

Editorial

Role of Blood Sampling Time and Dietary Factors in the Prediction of Cardiometabolic Risk among Patients with Obesity and/or Diabetes: Open Hypotheses

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Obesity prevalence is increasing at an alarming worldwide rate in all age-groups [1]. Concurrently, Type 2 Diabetes (T2D) is continuing to be an increasing public health burden. The worldwide prevalence of diabetes among adults (aged 20-79 years) will increase from 6.4% in 2010 to 7.7% by 2033 [2]. The exceeding of body weight associated with glucose intolerance and/or T2D is characterized by hyperinsulinemia, peripheral resistance to the action of insulin, hypertriglyceridemia, decreased High-Density Lipoprotein cholesterol (HDL) and other lipid and carbohydrate changes [3,4]. Diabetes and obesity affect functioning, quality of life and are significantly associated with worsening of many health issues especially, those related to cardiovascular events.

Assessment of health risks, including diabetes and obesity, are measured in clinical settings and epidemiological research by referring to fasting recommendations. However, the postprandial state, a period that comprises and follows a meal, is recently receiving increased attention. The postprandial state cumulatively includes approximately half of the nycthemeral period and involves numerous finely regulated motor, secretory, hormonal and metabolic events. It was about thirty-five years ago, when Zilversmit (1979) suggested that postprandial lipemia may have a role in atherogenesis and other cardiovascular problems [5].

Atherogenic dyslipidemia is a metabolic disorder characterized by low HDL levels, high triglycerides level, a large number of small, dense and oxidized Low-Density Lipoprotein (LDL) particles and high levels of apolipoprotein (apo) B.

The regulatory pathways of our metabolism are influenced by several factors; nutritional parameters modulated by dietary pattern and meal composition, life style conditions (physical activity, smoking, and alcohol consumption), physiological status (age, gender, and menopausal status), pathological conditions (diabetes, insulin resistance, and obesity) and genetics which may contribute to inter-individual variability on metabolic responses, and thereby, susceptibility to health complications [6].

The apo B to apo A1 ratio is supported by cumulative evidences as a practical risk predictor for cardiovascular events, particularly in

myocardial infarction, which can represent 66% of overall heart attack risk [7,8]. Furthermore, the total cholesterol (TC)/HDL ratio seems to be the most frequently used index that has a high association with cardiovascular diseases prediction than the usual lipid parameters. However, the association between dietary fat consumption intake components and lipid ratios (apo B/apo A1 and TC/HDL-c), is not as simple as it is thought [9]. For several decades, the causality association between diet and plasma lipid levels is receiving a close attention. This link is heavily influenced by the time of blood sampling as a powerful modifying factor. So should we rather refer to the fasting or the postprandial state?

Moreover, the saturated or unsaturated nature, as well as the number of carbon atoms in the chain, of the fatty acids consumed, is another powerful modifying factor. The high consumption of Saturated Fatty Acids (SFA) plays a non-negligible role in the genetic predisposition to obesity by modulating the relationship between “fat mass and obesity-associated protein” gene (FTO) rs9939609 and waist circumference. This may be a relevant explanation of the association between diet and metabolic diseases among individuals at increased cardio metabolic risk [10]. Overweight and obesity are accentuated among high-SFAs consumers. So, should we recommend diets with less SFAs for obese and/or diabetic patients? What are the most implicated saturated fatty acids in this relationship? Should we modify the proportions of some nutrients or replace some of them in order to lower the cardiovascular risk related to obesity and/or diabetes?

A couple of prospective epidemiological studies suggest that there is no significant evidence for concluding that dietary saturated fat is associated with an increased risk of cardiovascular diseases [11]. More data are also needed to elucidate whether CVD risks are likely to be influenced by the specific nutrients used to replace saturated fat.

Besides the importance of sampling time when measuring plasma lipids and their ratios, fat dietary intake, especially saturated ones, might be one of the more persuasive factors modifying the causal relationship between food habits and cardiovascular risk prediction. Likewise, T2D, overweight and obesity have relevant effects on TC/HDL and/or apo B/apo A1 levels as independent and advantageous risk predictors for CVD.

Until nowadays, the role of dietary factors and blood sampling time in the relationship between the physiological risk factors (obesity, diabetes) and the cardiovascular diseases prediction remains an open hypothesis that did not find convincing answers.

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