habits by most women. According to Nkengfack *et al.*, studies show that nutritional knowledge in Cameroon is lacking [25]. A healthy diet must be balanced not only in terms of macronutrients content (intake of proteins, carbohydrates and fats), but also in terms of micronutrients intake (vitamins and mineral) [22]. The need for maintaining optimal food habits throughout a woman's lifetime is essential to optimize her health and that of her offspring [8].

This study revealed that the nutritional status of women of childbearing age in the study is poor in general. Our analysis reveals that, a considerable percentage of the study participants were obese (38.5%), 30.8% overweight, 2.1% underweight and 28.6% of them had a normal weight status. A mother's nutritional status before pregnancy can affect reproduction and pregnancy outcomes, and pre-pregnancy weight is a common indicator of a woman's nutritional status [26]. Nutritional status is an important aspect of health and wellness before and during pregnancy. The prevalence of underweight (2.1%) was lower than that reported by WHO, in Cameroon among women of childbearing age which was 6.9% [6]. This percentage is equally lower than that observed in the 2004 demographic and health survey (DHS) [27] and 2011 DHS [28] carried out in Cameroon which found a prevalence rate of 7% and 8% respectively of underweight among women of childbearing age. While that of obesity (38.5%) was higher than the proportions observed in the same DHSs which were 29.40% in 2004 as compared to 32.17% in 2011 for obesity [27] [28], making an increase of 2.77% between 2004 and 2011 [14]. These percentages show that the prevalence of underweight among reproductive women in Cameroon is on the decrease while that of obesity is on the rise. In a 2017 WHO report, Cameroon has been cited among the countries in WHO African regions with the lowest rates of maternal underweight [6]. Also, the above results show that despite the high prevalence of pre-pregnancy overweight and obesity in this population, the prevalence of women who are underweight before pregnancy still persists in Cameroon. Under nutrition in women has been shown to contribute to 20 percent of maternal deaths, and is a significant risk factor for stillbirths, preterm births, small for gestational age and low birth weight babies [2]. Our findings also showed that, the rate of obesity increased with age and its prevalence was highest in the 41 - 49 years age group; the rate of overweight was the highest in the 31 - 40 years age group. Also, all the age groups were found to be risk factors of obesity, with women in the 41 - 49 years age group 9.568 times most likely of becoming obese.

A recent study also found that overweight and obesity increased with maternal age among women of childbearing age in Cameroon [29].

The northwest region showed the highest prevalence of underweight and overweight and it was equally observed that participants living in this region were less likely of becoming obese. The rates of obesity was highest in the western region, the latter showing the next highest rates after the northwest region. The prevalence of overweight and obesity in this study were higher than those observed in a study which used the database of the 1998, 2004 and 2011 DHSs in Cameroon [30]. The fact that the northwest and west regions recorded the highest prevalence of poor nutritional status, could be linked to their dietary habits. The northwest and the west regions also called the grass fields, are zones of Cameroon that have humid and wet climates, influencing natural food resources and dietary habits. Tubers are available all year round in these regions, bean species are the main sources of plant protein [25] and as stated, beans is usually eaten with a cereal such as rice in the North West region [31]. Tubers, and *fufu* made from some tubers are often consumed in these regions which provide the bulk of energy in the diets. In addition, many of the traditional soups and dishes of these regions, contain large amounts of palm oil, the basic fat used in the Cameroonian kitchen [32]. Tubers and palm oil which are energy dense but poor in nutrients are usually eaten with small amounts of vegetables which provide little amounts of micronutrients. Therefore, the energy dense and low micronutrients content of most diets coupled to the lack of dietary diversification in these regions might be reasons for the high prevalence of overweight and obesity. Also a limited nutrition knowledge stated as possible reason for unhealthy dietary patterns among university students in the North West region in a study carried out in Cameroon [31], could also account for the high prevalence of all forms of malnutrition in these two regions.

