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Literature Review on the Particularities of Dysmetabolism and T2DM in Libreville: Perspectives on Postprandial Glucido-Lipid Exposure

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Abstract

Dysmetabolism and T2DM are common in Libreville. Based on a literature review of study data, the aim was to identify the various salient findings and analyze these dysmetabolisms from the point of view of clinico-metabolic particularities, and to propose reinforcing their significance by postprandial dynamic explorations. In Gabon, all dyslipidemias are present in the population (moderate to severe hypercholesterolemia, high or low high-density lipoprotein cholesterol (HDL-C) and low-density lipoprotein cholesterol (LDL-C)). In connection with the studies by Ngou (1997) [1] on reference values for lipids in the population. Polymorphism of the low-density lipoprotein (LDL)-oxidizing enzyme paraoxonase-1 (PON-1) is associated with lower protective HDL in diabetics. Qualitative abnormalities in LDL with atherogenic potential, expressed as conjugated diene onset latency or PON-1 activity, were associated with chronic complications (hypertension, retinopathy, etc.). In Gabon, the interaction between metabolism and *Plasmodium falciparum* malaria leads to particularities of expression, given the endemic nature of the disease. These include the classic hypertriglyceridemia associated with malaria, high levels of free fatty acids (FAs) and hypoglycemia dissociated from high lactate levels. In Gabon, postprandial glycemia is used to diagnose pre-diabetic states. It may then be accompanied by a postprandial hyperinsulinism syndrome prefiguring insulin resistance. We had the opportunity to compare the results of T2DM patients in Libreville with those of Caucasians in Marseille, through the work we co-directed. Modalities of insulin resistance and morphotype are not always the same in the two races. Finally, an inadequacy in the monitoring of T2DM treatment by HbA1c (glycated haemoglobin) was highlighted in Li-

breville. In view of these numerous particularities of T2DM in African subjects, it has become necessary to conduct technical studies on the methodology and metabolic aspects of the postprandial space. Research into improving diagnosis and therapeutic strategy must be built today around specific knowledge of postprandial dyslipidemia and the dietary context.

Keywords

Dysmetabolism, T2DM Libreville Particularities, Dietary Dyslipidemia and Therapeutic Strategy

1. Introduction

The epidemiological transition, with the advent of metabolic pathologies (with a non-infectious profile), took place in Africa several decades ago.

It has been established that certain diseases, such as obesity, hypertension and T2D, associated with carbohydrate and lipid dysmetabolism, and secondarily protein dysmetabolism, are the source of increased cardiovascular risk. What's more, these various carbohydrate-lipid biochemical disorders (total cholesterol, LDL-C, HDL-C, lipoproteins, triglycerides, postprandial triglyceridemia, insulin resistance) will combine to form syndrome X or metabolic syndrome, which, together with abdominal obesity, will increase the risk of T2D and heart disease [Gamilia, IDF[1] , cited by Bongard, 2011] [2].

The lipid parameters of postprandial metabolism also require special attention and questioning. This is characterized by the fact that today's human being, with 3 to 5 food rations in a day, spends ¾ of his or her time in the postprandial state, which corresponds to a veritable traffic jam of exogenous and endogenous lipoproteins rich in chylomicrons, and therefore triglycerides (LRT), a source of strong metabolic provocations. The risk of chronic exposure to these metabolic disorders, linked to the duration of exposure over the day, is no less. Indeed, with 3 to 5 food intakes per day, the regularity of food intake periods ensures only incomplete clearance [3] [4].

According to the WHO, T2DM is defined as:

1) Fasting blood glucose > 1.26 g/L (normal 0.7 - 1 g/L) found twice in the blood;

2) A disease characterized by insulin resistance as a consequence of excess weight, leading to elevated blood sugar levels.

It's a formidable condition, with numerous complications (hypertension, retinopathy, nephropathy, macro-angiopathy).

Today, it is of the utmost importance to study the fluctuations and modulations of the postprandial phase, and to characterize the relationship between diet and health. The Monica study [2] found a cumulative incidence of mortality of 16.4%

[1]IDF: International Diabetes Federation.

in metabolic syndrome or X, associated with a dietary factor characterized by a low proportion of carbohydrates, polysaccharides, polyunsaturated fatty acids, milk, dairy products, fish, fruit and vegetables.

New insights are emerging into the pathophysiology of dyslipidemia in T2DM [5].

The aim is to find precise diagnoses in terms of dynamic information power (fluctuations and modulations) on biological functions and the functioning of metabolic organs in relation to different clinical-biochemical indications (healthy subjects, metabolic syndrome, risk situations for cardio-metabolic disease). This involves the methodological calibration of the postprandial dyslipidemia test (dietary tracers). The second phase of research concerns therapeutic strategy, leading to:

- Dietary advice and dietary therapy (foods characterized by a normal postprandial lipid purification profile;
- Production and promotion of therapeutic industrial foods for people with lipid purification anomalies.

Finally, as this is a literature review, the ethical provisions are notably linked to the rigorous choice of references for validated works.

2. Lipido-Glucid-Protein Metabolism and T2DM: A Brief Overview

These are primarily biochemical, physiological and metabolic organ events.

