Research Article

Lipid Profile Abnormalities Observed in Obese Cameroonian Adults do not Depend on Their BMI or Abdominal Circumference

Vicky Jocelyne Ama Moor[1,2*], Doris Bibi Essama[1], Batakeh B Agoons[1], Joel Cedric Bayem[4], Ntep Gwet Marie[3,4], Ahmadou Musa Jingi[5], Jan Rene Nkeck[1], Bernadette Ngo Nonga[1,2]

[1]Faculty of Medicine and Biomedical Sciences, University of Yaoundé I, Yaoundé, Cameroon

[2]Yaoundé University Hospital Centre, Yaoundé, Cameroon

[3]Yaoundé Central Hospital, Yaoundé, Cameroon

[4]Higher Institute of Medical Technology, Yaoundé, Cameroon

[5]Faculty of Health Sciences, University of Bamenda, Bamenda, Cameroon

***Corresponding author:** Vicky Jocelyne Ama Moor, Faculty of Medicine and Biomedical Sciences, University of Yaoundé I, Yaoundé, Cameroon.

Received: 09 December 2021; **Accepted:** 16 December 2021; **Published:** 05 January 2022

Citation: Vicky Jocelyne Ama Moor, Doris Bibi Essama, Batakeh B Agoons, Joel Cedric Bayem, Ntep Gwet Marie, Ahmadou Musa Jingi, Jan Rene Nkeck, Bernadette Ngo Nonga. Lipid Profile Abnormalities Observed in Obese Cameroonian Adults do not Depend on Their BMI or Abdominal Circumference. Archives of Clinical and Biomedical Research 6 (2022): 30-40.

Abstract

Background

Obesity and dyslipidemia are both lipid related diseases, and the management of obesity involves considering disorders of lipid metabolism. This study aimed to evaluate the relationship between obesity and lipid profile parameters in Cameroonians adults.

Methods

We conducted a cross-sectional study at two teaching hospitals in Yaoundé (Cameroon). Consenting adults with a BMI ≥25kg/m² were recruited. Participants taking lipid-modifying drugs were excluded. Total cholesterol, HDL, and triglycerides levels were assessed on a fasting blood sample using standardized and automated enzymatic methods.

LDL cholesterol was estimated using the Friedewald formula. Chi² test was used to compare proportions and seek for associations between lipid profile components and BMI, with a significance threshold of 0.05.

Results

136 participants (105 females, 77.3%) aged 58.8 ± 11 years on average were included. They were classified as overweight (44.1%), grade I (35.2%), grade II (13.2%), and grade III obesity (7.3%). Their main comorbidities were hypertension (63.9%) and diabetes (60.2%). Among them, 72.7% had a sedentary life style while 31.6% were alcohol consumers. The prevalence of dyslipidaemia was 52.9%, with main subtypes being HDL hypocholesterolaemia (54.4%) and hypertriglyceridemia (47.06%). There were no significant associations between BMI and lipid profile indices.

Conclusion

Half of the obese Cameroonians adults of the sample suffer from dyslipidemia, but it is not directly related to their BMI.

Keywords: Cameroon; Dyslipidemia; Obesity

1. Introduction

The World Health Organization (WHO) defines obesity as abnormal or excessive fat accumulation that presents a risk to health [1]. It is considered a BMI ≥ 30 kg/m^2. It is an increasing global public health issue and the second most common cause of preventable death after smoking [2]. Nearly one-third, or about 61.3 million adults over the age of 20 worldwide, are obese [3]. Obesity is a multifaceted and complex disease of genetic, socio-economic, environmental, and behavioral origin, and increases the risk of morbidity and mortality, significantly decreasing life expectancy by 5–10 years [4]. It is now clear that the prevalence of obesity has increased rapidly worldwide over the past two decades, especially in Sub-Saharan Africa, due to rapid modernization and diet changes [5]. This trend is not gender, age, or ethnic group specific. The conditions associated with obesity are numerous and varied and include insulin resistance, metabolic syndrome, and alterations in lipid metabolism [6]. The importance of an in-depth understanding of obesity and its effects on lipid metabolism is crucial.

