# Lipid Profile in Type 2 Moroccan Diabetic: Fasting Vs Non-fasting

Yousra Benabedlfedil<sup>1</sup>, Amal Moumen<sup>1</sup>, Mohamed Zraidi<sup>2</sup>, Sara Derrou<sup>1</sup>, Faycal El Guendouz<sup>1</sup>

<sup>1</sup>Endocrinology and Diabetology Department, Moulay Ismail Military Hospital of Meknes, Faculty of Medicine and Pharmacy, Biomedicine and Environment Laboratory, Sidi Mohamed Ben Abdellah University, Fes, Morocco. <sup>2</sup>Biology and Health Laboratory; Faculty of Sciences, Ibn Tofail University, Kenitra, Morocco. Address correspondence to: Faycal El Guendouz, Email: el.guendouz@gmail.com

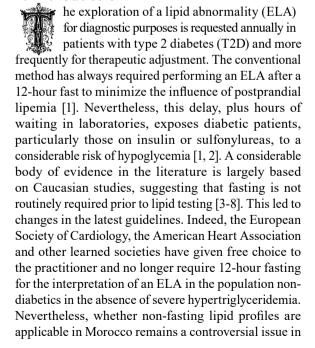
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## ■ Abstract

**Objective:** In the absence of a study of the applicability of postprandial lipid profiles in the Moroccan and Maghrebin population, we report a study comparing fasting and non-fasting lipid profile in a population of type 2 Moroccan diabetic. Our objective was to verify the applicability of postprandial lipid profiles in this population and secondarily determine the association of the non-fasting of lipid parameters with established cardiovascular disease. Patients and methods: In a prospective interventional before-and-after study, including type 2 Moroccan diabetic patients, aged over 18 years, received in endocrinology department of a tertiary care teaching hospital. Blood samples taken after a 12-hour fast were compared to those taken 2 to 3 hours after a standardized breakfast (postprandial test). The intraclass correlation coefficient (ICC) was used to compare the intraindividual lipid profiles. **Results:** 180 patients with Type 2 Diabetes Mellitus, were included in the study. The average age of patients was  $59.42\pm8.72$  years with a male predominance (56.1%). The mean differences between non-fasting and fasting total cholesterol, high-density lipoprotein, triglycerides, low-density lipoprotein, and non-HDL-C were -0.04 g/L, -0.0 g/L, +0.33 g/L, -0.05 g/L, and -0.03 g/L, respectively. A good ICC correlation >75% was approved for all lipid fractions. There is no association between non-fasting lipid profile and established cardiovascular disease. **Conclusion:** Non-fasting lipid profiles were applicable in this population. They are more comfortable and convenient for our diabetic patients, allowing to reduce waiting times and avoid fasting-related hypoglycemia.

**Keywords:** Fasting, Non-Fasting Lipid Profiles, Postprandial Lipid Profiles, Type 2 Moroccan Diabetic.

## 1. Introduction



the absence of a valid local study and in the presence of a different epidemiological situation from the West of type 2 diabetes (new global epicenter of increasing diabetes prevalence) and the very high level of cardiovascular risk. In addition, diabetic patients often have elevated triglycerides, the lipid fraction most affected by dietary intake, which could reduce the applicability of postprandial testing [1, 3, 4, 6]. The practice of an EAL after a balanced Moroccan breakfast will allow us to optimize turnaround time in laboratory and to reduce risk of hypoglycaemia. Moreover, some studies have suggested that non-fasting lipid levels (especially triglycerides) may predict cardiovascular risk better fasting lipid levels [9-11]. Here we report on a study comparing the fasting and non-fasting lipid profile in a population of type 2 Moroccan diabetic patients from a community setting. The main objective was to compare the lipid fractions (total cholesterol (TC), high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C), triglycerides (TG) and nonlipoprotein cholesterol of high density (Non-HDL-C)) made before and after a Moroccan breakfast (fasting vs non-fasting), to explore the applicability of the non-fasting lipid levels in this population and to determine whether non-fasting status of the lipid parameters may be associated with established cardiovascular disease (CVD).

