Original Article

Double Dengue Serotypes in Asymptomatic Populations Living in An Area of Thailand Endemic for Dengue Hemorrhagic Fever

Veerayuth Kittichai[1] Methee Sriprapun[2] Papawin Konklong[3] Nithita Thonsangin[3]

Jakkrawarn Chompoosri[4] Apiwat Tawatsin[4] Usavadee Thavara[4] Padet Siriyasatien[5,6*]

Abstract

Dengue virus infection remains a major health problem worldwide. Understanding dengue infection and characterizing circulating viruses are essential for disease prevention and control as well as vaccine development. In this study, we aimed to identify dengue virus in healthy people living in an area endemic for dengue disease. Blood samples were collected from 52 healthy local subjects living in a dengue-endemic area of Thailand. Viral RNA was detected using a nested reverse transcription polymerase chain (RT-PCR) that amplified the E gene. Phylogenetic trees were constructed by the neighbor-joining method using MEGA6.06. Dengue virus was detected in 5 of 52 samples (9.62%). Double dengue virus infection (Den 1&4 and Den 3&4) was found in two samples. Sequence analysis of the viruses showed that dengue serotype 1 belonged to sylvatic genotype. A mix of genotype I and II was found in a dengue serotype 3 sample while all dengue virus serotype 4 in this study belonged to genotype II. These preliminary results may provide better understanding of dengue infection and viral transmission between populations and mosquitoes. Therefore, it could be used for epidemiological studies and control of dengue hemorrhagic fever in the future.

Keywords: *Aedes aegypti*, asymptomatic infection, double dengue virus infection, endemic, phylogenetic tree

[1]*Medical Science Program, Faculty of Medicine, Chulalongkorn University, Bangkok 10300, Thailand*

[2]*Department of Clinical Microbiology, Faculty of Medical Technology, Huachiew Chalermprakiet University, Samut Prakan, 10540, Thailand*

[3]*Faculty of Medical Technology, Huachiew Chalermprakiet University, Samut Prakan 10540, Thailand*

[4]*National Institute of Health, Department of Medical Sciences, Ministry of Public Health, Nonthaburi 11000, Thailand*

[5]*Department of Parasitology, Faculty of Medicine, Chulalongkorn University, Bangkok 10330, Thailand*

[6]*Excellence Center for Emerging Infectious Disease, King Chulalongkorn Memorial Hospital, Thai Red Cross Society, Bangkok 10330, Thailand*

***Correspondence**: padet.s@chula.ac.th*

Introduction

Dengue viruses (DENV-1 to DENV-4) are transmitted to human hosts through bites of infected female *Aedes aegypti* and *Ae. albopictus* mosquitoes (Thavara et al., 2006). The infection can cause dengue fever (DF), dengue hemorrhagic fever (DHF), or dengue shock syndrome (DSS) (WHO, 2009). The disease is problematic worldwide, especially in tropical and subtropical areas. Of 390 million infected patients reported annually, 50-100 million dengue cases (10-15%) are symptomatic, approximately 500,000 develop DHF (Guzman et al., 2010), and up to 300 million are asymptomatic or mild cases (Bhatt et al., 2013). However, the relationship between asymptomatic and symptomatic infections and the role asymptomatic infection play in disease transmission remain unknown.

Thailand often experiences dengue outbreaks and epidemiological data report high annual levels of DHF and DSS. Based on historical dengue incidence data during 2007 and 2012 obtained from the Department of Disease Control in Thailand's Ministry of Public Health, provinces in the central region such as Samut Sakhon are at high risk of dengue outbreaks (Saungtho et al., 2013). According to cumulative provincial surveillance data, the 2012 incidence rate of DHF in Samut Sakhon was 252.26/100000, making it a severe epidemic year for dengue disease (90^{th} percentile) (Thavara et al., 2006; and Chompoosri et al., 2012). Several factors such as environmental conditions and human migration, particularly of foreign workers, contribute to increased dengue transmission (WHO, 2009). A previous study identified dengue infection by detecting immunoglobulin M and G (IgM and IgG, respectively) antibodies and an IgG and IgM immune response in adult residents of endemic areas (Barde et al., 2012). Various sample types, including white blood cells (Wang et al., 2000), platelets (Noisakran et al., 2009), saliva and urine (Ma et al., 2014), can be used to test for infection. As mentioned previously, the role of asymptomatic dengue infection in disease transmission and severity have not been fully evaluated; therefore, this study tested for presence of the virus in local adult volunteers with no reported dengue signs, symptoms, or diagnoses. These findings could provide a better understanding of dengue pathogenesis and the relationship between the virus and its hosts in the mosquito-human-mosquito cycle within endemic areas. This information could be of interest to epidemiological studies and inform development of effective dengue control measures.