Dyslipidemia is a metabolic abnormality leading to a persistent increase in plasma cholesterol and triglyceride levels. Approximately 60-70% of patients who are obese are dyslipidemic [7]. Multiple epidemiologic studies have shown obesity and dyslipidemia to be modifiable risk factors of coronary heart disease (CHD) [8-10]. Interestingly, despite the greater burden of CHD affecting low-income countries, the majority of these studies have been done in western settings [11]. Accruing evidence suggests an influence of geographical location on the prevalence and associated factors of obesity and dyslipidemia [12]; indeed in Cameroon, Ama Moor et *al*. found a high dyslipidemia prevalence of 56.4%, in patients with cardiovascular risk or disease [13], compared to 31.8% in China [14]. As CHD can result from obesity and dyslipidemia, adequate understanding of these in a sub-Saharan obese subpopulation can help in lowering the burden of heart disease in these regions. Currently, there is little information on the characteristics of the lipid profile of obese populations in sub-Saharan African studies. Understanding the clinical presentation of dyslipidemia in obesity is of paramount importance

for public health policies, especially in resource-limited countries. Thus, the objective of this study was to assess the relationship between obesity and lipid profile parameters in adult Cameroonians.

2. Methods

2.1. Ethical considerations

The study protocol was approved by the Ethical committee of the Faculty of Medicine and Pharmaceutical Sciences of the University of Douala, Cameroon (1458/CEI-UDm/04/2018/T). All study procedure was done in accordance with the 2013 revised Helsinki Declaration and the Good Clinical Practice guidelines of the International Conference on Harmonization. All participants provided a written informed consent form before beginning this study.

2.2. Study design and Setting

We carried out a cross-sectional study at the internal medicine units of two teaching hospitals in Yaoundé (Cameroon). Study participants were recruited from 1st February 2018 to 30th June 2018. Participants for this study were recruited from a pool of patients consulted at the two designated hospitals. All adult subjects aged 20 years and above, with a BMI ≥ 25 Kg /m^2, and who gave their consent were subsequently recruited. Subjects who refused to participate in the study, those taking lipid-modifying drugs such as statins, and those with hemolyzed blood serum found during laboratory analysis were excluded. Hemolysis has been proven to significantly interfere with blood lipid measurement [15].

2.3. Sample size estimation

The sample size was estimated for convenience. Our minimum sample size was 75 participants, estimated using the Cochran's formula [16].

3. Data Collection

3.1. Assessment of Covariates

Baseline characteristics including age, sex, race, marital status, smoking status, family history of obesity/dyslipidemia, and level of physical activity were obtained using standardized questionnaires. Physical activity was defined as participating in moderate-intensity sports or activities that cause an increase in breathing or heart rate for 10 minutes continuously.

3.2. Assessment of Obesity

All participants underwent a complete physical exam and their clinical variables were documented. Body mass index was assessed using the De Quetelet formula [17]. Then, participants were subclassified into the following quartiles according to their BMI values [17]:

Overweight: 25-29.9 kg/m²
Grade 1 obesity: 30-34.9 kg/m²
Grade 2 obesity: 35-39.9 kg/m²
Grade 3 Obesity; ≥40 kg/m²

3.3. Assessment of Lipid Profile

Blood was sampled from a vein in the antecubital fossa after proper site hygiene. To minimize the risk of hemolysis, a loose venous occlusion model was employed. The sample was immediately put in a non-anticoagulant (dry tube) tube and sent to the laboratory within 20 minutes of sampling. Prior to blood sampling, participants were asked to fast for 12 hours prior.

The following markers were used for lipid profile analysis: Total cholesterol (TC), HDL cholesterol (HDL-C), LDL cholesterol (LDL-C) and Triglycerides (TG). Analysis was done using the

CYAN START photometer of the CYPRESS Laboratory. Total cholesterol, HDL cholesterol, and triglycerides were determined using an enzymatic method found in commercial kits of the CYPRESS Laboratory (*Langdorp*, Belgium); the results were validated using a quantitative control serum. LDL-C was obtained using the Friedewald equation [18].

3.4. Operational terms

Obesity and overweight were defined in this study, according to the WHO BMI criteria [17]. Dyslipidemia was diagnosed by the presence of any lipid profile abnormality and was divided into hypercholesterolemia (total cholesterol ≥ 2 g/L), hyper-LDL cholesterolemia (LDLc ≥ 1.6 g/L), hyper triglyceridemia (triglycerides ≥ 1.5 g/L), and/or a hypo HDL cholesterolemia (HDLc < 0.4 g/L) [19]. The atherogenic index was calculated as the ratio of plasma concentration of total cholesterol to HDL-C and was considered high, if above >4.85.

