Enhanced Vector Competence of *Aedes aegypti* (Diptera: Culicidae) from the Torres Strait Compared with Mainland Australia for Dengue 2 and 4 Viruses

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ABSTRACT Australian *Aedes aegypti* (L.) mosquitoes colonized from the Torres Strait and three mainland localities (Charters Towers, Townsville, and Cairns) were fed on blood suspensions containing dengue virus type 2 (DEN-2) or dengue virus type 4 (DEN-4). Variation was found in oral susceptibility to DEN-2 (59–99% infection) and DEN-4 (28–79% infection) among *Ae. aegypti* assayed for virus at 8, 12, 16, or 20 d after ingestion of infected blood. Torres Strait *Ae. aegypti* were the most susceptible to DEN-2 and were significantly more efficient in transmission to capillary tube at 16 d (76% transmission) than mainland *Ae. aegypti* populations (20–28% transmission). Torres Strait *Ae. aegypti* were also the most susceptible to DEN-4, although transmission did not vary significantly from mainland populations at 16 d (12% compared with 0–4%) or 20 d (16% compared with 4–16%). Disseminated infection (i.e., leg infection) with either DEN-2 or DEN-4 was not an accurate predictor of transmission potential. This study demonstrates differences among Australian *Ae. aegypti* populations in vector competence for DEN-2 and DEN-4. Torres Strait *Ae. aegypti* were more frequently infected and able to transmit DEN-2 at higher rates than mainland populations. These data indicate that the Torres Strait region is potentially more receptive to dengue transmission than mainland localities, a finding discussed with respect to past outbreaks.

KEY WORDS *Aedes aegypti*, dengue, vector competence, virus, transmission

IN AUSTRALIA, MOST RECENT epidemic activity of dengue virus types 1, 2, 3, and 4 (DEN-1–4) has been limited to four regions of northern Queensland: the urban mainland centers of Townsville and Cairns, the inland town of Charters Towers, and the comparatively sparsely populated islands of the Torres Strait. In 1981–82, DEN-1 occurred at these four localities and other mainland towns, after a 26-yr absence of indigenous dengue transmission in Australia (Kay et al. 1984). Major morbidity occurred in Townsville and Charters Towers in 1992–93 and in the Torres Strait in 1996–97 with transmission of DEN-2, and in Cairns and nearby towns in 1997–99 with a DEN-3 epidemic. The latter outbreak, during which there was limited concurrent circulation of DEN-2, continued for 70 wk and resulted in 498 laboratory-confirmed cases and a 20% hospitalization rate (Hanna et al. 2001). A DEN-2 outbreak limited to a northern suburb of Cairns occurred in 2000 (Ritchie et al. 2001), followed by another small, focal epidemic in Townsville in 2001 (Hills et al. 2002). In early 2003, a DEN-2 outbreak was reported at Cairns with locally acquired cases also reported at Townsville. Over 400 cases were confirmed between January and May 2003 (S. A. Ritchie, personal communication).

Indigenous dengue transmission in Australia has been attributed to periodic reintroduction of virus by viremic travelers or residents returning from overseas (Mackenzie et al. 1998). Increases in global travel have resulted in greater human traffic to Australia from dengue-endemic countries, particularly from the Asia-Pacific region. *Aedes aegypti* (L.) is the only recognized vector of dengue viruses present on the Australian mainland, but *Aedes scutellaris* (Walker) also occurs on some Torres Strait islands (Lee et al. 1987). During the first half of the 20th century, the distribution of *Ae. aegypti* contracted from Western Australia, Northern Territory, and New South Wales to Queensland (Taylor 1943, O'Gower 1956), and this situation has since remained stable (Kay et al. 1983, Sinclair 1992).