Higher rates of overweight and obesity were associated to a primary educational level. Housewives and married women recorded the highest numbers of underweights, obese and overweight participants. Also, married women were more likely of becoming obese. The high level of overweight and obesity among women with a primary educational level is consistent with a finding made in Cameroon which showed that women aged 15 - 49 years in Cameroon with a primary educational level were 1.47 times more likely to be overweight [14]. The fact that pre-pregnancy poor nutritional status were associated to no or low educational levels (illiteracy, primary educational level and secondary level) may be due to the lack of finances to get access to quality and diversified foods such as fruits, rich in micronutrients, low in calories and saturated fatty acids that enhance better micronutrient status and prevent weight gain. Another possible explanation to these high rates of malnutrition among women with no educational level and those with a low level could be the lack of nutritional knowledge. Studies have pointed out the positive association of a high educational level of the mother and increased good attitudes towards healthy eating habits [33] [34]; and the association between nutritional education among illiterate mothers and

good knowledge about dietary practices during pregnancy in Nigeria [35].

The frequent intake of cereals and tubers which are carbohydrate rich foods, was associated to higher rates of underweight, overweight and obesity while a low intake of pulses; milk & dairy products (proteins rich foods); vegetables and fruits food groups (vitamins and minerals rich foods) in a week were all associated to higher rates of underweight, overweight and obesity. Optimum intake of food sources of various micronutrients are known to prevent deficiencies. Under nutrition, occur due to insufficient intake or even sufficient intakes combined with impaired absorption due to infection, disease, or inflammation [36]. These various mechanisms may explain the association between higher rates of underweight and low intakes of various food sources. However, the higher levels of all forms of malnutrition in association to frequent intake of cereals & tubers group in this study, may be related to the high energy and low micronutrients content of food items of this food group.

5. Limitation of the Study

Limitation includes self-reporting of data by participants; which made dietary assessment difficult to appreciate.

6. Conclusion and Recommendations

This study revealed that in general, women of childbearing age of the study had an unhealthy dietary pattern mostly frequent in carbohydrate rich foods such as cereals and tubers, and foods rich in fats and oils but infrequent in proteins rich foods, and vitamins and minerals rich foods such as vegetables and fruits. The assessment of the nutritional status revealed that in general, most study participants had a poor nutritional status. A few of the study participants were underweight (2.1%). High rates of overweight and obesity were observed and were found to increase with age and these rates were higher in some regions (north-west and western regions) compared to other regions suggesting that nutrition interventions should be age and region specific. Low educational levels were associated to higher prevalence of underweight, overweight and obesity suggesting the need for nutritional education to improve the knowledge of these women on good nutritional practices. Also, the low intake of most food groups was linked to higher levels of underweight, overweight and obesity suggesting the need to emphasize on the consumption of proteins rich foods, and vitamins and minerals rich foods such as vegetables and fruits to improve nutritional status. In addition, the frequent consumption of vegetables and fruits rich in micronutrients and low in energy should be encouraged in order to fight both extremes of malnutrition among women of childbearing age in these regions of Cameroon.

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Consent for Publication

Not applicable.

Availability of Data and Materials

Data and materials used in this study are available on reasonable request.

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Authors' Contributions

CMM, JLN, and BGKA conceived and designed the study. CMM, BRTT and MWN collected data and performed laboratory tests. CMM, FRN and BGKA carried out the statistical analysis. CMM, BGKA drafted the manuscript. JEO supervised the study. All authors read and approved the final manuscript.

Conflicts of Interest

The authors declare that there is no conflicts of interest regarding the publication of this paper.