In fact, carbohydrate-lipid metabolism connections are permanent in the body and present in all physiological situations (fed state, fasted state).

The main interactions that highlight this carbohydrate-lipid metabolic symbiosis through the existence of a community of organs, seats of this metabolism (liver, adipose tissue, muscle tissue, pancreas, brain, erythrocytes) are shown in Figure 1.

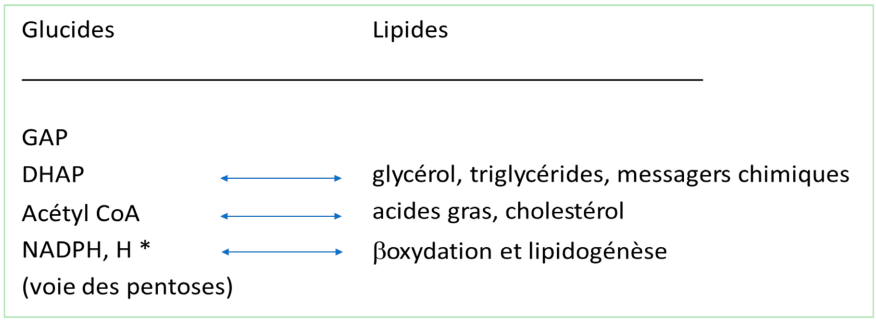

Figure 1. Carbohydrate-lipid metabolic pathways (Source: Hennen, 1998) [6].

In addition, carbohydrate-lipid pathways and the existence of amino acids with dual metabolic competence (ketoformers and glucoformers) complete these circuits by involving protein metabolism.

Similarly, following on from the description of the previous situations, the feed-

ing sequence in the nycthemer can be written as shown in Figure 2, with no less than five postprandial episodes, each of which cumulates the physiological mechanisms or disturbances during these postprandial phases with a chronic exposure profile in the nycthemer (Figure 2, Figure 3).

Figure 2. Dietary intake and episodes of postprandial lipemia (PPL).

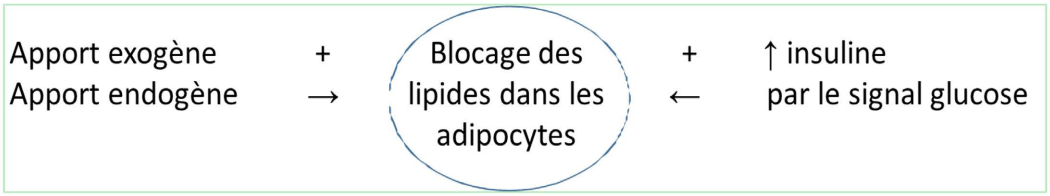

Figure 3. Mechanism of postprandial adipocyte lipid concentration.

Faced with this diversity of exogenous and endogenous metabolic loads, it is easy to understand the need for a controlled balance within the body. Hence the need for coordinated regulation (Table 1), from which the sequence of insulin actions and its role in carbohydrate-lipid metabolic organs can be deduced.

1) Inhibition of glucose output from the liver, thus of availability in the bloodstream.

2) Activation of glucose entry in liver, muscle and adipocytes.

3) Modulation of the activity of proteins already present (enzymes, transporters). Changes in the expression of specific genes.

Table 1. Regulation of carbohydrate-lipid energy metabolism.

Blood glucose	Hormones	Lipidemia
↓ **Blood glucose** ↑ **Glycytia**	← Insulin →	1) lipogenesis (↓ lipidemia and ↑ lipidocytia) 2) ↑ b oxidation TA → ↑ AG/glycerol for liver survival
Blood glucose	Glucagon ← Adrenalin → Cortisol TSH, FT4	1) ↑ Lipolysis 2) lipogenesis TA because DHAP, GAP, Ag are available
↓ **decrease,** ↑ **increase**		

With regard to this regulation, recent work has highlighted the novel role of insulin and cellular mechanisms in the liver, carried by the Sterol Regulatory Element Bindin Protein (SREBP). This protein controls the expression of genes involved in fatty acid (FA), triglyceride (TG) and cholesterol metabolism (Table 2)

[7].

Finally, it goes without saying that when these regulatory processes are disrupted, the risk of diabetes appears, including that of T2D, the focus of this work.

Table 2. Characteristics of the three SREBP isoforms (Source: Foufelle, 2005) [7].

Proteins	Gene	Location	Regulated genes
SREBP-1a	SREBP-1	Spleen, intestine, proliferating cells, cell lines	Enzymes of cholesterol and fatty acid biosynthesis
SREBP-1c	SREBP-1	Highly expressed in liver, adipose tissue, muscle	Enzymes of fatty acid biosynthesis
SREBP-2	SREBP-2	Weak expression in all cells	Cholesterol biosynthesis and capture enzymes

With regard to the exploration of dysmetabolism, we indicate the benchmarks for sampling. These benchmarks consist of a twelve (12)-hour fasting period reference zone. It is always worth pointing out that, within this zone, we encounter the dawn phenomenon [4] which is the difference between the minimum nocturnal blood glucose level and the pre-breakfast blood glucose level. Value greater than 10 mg/dl. It is seen in subjects of all ages, in 60% of T2DM. The debate today is whether the dawn phenomenon, in prolonged mode, has an impact on the glycemic balance of the T2DM patient, on glucose exposure and therefore on HbA1c levels [4] (**Figure 4**).