Vector competence, the intrinsic ability of a vector to become infected with, replicate, and transmit a pathogen, has been shown to vary among widely distributed populations of *Ae. aegypti* for dengue viruses (Gubler et al. 1979, Rosen et al. 1985, Tardieux et al. 1990). Intraregional variation also has been observed in *Ae. aegypti* populations from Ho Chi Minh City (Tran et al. 1999, Huber et al. 2002), Tahiti (Vazeille-

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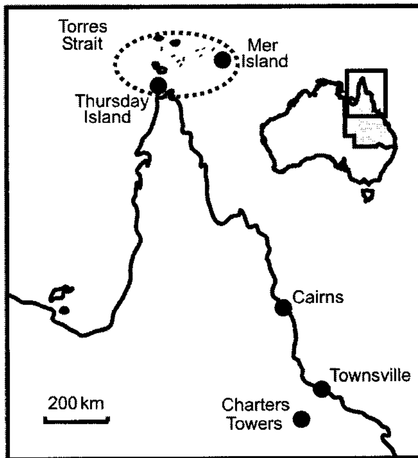

Fig. 1. Location of mosquito collection sites in northern Queensland, Australia.

Falcoz et al. 1999), South Africa (Jupp and Kemp 1993), and Mexico (Bennett et al. 2002). Such variation in *Ae. aegypti* is thought to be genetic, and related to barriers that limit or prevent viral infection of midgut cells (midgut infection barrier) and dissemination into the hemolymph (midgut escape barrier) (Bosio et al. 2000). Salivary gland infection and salivary gland escape barriers also have been identified in a number of other vector-arbovirus systems (Kramer et al. 1981, Paulson et al. 1989, Scott et al. 1990, Turell et al. 1999). However, these are yet to be demonstrated for dengue viruses and *Ae. aegypti,* and vector competence experiments generally rely on head or salivary gland infection as an indicator of transmission potential.

In Australia to date, only a single colonized strain of *Ae. aegypti* has been evaluated in terms of dengue vector competence (Watson and Kay 1999), although outbreaks are becoming more common in northern Queensland. Therefore, to determine whether *Ae. aegypti* from northern Australia vary in their ability to transmit dengue viruses, a series of vector competence experiments were conducted. We compared the susceptibility and transmission potential of four populations of *Ae. aegypti* from northern Queensland localities, where there has been major dengue morbidity (Charters Towers, Townsville, Cairns, and Torres Strait). Results are discussed with respect to the epidemiological pattern of dengue in Australia and implications for dengue disease prevention and control.

Materials and Methods

Mosquitoes. *Ae. aegypti* colonies from Charters Towers (20° 04′ S, 146° 15′ E), Townsville (19° 15′ S, 146° 49′ E), Cairns (16° 55′ S, 145° 46′ E), and Torres Strait (10° 00′ S, 142° 30′ E) (Fig. 1) were established at the Queensland Institute of Medical Research (QIMR) insectary. The Charters Towers colony was established in July 2000 from eggs collected in ovitraps set at various locations throughout Charters Towers. The Townsville colony was initiated in August 2000 from eggs collected in ovitraps and larvae collected from plant pot bases. The Cairns colony was established at QIMR in July 2000 from eggs from a colony initiated in 1995 by Scott Ritchie, Queensland Health, and supplemented by larvae in 1998. The Torres Strait colony was initiated in September 2000 from eggs collected in ovitraps on Thursday Island (10° 35′ S, 142° 13′ E) and Mer Island (9° 55′ S, 144° 03′ E).

Colonies were maintained at 12:12 (L:D) h, 28°C, and 70% RH. Each colony was located in a separate room to prevent cross-contamination. Eggs were hatched by submerging in deoxygenated water. First instar larvae were transferred to larval development trays (42 × 36 × 6 cm; Ilford, Cheshire, U.K.) containing 4 liters of tap water, and were provided with dried liver concentrate (Sigma-Aldrich, St. Louis, MO) ad libitum. Every 24 h, pupae were transferred to a dish enclosed in a large cage (46 × 46 × 46 cm) screened with 1-mm mesh. Adults were provided with 15% sucrose solution and fresh apple. Cairns, Townsville, and Charters Towers colonies were blood fed weekly on anesthetized guinea pigs (QIMR animal ethics protocol P361). As Torres Strait *Ae. aegypti* were collected from the Special Quarantine Zone in the Torres Strait, their colonization was subject to conditions imposed by the Australian Quarantine Inspection Service. As restrictions prohibited the direct or indirect exposure of any animals to the mosquitoes, these mosquitoes were fed heparinized (25 U/ml) rabbit blood once per week via a membrane feeder.