## 2. Materials and Methods

#### 2.1. Materials

This was a prospective interventional before-and-after study, conducted between June 2021 to January 2022. We enrolled adult outpatients (≥ 18 years old) with T2D from the endocrinology and diabetology consultation of the military hospital of Meknes (the only tertiary care teaching hospital in the city of Meknes). The research protocol was approved by the local ethics committee and complies with the ethical guidelines of the 1975 Declaration of Helsinki. All eligible patients agreed to participate in the study. The study received no funding. Patients were excluded if they had other types of diabetes, a known kidney failure, liver failure or chronic digestive disease, if they were pregnant or non-resident at the city of MEKNES or patients refusing to participate to the study.

## 2.2. Methods

Data collection was carried out using a preestablished exploitation sheet, administered face-toface, comprising the following elements:

Demographic data and CVR factor analysis: Anthropometric data and cardiovascular risk factors were compiled by patient response and clinical exam during a single routine health visit and from participants' medical records.

Hypertension was diagnosed if subjects were on drug treatment for hypertension or had a systolic blood pressure (SBP) of ≥ 140 mmHg and/or diastolic blood pressure of ≥90 mmHg. Obesity was diagnosed if patient had a body mass index (BMI)  $\geq$  30 kg/m<sup>2</sup>. Smoking was defined by active smoking or smoking cessation less than 3 years. Microalbuminuria was defined by an albuminuria/ creatininuria ratio (ACR) of 30-300 mg/g for a first morning void or in a spot urine sample. Macroalbuminuria was defined by an ACR greater than 300 mg/g. The estimated glomerular filtration rate (GFR) was calculated using the CKD-EPI (Chronic Kidney Disease - Epidemiology Collaboration) equation and chronic kidney failure was defined by GFR < 60 ml/min/1.73m<sup>2</sup>. Diabetic kidney disease (DKD) was defined by chronic kidney failure or and albuminuria confirmed twice. Assessment of cardiovascular risk was based on European Society of Cardiology (ESC) Guidelines on cardiovascular disease prevention in clinical practice of 2021 [12].

Established CVD was defined as a diagnosis of any of the following conditions in participants' medical records: cerebrovascular disease, coronary artery disease (CAD), heart failure, peripheral artery disease (PAD), or carotid artery disease. For analysis purposes, participants were stratified into two groups based on the presence (CVD group) or absence (non-CVD group) of established CVD.

Abbreviation	is:
ACR	Albuminuria/creatininuria ratio
BMI	Body mass index
CVD	Cardiovascular disease
LDL-C	Low density lipoprotein cholesterol
CI	Confidence interval
CKD-EPI	Chronic Kidney Disease -
	Epidemiology Collaboration
CAD	Coronary artery disease
DKD	Diabetic kidney disease
ESC	European Society of Cardiology
GFR	Glomerular filtration rate
HbAlc	Glycated haemoglobin
HDL-C	High density lipoprotein cholesterol
ICC	Intraclass correlation
Non-HDL-C	Non-lipoprotein cholesterol
	of high density
ELA	Exploration of a lipid abnormality
TC	Total cholesterol
T2D	Type 2 diabetes
TG	Triglycerides
SBP	Systolic blood pressure

Lipid levels determination: Each patient performed two ELA, one after 12 hours of fasting and a second postprandial. The postprandial tests were performed the same day 2 to 3 hours after a balanced meal recommended to the patient on a pre-established sheet: 80g of wholemeal bread, 2 to 3 large spoons of olive oil, or 30 to 40 g of industrial cheese, unsweetened tea or coffee in addition to taking the morning antidiabetic medication. Total cholesterol, HDL cholesterol, and triglycerides were determined by an enzymatic method on a fully automated biochemical analyzer run by a specialist who was unaware of the study. The LDL cholesterol level was calculated by Friedwald's formula if the triglyceride level is less than 4 g/L or measured by a direct assay if the triglyceride level is greater than 4 g/l. Non-HDL cholesterol was calculated by the difference between total cholesterol and HDL cholesterol. Dyslipidemia was defined by Total cholesterol > 2 g/l, LDL cholesterol > 1,6 g/l and/ or HDL cholesterol < 0.4 g/l for men and < 0.5g/l for women and /or triglycerides > 1.5 g/l or the use of lipid lowering drugs.