Materials and Methods

Sample collection: Adults between 20-60 years of age living in Ban Phaeo district in Samut Sakhon province with no history of dengue infection in the previous year were enrolled in the study. Blood samples were collected from individuals who had no clinical signs of dengue for at least 1 year before the study. Enrolled subjects signed consent forms for their participation in the study. The study was approved by the Ethic Committee of Research Affairs Unit, Faculty of Medicine, Chulalongkorn University (COA No. 328/2014, IRB No. 182/2014). Three milliliters of K_2EDTA-blood samples were collected from each subject. The blood samples were kept at 4^oC and transferred to the laboratory on the same day. Plasma was separated by centrifugation at 5,000 rpm for 10 min at 4^oC.

RNA extraction: The plasma samples were extracted for total RNA using an Invisorb® Spin Virus RNA Mini Kit (STRATEC Molecular GmbH, Birkenfeld, Germany) according to the manufacturer's instructions. RNA quality and concentration were determined using a NanoDrop™ 2000c Spectrophotometer (Thermo Fisher Scientific Inc., Massachusetts, USA). The extracted RNA samples were stored at -80^oC until further processing.

Amplification and sequencing of dengue virus envelope (E) gene fragment: A region of the dengue virus E gene was targeted for amplification in a nested reverse transcription polymerase chain reaction (RT-PCR). The nested RT-PCR was performed using type-specific oligonucleotide primers described previously (Yenchitsomanus et al., 1996). This technique was used to screen for dengue virus infection in the study samples. PCR was performed using One-Step RT-PCR Kit (Qiagen, Düsseldorf, Germany) and BIOTAQ™ PCR Kit (Qiagen, Düsseldorf, Germany), respectively, according to manufacturers' directions. Amplification was carried out on a Mastercycler® pro thermalcycler (Eppendorf, New York, USA) for cDNA synthesis and nested PCR that produced 641 bp and 434 bp products, respectively. The nested PCR was performed with 1 µL of 100-fold dilution (with ddH_2O) from the first reaction as template DNA. Samples positive for dengue in the nested reaction were then subjected to PCR with serotype-specific primers (Yenchitsomanus et al., 1996) to determine virus serotypes. The template DNA for this PCR was obtained by diluting the first-round PCR products (641 bp) as described previously. The dengue-specific primer sets produced 504, 346, 198, and 143 bp amplicons for serotypes DENV 1-4, respectively. The following positive controls and template concentrations were used: Hawaii strain (DENV-1), 5×10^5 PFU/mL; 16681 strain (DENV-2), 4.75×10^6 PFU/mL; H87 strain (DENV-3), 2.75×10^5 PFU/mL; and 814609 strain (DENV-4), 2.5×10^5 PFU/mL.

The amplified PCR products were then ligated into pGEM®-T Easy Vector Systems (Promega, Wisconsin, USA) and plasmids transformed into *Escherichia coli* (DH5α). The plasmids were extracted using the Invisorb Spin Plasmid Mini Two kit (STRATEC Molecular GmbH, Birkenfeld, Germany) according to manufacturers' directions. Plasmid concentration and purity were determined with a NanoDrop™ 2000c Spectrophotometer (Thermo Fisher Scientific Inc., Massachusetts, USA) before sequencing (ATI, Singapore) based on M13 primer.

Sequences were aligned using ClustalW (Bioedit v2.0.2). The aligned sequences matched, with the sequence histograms of the amplified region showing clear individual base peaks. The sequences were then compared with exiting reference nucleotides on the NCBI online webserver (http://blast.ncbi.nlm.nih.gov/Blast.cgi). The FASTA

sequences were deposited in GenBank and assigned accession numbers. Descriptive statistics, including percentage match to reference dengue sequences, were then calculated.

Phylogenetic tree construction: A phylogenetic tree was constructed using partial dengue virus E gene sequences by the neighbor-joining (NJ) method using Kimura's 2-parameter model and bootstrap with 1,000 replications in MEGA version 6.06. Sequences with GenBank accession numbers D00502 (DENV-1), JF967989 (DENV-2), JF968088 (DENV-3), and U18429 (DENV-4) were used as out-groups.

Results

A total of 52 subjects, including 15 males and 37 females between 26 and 60 years of age, enrolled in the study. Of the 52 samples, 5 (9.62%) were positive for dengue infection (Fig 1A). All dengue-positive samples were serotyped using techniques described previously. Samples S27 and S36 had double infection with DENV-1 (504 bp) and DENV-4 (143 bp), and DENV-3 (198 bp) and DENV-4 (143 bp), respectively (Fig 1B, Table 1). Single DENV-4 infection was found in samples S34, S43, and S51. The nucleotide sequences were deposited in GenBank with the following accession numbers: KM003963 (DENV-1) and KM003995-KM003998 and KM004003 (DENV-4). Since the DENV-3 nucleotide sequences were less than 200 bp in length, they were not accepted for deposit in the NCBI system.

