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European Journal of Experimental Biology, 2015, 5(6): 29-33

ISSN: 2248 –9215
CODEN (USA): EJEBAU

# Prevalence of *plasmodium vivax* and *plasmodium falciparum* in the city Aurangabad (MS) India: A case study

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**ABSTRACT**

*The present paper deals with incidence of malaria infection due to p vivax and p falciparum at Aurangabad city of Maharashtra. Total 4973 persons suffering from fever and suspected of malaria were examined. The study was carried out during Jun 2010 to Dec 2012. Out of 4973 the p vivax and p falciparum infected persons found to be 36(0.72%) and 18 (0.36%) respectively. The analysis of the data shows that during 2012 more incidences are observed. The maximum percent incidence is observed in the month of oct 2012. The overall incidence varies from 00% to 1.92%. It is also observed that p vivax is the main malaria causing species present in the Aurangabad city.*

**Keywords:** *p falciparum*; malaria incidence; *p vivax*; malaria causing species.

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## INTRODUCTION

The five primary species that infect humans are *P. falciparum*, *P. vivax*, *P. ovale*, *P. malariae* and *P. knowlesi*. Light microscopic examination of thick and thin blood films is considered the gold standard for diagnosis of acute or relapsing disease. This time-honored technique is dependent on the availability of skilled and experienced microscopists, good-quality reagents, and well-maintained microscopes. Malaria rapid diagnostic test (RDTs), which detect Plasmodium antigens (histidine-rich protein [HRP], lactate dehydrogenase or aldolase) have also been developed, and have been shown to be useful in both endemic and nonendemic settings. In general, malaria RDTs perform almost as well as microscopy for *P. falciaprum*, however, they do not fare so well for other species such *P. vivax* or *P. malariae*, especially when PCR is used as a comparator, and false-negatives may result from a prozone phenomenon with high levels of parsitemia or a lack of production of the target antigen (HRP 2 and 3 in certain *P. falciparum* variants). Molecular tests do not suffer from these limitations and therefore offer a viable diagnostic alternative in certain settings. In India , generally only two species are observed that is *p. vivax* and *p falciparum.* Therefore we dicided to study the prevalence of these species in the infectious patients. For this Aurangabad City is selected. Literature survey revealed that no such work is reported earlier for this geographical area.

## MATERIALS AND METHODS

For present work, blood samples of the patients coming with high fever were collected for the examination of malarial parasites. The apparatus used for the present study were sterilized previously. All chemicals used were of anlar grade wherever required distilled water is used Standard procedures are used for the identification of plasmodiums[1-2].

The parasites that cause malaria are found in the blood; part of their development takes place within the red blood cells. Malaria parasites are detected in blood films stained by the JSB or Giemsa stain.

The parasites are usually most numerous in the blood towards the end of an attack of fever. Therefore the blood samples were always collected before antimalarial drugs are given.

A drop of blood from the finger is placed on a slide, spread and dried. During staining of the drop of dried blood, the hemoglobin in the red blood cells dissolves and is washed out by the water in the staining solution. All that remain are the malaria parasites and the white blood cells which are observed under the microscope.

The thick film method makes it possible to find parasites more quickly and if there are only a few present

**Method:**

1. The finger tip to be pricked, was cleaned with spirit and dried with a piece of cotton wool. The finger was pricked at the side of tip with the needle or lancet. The blood is allowed to flow freely. The first drop of blood was discarded. The next drop was collected for examination.
2. Three drops of the blood was applied on the right hand quarter of the slide. With the corner of another clean slide, the blood is spread to an even thickness in a round form of about 1cm diameter this give thick film.
3. In order to prepare thin film of blood, a drop of blood was applied on the middle of the same slide. With the help spreader slide, the drop of blood is allowed to spread. The spreader was quickly pushed from the center to the left side of the slide, drawing the blood behind it. The film is allowed to dry in air. The film was labeled with patients number.
4. When both the thick and thin films are dried, the thin film is immersed only in jar 1 containing methanol and rapidly removed taking care not to let the methanol touch the thick film then dried thoroughly in air. The thick and thin films are dipped in jar 2 containing JSB Solution II for 1 – 2 seconds. Then the slide was dipped twice or thrice in jar 3 containing buffered water to remove excess of eosin stain. Both the thick and thin films are immersed in jar 4 containing JSB Solution 1 for 45 seconds. The slides are dipped three to four times in jar 5 containing buffered water to remove excess of blue stain. It is dried in air on a draining rack (with the side with the smear facing down). The slide is examined. The smear appeared mauve. This will enable a malaria trophozoite to be recognized.
5. (a) The thin film is fixed only by dipping in methanol for 2 – 3 minutes, dried in air. A 1 in 10 dilution of Giemsa stain is made. Mixed gently with a glass rod. The slides are placed across 2 glass rods. Covered them with diluted Giemsa stain. Allow to stand for 30 minutes. The stain was washed off with buffered water. The water is drained off. The slides are placed in a rack to dry, in a sloping position with the stained films facing downwards to protect them from dust in the air.

