Association of glucose-6-phosphate dehydrogenase (G6PD) expression and obesity: A systematic review

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Introduction: Several studies have associated glucose-6-phosphate dehydrogenase (G6PD) overexpression with weight gain and obesity due to its roles in proinflammatory signalling (1-3). However, a systematic review of the recent literature where the reported findings are synthesized and evaluated has not been conducted. This systematic review aimed at analysing and evaluating the latest 10-year publications to gain understanding on the associations of G6PD with adipose tissue inflammation and obesity.

Methods: The search strategy was performed using Pubmed and Medline databases. The following terms were used to search in titles and abstracts with Boolean operators: “G6PD,” “G-6-PD,” “G6PD deficiency,” “G-6-PD deficiency,” “G6PDD,” “Glucosephosphate dehydrogenase,” “Glucosephosphate dehydrogenase deficiency,” “BMI,” “body-mass-index,” “body weight,” “bodyweight,” “body mass index,” “obesity,” “adiposity,” obes*, adipo*. A filter to limit studies in the last 10 years was used. Articles were last searched on 25 April 2021. Duplicated studies from the two databases were checked.

Results: Two hundred fourteen records were identified, 135 of which were duplications. Sixty-nine records were excluded: 66 lacked association of G6PD expression with obesity or weight regulation, two could not be retrieved, and one was a commentary. Ten publications were included in the analysis with eight, two and one studies involving mice, human, and fish, respectively; four were randomized controlled trials. Reports suggested that G6PD activity or level is increased in the obese, and its expression or overexpression associates with increasing BMI and lipogenesis due to oxidative stress or chronic adipose tissue inflammation; in the same way, G6PD defect or deficiency was linked with insulin resistance amelioration and weight reduction. The role of G6PD in affecting redox balance and lipid accumulation as an NADPH-producing enzyme was found central to these findings. Four of the included studies involved diet inclusion or pharmacotherapeutic interventions with the use of reservatrol and nicotinamide to suppress G6PD activity, which may hence reduce weight.

Conclusion: All studies collectively indicated a positive association of G6PD overexpression with adipose tissue inflammation and obesity, with bidirectional repercussions: G6PD defect or deficiency associate with reduced weight. This may have implications on populations with high prevalence of obesity and/or G6PD deficiency, and in developing therapeutic interventions for obesity.

References

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