3.5. Statistical analysis

Data were encoded using the CS Pro software version 4.1. Statistical analysis was done using Epi Info 3.54 software and represented in tables using the Microsoft Excel 2010 software. The chi-square test was used to compare proportions. Difference was found to be significant if the p-value was less than 0.05.

4. Results

A total of 180 subjects were invited to take part in this study. Subjects with incomplete data and who refused to participate were excluded. Thus, 136 participants were enrolled finally. In this sample, heart failure, hypertension, and diabetes were the most common comorbidities.

4.1. Sociodemographic and lifestyle characteristics

As shown in table 1, this study population had a mean age of 58.9 ± 11.9years, with 105 (77%) being women. The most represented age group was 65-70 years of age (21.3 %). Mean BMI was 32.1 ±5.1 kg/m². When stratified by BMI, 10 participants (7.4%) were found to be morbidly obese. A total of 51 participants had a positive psycho-stimulant consumption history.

Variables		**Overall**
N (%)		136 (100)
Mean age, year, (SD)		58 (11.9)
Age, min-max, year		20-85
Comorbidities, n (%)		
	HTA	87(63.8)
	Type 2 diabetes	82(60.3)
	Heart Failure	6(4.4)
	Physical inactivity	99 (72.9)
Mean BMI, Kg/m² (SD)		23.8 (3.6)
BMI (Kg/m²), n (%)		
Overweight	25 – 30	60(44.1)

grade 1 obesity	30 – 35	48(35.3)
grade 2 obesity	35 – 40	18(13.2)
morbidly obese	>40	10(7.4)
Abdominal circumference (cm), SD		102.5 (11.5)
Physical inactivity		33 (22.9)
smokers, n (%)		8 (5.9)
Alcohol consumption, n (%),		43 (31.6)

Table 1: Characteristics of the sample.

4.2. Assessment of dyslipidemia

Lipid profile analysis in this cohort of participants are shown in Table 2. Seventy-two (72) participants had one or more forms of dyslipidemia, giving a prevalence of 52.9%. Analysis showed that the major form of dyslipidemia found was HDL hypocholesterolemia (54.4%). Atherogenic risk evaluation was found to be high in 26.4%. In Table 3, the presence of diabetes was found to be significantly associated with dyslipidemia.

	Lipid abnormalities			
	Yes	**No**	**TOTAL**	**p value**
Associated factors	n (%)	n (%)	n (%)	
High blood pressure				
Yes	43(49.4)	40(50.6)	83(64)	0.14
No	29(59.2)	20(40.8)	49(36)	
Diabetes				
yes	36(43.9)	46(56.1)	82(60.1)	0.004
No	36(66.67)	18(33.3)	54(39.9)	
Smokers				
Yes	5(62.5)	3(37.5)	8(5.9)	0.3
No	67(52.3)	61(47.7)	128(94.1)	
Alcohol consumption				
yes	25(58.14)	18(41.86)	43(31.6)	0.3
No	47(50.54)	46(49.46)	93(68.4)	
Physical activity				
Yes	20(54.1)	17(44.7)	37(27.2)	0.4
No	52(52.5)	47(47.5)	99(72.8)	
Gender				
Female	53(50.5)	52(49.5)	105(77.2)	0.14
Male	19(61.3)	12(38.7)	31(22.8)	

Table 2: Association of comorbidities and dyslipidemia.

Variables	Frequency (%)
Cholesterol Total (g/l)	
<1.4	22 (16.2)
[1.4; 2[	91 (66.9)
>2	23 (16.9)
Cholesterol HDL (g/l)	
<0.4	74 (54.4)
[0.4; 0.6[	51 (37.5)
>0.6	11 (8.1)
Cholesterol LDL (g/l)	
<1	69 (50.7)
[1; 1.6[	59 (43.4)
>1.6	8 (5.9)
Triglycerides (g/l)	
<1	23 (16.9)
[1; 1.5[	49 (36.0)
>1.5	64 (47.1)
Atherogenic index <4.5	100 (73.5)

Table 3: Values of the Lipid Profile of the Study Population.

4.3. Association between Lipid profile abnormalities and BMI

The presence of dyslipidemia was assessed according to increasing BMI subcategories. We observed that HDL and TG levels were higher in overweight subjects (32(43.2%) and 26(41.3%) respectively), while those of LDL and TC were higher in grade I obese subjects. However, results from our study population show that there exists no significant difference in lipid abnormalities with increasing BMI class. This is shown in Table 4.