For the analysis of DENV-1 and DENV-4 serotypes, evolutionary relationships based on the phylogenetic tree showed that the virus belonged to the sylvatic genotype (Fig 2) and genotype II (Fig 4), respectively. Interestingly, the DENV-3 tree showed that the virus belonged to genotypes I and II (Fig 3).

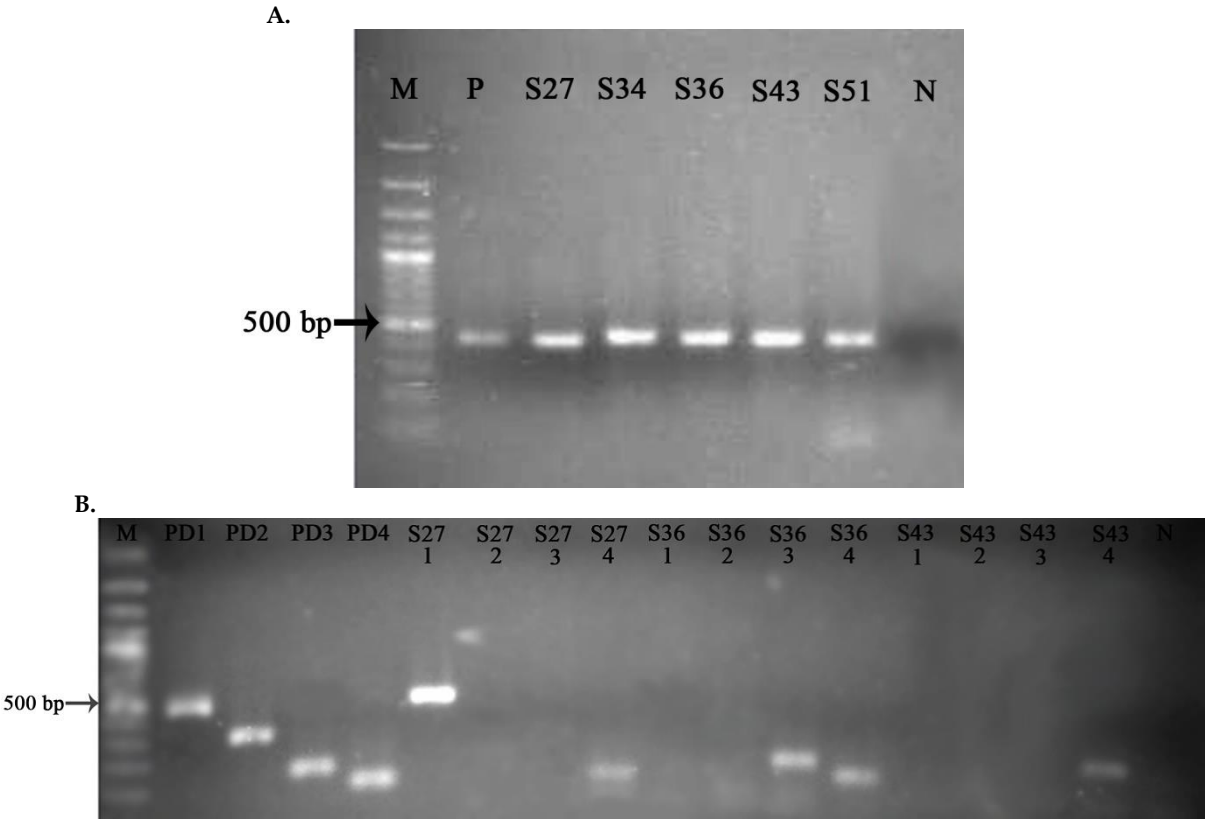

Figure 1 Dengue virus detection by nested RT-PCR amplification of a region of the E-gene. A) Plasma samples positive for dengue virus. M: 100 bp ladder, P: positive, S27: sample 27, S34: sample 34, S36: sample 36, S43: sample 43, S51: sample 51, N: negative control. B) Typing of dengue viruses from plasma samples. M: ladder, PD1-4: positive for DENV1-4 respectively, S27-1 to 4: typing of DENV1-4 respectively in sample 27, S36-1 to 4: typing of DENV1-4 respectively in sample 36, S43-1 to 4: typing of DENV1-4 respectively in sample 43, N: negative control.