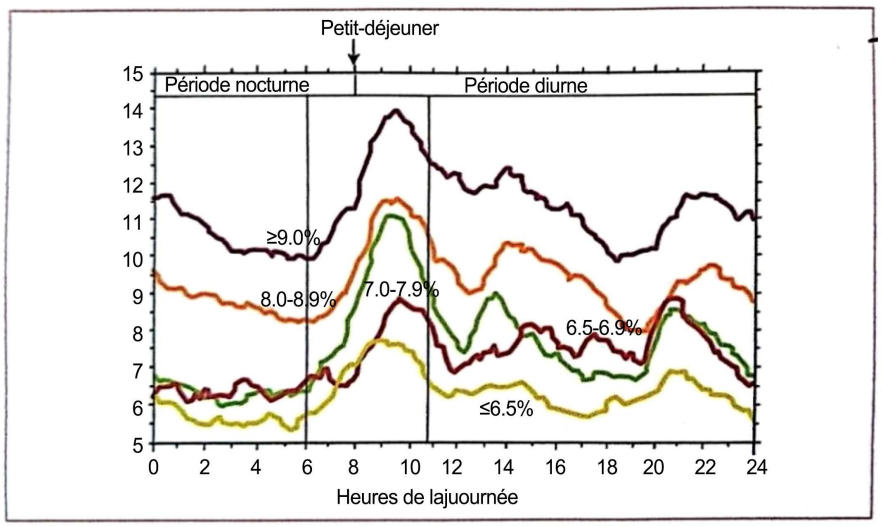

Figure 4. Dawn phenomenon (Source: Monnier, 2012) [4].

3. Literature Data from Studies Carried out in Libreville

3.1. Literature Review on T2DM

To build a new pathway on dysmetabolic studies and T2DM, we thought it would be useful to review the initial results we obtained in Libreville, and to point out the few opportunities we had to compare some with those of the Caucasians in Marseille.

The results of this review are those of a study we had planned on glucido-lipid dyslipidemia and T2DM in the Congo Basin. We began the study in Gabon, where we noted the high prevalence of T2D (10%) and the fact that our country was the 3rdmost affected sub-Saharan African country (with the highest rate of diabetics). In Libreville, the relationship between lipids and T2D is a matter of concern.

3.1.1. Epidemiological Data

These results cover various aspects:

– Aspects of the epidemiological transition and nutritional assumptions of the diet (Table 3 and Table 4).

It appears that our dietary traditions already included foods with "glucose potential" and assimilated (taro, yams...). Combined with the high glucose-lipid potential of the Western diet, this explains the sharp rise in the prevalence of T2D in Gabon (7% in 10 years) [8].

Table 3. Specific aspects of T2DM: nutritional hypothesis and diet.

Staple foods in the local diet	Epidemiological transition	Westernization of food
- **Tarots** - **Yams** - **Cassava (cyanogenetic glycosides)** - **Vegetable oil (Irvinga Gabonesis)** - **Palm oil**	→	- Butter - Animal oils - Delicatessen - Fast-absorbing sugar, etc.

Table 4. Evolution/projection of T2DM prevalence in Gabon (Source: Ntyonga Pono, 1996) [8].

1990	1994	2000	2003	2007	2010
0.3%	0.7%	<2%	2% - 5%	4% - 6%	5% - 7%
		7% in 10 years			

– Epidemiology of dyslipidemia in Gabon

In Gabon, all dyslipidemias are present in the population (moderate to severe hypercholesterolemia, high and low density lipoprotein cholesterol HDL-C and LDL-C high or low). In relation to the studies by Ngou, 1997 [1] on reference values for lipids in the population (Table 5).

Table 5. Epidemiology of dyslipidemia in Gabon (Source: Ngou, 1998) [9].

• General population: 31.88%/2074 subjects	
Moderate HCT	50%
Severe HCT	11.87%
• DT2 +	
Dyslipidemia HT	33.3%
Moderate HTC	2.67%

3.1.2. Pathophysiological and Clinical Data

– Pathophysiological features (Tables 6-8)

We have identified a series of clinico-metabolic events based on the work of Buresi, 1990 [10], Ngou and Perret/D These PEMBA, LBV, 1998: n˚ 309 [11].

An inadequate initial secretory profile in the black T2DM patient versus initial insulin resistance (hyperinsulinemia) with terminal insulinopenia in the Caucasian T2DM patient from Marseille, due to depletion of β (Beta) cells in the Langherans islets.

Diabetics from Libreville, often of normal weight, have rather low insulin levels compared with age-matched Caucasian T2DM from Marseille (56.64 < 75.61 pmol/L).