Variables	BMI (kg/m²) (n/%)				p-value
	25 – 30	30 – 35	35 – 40	>40	
Total cholesterol					
<1.4	9(40.9)	8(36.4)	3(13.6)	2(9.1)	0.7
[1.4; 2[	44(47.3)	29(31.2)	13(14.0)	7(7.5)	
>2	7(33.3)	11(52.4)	2(9.5)	1(4.8)	
HDL cholesterol					
<0.4	32(43.2)	26(35.1)	8(10.8)	8(10.8)	0.3
[0.4; 0.6[	22(41.5)	19(35.8)	10(18.9)	2(3.8)	
>0.6	6(66.7)	3(33.3)	0(0)	0(0)	
LDL cholesterol					
<1	28(40.6)	22(31.9)	11(15.9)	8(11.6)	
[1; 1.6[	30(50.8)	21(35.6)	7(11.9)	1(1.7)	0.15
>1.6	2(25.0)	5(62.5)	0(0)	1(12.5)	

Triglycerides					
<1	12(52.2)	8(34.8)	2(8.7)	1(4.3)	0.3
[1; 1.5[	22(44.0)	14(28.0)	7(14.0)	7(14)	
>1.5	26(41.3)	26(41.3)	9(14.3)	2(3.2)	

Table 4: Lipid profile as a function of the BMI of the study population.

5. Discussion

The objective of this study was to evaluate the relationship between obesity and lipid profile parameters in Cameroonian adults. The prevalence of dyslipidemia was 52.9%, the main subtypes being HDL, hypocholesterolemia, and hypertriglyceridemia. Diabetes was significantly associated with dyslipidemia (0.004). There was no relationship between obesity subtypes and different lipid profile parameters. Similarly, there was no significant difference between the atherogenicity indices of the different types of obesity. The dyslipidemia associated with obesity predicts the majority of the increased CV risk seen in obese subjects. We found that the most frequent lipid abnormality was HDL hypocholesterolemia, with a percentage of 54.41%. The mean HDL cholesterol concentration was 0.40 ± 0.11. Stephien et al. reported a similar result after a cross-sectional study of obese nondiabetic patients. They showed that obesity is associated with lipid disturbances, in particular a reduction in HDL-C levels [20]. High-density lipoprotein cholesterol (HDL-C) has a prominent place among the various lipid components; hence the name "good cholesterol", which has been attributed to it. The decrease in HDL-C concentration is an important risk factor for cardiovascular disease [21]. Several studies have reported the inverse relationship between HDL-C and obesity [22, 23]. Further studies are needed to evaluate the extent to which obesity contributes to the decrease in HDL-C levels.

In this study, no significant correlation was found between each of the lipid profile parameters and obesity based on BMI. Stepien et al. reported a different result. They found a negative correlation between: BMI and CT, LDL, and HDL (r=-0.291; r=-0.310, r=-0.240, respectively). The negative correlations observed in their study between BMI, CT, and LDL-C would result from lower LDL-C levels in morbidly obese patients, as reported by other authors [24, 25]. Many scientists who have conducted studies in the general population have reported increased BMI and decreased HDL-C and increased TG [26]. Howard et al. found that the relationship between BMI and serum LDL-C levels is much more complex and would depend on unmodifiable factors, particularly age or gender. The Strong Heart study of 773 women and 739 men of American Indian origin reported a similarly different result from our study, in which morbidly obese patients had lower LDL-C levels than other patients, which was statistically significant [24,25,27]. We found a significant association between diabetes and dyslipidemia (p=0.004), confirming the findings of other studies evaluating dyslipidemia as a common condition of diabetes [28-30]. A few elements have been questioned as contributing to the alterations in lipid metabolism frequently observed in diabetes: insulin deficiency or resistance, adipocytokines and