Discussion

Dengue viruses were detected in plasma of individuals based on the nested RT-PCR, which amplified part of the E gene, with reported high detection sensitivity of 0.1-1.0 PFU/ mL (Yenchitsomanus et al., 1996). DENV-1, DENV-3, and DENV-4 infections with both single and double infections were identified from the plasma samples collected in the present study. To our knowledge, this is the first report of DENV-1 & 4 and DENV-3 & 4 co-infections in asymptomatic individuals in Thailand (Figure 1B, Table 1). Recently, a DENV-1 and 2 co-infection has been described in a Belgian resident returning from Thailand (Cnops et al., 2014). It is possible for humans to acquire dengue infections via multiple bites from vector mosquitoes carrying single or multiple viruses (Thavara et al., 2006). Additionally, co-infection probably depends on host immune status which affects viral replication (Steinhauer and

Holland, 1987). However, the subjects with co-infections in this study did not show clinical evidence of dengue infection. Some survey studies reported similar infection incidence rates between asymptomatic and symptomatic children in primary schools (Endy et al., 2002; Yoon et al., 2012). The reports also suggested that asymptomatic dengue infection could play an important role in dengue virus transmission. Longitudinal studies found symptomatic and asymptomatic dengue infection prevalence of 18 and 56 cases per 1,000 person-years, respectively, in adult residents of the endemic area of Bandung, in west Java, Indonesia (Porter et al., 2005). However, co-infection in patients with acute-phase illness during outbreaks was reported (Figueiredo et al., 2011; Colombo et al., 2013). Previous studies reported that the virus could be detected in white blood cell (Wang et al., 2000), platelets (Noisakran et al., 2009), saliva, and urine (Ma et al., 2014) samples collected from patients during both acute and convalescent phases. However, no studies have suggested that these viruses are attenuated or virulent strains. This question should be addressed in further research. Moreover, patients in the convalescence phase of the disease could test positive for dengue by RT-PCR due to detection of lingering antibodies to the virus (Figueiredo et al., 2011). We ensured that the results of nested RT-PCR were not false positive findings through the pre- and post-analytical testing, which was performed based on standard PCR criteria to minimize false negative findings (Kwok and Higuchi, 1989). The samples with multiple infections were tested in triplicate with re-extracted template material. Long-term storage could degrade RNA or the sample itself might contain virus concentrations lower than the threshold for detection. High sensitivity methods such as the nested RT-PCR could be useful for detecting the virus in these cases. Therefore, long-term asymptomatic infection may be a viral adaption or its way of escaping the host's immune response.

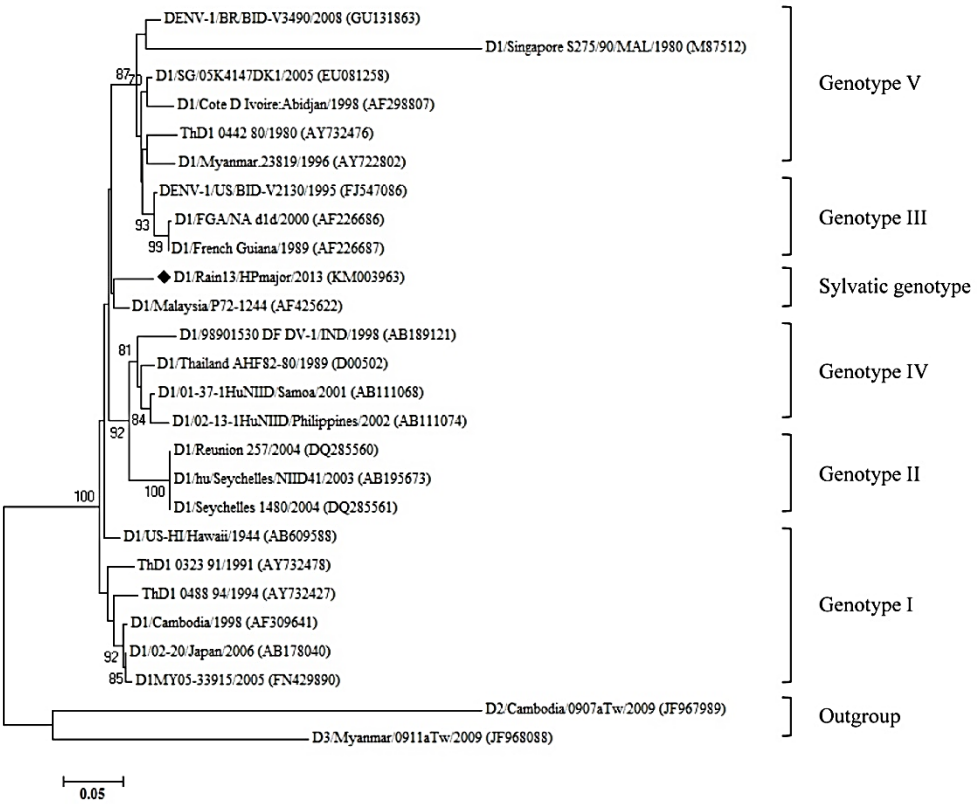

Figure 2 A phylogenetic tree of DENV-1 based on partial E gene sequences. The tree was constructed using 26 DENV-1 sequences, including 25 references and one sequence from studied participants (black solid diamond).