In Gabon, postprandial hyperinsulinism syndrome on the 2-hour postprandial glucose test is diagnostic of prediabetic states and precedes the onset of insulin resistance, which is brief in blacks due to pancreatic exhaustion and/or diabetic pancreatopathy.

Malnutrition is present in black T2DM, specifically protein malnutrition (Table 7).

Similarly, Methionine and Taurine were significantly lower in 19.92% and 29.79% of diabetics versus controls in Libreville respectively (Methionine 26.87/34.87; t = 3.03; Taurine 186.9/234.9; t = 2.29).

All these points to the nutritional hypothesis of malnutrition-related diabetes mellitus (MRDM), but not as an exclusive factor. Diabetic pancreopathy, possibly linked to the nutritional hypothesis, leads to a decrease in the functional mass of β-cells in the Langerhans islets of Black Africans.

The frequency of cardiovascular disease, such as Myocardial Infarction (MI) is relatively low in Black vs. Caucasian subjects. On the other hand, it should be noted that cardiovascular accidents (CVA) are more frequent in black African vs. Caucasian T2DM subjects.

Table 6. Comparative pathophysiology of the Black African vs. Caucasian population (Source: Buresi, 1990) [10].

Black African subjects	Caucasian subjects
- **Global malnutrition**	
- **Specific malnutrition (A.A. decline)**	
- **I.M.C < 19 kg/m²**	BMI
- **Diabetic NAI/DSLM pancreatopathy**	Initial insulin resistance
- **Decrease in IL β-cell functional mass**	Hyperinsulinism
- **Initial secretory insulinopenia**	MCV
- **Relative weakness MCV/DT2**	Advanced insulinopenia

- **BMI Caucasian profile can be seen**	

Table 7. T2D/AA/Nutritional Hypothesis.

	DT2 LBV POP. Black	T2DM Marseille Caucasian	
Met (M) g/L	26.87	31.3	Deviation s
Cys g/L (C)	2.04	10.56	Deviation s
Taurine (g/L)	186.9	234.9	Deviation s
Lys g/L (K)	311.4	281.6	Deviation s

These PEMBA L. LBV
Ngou and Perret/D, 1998: n˚ 309 [12]

Table 8. T2DM/BMI and postprandial hyperinsulinism syndrome (Insulin resistance?).

DT2 Libreville Black population	DT2 Marseille causasien
BMI N or low	FMC N or
Insulin level: 56.64 pmol/L	Insulin 75.61 pmol/L

These PEMBA LBV
Ngou and Perret/D, 1998: n˚ 309 [12]

– Morphological features and characteristics of dyslipidemia.

The main results, based on a review of the work of Reaven, 1998 [12]; Cuisinier-Raynal, 1985 [13]; Ngou, 1995 [14]; Faucher and Ngou, 2003 [15]; Planche and Ngou, 2005 [16]; Ovono and Ngou, 2012 [17], focus on the particularities of the black race and Caucasians.

There are many uncertainties about the definition and decision thresholds for metabolic syndrome in the black African population, particularly in Libreville. These uncertainties are due to:

- Lower HDL-C in T2DM patients (PON-1 polymorphism);
- The problem of LDL-C target values in the African context;
- To the high carbohydrate tolerance in the African Libreville subject;
- The notion of being overweight, which is not systematic among T2DM patients in Libreville, with a Body Mass Index (BMI) that is not systematically high;
- The low prevalence of syndrome X estimates (7% in Libreville, 5% in Abidjan).

And above all, the lack of validation of the target values of the various variables by clinical-biochemical consensus in relation to the reference values established in the Gabonese population. All this explains the difficulty of comparing threshold values with those of Caucasians (**Table 9** and **Table 10**). To shed light on this issue, we present the results of a reference study on this population of T2DM patients [17] (**Tables 10-12**).

Table 9. Lipid and morphological particularities and metabolic syndrome (Sources: Reaven, 1998 [12], Bongard, 2011 [2]).

Caucasians	Blacks/Afr/Gabon
TG > 1.7 mmol/L	TG Malaria, diet, tolerance?
CHDL < 1.03 40 mg/dl ♂ **< 1.29 50 mg/dl ♀**	CHDL (lower HDL-C in T2DM) LDL-C target value problem (no clinico-biochemical consensus study)
Systolic hypertension ≥ 130 mmHg **diastolic ≥ 85 mmHg**	High blood pressure, the problem of thresholds and clinico-biochemical consensus
Blood glucose ≥ 5.6 mmol/L **[oral test recommended].**	Glycemia: carbohydrate tolerance ↑ in the black population of Gabon
BMI > 30 kg/m², Waist circumference	Non-systematic BMI? Obesity > 30 Kg/m²
Prevalence♂ 22.5%, 18.5% ♀ **Prevalence ↑ but evolving clinical concept**	Atherogenic profile, syndrome X Ivory Coast: 5%. Gabon: 7%.
1950, 1980, 1988, 1998, 2001, 2005	
Reaven OMS EU FID	

Table 10. Average parameters of the study population.

