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Expression and evaluation of a 24-kDa recombinant protein of the N-terminal E2 glycoprotein of chikungunya virus

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ABSTRACT

Chikungunya is an acute febrile illness caused by chikungunya virus (CHIKV). In this study, a short 24-kDa N-terminal of E2 glycoprotein of chikungunya virus was cloned and expressed in *E.coli* expression system. The E2 recombinant protein was expressed as a fusion protein to 6-Histidine for ease of purification. The expression of the 24-kDa recombinant protein was detected by SDS-PAGE and the protein reactivity was evaluated by western blot analysis. The immunogenicity of the 24 kDa protein was further tested against human positive and negative sera for chikungunya and dengue. The results showed that the recombinant antigen was able to detect CHIKV positive sera and no cross reactivity was observed with dengue virus positive serum. © 2015 Trade Science Inc. - INDIA

KEYWORDS

Chikungunya virus; Glycoprotein; Recombinant antigen; Immunoblot assay; E.coli expression system.

INTRODUCTION

Chikungunya fever is an acute illness caused by chikungunya virus (CHIKV), an alphavirus of the family *Togaviridae*. CHIKV is transmitted to humans by mosquitoes of the genus *Aedes*, particularly *Aedes aegypti* and *Aedes albopictus*. The hallmark of CHIKV infection is a long lasting polyalthralgia, which may persist for months or even years^[1]. Though generally a non-fatal condition, CHIKV infections may rarely be associated with complications such as encephalopathy and hepatic failure^[2] and occasional deaths have been reported over the last decade^[3]. The clinical illness is often associated with prolonged morbidity, which

can impose enormous social and economic disadvantages on affected communities^[4]. The first formal description of the disease was during an outbreak of chikungunya fever in 1952 in Tanzania^[5] and the subsequent isolation of CHIKV^[6]. The first outbreak in Asia was documented in Bangkok, Thailand in 1958 and since then, outbreaks have been reported in Cambodia, Vietnam, Laos, Myanmar, Malaysia, Singapore, the Philippines, and Indonesia^[7]. Malaysia reported its first outbreak between December 1998 and February 1999^[8]. There are three distinct lineages of CHIKV, a West African, an East Central and South African (ECSA) and an Asian lineage^[9]. In 2004-2005, CHIKV of the ECSA lineage caused massive outbreaks in the