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Expression of Recombinant E1 Glycoprotein of Chikungunya Virus in Baculovirus Expression System

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HISTORY	ABSTRACT
Received: 10 th July 2015 Received in revised form: 21st of June 2015 Accepted: 15th of September 2015	Chikungunya is an acute febrile illness caused by chikungunya virus (CHIKV). In this study, the envelope E1 gene of CHIKV was cloned and expressed in a baculovirus system. The recombinant E1 protein with N-term 6-His residues protein was successfully expressed and
KEYWORDS	purified as confirmed by SDS-PAGE and western blot analysis. The seroreactivity of the recombinant protein was evaluated in immunoassay for anti-CHIKV IgM and IgG antibodies.
Chikungunya virus E1 glycoprotein baculovirus system anti-CHIKV IgM immunoassay	The recombinant antigen showed 69% sensitivity and 100% specificity for anti-CHIKV IgG by dot blot assay. Detection of anti-CHIKV IgM by dot assay showed 79% sensitivity and 100% specificity. No cross reactivity of the antigen was observed with anti-dengue virus serum samples. The results strongly support that the recombinant E1 protein has potential to be used as diagnostic antigen. The used of the antigen in a dot blot assay gives an advantage for laboratory detection without the need of any specialised equipment.

INTRODUCTION

Chikungunya virus (CHIKV) is an arthropod-borne virus of the Alphavirus genus and the family Togaviridae. It is transmitted to humans by Aedes mosquitoes and the infection is often characterized by sudden onset of fever, skin rash and arthralgia that could persist for several months. In outbreaks that are more recent however, CHIKV was also associated with severe neurological disease and even fatalities [1]. Although was first isolated in Tanzania in 1953 [2], CHIKV infection has only received considerable attention in recent years with major outbreaks in the Indian Ocean region, India, Southeast Asia and especially with the explosive epidemics in Reunion Island from 2005-2006 [3]. The re-emergence of the CHIKV since then had not only confined to the Asian region but had continued to spread to the Western world including a first outbreak in Europe reported in 2007 in Italy [4] and autochthonous transmission events in France in 2010 [5]. Many factors had influent the geographical spread of CHIKV, including vector distribution, human travel, urbanization and climatic changes. The introduction of the new strain from the epidemic on the Reunion Islands with the mutation at codon 226 of the envelope protein E1 from alanine to valine, too had contributed to the unprecedented worldwide spread of CHIKV [6]. The CHIKV

genome consists of a linear, single-stranded positive sense RNA of approximately 11.8 kb. The structural genes encode three structural proteins, E1 and E2 of the envelope and the nucleocapsid protein [7]. The envelope proteins formed triplets of heterodimer of E1 and E2 glycoproteins, which cover the viral surface in the form of membrane-anchored spikes. The viral spike proteins facilitate attachment to cell surfaces and viral entry into the cells [8]. E1 contains more conserved cross-reactive epitopes whereas E2 is the site of neutralizing epitopes [9].

Currently there is no effective antiviral treatment for CHIKV infection. Therefore, reliable laboratory confirmation of CHIKV infection is important for timely vector control. Laboratory conformation is even more critical in dengue endemic areas, as the clinical management is different for these infections even though their manifestations can be similar. Currently the gold standards of CHIKV diagnosis are virus culture, and molecular detection using reverse transcriptase polymerase chain reaction (RT-PCR) [10]. Both methods require specialised equipment, facilities and skills, which are limited and can be too costly for developing countries. Diagnoses are also done by detecting the present of CHIKV specific antibodies in patients by an enzyme immunoassay or