Parameters	Averages	Standard deviations
Age (years)	53.3	10.9
Weight (kg)	76.4	4.1
Systolic pressure (mmHg)	157	24
Diastolic pressure (mmHg)	104	10
Body mass index (kg/m²)	26.5	6.3
Latency (seconds)	74	4
PON-1 activity (mU/mL)	0.32	0.11
Total cholesterol (mmol/L)	5.2	0.6
HDL cholesterol (mmol/L)	1.5	0.3
LDL cholesterol (mmol/L)	3.2	0.9
Triglycerides (mmol/L)	2.0	0.9
Blood glucose (mmol/L)	8.2	3.0
HbA1c (%)	8.3	3.0
Glomerular filtration rate (ml/min)	95	16
Plasma creatinine (mmol/L)	143	23

Table 11. Main atherogenic profiles found in the T2DM population studied (Source: Ovono, 2012) [17].

Main profiles and results DT2 Libreville		
Anomalies	**Number of cases**	**percentages**
High LDL	344	65.2
High triglycerides	88	16.7
Low HDL	336	63.6
High LDL and triglycerides	64	12.1
Low HDL and high triglycerides	44	8.3
High LDL, low HDL and high triglycerides	28	7.0
High LDL and low HDL	168	31.8
T2D complications		18% - 21%
Kinetics of conjugated diene appearance (Latency) **DT2 > DT2/HTA > DT2/Nephropathy > DT2/Retinopathy > DT2/Macroangiopathy**		
Activity PON1 **DT2 > DT2/Nephropathy > DT2/HTA > DT2/Retinopathy > DT2/Macroangiopathy**		

Table 12. dLDL oxidation lag times and PON1 enzyme activities (mean + standard deviation).

Pathophysiological condition	Number (%)	LDL oxidation (s)	PON-1 (mUI/mL)
Diabetes	120 (22.7)	80 ± 4	0.42 ± 0.11
Diabetes + hypertension	112 (21.2)	71 ± 6	0.28 ± 0.16
Nephropathy	100 (18.9)	81 ± 3	0.38 ± 0.05
Retinopathy	100 (18.9)	73 ± 4	0.25 ± 0.08
Macroangiopathy	96 (18.2)	67 ± 2	0.24 ± 0.06
Total	**528 (100)**	**74 ± 6**	**0.32 ± 0.11**

– Particularities of lipid metabolism in *Plasmodium falciparum* malaria

With regard to the glucido-lipidic observations in Table 13, we note that malaria induces several mechanisms in dyslipidemia which may even appear discordant. We prefer to use the term "particularities". Compared with Table 10, we find the classic hypertriglyceridemia of malaria. At the same time, a decrease in lipoprotein lipase (probably hepatic) is suggested. We also note hypoglycemia (classic in malaria), while at the same time lactates are elevated. Is there a problem with the Cori cycle (persistence of parasites and malarial pigments in the liver)?

Table 13. Malaria and dyslipidemia (Sources: Cuisinier-Raynal, 1985 [13]; Ngou, 1995 [14]; Faucher, 2003 [15]; Planche, 2005 [16]).

Malaria and dyslipidemia
Hyper TG (true vs pseudo) factor 4
Lower lipoprotein lipase
AGNE (rate x 2)
Hypoglycemia
Lactates

3.1.3. Analysis of Clinico-Biochemical Features

1) Dyslipidemia in diabetic adults

Looking at the discriminating and qualifying markers of dyslipidemia observed in Libreville, we note that HDL-C (protective) is low and LDL-C (bad cholesterol) is high.

In T2DM, this atherogenic profile is observed in 31.8% of the population [17]. Of course, TGs were elevated. In the same study, the onset time of conjugated dienes and $PON_{(1)}$ (paraoxonase 1) activity, indicators of chronicity and complications, were also disturbed. But especially in diabetics with the PON_1polymorphism (−107.55 and 192), the differential distribution of alleles, T, M, R is associated with the difference in enzymatic activities. The frequent allele is associated with low enzymatic activity and consequently low HDL-C.

These metabolic peculiarities led us to envisage predictive studies, imagining the extrapolation of metabolic disturbances at birth or in children into adulthood.

Thus, working on adiponectin, known to be a postprandial lipid-cleansing adipokine, we observed exaggerated fetal weight gain during pregnancy and at birth.

At the end of pregnancy, we noted an exaggerated transfer of nutrients from the mother to the fetus until birth. Adiponectin levels in the baby were 3 times higher than in the mother. This decrease in the mother's adiponectin level favored fetal lipid utilization, but highlighted the risk of macrosomia if storage was very high compared with synthesis. Perhaps a likely predictor of diabetes in adulthood in the absence of lifelong dietary control.

We also demonstrated that maternal weight gain during pregnancy was directly correlated with the concentration of LDL-C apolipoprotein B100 in umbilical cord venous blood ($r = 0.193$; $p = 0.017$). Thus, maternal weight gain responsible for disturbances in glycoregulatory balance and lipid disturbances both quantitative and qualitative, would be at the origin of exaggerated weight gain during pregnancy and at birth [18].

In situations of pre-diabetes 2, we noted a syndrome of postprandial hyperinsulinism characterized by the fact that baseline fasting blood glucose was lower than postprandial blood glucose at 2 h. The subject had prediabetes and incipient insulin resistance [19].