It is possible that some local residents were infected with homologous or heterologous viruses via single or multiple bites from vector mosquitoes (Thavara et al., 2006). Evidence of more than one dengue virus strain was reported in mosquito vectors collected from Thailand (Thavara et al., 2006). These situations might lead to persistent infections that may develop into secondary infections when exposed to heterologous dengue infection (Bhatt et al., 2013).

The phylogenetic tree analysis revealed sylvatic genotypes. These genotypes have been circulating in the local populations and represent an attenuated strain (Rico-Hesse, 2003). The strain is part of an enzootic cycle and might be transmitted to local humans through a bite from an infected non-local mosquito (Wang et al., 2000). This genotype might also be introduced to the area via a local mosquito transmitting the virus from an infected 'working monkey' (used to pick coconuts) or foreign workers such as Burmese laborers who commonly work in the local industry. Although there is no evidence for sylvatic DENV emergence into human transmission, a newly arrived sylvatic DENV strain could be carried by non-human primates (NHP) and local competent

vectors and spread a new cycle of transmission (Hanley et al, 2013). However, Vasilakis and Weaver (2008) stated that the sylvatic DENV emergence was found in Asia and West Africa and might remain a potential source of re-emergence since the mosquito vector and NHP were not responsive to interventions. Two DENV-3 genotypes might represent viral diversification and contribute to virus survival (Wang et al, 2000; Lin et al, 2004).

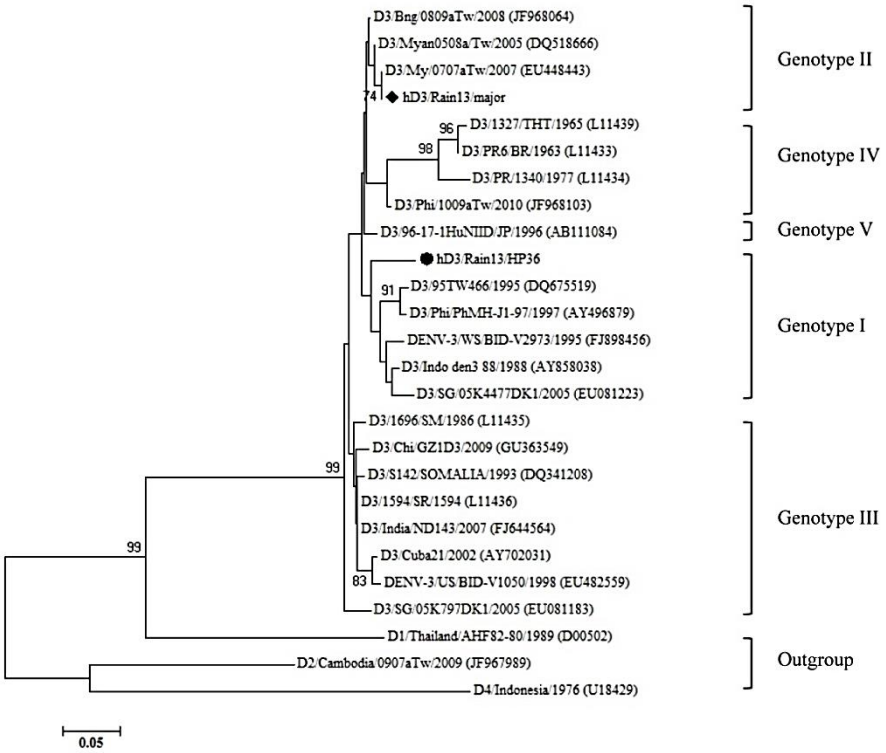

Figure 3 A phylogenetic tree of DENV-3 based on representative partial sequences of the dengue virus E gene. The tree was constructed with 36 DENV-3 sequences including 24 reference sequences and two DENV-3 sample sequences (black solid diamond and black solid circle), which belonged to genotype I and genotype II, respectively.