Statistical analysis: Data were analyzed by SPSS software. Quantitative variables were expressed as mean ± standard deviation (age, total cholesterol, LDL cholesterol, non-HDL cholesterol) or median and interquartile range (diabetes duration, HDL cholesterol, triglycerides, HbA1c, BMI). Qualitative variables were expressed as numbers and percentages. The comparison between the fasting and non-fasting lipid profile was made using the Student's t test for paired samples for the means, the Wilcoxon test for the medians and the McNemar test for the proportions. The comparison of patients with or without CVD was done by Student's t test for means, by non-parametric tests (Mann Whitney test) for the medians and by the

Chi-square test and Fisher's exact test for proportions. The statistical significance level was set at 5%. For comparison of the agreement of two continuous measures, fasting and non-fasting lipids from the same individual, we used the intraclass correlation (ICC). We estimated the ICC to be  $\pm 0.025$ , 95% CI width=0.05. ICC values  $\geq 0.71$  indicate a satisfactory correlation,  $\geq 0.81$  a good correlation, and  $\geq$  0.91 a very good correlation.

### 3. Results

180 T2D patients were enrolled in the study. The mean age of the patients was  $59.42 \pm 8.72$  years with male predominance (56.1%) and the median duration of diabetes was 10 (7;16) years. The anthropometric characteristics, the different CVR factors, the degenerative profile and the level of cardiovascular risk of the patients are summarized in Table 1.

**Table 1:** Baseline Characteristics of Patients (n=180).

Characteristics  ge (years)* ender\$ en omen uration of diabetes° tabetes treatments \$ sulin etformin tlifonamides DP4 inhibitors	(n=180) 59,42 ± 8,72 101 (56.1%) 79 (43.9%) 10 (7;16) 119 (66.1%) 126 (70%) 39 (21.7%)
en omen uration of diabetes° iabetes treatments§ sulin etformin ulfonamides	79 (43.9%) 10 (7; 16) 119 (66.1%) 126 (70%)
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etformin ilfonamides	126 (70%)
ulfonamides	
	39 (21.7%)
DP4 inhibitors	
	9 (5%)
LP-1 agonists	7 (3.9%)
GLT2 inhibitors	3(1,7%)
bA1C°	8.15 (7.28; 9.68)
ycemic control	
7%	31 (17.2%)
etween 7 and 8%	49 (27.2%)
etween 8 and 10%	63 (35%)
10%	37 (20.6%)
MI °	26.7 (24.2; 29.3)
eight	
ormal	58 (32.2%)
verweight	85 (47.2%)
osity	37 (20.6%)
noking <sup>§</sup>	14 (7.8%)
TA§	81 (45%)
ntihypertensive treatment	
CE I	54 (30%)
RBs	25 (13.9%)
CB	10 (5.6%)
uretic	37 (20.6%)
B	12 (6.7%)
pirinolacton	3 (1.7%)
yslipidemia§	145 (80.6%)
pid-lowering therapy §	101 (70 10/)
one	101 (56.1%)
ow-intensity statin	34 (18.9%)
igh-intensity statin oderate intensity statin + fibrates	45 (25%)
oderate intensity statin + norates ntiagrégant treatment	2 (1.1%) 20 (11.2%)
ascular disease hereditary §	18 (10%)
ardiovascular Risk Levels §	18 (1070)
oderate	3 (1,7%)
oderate igh	59 (32.8%)
ry high	118 (65.6%)
ardiovascular disease §	24 (13.3%)
abetic kidney disease §	35 (19.4%)
buminuria §	29 (16.1%)
nronic renal failure §	20 (10.170)
oderate	10 (5.6%)
evere	3 (1.7%)
pressed as mean ± standard deviation;	9 (1.170)

Comparing the lipid profile performed in the fasting and postprandial state, the mean difference between TC, HDL-C, TG, LDL-C and Non-HDL-C in the fasting and postprandial state was -0.04 g/l, -0.0 g/l, +0.33g/l, -0.05 g/l and -0.03 g/l respectively. There was no significant difference in total cholesterol level (1.61  $\pm 0.39$  g/l vs  $1.64 \pm 0.35$  g/l; p = 0.127), Non-HDL-C level  $(1.16 \pm 0.37 \text{ g/l vs } 1.19 \pm 0.33 \text{ g/l}; p = 0.085)$  and HDL-C level (0.42 g/l (0.36; 0.51) vs 0.42 g/l (0.35; 0.52); p = 0.505). In addition, after food intake, LDL-C decreased significantly (0.84  $\pm$  0.29 g/l vs 0.89  $\pm$  0.32 g/l; p < 0.001) while TG levels increased significantly (1.31 g/l (0.96; 1.76) vs 1.64 g/l (1.2; 2.18); p < 0.001),Table 2 and Figure 1 summarize the average difference between the different lipid parameters collected in the fasting and non-fasting state.

