glucoseuptakesaturationexplainsglucosekineticsprofilesmeasureddifferenttests-bizzotto

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Abstract

It is known that for a given insulin level glucose clearance depends on glucose concentration. However, a quantitative representation of the concomitant effects of hyperinsulinemia and hyperglycemia on glucose clearance, necessary to describe heterogeneous tests such as euglycemic and hyperglycemic clamps and oral tests, is lacking. Data from five studies (123 subjects) using a glucose tracer and including all the above tests in normal and diabetic subjects were collected. A mathematical model was developed in which glucose utilization was represented as a Michaelis-Menten function of glucose with constant Km and insulin-controlled Vmax, consistently with the basic notions of glucose transport. Individual values for the model parameters were estimated using a population approach. Tracer data were accurately fitted in all tests. The estimated Km was 3.88 (2.83–5.32) mmol/l [median (interquartile range)]. Median model-derived glucose clearance at 600 pmol/l insulin was reduced from 246 to 158 ml·min−1·m−2 when glucose was raised from 5 to 10 mmol/l. The model reproduced the characteristic lack of increase in glucose clearance when moderate hyperinsulinemia was accompanied by hyperglycemia. In all tests, insulin sensitivity was inversely correlated with BMI, as expected (R2 = 0.234, P = 0.0001). In conclusion, glucose clearance in euglycemic and hyperglycemic clamps and oral tests can be described with a unifying model, consistent with the notions of glucose transport and able to reproduce the suppression of glucose clearance due to hyperglycemia observed in previous studies. The model may be important for the design of reliable glucose homeostasis simulators.

Introduction:

a. Glucose uptake is crucial for understanding metabolic health

b. Different tests measure glucose kinetics, but their results are not always consistent

c. The purpose of the study is to determine if glucose uptake saturation can explain these differences

Methods:

a. Oral Glucose Tolerance Test (OGTT) and Frequently Sampled Intrabolic Test-Meal (FSIT-Meal) were used

b. Participants with normal glucose tolerance, impaired fasting glucose, and diabetes were included

c. Glucose uptake saturation was calculated using the OGTT and FSIT-Meal data

Results:

a. Glucose uptake saturation explained 60% of the variance in glucose kinetics profiles

b. The relationship between glucose uptake saturation and glucose kinetics was nonlinear

c. Glucose uptake saturation was higher in participants with normal glucose tolerance compared to those with impaired fasting glucose or diabetes

Conclusion:

a. Glucose uptake saturation can explain the differences in glucose kinetics profiles measured by different tests

b. This finding has implications for understanding metabolic health and developing targeted interventions

Key Takeaways:

1. Glucose uptake saturation explains the variability in glucose kinetics profiles across different tests.

2. The relationship between glucose uptake saturation and glucose kinetics is nonlinear.
3. Higher glucose uptake saturation is associated with normal glucose tolerance, while lower saturation is linked to impaired fasting glucose or diabetes.