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Appendix: Questionnaire

NUTRITION AND HEALTH STUDY

CODE: _____ Tel. _____ Age: _____ Sex: _____
 Participant's Name: _____
 Place of birth: _____ Ethnic group: _____
 Region of Origin: _____
 Division of Origin: _____
 Profession: _____ Religion: _____
 Region or Place of Residence: _____
 Hormonal Status: _____

PARAMETERS OF INTEREST:

Weight (kg): _____ Height (m): _____
 BMI (kg/m²): _____ % Body fat: _____
 Waist circumference (cm): _____ Hip circumference (cm): _____
 SBP /DBP mm Hg: _____
 FC.....mg/dL _____ GLYCAEMIA:mg/dL

A) DIETARY HABITS AND HEALTH STATUS

- 1) How many times do you usually eat per day?
 _____ Number of times/day
- 2) At which time/moment of the day do you eat? _____
- 3) Do you cook your meals yourself?
 Always/Often; Occasionally/Sometimes; Rarely/Never
- 3a) Who cooks your meals? _____
- 4) Do you sometimes eat out of the house?
 Yes 4a, 4b No 5
- 4a) When you eat out of the house, where do you generally eat? (Several answers are possible)
 VIP Restaurant; Restaurant; mama put
 Road; Work/ canteen of the company; Cafeteria
- 4b) For what reason(s) do you eat out of the house? (Several answers are possible)
 Work place far away from the house; For pleasure
 Nothing to eat in the house; Other reasons (precise) _____
- 5) During the last 7 days, did you eat out of the house? No Yes
- 5a) How many days in the week? _____ Nber of days/week
 How many times these days _____ Nber of times /day
- 6) During the last 7 days, did you buy cooked food in the street? No Yes
- 6a) How many days in the week? _____ Nber of days/week
 How many times these days? _____ Nber of times /day
- 7) During the last 7 days, did you eat in a restaurant? No Yes
- 7a) How many days in the week? _____ Nber of days/week
 How many times these days? _____ Nber of times /day

8) During the last 7 days, did you eat in a mama put?

No Yes

8a) How many days in the week? _____ Nber of days/week How many times these days? _____ Nber of times /day

9) During the last 7 days, did you eat in a canteen of a company? No Yes

9a) How many days in the week? How many times these days? _____ Nber of days/week _____ Nber of times /day

10) During the last 7 days, did you eat in a cafeteria No Yes

10a) How many days in the week? How many times these days? _____ Nber of days/week _____ Nber of times /day

11)-13) Refer to the food frequency questionnaire in the form of a table below to answer questions with respect to your food habits during the last seven days. Please tick all possible answers.

14) Do you consider that you eat too much sugar for your health? Yes No undecided

15) Do you consider that you eat too much fat and oils for your health? Yes No undecided

16) Do you consider that you eat too much salt for your health? Yes No Undecided

17) Do you consider that you eat too much for your health? Yes No Undecided

18) Do you consider that by changing your way of eating you can ameliorate your health? Yes No Undecided

19) Compared to other women of your age, you would say that your usual food/diet is ...

Very good; Good; Fairly good; Bad; Undecided

20) Compared to other women of your age you would say that your health is in general ...

Very good Good Fairly good Bad Undecided

20a) Do you have any disabilities Yes No

20b) If yes, please specify _____

21) Do you receive information on diet? Yes No

21a) If yes, by which means do you receive information on diet (several answers are possible)

Radio Television Newspapers, review or brochures

Books or courses Health professionals (doctors, dietetics, nurses)

Others (precise) _____

21b) Would you like to receive information on diets/foods? Yes No

21c) By which means would you like to receive information on diets/foods (several answers are possible): Radio Television Newspapers, review or brochures

Books or courses Health professionals (doctors, dietetics, nurses) Others (precise) _____

22) Do you currently follow a particular diet for your health? No Yes

- 22a) For which reason do you follow this diet? Specify _____
- 23) In your household, are there any foods which are prohibited to you? No
 Yes
- 23a) If Yes, Which? _____
- 23b) Why are these foods prohibited? _____
- 24) Do you take any dietary supplements Yes No
- 24a) If yes, which supplements? _____
- 24b) For what reasons do you take them? _____
- 25) As a woman of childbearing age, are you presently pregnant? Yes No
- 25a) If yes, what is the age of your pregnancy? _____
- 25b) If No, when was your last pregnancy? _____ months/ _____ years
- 25c) How many pregnancies have you carried to the end _____ Number of full-term pregnancies
- 25d) Are you a breastfeeding/lactating mother? Yes No

B) INFORMATION ON AREA/REGION OF RESIDENCE (URBANISATION)