Dengue Viruses. DEN-2 (92T) and DEN-4 (97B) virus isolates were obtained from Queensland Health Scientific Services, Coopers Plains, Brisbane, and were passaged in *Aedes albopictus* (Skuse) salivary gland (C6/36) cells in cell culture media (RPMI 1640, with 10% vol:vol fetal bovine serum [FBS], 2 mM L-glutamine, 200 μg/ml benzylpenicillin, and 200 μg/ml streptomycin). DEN-2 was isolated in 1992 from human serum collected from a patient from Townsville with clinical signs of dengue infection. DEN-2 virus stock (C6/36 passage 3) had a titer of $10^{8.1}$ cell culture infectious dose, 50% endpoint ($CCID_{50}$)/ml. DEN-4 virus was isolated in 1997 from human serum collected from a patient with clinical signs of dengue infection. The patient was thought to have been infected in Bali, Indonesia. DEN-4 virus stock (C6/36 passage 2) had a titer of $10^{8.6}$ $CCID_{50}$/ml.

Infection of Mosquitoes. For each of the DEN-2 and DEN-4 experiments, ≈750 adult mosquitoes (2–5 d old) from each colony were divided evenly into five gauze-topped, cylindrical plastic containers. Mosquitoes were starved for 24 h, and four containers were exposed to a feeding solution of virus in rabbit blood of $10^{6.4}$ $CCID_{50}$/ml for DEN-2 and $10^{7.0}$ $CCID_{50}$/ml for DEN-4. This was maintained at 37°C for 1 h using membrane feeders covered with a porcine intestinal membrane (Rutledge et al. 1964). Samples of feeding solutions were taken before and after feeding, and were titrated to confirm there was no loss in infectivity of virus compared with titers determined by dilution of viral stocks of known titer. Preliminary feeds using

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lithium heparin (25 U/ml) and sodium citrate (3.2 mg/ml) anticoagulants indicated significant loss of infectivity of DEN-4 (>10^5 $CCID_{50}$) in heparinized blood over the feeding period. As a result, lithium heparin was used in DEN-2 feeds and sodium citrate in DEN-4 feeds. The fifth container was exposed to rabbit blood with no virus for negative controls.

After feeding, mosquitoes were anesthetized with CO_2 and sorted on a chill table (Industrial Inventions, Monmouth Junction, NJ), and partially and nonengorged mosquitoes were discarded. Fully engorged females were maintained on a 15% sucrose solution under QIMR insectary conditions (as above) for 8, 12, and 16 d (DEN-2), or 8, 12, 16, and 20 d (DEN-4).

Processing of Mosquitoes. To determine DEN-2 and DEN-4 infection and transmission rates, bodies, legs, heads, and saliva of 20 or 25 mosquitoes were processed separately for each sampling period. Mosquitoes were anesthetized with CO_2, the legs and wings were removed, and legs were transferred to a microfuge tube containing 100 μl of grinding media (cell culture media containing 2 μg/ml fungizone [Apothecon, Princeton, NJ]). Saliva was collected based on the in vitro feeding technique developed by Aitken (1977) in which the proboscis of a mosquito was inserted into a capillary tube (75 mm long and <1.1 mm diameter) containing ≈5 μl of FBS. After 15 min, the FBS was ejected using a micropipette into a microfuge tube containing 100 μl of grinding media. The head of each mosquito then was severed from the body (thorax/abdomen), and both segments were transferred to separate microfuge tubes, each containing 100 μl of grinding media. Control mosquitoes were processed 16 d after feeding on blood with no virus. All samples were stored at −60°C until they were tested for virus.

