Case Study

# Ambient temperature-related exaggerated post-prandial insulin response in a young athlete: a case report and implications for climate change

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The objective is to present the case of a 21-yr-old athlete observed with non-physiological immediate post-prandial insulin response (1162 pmol/l; normal glucose excursion: 6.6 mmol/l), in a warm environment. No suspicion or evidence of any underlying pathology was found in this well-trained Afro-Caribbean male runner. He never reported any hypoglycemic episode. When performing the same protocol performed in a cooler environment (21.0°C vs 30.3°C), only physiological responses were observed. We conclude that 1) youth, leanness and regular exercise training are not absolutely protective against glucose metabolism impairment in apparently healthy subjects; 2) ambient temperature should be regarded as a potential source of glucose metabolism impairment.

**Key Words: environment, heat, diabetes, case study, exercise training**

## INTRODUCTION

Insulin resistance is a metabolic condition known to be associated with diabetes and its macro- and microvascular complications. Although exact causes are not completely understood, experimental and epidemiological evidence incriminates excess weight and physical inactivity as major contributors to insulin resistance development.[1] Here we report the unexpected case of a fit middle-distance runner considered as healthy who presented non-physiological insulinemia.

## CASE REPORT

An athlete who had been included in one of our nutrition studies presented exaggerated post-prandial insulin response (1162 pmol/L; electrochemiluminescence immunoassay method; sample double-checked) during his participation in a nutrition research protocol previously published.[2] Plasma glucose remained normal (6.6 mmol/L). After a fast of approximately 10 hours, he arrived at 6.45AM at the laboratory which was deliberately heated (30.3°C) for the experiment. He had to rest in a semi-recumbent position for 90 minutes. Then, he sat 30 minutes alone in a non-stimulating environment for an ad libitum meal of small ham and cheese sandwiches (799 kcal, 25.7% proteins, 33.2% carbohydrates, 41.0% lipids), widely appreciated in Guadeloupe. He finished his meal within 20 minutes. The food was proposed in abundant quantity, on a plate weighted before and after the meal, the participant being unaware of the fact that food intake was a study outcome. The water intake during the meal was 275 mL (4 mL/kg). He had blood sampling at different time points of the session before the meal, and 35 minutes after starting the meal.

The case is a healthy 21-yr-old Afro-Caribbean middle-distance runner (800 m-1500 m) performing at the regional level. He is 1.84 m tall, 68.8 kg body mass and 9.5% body fat mass (Multi-frequency bioelectrical impedance analysis: InBody S10®, Biospace Inc, Japan). He had met all eligibility criteria of the study: absence of identified chronic or acute pathology, any other limitation to exercise, absence of any food allergy or eating disorder (normal score for the three-factor eating questionnaire), acclimatisation to the tropical climate, regular physical activity (3650 METs/min/week), normal birth weight, <2 kg body mass variation in the previous 6 months. For the study, he had a general dietary assessment and underwent a cardiopulmonary exercise test. His dietary pattern was characterized by a daily intake of processed energy-dense food high in fat and low in dietary fiber, with large amounts of carbohydrate – in particular with a high glycemic index. He had been training regularly and his max imal oxygen uptake was normal (52.3 $mLO_2$/min/kg,

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108.3% of the predicted maximal in sedentary) with excellent endurance evident from his ventilatory threshold (67.3% of maximal oxygen uptake). Other biological and clinical assessments were made. Blood pressure, fasting glucose, insulin, lipid profile, lactate, CRPus, cortisol, IL6, glucagon, leptin, ghrelin, CCK, PP, and body water distribution were normal. There was no evidence of acanthosis nigricans or any hepatic or thyroid disease. As an athlete, he reported no episode of hypo or hyperglycemia. He denied doping and was not aware of any direct relative with diabetes. Other markers and determinants of insulin and glucose metabolism and function such as serum c-peptide or HbA1c were not evaluated. He declined an oral glucose tolerance test to further investigate potential metabolic disturbance. However, he was re-interviewed 2 years later and found to be in good health and performance status.

Interestingly, a few weeks after the occurrence of the odd post-prandial response, the athlete performed the same session with a 21.0°C ambient temperature (the time pattern, food intake for the last meal of the day preceding the test were like that when hyperinsulinemia was found). His post-prandial response was completely normal in terms of insulin (416 pmol/L) and blood glucose (5.5 mmol/L), despite the ingestion of a larger quantity of sandwiches (1182 kcal) during the meal on this occasion (Figure 1).