(b) The slides are picked up with forceps one by one and slotted them into the rack of the staining trough, in a Z pattern. The staining trough is filled with stain slowly and left for 30 minutes out of the sunlight. The cover is remade poured clean water from a beaker into the trough to remove the deposit on the surface of the staining solution. Gently paired off all the staining solution from the trough. The staining trough was filled with buffered water. The slides were taken out one by one, using forceps. Dipped each slide in a beaker of ordinary water, gently, so that the stained preparation does not become unstuck. The slides are drained, placed them in the rack to dry (the slide with the blood film facing downwards).

## RESULTS AND DISCUSSION

For the present investigation total 4973 persons suffering from fever and suspected of malaria were examined. The study was carried out during Jun 2010 to Dec 2012. Out of 4973 the *p vivax* and *p falciparum* infected persons found to be 36(0.72%) and 18 (0.36%) respectively. The Month wise prevalence is given in table 1. The analysis of the data shows that during 2012 more incidences are observed. The maximum percent incidence is observed in the month of oct 2012. The overall incidence varies from 00% to 1.92% These are shown in Fig 1,2 and 3.It is also observed that *p vivax* is the main malaria causing species present in the Aurangabad city.

Globally, *Plasmodium falciparum* is responsible for the majority of uncomplicated febrile illness, as well as severe and fatal malaria and has therefore overshadowed the clinical and public health importance of *vivax* malaria. Despite this, *Plasmodium vivax* is a major cause of morbidity, accounting for almost half of all malaria cases outside of Africa. Each year, there are between 10 and 390 million clinical *vivax* infections.

Because individual clinical episodes of malaria due to *P. vivax* infection were less likely to cause severe illness than *P. falciparum*, *vivax* malaria are historically described as benign *tertian* malaria. However this relative term is misleading since *P. vivax* was and remains responsible for major morbidity and attributable mortality in vivax-endemic areas. The spectrum of disease associated with *P. vivax* infection ranges from asymptomatic parasitaemia and uncomplicated febrile illness through to severe and fatal malaria.[3]

**Table 1: The positive cases of *p. vivax* and *p. falcipurum* during 2010**

| Sr No | Month | 2010 | | | | 2011 | | | | 2012 | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Total Samples collected | pv | pf | total | Samples collected | pv | Pf | Total | Samples collected | pv | pf | Total |
| 1 | Jan | | | | | 107 | 1 | 0 | 1 | 127 | 2 | 1 | 3 |
| 2 | Feb | | | | | 180 | 2 | 1 | 3 | 125 | 2 | 1 | 3 |
| 3 | Mar | | | | | 106 | 0 | 1 | 1 | 199 | 2 | 0 | 2 |
| 4 | Apr | | | | | 124 | 1 | 1 | 2 | 132 | 2 | 0 | 2 |
| 5 | May | | | | | 154 | 1 | 2 | 3 | 140 | 1 | 1 | 2 |
| 6 | Jun | 210 | 1 | 1 | 2 | 223 | 2 | 1 | 3 | 205 | 1 | 0 | 1 |
| 7 | Jul | 198 | 1 | 1 | 2 | 245 | 1 | 2 | 3 | 160 | 1 | 1 | 2 |
| 8 | Aug | 148 | 1 | 0 | 1 | 233 | 2 | 1 | 3 | 155 | 0 | 0 | 0 |
| 9 | Sep | 102 | 1 | 0 | 1 | 248 | 2 | 1 | 3 | 128 | 1 | 1 | 2 |
| 10 | Oct | 123 | 1 | 1 | 2 | 172 | 2 | 0 | 2 | 156 | 2 | 1 | 3 |
| 11 | Nov | 128 | 0 | 0 | 0 | 152 | 2 | 1 | 3 | 175 | 0 | 1 | 1 |
| 12 | Dec | 102 | 1 | 0 | 1 | 132 | 1 | 0 | 1 | 180 | 2 | 1 | 3 |