Processing of Samples for Virus. Titration of dengue virus stocks and blood meals, and testing of mosquito samples were performed using cell culture and an enzyme-linked immunosorbent assay (ELISA) method similar to that described by Broom et al. (1998). Mosquito samples were homogenized using sterile micropestles and a cordless motor (Quantum Scientific, Brisbane, Australia); 600 μl of grinding media was added; and tubes were centrifuged at 10,000 × *g* for 10 min at 4°C. Confluent monolayers of C6/36 cells in 96-well plates were inoculated with 50 μl/well of virus stock dilutions or ground mosquito homogenates. Negative controls were included on each plate (cell culture media or uninfected mosquito grinds). Plates were incubated at 28°C with 3–5% CO_2 for 5 d, except for saliva samples, which were incubated for 10 d. For mosquito assay and virus titration, fixed cell monolayers were examined for dengue virus antigens using a mixture of three flavivirus cross-reactive monoclonal antibodies (4G4, 3H6, and 1E7) and a dengue group cross-reactive monoclonal antibody (2H2). To confirm virus identity, DEN-2- and DEN-4-specific monoclonal antibodies were used (3H5 and 1H10, respectively). Antibodies originally were obtained from the American Type Culture Collection (Manassas, VA) (2H2, 3H5, and 1H10) and TropBio (Townsville, Australia) (1E7), or were produced in the Molecular Virology Laboratory, University of Queensland, Australia (4G4 and 3H6) (Hall et al. 1990).

Horseradish peroxidase-labeled goat anti-mouse IgG conjugate (DAKO, Carpenteria, CA) was added with enzyme activity visualized using ABTS substrate. Absorbance was measured at 405 nm in a Multiskan EX microplate reader (Thermo Labsystems Oy, Helsinki, Finland) to determine optical density (OD). A positive result was recorded if virus was detected in at least one test well for saliva samples, or more than half of test wells for other samples, as indicated by an OD greater than twice (or three times for saliva assays) the mean OD of all uninfected control wells of the same plate.

Infection and transmission rates were calculated by dividing the number of samples positive for DEN-2 or DEN-4 by the total number of mosquitoes exposed to virus. Oral susceptibility of a strain was indicated by the body infection rate, with the virus dose per mosquito based on the assumption of an average *Ae. aegypti* bloodmeal size of 0.002 ml. Disseminated infection, presence of virus in the hemolymph of the mosquito (Turell et al. 1984), was indicated by recovery of virus from the legs of a mosquito. Head and leg samples were processed separately to provide comparative data. Virus detection (using the above method) in FBS from capillary tubes was considered evidence of dengue virus transmission.

Results

Oral susceptibility, defined as the proportion of mosquitoes in which virus was detected in the body varied among the four northern Queensland *Ae. aegypti* populations for both DEN-2 ($\chi^2 = 39.48$; $P < 0.001$) and DEN-4 ($\chi^2 = 79.62$; $P < 0.001$). Infection rates in *Ae. aegypti* ranged from 52 to 100% for DEN-2 (Table 1) and from 12 to 84% for DEN-4 (Table 2) in response to infectious meals of $10^{3.7}$ and $10^{4.3}$ $CCID_{50}$ virus per mosquito, respectively after incubation at 28°C for 8, 12, 16, or 20 d. Infection of *Ae. aegypti* populations did not vary with incubation period ($P > 0.05$). Torres Strait *Ae. aegypti* were more susceptible to oral infection than the three mainland populations for both DEN-2 (99% versus 59–81%) and DEN-4 (79% versus 28–29%).