With regard to the particularities of insulin resistance (elevated insulinemia), we had already pointed out that, compared with Gabonese T2DM patients, Caucasians from Marseille showed a greater amplitude of insulinemia, a phenomenon consistent with BMI values and the respective trends of the Gabonese morphotype (BMI, most often normal = normal morphotype) or lower insulinemia amplitude in overweight Gabonese T2DM or pre T2DM versus permanent overweight with high insulinemia amplitude in Marseilles. In the MONICA study [2], we zoomed in on metabolic syndrome, with a detailed clarification of the data.

The metabolic syndrome (**Table 14**) brings together a maximum number of pathophysiological elements, enabling us to better identify this metabolic entity, study postprandial behavior and better understand the transition to T2DM.

Table 14. Metabolic syndrome (IDF cited by Bongard, 2011 [2]).

The presence of 3 criteria defines (poly) (dys)metabolic syndrome:

- Abdominal obesity (mandatory criterion)

Waist circumference > 54 cm ♂
84 cm ♀

+2 of the four criteria listed below:

- High triglycerides: triglyceride levels equal or exceed 1.7 mmol/L, equivalent to 150 mg/dL.
- Low HDL ("good") cholesterol: HDL cholesterol levels are below 1.03 mmol/L (40 mg/dL) in men and 1.29 mmol/L (50 mg/dL) in women.
- Hypertension: blood pressure, also known as arterial "pressure", is greater than or equal to 130 mmHg for systolic blood pressure and 85 mmHg for diastolic blood pressure.
- High venous blood glucose: fasting venous blood glucose equal to or greater than 5.6 mmol/L (100 mg/L).

These peculiarities may also be explained by the high carbohydrate tolerance of black Africans. In Africa, we see clients standing with 40 mmol/L of glucose in the blood. Similarly, and still in Africa, glycosuria only appears above 8 mmol/L of glucose in the blood (personal observations based on forty years' practice at the Libreville Faculty of Medicine). And lastly, we have observed that the monitoring of T2DM is inadequate for A1c glycated Hb [20]. Clinico-biochemical consensus is therefore becoming a major challenge (Biochemistry-Cardiology-Endocrinology departments, etc.).

2) Special features in children

Hypertriglyceridemia has also been described and analyzed, and the mechanisms evoked [13] [21]. Moreover, endothelial dysfunction, associated with a disturbance in glucido-lipid metabolism, is usually associated with an increase in cardiovascular risk. This is the case with *Plasmodium falciparum* malaria in Gabon, the only species responsible for pernicious attacks. In this host-parasite relationship, we demonstrated a decrease in both plasma and membrane phospholipids in children infected with *Plasmodium falciparum.* This decrease was correlated with parasitaemia, and would be responsible for the fragilization of the red blood cell, and hence hemolysis and lactic acidosis.

This interaction may also be responsible for the increase in triglycerides and the drop in HDL cholesterol in children with malaria. In a study carried out in collaboration with the parasitology department of the Faculty of Medicine, we demonstrated the correction of these lipid parameters in children suffering from *Plasmodium falciparum* malaria and treated with the sulfadoxine-pyrimethamine combination. This hypertriglyceridemia is true, not dependent on glycerol elevation [15].

The persistence of this dyslipidemia is rapid under treatment. This correction is rapid for total, HDL and LDL cholesterol, but takes three to fifteen days for triglycerides. In fact, after clearance of the parasite load, we observed a variation in plasma lipid parameters CT and HDL-C significantly high ($p < 0.001$) LDL-C and TG significantly low ($p = 0.93$ and $p = 0.04$ respectively). This dyslipidemia in children could be associated with other perinatal factors, such as pregnancy

with disorders of glycoregulation. Could the high status of T2DM in the Gabonese population be linked to these fluctuations in glycoregulation throughout life, given that malaria is endemic in Gabon?

Other studies on malaria during gestational diabetes have highlighted either macrosomia or low birth weight in children [22].

4. The Value of Exploring Postprandial Dysmetabolism: Pathophysiology and Review of the Literature, Outlook

It is now accepted that fasting lipid data alone, without any exploration of postprandial lipid metabolism, are no longer sufficient to assess the full range of metabolic excursions that are the cause of cardiovascular damage and various risks.

This message also applies to the exploration of carbohydrate metabolism in the fasting state, but also in dynamic form due to the relatively short exploration period (2 - 3 h) of the orally induced hyperglycemia (OIGH) test.

With regard to lipid metabolism, Martine LAVILLE, 2013 [23] defines the postprandial state as a dynamic, non-equilibrium state, characterized by a prolonged increase in the concentration of chylomicron-rich lipoproteins (CRL) of exogenous and endogenous origin, accompanied by remodeling of LDL and HDL, and the strong clinical and metabolic implications on which their complex and comprehensive study is based.

- Key blood dysmetabolic varieties to be studied in relation to health harms (cardiovascular risks and cardiovascular disease and diabetes 2) [24].
- Lipotoxicity-glucotoxicity cellular, tissue, endothelial dysfunction, carotid intima [23].
- The involvement of HGPP and HLPP in risk situations [23].