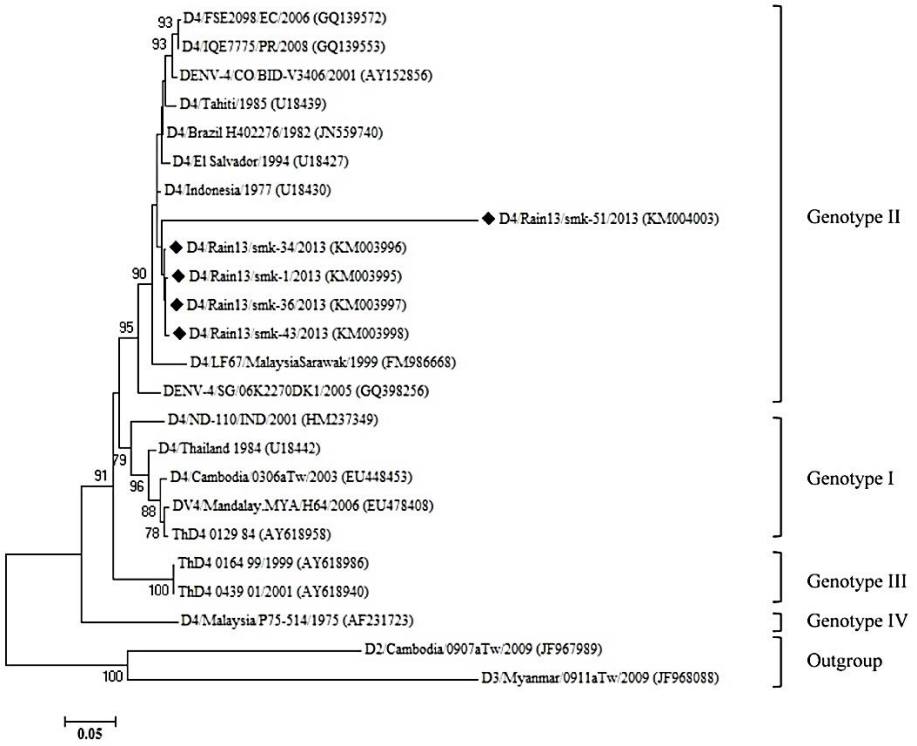

Figure 4 A phylogenetic tree of DENV-4 based on partial E gene sequences. The tree was constructed using 24 DENV-4 sequences, including 19 reference sequences and five DENV-4 sample sequences (black solid diamonds).

In summary, we identified asymptomatic dengue virus infection with both single and co-infections in a highly endemic area of Thailand. The study of subclinical infections could provide better understanding of disease pathogenesis in the human host. Moreover, basic knowledge of co-infection and concurrent transmission in endemic areas are key factors for epidemiological design of effective control programs. Additionally, data on variations in viral sequences could be used to support future development of diagnostic and treatment approaches as well as vaccine design.

Table 1 Results of RT- and nested RT-PCR amplification of the dengue virus E gene amplification from plasma samples. The samples were collected from local asymptomatic populations without history of dengue infection during the study period.

No. (Samples)	Sex	Age	E gene (Typing)	Accession numbers
1 (S27)	Female	59	DENV-1	KM003963
			DENV-4	KM003995
2 (S34)	Female	59	DENV-4	KM003996
3 (S36)	Female	60	DENV-3	-
			DENV-4	KM003997
4 (S43)	Female	45	DENV-4	KM003998
5 (S51)	Female	27	DENV-4	KM004003

Acknowledgements

This study was supported by His Majesty King Bhumibhol Adulyadej's 72nd Birthday Anniversary Scholarship, Graduate School, Chulalongkorn University; the Ratchadapisek Sompoch Fund, Faculty of Medicine, Chulalongkorn University (RA 57/099); and National Science and Technology Development Agency (Thailand) for a Research Chair Grant.

References

Barde PV, Godbole S, Bharti PK, Chand G, Agarwal M and Singh N 2012. Detection of dengue virus 4 from central India. Indian J Med Res. 136: 491-494.

Bhatt S, Gething PW, Brady OJ, Messina JP, Farlow AW, Moyes CL, Drake JM, Brownstein JS, Hoen AG, Sankoh O, Myers MF, George DB, Jaenisch T, Wint GR, Simmons CP, Scott TW, Farrar JJ and Hay SI 2013. The global distribution and burden of dengue. Nature**.** 496: 504-507.

Chompoosri J, Thavara U, Tawatsin A, Anantapreecha S and Siriyasatien P 2012. Seasonal monitoring of dengue infection in *Aedes aegypti* and serological feature of patients with suspected dengue in 4 central provinces of Thailand. Thai J Vet Med. 42:185-193.

Cnops L, Domingo C, Van den Bossche D, Vekens E, Brigou E and Van Esbroeck M 2014. First dengue co-infection in a Belgian traveler returning from Thailand, July 2013. J Clin Virol. 61: 597-599.

Colombo TE, Vedovello D, Mondini A, Reis AFN, Cury AAF, Oliveira FHD, Cruz LEAA, Bronzoni RVdM and Nogueira ML 2013. Co-infection of dengue virus by serotypes 1and 4 in patient from medium sized city from Brazil. Rev Inst Med Trop Sao Paulo. 55: 275-281.

Endy TP, Chunsuttiwat S, Nisalak A, Libraty DH, Green S, Rothman AL, Vaughn DW and Ennis FA 2002. Epidemiology of inapparent and symptomatic acute dengue virus infection: a prospective study of primary school children in Kamphaeng Phet, Thailand. Am J Epidemiol. 156: 40-51.

Figueiredo RM, Naveca FG, Oliveira CM, Bastos MdeS, Mourão MP, Viana SdeS, Melo MdoN, Itapirema EF, Saatkamp CJ and Farias IP 2011. Co-infection of Dengue virus by serotypes 3 and 4 in patients from Amazonas, Brazil. Rev Inst Med Trop Sao Paulo. 53(6): 321-323.