In contrast to body infection rates, which did not vary with time, disseminated infection (leg infection) rates were higher in mosquitoes tested 12–20 d after ingestion of virus, than at 8 d ($\chi^2 = 43.65$; $P < 0.001$). Although this trend was consistent for both DEN-2 and DEN-4, 97% of DEN-2-infected mosquitoes had disseminated infections at 12 d, compared with only 29% of DEN-4-infected mosquitoes. In fact, at both 16 and 20 d after feeding, 21% of DEN-4-infected mosquitoes did not have a disseminated infection. This indicated that virus infection probably was limited to the midgut in these mosquitoes.

Leg and head samples from individual mosquitoes gave similar assay results with 95% consistency for DEN-2 and 98% for DEN-4. Therefore, the testing of

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Table 1. Percentage of infection and transmission of dengue 2 virus on days 8–16 ($n = 25$) postingestion of $10^{3.7}$ $CCID_{50}$ by northern Queensland *Ae. aegypti*

Population	Period of incubation											
	8 days				12 days				16 days			
	BIR[a]	LIR[a]	HIR[a]	TR[a]	BIR	LIR	HIR	TR	BIR	LIR	HIR	TR
Charters Towers	52	36	36	8	60	60	60	4	64	64	64	24
Townsville	72	20	16	0	90[b]	85[b]	85[b]	0[b]	92	92	92	28
Cairns	80	60	60	8	84	80	80	4	80	80	80	20
Torres Strait	96	48	32	0	100	100	96	8	100	100	100	76

[a] BIR, body infection rate; LIR, leg infection rate; HIR, head infection rate; TR, transmission rate.
[b] $n = 20$.

either a leg or head sample was equally indicative of a disseminated infection.

Transmission of DEN-2 was first observed at 8 d after feeding (Charters Towers and Cairns *Ae. aegypti*), but the highest rates were not obtained until 16 d (Table 1). Torres Strait *Ae. aegypti* were significantly more efficient at transmitting DEN-2 at 16 d (76%) than compared with the three mainland *Ae. aegypti* populations (20–28%). There was no significant difference in the DEN-4 transmission rates among *Ae. aegypti* strains at any incubation period (Table 2); however, this may have been because of the small number of saliva samples that were positive for virus.

There was no association between disseminated infection and transmission rates of mosquitoes fed either DEN-2 or DEN-4. For example, 100% of Torres Strait *Ae. aegypti* had a disseminated infection at both 12 and 16 d after ingestion of DEN-2, whereas 8% were observed transmitting at 12 d and 76% at 16 d. In this case, using disseminated infection as an indicator of transmission potential would have overestimated transmission by 92% at 12 d and by 24% at 16 d. At 20 d, Townsville and Torres Strait *Ae. aegypti* transmitted DEN-4 at the same rate (16%), despite differences in disseminated infection rates (28 and 60%, respectively). Therefore, disseminated infection was shown to be a poor predictor of transmission potential.

Discussion

To date, the only vector competence analysis of Australian *Ae. aegypti* for dengue viruses involved a colonized strain of *Ae. aegypti* from Townsville (Watson and Kay 1999). Therefore, the research reported in this work represents the first comparative analysis of the susceptibility to oral infection, dissemination, and transmission of dengue viruses by Australian geographic populations of *Ae. aegypti.* Variation was observed in the oral susceptibility of four northern Queensland populations of *Ae. aegypti* to DEN-2 and DEN-4. Torres Strait mosquitoes were found to be: 1) more susceptible to oral infection with both DEN-2 and DEN-4; 2) more susceptible to dissemination of DEN-2 and DEN-4; and 3) significantly better transmitters of DEN-2 than mainland *Ae. aegypti* populations.