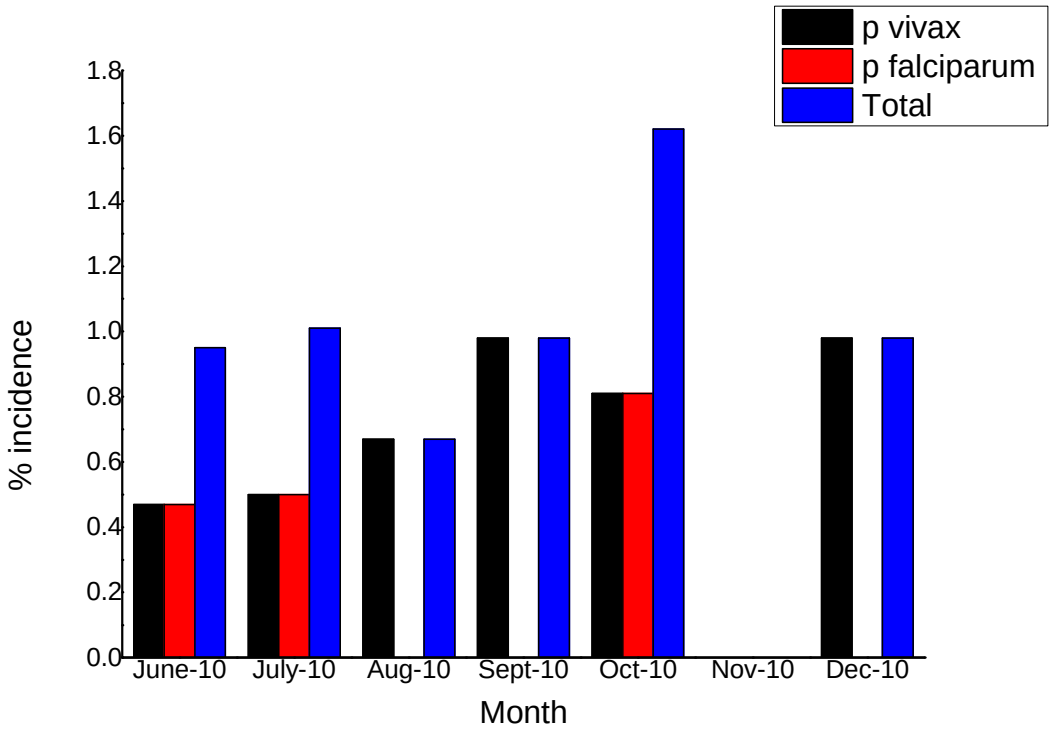


**Fig.1 Percentage incidence of Malarial parasites during the year 2010**

Anti-malarial drugs have an important role in reducing transmission of malaria. However, drug discovery and development of novel agents has focused almost entirely on *Plasmodium falciparum*. Little priority has been given to development of compounds especially for *P. vivax*. These are several reasons for this. First, blood schizontocidal drugs that are active against *P. falciparum* are also active against the blood stages of *P. vivax*. Second, our inability to sustain *P. vivax* in continuous ex vivo culture significantly restricts high-throughput in vitro drug-susceptibility testing. Third, there is no standardized approach to assay anti-malarial activity against hypnozoites. *Plasmodium vivax* has long less regarded as a benign infection and, therefore, has attracted less attention than its more virulent cousin *P. falciparum*. However, recent studies have challenged this paradigm, high-lighting *P. vivax* as a significant cause of morbidity and mortality and socioeconomic disruption. It is clear that the long-standing neglect of this species is no longer tenable.

During the 1920s, *P. vivax* was used extensively for malaria therapy of patients with neurosyphilis. Many of these patients, as well as ‘volunteers’ from penitentiaries, were enrolled in trials of early anti-malarial compounds, if the mosquito subsequently takes another blood meal, these infectious sporozoites will be inoculated into the subcutaneous tissue, thus initiating a new human *P. vivax* infection.[4]

In *P. vivax*, *Plasmodium ovale* and *Plasmodium malariae* infections, a proportion of erythrocytic merozoites will begin replicating sexually to produce male and female offspring immediately after release from the liver. In clinical trials of *P. vivax* therapy, the proportion of patients who are gametocytaemic on enrolment typically varies from 60 to 95%. By contrast, gametocytogenesis in *P. falciparum* infections is delayed by 1 or 2 weeks with respect to the initiation of blood-stage asexual parasite replication. Consequently, *P. vivax*, but not *P. falciparum* infections, are usually transmissible before symptom onset, and thus before anti-malarial treatment is commenced.[5-6]