<sup>§</sup>Expressed in numbers (percentages);

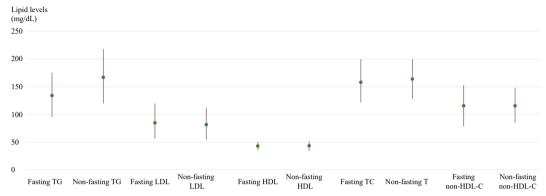
<sup>&</sup>lt;sup>e</sup>Expressed in median (interquartile)

**Table 2:** Comparison of the Lipid Profile Performed Fasting and Not Fasting in a Moroccan Population with Type 2 Diabetes.

	Fasting	Not fasting	P
Total Cholesterol (g/l) *	$1.61 \pm 0.39$	$1.64 \pm 0.35$	0.12
LDL Cholesterol (g/l) *	$\boldsymbol{0.89 \pm 0.32}$	$\textbf{0.84} \pm \textbf{0.29}$	< 0.001
HDL Cholesterol (g/l) °	0.42 (0.36; 0.51)	0.42 (0.35; 0.52)	0,5
Non-HDL Cholesterol (g/l) *	$1.16 \pm 0.37$	$1.19 \pm 0.33$	0,085
Triglycerides (g/l)°	1,31 (0,96; 1,76)	1,64 (1.2; 2.18)	< 0.001

<sup>\*</sup>Expressed as mean ± standard deviation;

<sup>&</sup>lt;sup>e</sup> Expressed as median (interquartiles)



**Figure 1:** Comparison Between an EAL Performed Fasting and Non-fasting in the Same Patients in a Moroccan Population with Type 2 Diabetes.

The intraclass correlation (ICC) measure (95% CI) of non-fasting and fasting TC, HDL-C, TG, LDL-C, and Non-HDL-C was respectively 0.893 (0.857–0.920), 0.913

 $\begin{array}{l} (0.884-0.935),\, 0.803\,\, (0.444-0.905),\, 0.909\,\, (0.867-0.936),\\ \text{and }0.892\,\, (0.856-0.920)\,\, (\text{see Table 3}). \end{array}$ 

**Table 3:** Intraclass Correlation Agreement (ICC) of Fasting and Non-fasting Lipids. An ICC Value  $\geq 0.75$  Indicates a Good Correlation.

Parameters studied ICC (95% CI)	ICC (95% CI)	Interpretation of the correlation
Fasting and non-fasting CT	$0,893 \ (0,857 - 0,920)$	Good
Fasting and non-fasting HDL C	0.913 (0.884 - 0.935)	Very good
Fasting and non-fasting TG	$0,803 \ (0,444 - 0,905)$	Good
Fasting and non-fasting LDL CT	$0,909 \ (0,867 - 0,936)$	Good
Fasting and non-fasting Non-HDL	0,892 (0,856 - 0,920)	Good

On average, TG level increased by 0.33 g/l two to three hours after a balanced breakfast (Table 2 and figure 1). There was also a strong, statistically significant

positive correlation between fasting and non-fasting TG levels (r= 0.816; p < 0.001) (Figure 2).

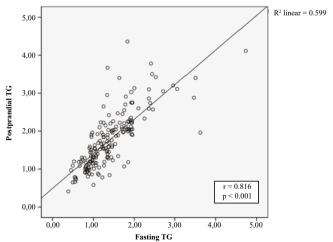


Figure 2: Correlation Study Between the Fasting TG Level and the Postprandial TG Level Carried Out in the Same Patients.

