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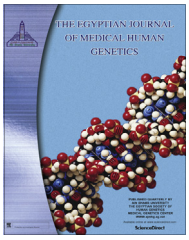

REVIEW

Prevalence of glucose-6-phosphate dehydrogenase deficiency in India: An updated meta-analysis

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KEYWORDS

G6PD deficiency;
Hemolytic anemia;
Meta-analysis;
Prevalence

Abstract *Background:* Glucose-6-phosphate dehydrogenase (G6PD) is a house keeping enzyme which catalyzes the first step in the hexose monophosphate pathway of glucose metabolism. G6PD deficiency is the commonest hemolytic X-linked genetic disease, which affects approximately 400 million people worldwide. The prevalence rate of G6PD deficiency varies worldwide with a higher prevalence in malarial endemic population. In India several studies were published and reported with varying incidences of this disease in different populations.

Objective: The aim of the present study was to assess the overall frequency of G6PD deficiency in the Indian population using meta-analysis.

Methods: PubMed, Science Direct, Google Scholar and Springer Link databases were searched for studies that investigated G6PD deficiency in Indian population. If any author studied different sub-populations we treated the study as an independent study.

Results: A total of 72 studies with a total sample size of 38,565 and 2,623 G6PD deficient subjects were included in the present meta-analysis. Meta-analysis was performed in both fixed and random effect models. Meta-analysis with random model showed an overall prevalence proportion as 0.085 (95% CI = 0.070–0.103; $p = 0.000$; tau = 0.826; $I^2 = 0.486$; Cochran $Q = 0.999$).

Conclusion: In conclusion the present meta-analysis confirms the overall magnitude of the frequency of G6PD deficiency (8.5%) in the Indian population.

Contents

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1. Introduction

Glucose 6-phosphate dehydrogenase (G6PD) deficiency was discovered half a century ago and is still the most common inherited enzymopathy. Clinically, deficiency of this enzyme affects as many as 400 million individuals worldwide [1]. This inherited deficiency causes neonatal hyperbilirubinemia and chronic hemolytic anemia. Although most affected individuals are asymptomatic, exposure to oxidative stresses such as certain drugs or infection, can elicit acute hemolysis. G6PD deficiency was first identified in American blacks (African and Asian descent) in the course of studies of sensitivity to the hemolytic effect of primaquine [2]. Soon after G6PD deficiency was also reported from Mediterranean populations and it became apparent that the enzyme deficiency in the Mediterranean population was much more severe than the prototype deficiency that had been found in American blacks [3].

In 1986, the G6PD gene was cloned independently by Persico et al. [4] and Takizawa et al. [5]. G6PD gene is located on the long arm of the X chromosome (Xq28), and consists of 13 exons [6]. G6PD locus is thought to be one of the most polymorphic loci among humans with almost 300 allelic variants reported [7]. The G6PD enzyme monomer consists of 515 residues with over 59 kDa molecular weight. It was reported that the enzymatically active form of G6PD is either a dimer or tetramer of a single polypeptide subunit according to cellular pH [8].

2. Methods

2.1. Searched strategy and identification of studies

For the present meta-analysis PubMed, Science Direct, Springer link and Google scholar databases were searched for suitable articles using keywords "G6PD deficiency" and "Glucose 6 phosphate dehydrogenase deficiency". Since the retrieved article list was too long only the studies carried out in India were taken into consideration. The included articles were also hand searched for additional studies which can be included in this study.

2.2. Inclusion and exclusion criteria

The inclusion criteria for the studies were as follows: studies: (1) should be original and published, (2) that used only Indian samples. Studies were excluded if they were: (1) molecular analyses, (2) case reports, and (3) reviews and editorials.

2.3. Data extraction

From all the eligible studies the following information was extracted: first authors' family name, year of publication, population/ethnic group, the number of samples analyzed, the number of G6PD deficient subjects and journal name. If in any study samples were taken from multiple caste/race then information was abstracted separately for each caste.

2.4. Statistical analysis

Prevalence proportion (PP) was computed from the number of deficient and sample sizes (N) with the corresponding 95% confidence interval (CI) from each study. A pooled PP was then estimated on the basis of the individual PPs. The PP was estimated either by using fixed effects [9] or random effects [10] model depending upon heterogeneity. The heterogeneity between studies was tested using the Q-statistics and quantified using the I^2 statistic [11]. If $I^2 > 50\%$ then random effect model was used [12]. Publication bias was investigated by using the funnel plots. All p values are two tailed with a significance level at 0.05. All statistical analyses were undertaken by computer program Meta-analyst.

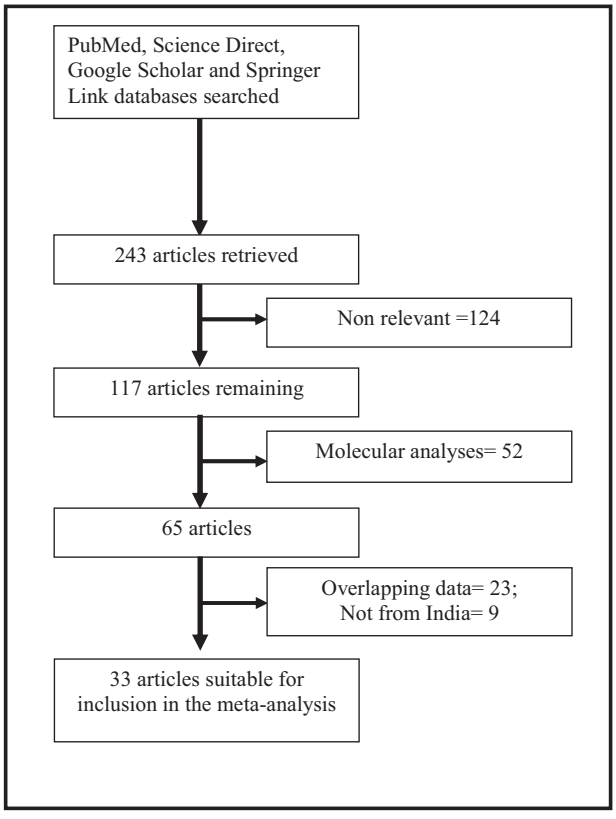

Figure 1 Flow diagram of selection of studies.