Guzman MG, Halstead SB, Artsob H, Buchy P, Farrar J, Gubler DJ, Hunsperger E, Kroeger A, Margolis HS, Martínez E, Nathan MB, Pelegrino JL, Simmons C, Yoksan S and Peeling RW 2010. Dengue: a continuing global threat. Nat Rev Microbiol. 12: 7-16.

Hanley KA, Monath TP, Weaver SC, Rossi SR, Richman RL and Vasilakis N 2013. Fever versus Fever: the role of host and vector susceptibility and interspecific competition in shaping the current and future distributions of the sylvatic cycles of dengue virus and yellow fever virus. Infect Genet Evol. 19: 292-311.

Kwok S and Higuchi R 1989. Avoiding false positives with PCR. Nature. 339: 237-238.

Lin SR, Hsieh SC, Yueh YY, Lin TH, Chao DY, Chen WJ, King CC and Wang WK 2004. Study of Sequence Variation of Dengue Type 3 Virus in Naturally Infected Mosquitoes and Human Hosts: Implications for Transmission and Evolution. J Virol. 78: 12717–12721.

Ma X, Zhen W, Yang P, Sun X, Nie W, Zhang L, Xu H and Hu K 2014. First confirmation of imported dengue virus serotype 2 complete genome in urine from a Chinese traveler returning from India. J Virol. 11: 56.

Noisakran S, Gibbons RV, Songprakhon P, Jairungsri A, Ajariyakhajorn C, Nisalak A, Jarman RG, Malasit P, Chokephaibulkit K and Perng GC 2009.

Detection of dengue virus in platelets isolated from dengue patients. Southeast Asian J Trop Med Public Health. 40: 253-262.

Porter KR, Beckett CG, Kosasih H, Tan RI, Alisjahbana B, Rudiman PI, Widjaja S, Listiyaningsih E, Ma'Roef CN, McArdle JL, Parwati I, Sudjana P, Jusuf H, Yuwono D and Wuryadi S 2005. Epidemiology of dengue and dengue hemorrhagic fever in a cohort of adults living in Bandung, West Java, Indonesia. Am J Trop Med Hyg. 72: 60-66.

Rico-Hesse R 2003. Microevolution and virulence of dengue viruses. Adv Virus Res. 59: 315-341

Steinhauer DA and Holland JJ 1987. Rapid Evolution of RNA Viruses. Annu Rev Microbiol. 41: 409-433.

Suangtho P, Kaewnorkkao W and Sayumpurujinun S 2013. 506 Surveillance weekly summarized reports of dengue virus infection during 1 January to 18 April 2013. Bureau of Epidemiology, Department of Disease Control, Ministry of Public Health, Thailand. Available: http://203.157.15.4/wesr/file/y56/F56151_1353.pdf. Accessed May 25, 2014.

Thavara U, Siriyasatien P, Tawatsin A, Asavadachanukorn P, Anantapreecha S, Wongwanich R and Mulla MS 2006. Double infection of heteroserotypes of dengue viruses in field populations of *Aedes aegypti* and *Aedes albopictus* (Diptera: Culicidae) and serological features of dengue viruses found in patients in southern Thailand. Southeast Asian J Trop Med Public Health. 37: 468-476.

Vasilakis N and Weaver SC 2008. The history and evolution of human dengue emergence. Adv Virus Res. 72: 1-76.

Wang HL, Lin KH, Yueh YY, Chow L, Wu YC, Chen HY, Sheu MM and Chen WJ 2000. Efficient diagnosis of dengue infections using patients' peripheral blood leukocytes and serum/plasma. Intervirology. 43: 107-111.

Wang E, Ni H, Xu R, Barrett AD, Watowich SJ, Gubler DJ and Weaver SC 2000. Evolutionary relationships of endemic/epidemic and sylvatic dengue viruses. J Virol. 74: 3227-3234.

World Health Organization, 2009. Dengue: Guidelines for diagnosis, treatment, prevention and control. New ed. Geneva.

Yenchitsomanus PT, Sricharoen P, Jaruthasana I, Pattanakitsakul SN, Nitayaphan S, Mongkolsapaya J and Malasit P 1996. Rapid detection and identification of dengue viruses by polymerase chain reaction (PCR). Southeast Asian J Trop Med Public Health. 27: 228-236.

Yoon IK, Rothman AL, Tannitisupawong D, Srikiatkhachorn A, Jarman RG, Aldstadt J, Ananda N, Mammen MP, Thammapalo S, Green S, Libraty DH, Gibbons RV, Getis A, Endy T, Jones JW, Koenraadt CJM, Morrison AC, Fansiri T, Pimgate C and Scott TW 2012. Underrecognized Mildly Symptomatic Viremic Dengue Virus Infections in Rural Thai Schools and Villages. J Infect Dis. 206(3): 389–398.