The number of participants controlled according to the LDL targets of step 2 of the 2021 ESC recommendations was 40 (22.2%) using fasting LDL versus 35 (19.4%)

using non-fasting LDL and the difference was not significant (Table 4).

**Table 4:** Comparison Between the Use of Fasting LDL Versus Non-fasting LDL to Define the Number of Patients Controlled on Statins According to the Level of VCR.

	Fasting LDL	Non-fasting LDL	P
Number of patients with controlled LDL according to RCV	40 (22.2%)	35 (19.4%)	0.227

The search for an association between a non-fasting lipid parameter and the presence of established cardiovascular disease was negative, particularly for

non-fasting TG compared to fasting TG: 1.79 (1.26; 2.08) vs 1.61 (1.19; 2.2); P = 0.928 (Table 5).

**Table 5:** Association Between the Various Fasting and Non-fasting Lipid Parameters and the Presence of Established Cardiovascular Disease (CVD).

	Without CVD (n=156)	With CVD (n=24)	P
Fasting CT (g/l) *	$1.62 \pm 0.39$	$1.53 \pm 0.37$	0.305
Fasting LDL (g/l) *	$0.91 \pm 0.32$	$0.81 \pm 0.3$	0.162
Fasting Non-HDL (g/l) *	$1.18 \pm 0.38$	$1.08 \pm 0.38$	0.265
Fasting HDL (g/l) °	0.42 (0.35; 0.52)	0.43 (0.36; 0.51)	0.833
Fasting TG (g/l) °	1.28 (0.95; 1.78)	1.34 (1.05; 1.71)	0.755
Non-fasting CT (g/l) *	$1.65 \pm 0.34$	$1.57 \pm 0.4$	0.344
Non-fasting LDL (g/l) *	$0.85 \pm 0.28$	$0.76 \pm 0.3$	0.183
Non-fasting Non-HDL (g/l) *	$1.21 \pm 0.33$	$1.12 \pm 0.34$	0.224
Non-fasting HDL (g/l) °	0.42 (0.35; 0.52)	0.45 (0.4; 0.51)	0.634
Non-fasting TG (g/l) °	1.61 (1.19; 2.2)	1.79 (1.26; 2.08)	0.928

<sup>\*</sup> expressed as mean ± standard deviation;

## 4. Discussion

Our study revealed that, in a Moroccan population with T2D, the lipid profile changed slightly after a test meal and the difference considered significant for TG and LDL-C was very small, ranging from + 0.33 g/l for TG to -0.05 g/l for LDL-C. In our data we found that outsid minor increases in plasma TG and minor decreases in LDL-C comparable results are obtained in measuring total cholesterol, HDL-C and non-HDL-C whether the patient is fasting or not. These minor and transient changes in lipid concentrations appear to be clinically insignificant.

Our results were remarkably similar to those reported in the literature. In a large series from Copenhagen (n=108245) on a Danish population, the difference was significant for TC, LDL-C and TG, with an even greater mean difference in LDL-C relatively (respectively -0.20 mmol/1 vs - 0.13 mmol/1), despite the greatest increase in TG (+ 0.30 mmol/1 vs + 0.37 mmol/1 respectively) [3, 6]. The same findings were observed in the US Women's Health Study (n=26,330), the US National Health and Nutrition Survey (n=12,744), and from the Calgary Laboratory Services series in Canada (n=209,180) [10, 13, 14].

The calculation of the ICC showed a good correlation between the values of the different non-fasting and fasting lipid fractions: TC, TG, LDL-C, Non-HDL-C, whereas HDL-C had a very good correlation. In an Asian series of 470 cases, the correlation was good for TC, HDL-C and non-HDL-C while it was satisfactory for LDL-C (0.71-0.80) and poor for TG (0.51-0.60) [15].

It is important to note that the change in TG will depend on baseline triglyceride levels, the presence of diabetes, fat intake, and time since last meal [4, 5, 7]. Since all the patients in our study were diabetic (compared to the Danish study), we expected a higher increase in TG, with an almost similar mean time from last meal to sampling in the Copenhagen series (2.46 h) [3]. The main factor that remains to explain the difference is the amount of fat present in the meal. Thus, the test meal used had a very high fat content [3] and we believe that the typical breakfast proposed to the participants in our study was much lower in fat, which is why we did not reproduce the difference in expected TG increase.