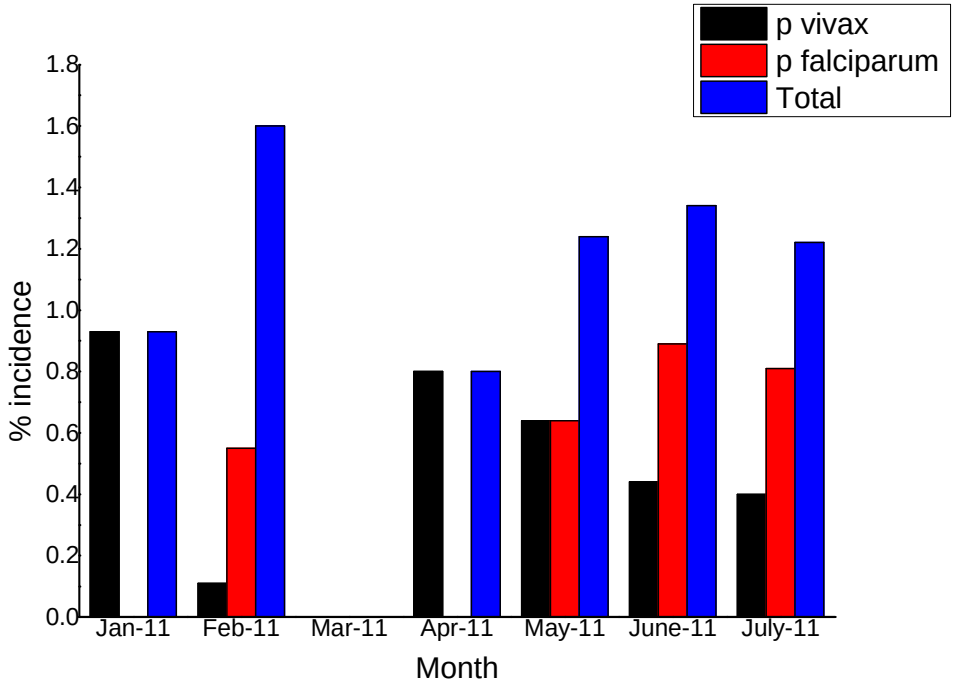


**Fig.2 Percentage incidence of Malarial parasites during the year 2011**

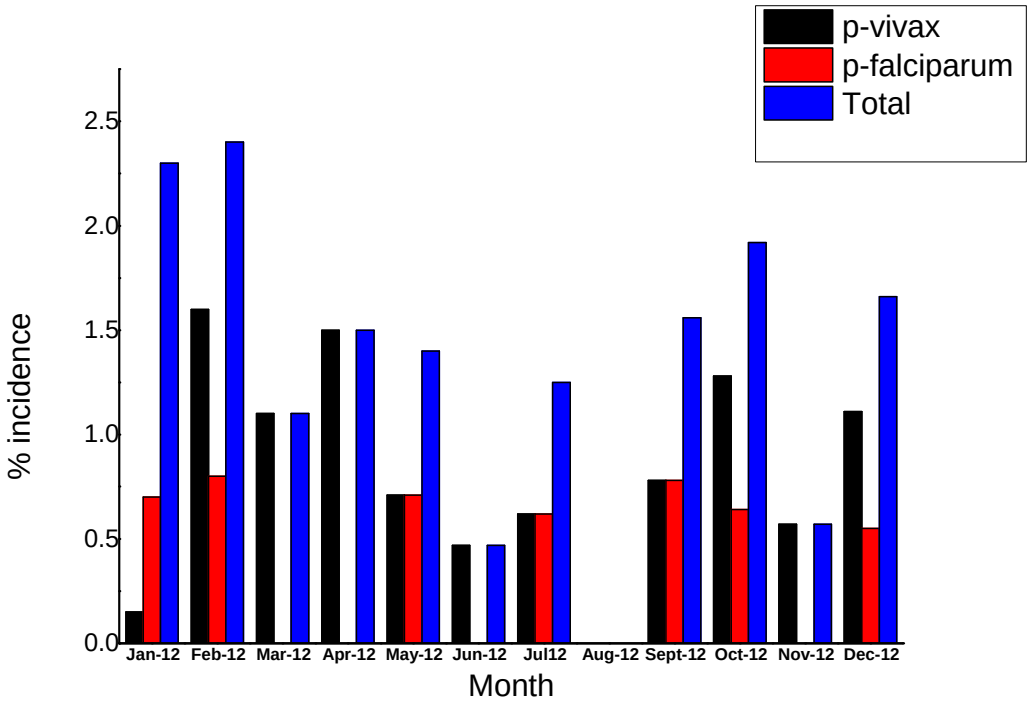


**Fig.3 Percentage incidence of Malarial parasites during the year 2012**

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