hyperglycemia [31]. Dyslipidemia in type 2 diabetes in particular is characterized by a decrease in high-density lipoprotein (HDL) cholesterol and an increase in triglyceride levels, all of which usually occur several years before the onset of diabetes [32, 33]. Recently published evidence indicates that low HDL cholesterol alone is a factor in the development of diabetes. It is therefore crucial to act by regulating the lipid profile of patients at risk. Taking action through lifestyle modification is paramount in the management of diabetes-related dyslipidemia and includes diet modification, weight loss, and exercise [34]. Notice that regarding physical activity, only 37(27.21%) of our subjects included practiced a minimum physical activity of 30 minutes for at least 5 times a week as recommended. After the age of 70, both free fat mass and fat mass decrease together [35]. The most represented age group in our population was 65-70 years old, with an average BMI of 32.09 ±5.11. In addition, 77% of the population had a lower atherogenic index, indicating a protective role of high BMI in the elderly. An observation that has already been reported in previous studies and meta analyses [36] The present study has some limitations. This was a cross-sectional study and therefore it is subject to the issues of unmeasured, residual confounding and reverse causation. Hence, this limits our ability to establish causality between obesity and the dyslipidemic indices we studied. Moreover, the possibly small sample size could have impacted the ability of this study to detect significant differences. However, the findings presented in this study provide preliminary data to guide lipid research in sub-saharan Africa.

6. Conclusion

Half of the obese Cameroonians adults of the sample suffer from dyslipidemia, but it is not directly related to their BMI. The prevalence of dyslipidemia was 52.94%; distributed in decreasing order of frequency in hypo HDL cholesterolemia, hypertriglyceridemia, total hypercholesterolemia and LDL hypercholesterolemia.

Conflict of Interest

The authors declare no conflict of interests.

Acknowledgements

We are grateful to the staff of the Yaoundé Central Hospital and Biyem-assi District Hospital for their help in the recruitment of study participants. Additionally, we thank the participants for their contribution to our study.

Authors' contribution

VJAM, NGM, BNN designed the study

JCB, DBE, BBA, AMJ, JRN collected data.

VJAM, DBE, BBA, AMJ, JRN analyzed data

VJAM, DBE, JCB, BBA, AMJ, JRN, NGM built the manuscript

All authors revised the manuscript

The study was done under the supervision of BNN.

All authors read and approved the final manuscript.

References

1. Obesity and Over view. Disponible sur: https://www.who.int/westernpacific/health-topics/obesity
2. Fruh SM. Obesity: Risk factors, complications, and strategies for sustainable long-term weight management. J Am Assoc Nurse Pract 29 (2017): 3-14.
3. Rubenstein AH. Obesity: a modern epidemic. Trans Am Clin Climatol Assoc 116 (2005):103-111.

4. Berrington de Gonzalez A, Hartge P, Cerhan JR, et al. Body-mass index and mortality among 1.46 million white adults. N Engl J Med 363 (2010): 2211-2219.
5. Popkin BM, Adair LS, Ng SW. NOW AND THEN: The Global Nutrition Transition: The Pandemic of Obesity in Developing Countries. Nutr Rev 70 (2012): 3-21.
6. Jung UJ, Choi M-S. Obesity and Its Metabolic Complications: The Role of Adipokines and the Relationship between Obesity, Inflammation, Insulin Resistance, Dyslipidemia and Nonalcoholic Fatty Liver Disease. Int J Mol Sci 15 (2014): 6184-6223.
7. Feingold KR. Obesity and Dyslipidemia. In: Feingold KR, Anawalt B, Boyce A, et al. éditeurs. Endotext. South Dartmouth (MA): MDText.com Inc (2000).
8. Hajar R. Risk Factors for Coronary Artery Disease: Historical Perspectives. Heart Views 18 (2017): 109-114.
9. Assmann G, Cullen P, Jossa F, et al. Coronary Heart Disease: Reducing the Risk. Arteriosclerosis, Thrombosis, and Vascular Biology. American Heart Association; 1 août 19 (1999): 1819-1824.
10. Hedayatnia M, Asadi Z, Zare-Feyzabadi R, et al. Dyslipidemia and cardiovascular disease risk among the MASHAD study population. Lipids in Health and Disease 19 (2020): 42.
11. Gaziano TA, Bitton A, Anand S, et al. Growing Epidemic of Coronary Heart Disease in Low- and Middle-Income Countries. Curr Probl Cardiol. Févr 35 (2010): 72-115.
12. Yusuf S, Hawken S, Ounpuu S, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. Lancet 364 (2004): 937-952.
13. Ama Moor VJ, Ndongo Amougou S, Ombotto S, et al. Dyslipidemia in Patients with a Cardiovascular Risk and Disease at the University Teaching Hospital of Yaoundé, Cameroon. International Journal of Vascular Medicine 2017 (2017): 6061306.
14. Xi Y, Niu L, Cao N, et al. Prevalence of dyslipidemia and associated risk factors among adults aged ≥35 years in northern China: a cross-sectional study. BMC Public Health 20 (2020): 1068.
15. Koseoglu M, Hur A, Atay A, et al. Effects of hemolysis interference on routine biochemistry parameters. Biochem Med. Croatian Society of Medical Biochemistry and Laboratory Medicine 21 (2011): 79-85.
16. Woolson RF, Bean JA, Rojas PB. Sample size for case-control studies using Cochran's statistic. Biometrics 42 (1986): 927-932.
17. Nuttall FQ. Body Mass Index. Nutr Today 50 (2015): 117-128.
18. Knopfholz J, Disserol CCD, Pierin AJ, et al. Validation of the Friedewald Formula in Patients with Metabolic Syndrome. Cholesterol 2014 (2014).
19. Soran H, Adam S, Mohammad JB, et al. Hypercholesterolaemia – practical information for non-specialists. Arch Med Sci 14 (2018): 1-21.
20. Stępień A, Stępień M, Wlazeł RN, et al. Assessment of the Relationship between Lipid Parameters and Obesity Indices in