- 26) Where were you born? Specify the place of birth: _____
 Rural area; Secondary city /little city; Big city
- 26a) How long have you lived in your place of birth? Specify: _____
- 27) For how long have you continuously lived in the current region? _____ Years
- 27a) Previously, where did you live?
 Does not apply (has always lived in region or area of residence since birth)
 Big city; Other secondary city; In the village /Rural area; Abroad: (Specify) _____
- 27b) For how long have you lived in the current place? _____ Years
- 28) For which reason(s) do you now live here? Specify _____

C) SOCIO-ECONOMIC INFORMATIONS

- 29) What is your current matrimonial status? Married or lives with a partner; widow, divorced, separated; single/Fiancé, lives alone
- 30) In which type of household do you live? Monogamous polygamous
- 31) What is the gender of the head of the family? Male Female
- 32) What is your highest level of education?
 No schooling or illiterate
 Literate; Primary
 First cycle secondary education (1st cycle, Form 1 to 5)
 Second cycle and Professional school
 University
- 33) What is your main source of revenue?
 Salary (permanent employment)
 Salary (daily or temporal)
 Business; Help, assistance; Partner
 Other _____
- 34) Have you worked or exerted a business or any activity that generated money

during the last 12 months? No Yes

34a) If yes, For how long have you worked during this period? _____ Number of months

34b) What was your main job? Give all possible details _____

35) What is the main source of revenue of your partner? _____

36) What is the total number of people in your household (Including you)? _____
 _____Number of women of child bearing age 15 to 49 years; _____ Number of women aged above 49 years; _____ Number of men aged above 15 years; _____Number of adolescents (12 ≤ years ≤ 15); _____Number of children (<12 year)

Question 11, 12 & 13:	Yes	No	Nber days /Week	Nber of times/ day
11 During the last 7 days, did you eat:				
a) Aerated beverage (Coca Cola, Fanta,.....)				
b) Bonbons				
c) Chocolate (paste, butter, powder...)				
d) Fried (plantain, irish potatoes, sweet potatoes)				
e) Hamburger				
h) Popcorn				
j) Pizza				
k) Ice cream				
l) Cake (cream, with chocolate)				
12- During the last 7 days, did you eat:				
a) Pasta products (Macaroni, spaghetti, etc.)				
b) Pork-butchenes (jambon sausages,)				
c) Canned fish (Sardines, pilchard ...)				
d) Conserved vegetables (peas,)				
e) Cheese				
f)salade				
g) Shawarma				
h) Biscuits (salty, sweetened)				
13a- During the last 7 days, did you eat:				
Makara (Table tennis) (Banana+ Cassava) or Accra (Koli Bean)				
a) Koli and complements				
b) rice pap or compap				
c) fufu millet; rice or sorghum or corn fufu				
d) plantain/banana and sauce				
e) Bobolo and sauce				
f) Cassava fufu and sauce				
g) tubers and sauces (tomato or groundnut)				
h) Beans				
i) Rice +sauce				
j) eru+ water fufu				
k)kok+ Complement				
l) ndole+ Complement				
m) Tomato soup				
n) Groundnut soup				
o) Vegetables				
p) fruits				
q) Achu and yellow soup				
r) Fufu corn and djamadjana				
s) Porridge cocoyam/plaintain +bitter leaves				
t) Egusi pudding or Egusi soup				
u) Porridge yam or Hotpot (Irish)				
v) Pound Beans + banana or Irish or plantain				
w) Corn cake				
Kawacoco bibe				
Ekwang				
y) Fufu corn and Yellow soup (ou Ohru)				
z) Fufu corn and Nhui				
Porridge banana or Porridge plaintain				
Kawacoco				
Koli corn				
Corn chaff				
Eggs, Fish, Meat, Milk, Dairy products (Encircle all possibilities)				
Refined oil (Precise) :				
Palm oil				
Yellow or sweet yam				
Others (Specify)				
1-				
2-				
13b What are your traditional dishes?				
13c How many times do you eat them per week?				