This means using relevant, integrative parameters and markers of the postprandial response, discriminating metabolites and discriminating times. Those that enable us to understand post-meal lipid and carbohydrate responses and their consequences.

We can also remind you of the parameters and times of exploration in the interpretation of postprandial lipid metabolism.

Discriminating metabolites:

- TG, palmitate, retinol (vitamin tracer of intestinal lipoproteins) ApoB48.
- Discriminating times.
    - Lipid peak.
    - Late lipid elimination times.

Many authors have worked on the response to nutrients during postprandial lipemia in subjects [3] [23] prediabetics (syndrome X) and diabetics [2] [5] [25] to determine the kinetic modulation of metabolites, the kinetics of markers, the nature of nutrients, cardiovascular and atherogenic impacts, consequences and risks, and the vision of therapeutic strategy (prevention, food and drug industries). We have summarized all the metabolic events and the various health impact markers in the form of tables, with bibliographical references (**Tables 15-17**).

Table 15. Pathophysiological summaries and exploration of postprandial hyperglycemia and lipemia.

Metabolic event	Meaning Impact biosanté	Marker exploration
Postprandial phase modulation Postprandial excursions during the day	Lipotoxicity Cellular and tissue glucotoxicity Artery Diabetes risk MCV	Fasting dosages EPP, EPA Postprandial phase modulation Effects of different nutrients to be characterized
Glycemic homeostasis • G exogenous • Endogenous production G • Non-ID use • Use ID	Pool glycemia controlled in the 4 mechanisms and insulin-dependent organs liver, kidney Cloudy revelation	Blood glucose/isotopes Track exploration Intestinal absorption Production, use of metabolic organs Insulin (resistance)
Dietary glycemic response = GI (hyperglycemic power) Glycemic index X dietary carbohydrate quantity → glycemic load [23]	Knowledge Glycemic load food Food selection	Glycemic load
Postprandial hyperglycemia + Hyperlipidemia PP + Hyperinsulinism	Complications of T2DM and CVD Endothelial dysfunction Oxidative stress	Biochemical exploration Syndrome X
Postprandial lipemia Metabolic organ marker kinetics (liver, BP, muscles)	Cellular utilization from lymph/blood to cells and tissues	CM: Lipoprotein-rich TG Apo B48 Apo B10C VLDL IDL, LDL Isotopic tracers Kinetic parameters, appearance time • Pic TG • Area under the curve • Maxima, minima values • Time to return to basal state
Storage obesity - Activated adipocytes. incoming lipid flow - Exogenous + inhibition of adipocyte lipolysis + effect of meal glucose on insulin stimulation	Involvement of events in atherogenesis [23] Stroke risk [23] Coronary risk relationship carotid intima/PP lipemia [23] Cytotoxic/atheromatous plaque risk and↗ cholesterol-enriched LRT [23] Modified postprandial response exacerbated in prediabetic (syndrome X) LRT↗ [23]	Postprandial lipid markers
Modulation of metabolic kinetics in the postprandial phase	Lipid clearance capacity and threshold 20 - 30 g fat →↗ significant TG_{PP} Lipids ↗↗→ very long PP period 8 - 12 h [3] Variable lipemic kinetic profile depending on the amount of lipids ingested (numerous peaks, different amplitudes, plateau zone) If LRT (rich in TG) remain PP, independent CVD risk [23]	Amount of fat Nature of lipids Profile layout PP - Woodpecker - Air under the curve - Return time
Nature of AGMs	Nature AG Short-chain AGS→ low PP response as absorbed in door circulation.	

Table 16. Anomalies in T2DM dyslipidemia (Source: Vergès, 2019 [5]).

Type of event	Expression	Observation
Quantitative lipoprotein abnormalities	TG ↗ HDL-C ↘	Predictable atherogenic and cardiovascular risk
Qualitative and kinetic anomalies with atherogenic potential	↗ Large VLDL / CE, TG, small and dense LDL ↗ Breast TG LDL and HDL, glycation, Apo ↗ LDL susceptibility to oxidation	Atherogenic and cardiovascular risk
Insulin resistance ()		
Adipokines Retinol binding Protein 4	Leptin, adiponectin Postprandial lipid purification	Cardiovascular disease in the event of purification failure
Loss of anti-atherogenic properties	↗ TG in HDL	HDL dysfunction

Table 17. Factors modulating lipidemia PP (Source: Lairon, 2008 [3]).

Nature	BVG response	Comments
Food Lipid type Sugars Fibers Alcohol		
Physical/aerobic activity during the previous 24 hours	↘–24% - 35%	
Smoking	↗ +50%	Habitual smokers vs. non-smokers
Alcohol	↗ +60%	Alcohol addition mixed meal
Age	↗ with age	To be well defined
Gender and menopausal status	↗ more for♂ for the same meal ↗♀ menopausal vs. reproductive	
Obesity	abdominal obesity	Compared with normal-weight subjects
HIG	↗ HTG fasting	
Genetics	Gene polymorphism and response	Apo A_1, IV, A, V, B, E, C_1, C_3

Finally, in this pathophysiological review, the omnipresence of diabetic 2, cardiovascular and cardiometabolic risk situations are such that research in people living with the human immunodeficiency virus (PLHIV) under antiretroviral treatment (protease inhibitor) revealed lipid and carbohydrate disorders at cardiovascular and diabetic risk:

- CT ↗
- LDL-C ↗
- HDL-C ↘

These anomalies were found respectively with a prevalence of 44.4%, 23.5%, 17.4% [26].

