

Glucose-6-phosphate dehydrogenase deficiency associates with lower BMI in males with Type 2 diabetes

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Introduction

Glucose-6-phosphate dehydrogenase (G6PD) deficiency, the most common human enzyme defect, is inherited in an X-linked recessive pattern hence affects males more severely. G6PD potentiates generation of reactive oxygen species (ROS) in the pentose phosphate pathway which are implicated in obesity. However, studies on the associations of G6PD deficiency and obesity are limited.

In this retrospective study, we sought to compare the BMI of males with T2D and G6PD deficiency (Study Group, SG); and males with T2D and normal G6PD levels (Control Group, CG) seen at Imperial College London Diabetes Centre (ICLDC).

Methods

Information on patients tested for G6PD levels were reviewed from the centre's database. Data retrieved for the SG and age-matched CG included age, sex, age at T2D diagnosis, and BMI. Statistical analyses were performed using SPSS version 24.

Results

A total of 6693 patients were tested for G6PD levels; out of which, 1679 (25.1%) were found to be G6PD-deficient. 171 (21.5%) of the 794 G6PD-deficient males also had T2D (SG); 180 males composed the CG. Majority of the subjects were Emirati (88.0% for both the SG and CG).

There was no significant difference with the age of T2D diagnosis between the two groups ($p>0.05$). BMI in the SG was found to be significantly lower in the SG than the CG ($p<0.01$). (Table 1)

Parameter	Group	N	Median	IQR	p-Value
Age (years)	SG	171	55.0	47.0-65.0	.653
	CG	180	56.0	50.0-64.0	
Age at T2D Diagnosis (years)	SG	171	44.0	36.0-53.0	.961
	CG	180	44.3	35.0-51.6	
BMI (kg/m ²)	SG	171	27.4	25.1-29.6	.000
	CG	180	28.7	26.2-32.0	

Table 1. Patient characteristics of the study and control groups compared using Mann Whitney U-test. SG= Study Group (G6PD-Deficient with T2D); CG=Control Group (G6PD-Normal with T2D); IQR=Interquartile Range; T2D=Type 2 Diabetes

Conclusion

This is the first report on the association of G6PD deficiency with obesity, to the best of our knowledge, which corresponds to the reported mechanistic role of G6PD in driving adipose tissue inflammation in obesity. Prospective studies in larger cohorts and different ethnic groups are needed to establish how G6PD moderates weight and T2D. Potential therapeutic implications may be explored.