Climate change and human health: Malaria and glucose-6-phosphate dehydrogenase deficiency

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Abstract: Global warming has serious consequences on climate as it modifies rainfall patterns, amplifies coastal flooding and erosion, lengthens the growing season, melts glaciers and alters the range of some infectious diseases. As the precipitation patterns change storms floods and droughts will be more severe and an increased frequency of storms, flood, drought and intense rain events have been already observed in all parts of the world in last few decades. More intense rains and hurricanes and rising sea levels will lead to more severe flooding and potential loss of property and life.

Key words: Global warming, Climate change, malaria, G6PD deficiency

Introduction

Global warming is the unusual rapid increase in the earth's average surface temperature over the past century primarily due to the green house gases released as people burn fossil fuels. The rate of temperature increase has nearly doubled in the last five decades (IPCC, 2007) and Intergovernmental Panel on Climate Change (IPCC) predicted that the average global surface temperature could rise 1.4-5.8°C by the year 2100 (McMichaël et al., 2006). The change in temperature has shifted the growing season in different parts of the northern hemisphere. The shift in growing season due to warmer temperature affects the ecosystem. The changes in climate and ecosystems will have a direct affection on the people. As tropical temperature zone expand the reach of some infectious diseases such as malaria, will change (Riebeek, 2010). The abundance and distribution of disease vectors like mosquito is primarily influenced by global warming, and climate change will certainly affect the expansion of these vector populations into new geographical areas. Various publications and mathematical models have predicted how climate change will affect the future transmission and prevalence of malaria in different geographic regions (Reiter et al., 2004). Altitudes that were once safe from mosquitoes will be at risk for epidemics (Cook, 1996). The Fourth Assessment Report of IPCC (2007) has clearly highlighted the possible increase in vector-borne diseases spatially and temporally but the relation between climate and malaria transmission is highly complex and varies according to location (Reiter, 2001).

Diseases Due to Environmental Factor

In developing countries like India there are social, economic and environmental factors which affect occurrence of disease. In India it has been found that northern states like Jammu and Kashmir, Himachal Pradesh, Punjab, Haryana, Uttarakhand and Uttar Pradesh, and these states are also more vulnerable to climate change (Dhiman et al., 2003; Bhattacharya et al., 2006). A study conducted by Bhattacharya et al. (2006) indicates the dominant role of temperature and relative humidity on vector production and malaria transmission in India. Using their transmission window criteria, to link the climate change projections with the likely spatial distribution of malarious regions in India, they found that, malaria is endemic in the central and eastern Indian regions of the country for the current climate. However, it may shift from the central Indian region to the south western coastal states and the northern states, including Himachal Pradesh may become malaria prone in the future climate change regime (Bhattacharya et al., 2006).

Primaquine is used in the treatment of malaria and has been linked to acute hemolysis in Glucose-6-phosphate dehydrogenase deficient individuals. Glucose-6-phosphate dehydrogenase deficiency is characterized by abnormally low level of Glucose-6-phosphate dehydrogenase enzyme in red blood cells. Glucose-6-phosphate dehydrogenase deficiency (EC 1.1.1.49) is a house keeping enzyme that catalyzes the first step in pentose phosphate pathway providing reducing power in the form of NADPH, and helps red blood cells to function normally. G6PD deficiency is the most common human innate error of metabolism, an X-linked genetic disorder (Beutler, 1994). There are approximately 3, 90,000 children born with this disorder every year in India, causing significant morbidity and mortality due to prolonged neonatal jaundice (NNJ) and acute haemolytic anaemia (Nair, 2009). Individuals deficient in G6PD are generally asymptomatic; however, in some cases ingestion of fava beans, exposure to chemicals (eg. naphthalene) and drugs (eg. sulfamides, antipyretics, primaquine and chloroquine) can induce massive intravascular hemolysis. It affects around 400 million people worldwide with a global prevalence of 4.9%. The highest frequencies are reported in malaria endemic areas like in tropical Africa and tropical and subtropical Asia (Cappellini and Fiorelli, 2006).

In India highest frequency (27.94%) has been reported from Vataliya prajapati from Surat, Gujarat (Gupte et al., 2005). If managed inadequately, it can cause death or permanent neurological damage. The most effective management strategy is to prevent haemolysis, by avoiding oxidativé stressors. The high prevalence of G6PD deficiency in malaria-endemic areas implies that many patients that are treated for malaria with an artemisinin-primaquine combination will suffer severely. To prevent this, patients should be tested for G6PD deficiency before treatment with these anti-malaria drugs (Cappellini and Fiorelli, 2008). When severe G6PD deficiency complicates malaria infection, treatment with primaquine can lead to life-threatening acute intravascular hemolysis followed by anaemia and acute renal failure. Glucose-6-phosphate dehydrogenase deficiency screening programmes are already running in the countries like USA, Malaysia, Singapore, Taiwan, Hong Kong, Philippines and in most part of the Europe. The U.S. Army recently...
The aim of the present study was to carry out screening of the Yadav population of eastern Uttar Pradesh for Glucose-6-phosphate dehydrogenase deficiency and to find out the distribution of the Glucose-6-phosphate dehydrogenase enzyme phenotypes and their allele frequencies in OBC. This group practices strict endogamy, which substrat it as separate gene pool in UP Population that extends to the social, occupational, regional and subregional levels and may be considered a genetic isolate. Screening for G6PD deficiency was performed by employing methemoglobin reduction test (Brewer et al., 1962). Ethical clearance was taken from institutional ethics committee of VBS Purvanchal University Jaunpur, for the present study. Informed written consent was taken prior blood sample collection from each subject. 5 ml peripheral blood samples were collected in ACD coated vacutainer from total 200 unrelated randomly selected individuals belonging to Yadav community, which included 107 males and 93 females. After collection, blood samples were carried to Human Molecular Genetics Laboratory, Department of Biotechnology, VBS Purvanchal University, Jaunpur, in ice bucket, where each sample was analyzed for glucose-6-phosphate dehydrogenase deficiency. Collected blood was stored at 4°C until tested. Allele frequencies were calculated by simple gene count method. Out of total 200 samples analyzed, 07 samples were G-6-PD deficient, 11 were carrier and 192 were normal (Table 1). The frequency of G6PD deficient and carrier were 13% and 3.5% respectively. The prevalence rate of G6PD deficiency of our study was similar to that observed for different caste and populations of India in earlier studies (Bhasin, 2006).

In conclusion persons tested positive for G6PD deficiency should be given a card indicating their deficiency status and containing a list of drugs and chemicals to be avoided. Specific G6PD deficiency sensitive training for doctors in all malaria-affected districts of Uttar Pradesh should be offered to raise awareness about this issue. Early detection of the deficiency by screening and avoidance of oxidative stressors is the key strategy to successful management and control of G6PD deficiency (Cappellini and Fiorelli, 2006). Genetic counselling, prenatal diagnosis and health education and public awareness can provide benefits by way of preventive genetics to the affected individuals and their families.

Table 1: Distribution of the glucose-6-phosphate dehydrogenase enzyme deficiency phenotypes and their allele frequencies among OBC samples (sample size=200)

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Heterozygous</th>
<th>Deficient</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males</td>
<td>101</td>
<td>0</td>
<td>6</td>
<td>107</td>
</tr>
<tr>
<td>Females</td>
<td>81</td>
<td>11</td>
<td>1</td>
<td>93</td>
</tr>
<tr>
<td>Total</td>
<td>192</td>
<td>11</td>
<td>07</td>
<td>200</td>
</tr>
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**References**