All in all, there's a biochemical black hole between the punctual lipid results obtained after the 8:30 a.m. sample at D0 and the fluctuating situations since 8:30 a.m. at D-1 or since the last meal of the night.

In fact, just as postprandial glycaemia enables us to appreciate the dynamic and kinetic nature of glymoregulation during the exploration of glycaemic balance (even if there are those who deplore the short 2 - 3 h postprandial duration), so many authors are increasingly considering the dynamic study of postprandial lipemia in fat metabolism. This parallelism of diagnostic tests is not accidental, given the close interaction between the metabolisms of the two classes of nutrients mentioned above.

Furthermore, the difficulties of measuring lipoproteins of intestinal origin, the glycation of apolipoproteins [27], the dawn phenomenon, the problems of threshold values for postprandial hyperglyceridemia (TGPP 3h < 1 g/L and TGPP 4h < 2.6 g/L) [19] lead us to prefer the notion of dynamic postprandial lipidemia to postprandial lipidemia in punctual dosage, as the latter does not account for the dynamics of disorders.

Clearly, this literature review on pathophysiology and exploration sheds light on the stakes, the vision and the prospects for the management of these frequent pathologies—T2D and cardiovascular disease—as well as their interactions. It reveals the biological and technical black holes and opens up new avenues of research.

5. Conclusions

It is customary to study lipids and carbohydrates under fasting conditions. This framework facilitates static diagnoses to assess the level of normality or elevation of carbohydrate-lipid metabolism markers. However, it does not allow us to study load dynamics in relation to deviant, unregulated metabolic behaviours, so that we can index all dietary metabolic provocations in relation to pathological states, investigate cellular disorders and envisage therapeutic management.

Today, the interaction between diet and health has become a major issue for experts in biological functions (during metabolism) and the functioning of metabolic organs (liver, pancreas, muscles, erythrocytes, adipose tissue, etc.).

Punctual marker assays have become insufficient, and the current option is dynamic explorations, including postprandial lipemia. This no longer involves a single punctual result, but the monitoring of fluctuations and modulations in the body's metabolic response to a food load (or food tracers administered under specific technical conditions) by means of discriminating metabolites in the blood, qualification of the lipid peak(s) (intensity, height, surface area under the layer) and lipid elimination and purification times (early or late).

Today's challenges are:

1) Diagnostics: improving dynamic mode diagnostics (postprandial lipemia) to enable informative power on fluctuations and modulations on biological functions and metabolic organs. The methodology must therefore be calibrated.

2) Treatment strategy.

Multicenter studies involving teams of specialists in biochemistry, physiology, nutrition, endocrinometabolism, cardiology and diet therapy need to be launched, sometimes in specific areas such as the Congo Basin. They aim to:

- Dietary advice and diet therapy (foods characterized by a normal postprandial lipid purification profile);
- Production and promotion of therapeutic industrial foods for people with lipid purification disorders.

Conflicts of Interest

The authors declare that they have no conflicts of interest in relation to this article.

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Glossary

ACAT	Acyl coa cholesterol acyl transferase
AGL	Free fatty acids
Apo	Apoprotein
ASC	Area under the curve
AUC	Area under the curve
CETP	Cholesterol ester transfer protein
CL	Free cholesterol
HPLC	High-performance liquid chromatography
CM	Chylomicron
eNOS	Endothelial NO synthase
EOHF	Endothelium derived hyperpolarizing factor
HGPO	Orally-induced hyperglycemia
HGPP	Postprandial hyperglycemia
HOL	High density lipoprotein
HOMA	Homeostasis model assessment
ICAM	Intercellular cell adhesion molecule
IDL	Intermediate density lipoprotein
BMI	Body mass index
ITG	Glucose intolerance
LCAT	Lecithin cholesterol acyl transferase
LE	Endothelial lipase
L-NMMA	N^G-monomethyl-L-arginine
LOL	Low density lipoprotein
LPL	Lipoprotein lipase
LRP	LDL receptor related protein
LRT	Triglyceride-rich lipoproteins
LSR	Lipolysis stimulated receptor
nCM	No chylomicron
NO	Azode monoxide (nitric oxide)
PAl1	Plasminogen activator inhibitor
PGH2	Prostaglandin
PGl_2	Prostacyclin
PL	Phospholipid
PLTP	Phospholipid transfer protein
RE	Retinol ester
RP	Retinol palmitate
TG/PP	Triglyceride/postprandial
TGLH	Hepatic triglyceride lipase
TXA_2	Thromboxane A2
VCAM	Vascular cell adhesion molecule
VLDL	Very-low density lipoprotein