Non-Diabetic Obese Patients: A Preliminary Report. Med Sci Monit 20 (2014): 2683-2688.

21. Petrenya N, Brustad M, Dobrodeeva L, et al. Obesity and obesity-associated cardiometabolic risk factors in indigenous Nenets women from the rural Nenets Autonomous Area and Russian women from Arkhangelsk city. Int J Circumpolar Health 73 (2014): 23859.
22. Lichtash CT, Cui J, Guo X, et al. Body adiposity index versus body mass index and other anthropometric traits as correlates of cardiometabolic risk factors. PLoS One 8 (2013): 65954.
23. National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. Circulation 106 (2002): 3143-3421.
24. Howard BV, Ruotolo G, Robbins DC. Obesity and dyslipidemia. Endocrinol Metab Clin North Am 32 (2003): 855-867.
25. Hu D, Hannah J, Gray RS, et al. Effects of obesity and body fat distribution on lipids and lipoproteins in nondiabetic American Indians: The Strong Heart Study. Obes Res 8 (2000): 411-421.
26. Unek IT, Bayraktar F, Solmaz D, et al. The levels of soluble CD40 ligand and C-reactive protein in normal weight, overweight and obese people. Clin Med Res 8 (2010): 89-95.
27. Gruson E, Montaye M, Kee F, et al. Anthropometric assessment of abdominal obesity and coronary heart disease risk in men: the PRIME study. Heart 96 (2010): 136-140.
28. Schofield JD, Liu Y, Rao-Balakrishna P, et al. Diabetes Dyslipidemia. Diabetes Ther 7 (2016): 203-219.
29. Chapman MJ, Ginsberg HN, Amarenco P, et al. Triglyceride-rich lipoproteins and high-density lipoprotein cholesterol in patients at high risk of cardiovascular disease: evidence and guidance for management. Eur Heart J 32 (2011): 1345-1361.
30. Sheth J, Shah A, Sheth F, et al. The association of dyslipidemia and obesity with glycated hemoglobin. Clin Diabetes Endocrinol (2015): 1.
31. Taskinen MR. Diabetic dyslipidaemia: from basic research to clinical practice. Diabetologia 46 (2003): 733-749.
32. Dean JD, Durrington PN. Treatment of dyslipoproteinaemia in diabetes mellitus. Diabet Med. Avr 13 (1996): 297-312.
33. Haffner SM, Stern MP, Hazuda HP, et al. Cardiovascular risk factors in confirmed prediabetic individuals. Does the clock for coronary heart disease start ticking before the onset of clinical diabetes? JAMA 263 (1990): 2893-2898.
34. Warraich HJ, Wong ND, Rana JS. Role for combination therapy in diabetic dyslipidemia. Curr Cardiol Rep 17 (2015): 32.
35. St-Onge MP, Gallagher D. Body composition changes with aging: The cause or the result of alterations in metabolic rate

and macronutrient oxidation? Nutrition 26 (2010): 152-155.

36. McKee A, Morley JE. Obesity in the Elderly. In: Feingold KR, Anawalt B, Boyce A, Chrousos G, de Herder WW, Dungan K, et al., éditeurs. Endotext [Internet]. South Dartmouth (MA): MDText.com, Inc (2000).