## DISCUSSION

Fitness and leanness are considered as protective against insulin resistance. Here is described a counterexample, in which post-prandial insulinemia is not physiological in a young well-trained athlete with low percent body fat.

None of the criteria used to define the metabolic syndrome were met, whatever the chosen definition.[3] In this patient, the exaggerated post-prandial insulin response did not occur concomitant with isolated hypoglycemia. Hypoglycemia is listed in the differential diagnosis of most insulin-secreting islet cell tumors and other pathological conditions leading to hyperinsulinemia. Acute and chronic diseases that affect the pancreas, liver, kidneys, thyroid and adrenal glands would be compatible with transient exaggerated post-prandial insulinemia. In particular, insulinoma and other endocrinopathies could be suspected. However, none of these possible diagnoses was evident given the overall clinical picture along with the consistency and concordance of otherwise normal investigations and biomarkers. For example, fasting glycaemia and insulinemia were measured at rest 12 times on 4 separate days. Only physiological values were observed (minimum and maximum were 3.7 and 4.6 mmL/L and 34 and 179 pmol/L for glucose and insulin, respectively). Based on available data and on the absence of development of later observable disease, our interpretation is that he had no underlying pathology of a known kind. Environmental temperature is the only identified factor identified which might have contributed to the abnormal profile described here. Increased glycaemia and/or insulinemia, specifically in ambient temperatures above 25°C have been previously reported in healthy subjects[4,5] and pregnant women.[6] This athlete participated in a study which, it appears, induced a higher postprandial glucose and insulin with heat exposure.[2] Although he was the only one to present a quasi-pathological insulin response in the session performed in a warm environment, the increase in insulin and glucose was observed in most subjects. In the same study, but with another group of participants performing a standard glucose tolerance test, the glucose load elicited an exaggerated increase in blood glucose in a warm environment. We therefore suggest that this case is additional evidence supporting the argument that a warm environment is increase the risk of impaired glucose metabolism. This point is of potentially wider clinical relevance since it could help to explain the increased diabetes prevalence in warm regions like India or the Pacific islands. To date, the increased prevalence of metabolic pathologies in these regions has been attributed to genetic factors along with poor dietary and physical activity behaviors. If confirmed, this phenomenon calls for better standardization of environmental temperature to improve the accuracy of glu-

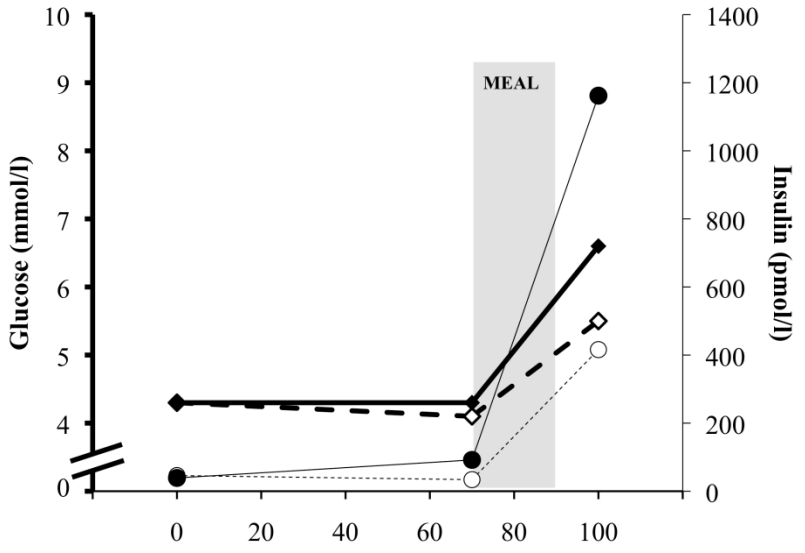


**Figure 1.** Time course of blood glucose (thick lines, diamond markers) and insulin (thin lines, circle markers) in warm (30.3°C, continuous line, closed markers) and cooler (21.0°C, discontinuous line, open markers) environmental temperature

cose tolerance tests. It also should be considered in the context of climate change where it may be a risk multiplier as well as a trigger of changes in global health.[7] Disturbance of glucose metabolism is, therefore, a putative contributor to the growth health burden evident with climate change.

In conclusion, the present case indicates that fitness and leanness may not be systematically protective against acquired glucose impairment. That high ambient temperature may contribute to transient or even chronic glucose intolerance and/or insulin resistance deserves serious consideration in regard to living conditions, global warming and climate change.

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