The maximum mean changes in the literature were observed in a multi-ethnic Asian population with T2D and dyslipidemia on a stable statin dose, they concerned TC, LDL-C and TG (+ 0.04 mmol/l, - 0.15 mmol/l and + 0.48 mmol/l respectively) [13]. In this Singaporean study we noticed the same trend of TC, LDL-C and TG but with higher differences that could be explained by ethnic and nutritional factors [10, 13].

An unchanging observation in all the studies performed is that HDL-C as well as non-HDL-C are not affected by a non-fasting blood sample [3, 5-8, 16].

Regarding the non-fasting TG level, we noticed that there is a statistically significant positive correlation

<sup>§</sup> expressed in numbers (percentages);

<sup>°</sup>expressed in median (interquartiles)

with the fasting TG level (r=0.816; p<0.001) (Figure 2). This means that as fasting TG increased, the range of TG concentration variation increased significantly and became quite wide above 4.5 mmol (4 g/L). These data agree with the literature and suggest avoiding the use of a non-fasting lipid profile in patients with TG levels above 4.5 mmol (4 g/L) [1, 5, 6, 16].

To explain the reduction of LDL-C in non-fasting samples, most authors have put forward a single hypothesis: dilution from hydration [17, 18]. This hypothesis was not verified in our series by evaluating the hydration status of patients with serum albumin or hematocrit and we did not restrict water intake before fasting sampling. In the Danish study, the decrease in LDL cholesterol observed in the participants became insignificant after adjusting for plasma albumin (marker of hemodilution) [3, 6].

Looking at the growing body of evidence [3-9], the American College of Cardiology, the American Cardiovascular Association, the European Society of Cardiology, and the Canadian Cardiovascular Society as well and other learned societies have given practitioners a free choice and no longer require a 12-hour fast for the interpretation of an EAL in the absence of severe hypertriglyceridemia [12, 14, 16, 19, 20].

Another easy way to compare the two means of fasting and non-fasting monitoring is to define the number of patients controlled by lipid-lowering treatment. In our study, the number of participants controlled according to the step 2 LDL targets of the ESC 2021 guidelines was higher using fasting LDL but the difference was not significant (table 4). This finding has not been studied in the other series, and we believe that the reduction in patients controlled on statins is an argument in favor of the use of non-fasting sampling since it will lead to intensification of therapy in this population of diabetics mainly at high risk or at very high CV risk (98%).

Theoretically, the non-fasting period could better reflect the current atherogenic burden than the fasting period [21, 22], which is why studies have focused on

investigating the contribution of non-fasting sample in the prediction of CVR [9, 10, 23, 24]. The study by Tada et al revealed an increase in the positive predictive value of the TG level when it is performed in the non-fasting state [11]. In this sense, we fail to find statistical significance between non-fasting lipid levels and established CVD (Table 5).

## 4.1. Strengths and Limitations of the Study

One of the strengths of our study is that the assessment of fasting and non-fasting lipid profiles was performed in the same individual, on the same day, prospectively with a standardized meal.

This reduced potential inter-individual and inter-day variations. In addition, we focused on diabetics who are particularly exposed to hypoglycemia and at very high cardiovascular risk [3, 25].

Our study was limited by the lack of measurements of apolipoprotein B and apolipoprotein A1 as well as lipoprotein (a) in participants, which are not available in our laboratories. Nevertheless, these assays are only occasionally requested in routine practice. The small sample size of the study, the mono-centric nature and the lack of verification of compliance with the proposed meal are all limiting factors and we believe, that this does not significantly affect the conclusion of the study. however, a larger multi-center study might help to get better results.

#### 4.2. Conclusion

It seems therefore important that we consider what is most practical for our diabetic patients. The results demonstrated a good ICC between a non-fasting and a fasting lipid profile in a Moroccan adult with T2D. In the light of the results provided by our study and looking at the risk of hypoglycemia in diabetic patients, particularly those on insulin or hypoglycemic sulfonamides, the added stress, poor compliance with medication taken the morning of the sample, the congestion observed in the medical analysis laboratories due to the increasing demands, we suggest the use of non-fasting lipid profiles in the absence of elevated TG level.

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