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

RUNNING TITLE: A case of post-prandial hyperinsulinemia

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This manuscript is original.

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ABSTRACT

1 The objective is to present the case of a 21-yr-old athlete observed with non-physiological
2 immediate post-prandial insulin response (1162pmol/l; normal glucose excursion:
3 6.6mmol/l), in a warm environment. No suspicion or evidence of any underlying pathology
4 was found in this well-trained Afro-Caribbean male runner. He never reported any
5 hypoglycemic episode. When performing the same protocol performed in a cooler
6 environment (21.0°C vs. 30.3°C), only physiological responses were observed. We conclude
7 that 1) youth, leanness and regular exercise training are not absolutely protective against
8 glucose metabolism impairment in apparently healthy subjects; 2) ambient temperature
9 should be regarded as a potential source of glucose metabolism impairment.

Key words: environment; heat; diabetes; case study; exercise training

10 Odd post-prandial insulin response in a young athlete: a case report

11

12 INTRODUCTION

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

19

20 CASE REPORT

21 An athlete who had been included in one of our nutrition studies presented
22 exaggerated post-prandial insulin response (1162pmol/l; electrochemiluminescence
23 immunoassay method; sample double-checked) during his participation in a protocol research
24 in nutrition with published results.² Plasma glucose remained normal (6.6mmol/l). After
25 approximately 10 hours of fasting, he arrived at 6.45AM at the lab deliberately heated
26 (30.3°C) for the purpose of the experiment. He had to rest down in a semi-recumbent position
27 during 90 minutes. Then, he sat 30 minutes alone in a non-stimulating environment for an *ad*
28 *libitum* meal of small ham and cheese sandwiches (799kcal, 25.7% proteins, 33.2%
29 carbohydrates, 41.0% lipids), widely appreciated in Guadeloupe. He finished his meal within
30 20 minutes. The food was proposed in abundant quantity, on a plate weighted before and
31 after the meal, the participant being unaware of the fact that food intake was a study outcome.
32 The water intake during the meal was 275mL (4ml/kg). He had blood sampling at different

33 time points of the session before the meal, and 35 minutes after starting the meal.

34 The patient is a healthy 21-yr-old Afro-Caribbean middle-distance runner (800m-
35 1500m) performing at the regional level. He is 1.84m tall, 68.8kg body mass and 9.5% body
36 fat mass (Multi-frequency bioelectrical impedance analysis: InBody S10®, Biospace Inc,
37 Japan). He had met all eligibility criteria of the study: absence of identified chronic or acute
38 pathology, any other limitation to exercise, absence of any food allergy or eating disorder
39 (normal score at the three-factor eating questionnaire), acclimation to tropical climate, regular
40 physical activity (3650METs/min/week), normal birth weight, <2kg body mass variation in
41 the previous 6 months. For the purpose of the study, he underwent a cardiopulmonary
42 exercise test and a general dietary investigation. He had been training regularly and his
43 maximal oxygen uptake is normal (52.3mlO₂/min/kg, 108.3% of the predicted maximal in
44 sedentary) with excellent endurance objectivized by the ventilatory threshold (67.3% of the
45 maximal oxygen uptake). His dietary pattern is characterized by a daily intake of processed
46 energy-dense food high in fat and low in fiber, and large amounts of carbohydrates – in
47 particular with a high glycemic index.

48

49 Further biological and clinical explorations were performed. All values for blood
50 pressure, fasting glucose, insulin, lipid profile, lactate, CRPus, cortisol, IL6, glucagon, leptin,
51 ghrelin, CCK, PP, body water distribution were normal. There was no evidence of *acanthosis*
52 *nigricans* nor any hepatic or thyroid disease. The athlete reported no episode of hypo or
53 hyperglycemia. He denied doping and was not aware of direct ascendant with diabetes. Other
54 markers and determinants of insulin and glucose metabolism and function such as serum c-
55 peptide or HbA1c were not evaluated and the athlete was not interested in undergoing an oral
56 glucose tolerance test to further investigate potential metabolic disturbance. However, he was
57 re-interviewed 2 years later and declared good health and performance status.

58

59 Interestingly, a few weeks after the occurrence of the odd post-prandial response, the
60 athlete performed the same session with 21.0°C ambient temperature (similar time pattern,
61 similar food intake for the last meal of the day preceding the test). His post-prandial response
62 was completely normal in terms of insulin (416pmol/l) and blood glucose (5.5mmol/l),
63 despite the ingestion of a larger quantity of sandwiches (1182 kcal) during the meal.

64

65

66 **DISCUSSION**

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

70 None of the criteria used to define the metabolic syndrome were met, whatever the
71 chosen definition.³ In this patient, the exaggerated post-prandial insulin response does not
72 occur concomitantly with isolated hypoglycemia. Hypoglycemia is listed in the differential
73 diagnosis of most insulin-secreting islet cell tumors and other pathological conditions leading
74 to hyperinsulinism. Acute and chronic diseases that affect the pancreas, liver, kidneys,
75 thyroid and adrenal glands would be compatible with transient exaggerated post-prandial
76 insulinemia. In particular, insulinoma and other endocrinopathies could be suspected.
77 However, none of the conditions to consider their diagnosis was present given the
78 convergence of normal values for the biomarkers and the absence of symptom. For example,
79 fasting glycaemia and insulinemia was measured at rest 12 times on 4 separate days. Only
80 physiological values were observed (minimum and maximum were 3.7 and 4.6 mml/l and 34
81 and 179pmol/l for glucose and insulin, respectively). Based on available data and on the
82 absence of development of later observable disease, our interpretation is to discard

83 underlying pathology. We are not aware of any bias that would explain the observation.
84 Subsequently, environmental temperature is the only identified factor likely to contribute to
85 the abnormal profile described here. Increased glycaemia and/or insulinemia, specifically in
86 ambient temperature above 25°C have been previously reported in healthy subjects^{4,5} and
87 pregnant women.⁶ Also, this athlete participated in a study which evidenced higher
88 postprandial glucose and insulin levels with heat exposure.² Although he was the only one to
89 present a quasi-pathological insulin response in the session performed in a warm
90 environment, the increase in insulin and glucose was observed in most subjects. In the same
91 study but with another group of participants performing a standard glucose tolerance test, the
92 glucose load elicited an exaggerated increase in blood glucose in a warm environment. We
93 therefore suggest that this case is an additional piece of evidence supporting that a warm
94 environment is likely to impair glucose metabolism. This point is of potentially wide clinical
95 relevancy since it could contribute to explain the diabetes burden in warm regions like India
96 or the Pacific islands, where the overprevalence of metabolic pathologies is mostly attributed
97 to genetic factors, jointly with poor diet and physical activity behaviors. Also, if confirmed,
98 this phenomenon calls for better standardization of environmental temperature to improve the
99 accuracy of the analyses of glucose tolerance. It furthermore has to be considered in the
100 context of climate change that has been conceptualized as a risk multiplier as well as a trigger
101 of primary effects on global health.⁷ Disturbance of glucose metabolism is a putative
102 additional piece in the burden of disease of climate change.

103

104 In conclusion, in light of the present case, fitness and leanness appears as not
105 systematically protective against acquired glucose metabolism impairment. The eventuality
106 that high ambient temperature contributes to transient or even chronic glucose intolerance
107 and/or insulin resistance deserves to be seriously explored.

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FIGURE LEGENDS

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.