บทคัดย่อ

การติดเชื้อไวรัสเดงกีร่วมในผู้ที่ไม่แสดงอาการของโรคที่อาศัยอยู่ในพื้นที่ระบาดของโรคไข้เลือดออกของประเทศไทย

วีรยุทธ กิตติชัย[1] เมธี ศรีประพันธ์[2] ปภาวิน คนคล่อง[3] นิธิตา โทนสังข์อินทร์[3] จักรวาล ชมภูศรี[4] อภิวัฏ ธวัชสิน[4] อุษาวดี ถาวระ[4] เผด็จ สิริยะเสถียร[5,6]*

การติดเชื้อไวรัสเดงกียังถือเป็นปัญหาสาธารณสุขหลักที่สำคัญของโลก ความเข้าใจลักษณะการติดเชื้อและการระบุไวรัสที่ยังคงในอยู่สิ่งแวดล้อม ณ ขณะนั้นถือว่ามีความจำเป็นสำหรับการวางแผนควบคุมและป้องกันการติดเชื้อตลอดจนการพัฒนาวัคซีน สำหรับการศึกษานี้คณะผู้วิจัยมีจุดประสงค์ที่จะศึกษาการติดเชื้อไวรัสเดงกีในผู้ที่ไม่แสดงอาการที่อาศัยในพื้นที่ระบาดของโรคจากอาสาสมัครทั้งหมด 52 คน โดยการตรวจการติดเชื้อนี้อาศัยวิธีเพิ่มปริมาณยีนห่อหุ้มด้วยปฏิกิริยาลูกโซ่โพลีเมอร์เรสแบบ nested reverse transcription และศึกษาวิวัฒนาการของเชื้อไวรัสเดงกีด้วยวิธี neighbor-joining ด้วยโปรแกรม MEGA6.06 จากการศึกษาพบว่าอาสาสมัครติดเชื้อ 5 คน คิดเป็นร้อยละ 9.62 และสามารถตรวจพบการติดเชื้อร่วมสายพันธุ์ของเชื้อไวรัส (ซีโรทัยป์1-4 และ ซีโรทัยป์ 3-4) ในตัวอย่างพลาสมาของอาสาสมัครได้ การวิเคราะห์ข้อมูลลำดับสารพันธุกรรมของไวรัสพบว่าไวรัสเดงกีซีโรทัยป์ที่หนึ่งเป็น sylvatic genotype มีการพบไวรัส genotype I และ genotype II ในตัวอย่างของไวรัสซีโรทัยป์ที่สามจากตัวอย่างเดียวกัน ในขณะที่ไวรัสไข้เลือดออกซีโรทัยป์ที่สี่ของการศึกษานี้จัดอยู่ในกลุ่ม genotype II จากผลการศึกษาข้างต้นนี้อาจนำมาซึ่งการสร้างความเข้าใจการติดเชื้อตลอดจนการถ่ายทอดเชื้อไวรัสระหว่างยุงพาหะและประชากรในท้องถิ่นได้ และอาจจะใช้ในการศึกษาระบาดวิทยาและการควบคุมโรคไข้เลือดออกในอนาคตต

คำสำคัญ: ยุงลายบ้าน การติดเชื้อแบบไม่มีอาการแสดงของโรค การติดเชื้อไวรัสเดงกีร่วม โรคประจำท้องถิ่น แผนภูมิวงศ์วานวิวัฒนาการ

[1]หลักสูตรวิทยาศาสตร์การแพทย์ จุฬาลงกรณ์มหาวิทยาลัย กรุงเทพฯ 10330

[2]ภาควิชาจุลชีววิทยาคลินิก คณะเทคนิคการแพทย์ มหาวิทยาลัยหัวเฉียวเฉลิมพระเกียรติ จ.สมุทรปราการ 10540

[3]คณะเทคนิคการแพทย์ มหาวิทยาลัยหัวเฉียวเฉลิมพระเกียรติ จ.สมุทรปราการ 10540

[4]สถาบันวิจัยวิทยาศาสตร์สาธารณสุข กรมวิทยาศาสตร์การแพทย์ กระทรวงสาธารณสุข จ.นนทบุรี 11000

[5]ภาควิชาปรสิตวิทยา คณะแพทยศาสตร์ จุฬาลงกรณ์มหาวิทยาลัย กรุงเทพฯ 10330

[6]ศูนย์ความเป็นเลิศทางด้านโรคติดเชื้ออุบัติใหม่ โรงพยาบาลจุฬาลงกรณ์ สภากาชาดไทย กรุงเทพฯ 10330

****ผู้รับผิดชอบบทความ** E-mail: padet.s@chula.ac.th*