Geographic variation in vector competence of *Ae. aegypti* for dengue viruses is well known. For example, a recent study found that *Ae. aegypti* populations from the Yucatán region exhibited greater vector competence for DEN-2 than those from other areas of Mexico (Bennett et al. 2002). Our data on DEN-2 oral susceptibility of Australian *Ae. aegypti* are similar to those recently reported for various populations from the Pacific (Vazeille-Falcoz et al. 1999), Southeast Asia (Tran et al. 1999, Huber et al. 2002, Vazeille et al. 2003), and the Americas (Fouque et al. 2001, Bennett et al. 2002). Watson and Kay (1999) observed maximum infection rates of 68 and 60% for DEN-2 and DEN-4, respectively, for a Townsville *Ae. aegypti* colony established in 1990. The maximum DEN-2 infection rate observed for a different Townsville *Ae. aegypti* colony in our study was higher (92%). A lower infection rate than that of the earlier study was found for DEN-4 (40%). Although few other vector competence analyses have been conducted for DEN-4, our northern Queensland populations had greater susceptibilities than those tested from the South Pacific, Africa, and Asia (Gubler et al. 1979).

Data from investigations employing different experimental protocols do not necessarily indicate the

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Table 2. Percentage of infection and transmission of dengue 4 virus on days 8–20 ($n = 25$) postingestion of $10^{4.3}$ $CCID_{50}$ by northern Queensland *Ae. aegypti*

Population	Period of incubation															
	8 days				12 days				16 days				20 days			
	BIR[a]	LIR	HIR	TR	BIR	LIR	HIR	TR	BIR	LIR	HIR	TR	BIR	LIR	HIR	TR
Charters Towers	36	0	0	0	16	4	4	0	28	16	16	0	32	12	12	8
Townsville	12	0	0	0	28	8	8	0	40	40	32	0	32	28	32	16
Cairns	16	0	0	0	28	8	4	0	36	24	24	4	36	36	36	4
Torres Strait	80	8	12	0	80	24	24	0	84	68	68	12	72	60	60	16

[a] As for Table 1.

comparative vector competence of populations. For instance, oral susceptibility of *Ae. aegypti* for dengue viruses varies with virus serotype, virus strain, and titer of the infectious bloodmeal (Gubler et al. 1979, Bennett et al. 2002), and is influenced by environmental and other experimental effects in the insectary (Bosio et al. 2000). Sensitivity of virus/antigen detection methods may also affect results. As we used different anticoagulants for DEN-2 and DEN-4 feeding solutions, and as there is no published data on the effect of anticoagulant on the growth characteristics of dengue viruses in *Ae. aegypti,* direct comparisons of DEN-2 and DEN-4 infection and transmission rates have not been made.

The maximum rates of DEN-2 transmission observed for Australian mainland *Ae. aegypti* (20–28%) were comparable to those found with South African *Ae. aegypti* populations using a similar capillary tube transmission model (18–34%) (Jupp and Kemp 1993). Indonesian *Ae. aegypti* transmitted DEN-2 to droplets at a comparable rate also (19%); however, Kenyan *Ae. aegypti* were less efficient transmitters at 7% (Gubler et al. 1979). No transmission rate as high as the 76% observed in our study for Torres Strait *Ae. aegypti* has been reported for any dengue virus.

In previous dengue studies, the term extrinsic incubation period (EIP) has been used to describe the duration between ingestion of virus and first transmission. In their studies of transmission of dengue to human volunteers by *Ae. aegypti,* Siler et al. (1926) reported an EIP of between 10 and 14 d under the ambient conditions of Manila in the Philippines. With *Ae. aegypti* from Thailand, Watts et al. (1987) found an EIP of 7 d with incubation at 32–35°C, and of 12 d with incubation at ≤30°C for DEN-2, with transmission to rhesus monkeys. However, in these studies, groups of mosquitoes were examined for virus transmission, whereas in the current study individual mosquitoes were tested. Based on our results, one could argue whether the time to first transmission should be used as an indication of transmission potential. For example, although Charters Towers and Cairns *Ae. aegypti* transmitted DEN-2 after 8-d incubation, virus was isolated from only two saliva samples for each population (8%). Similar results were found at 12 d; however, transmission increased to 20–76% at 16 d. The low number of *Ae. aegypti* observed transmitting at the earlier incubation periods indicated that the time of first transmission was not a good indicator of the comparative vector competence of populations, and that the proportion of mosquitoes able to transmit at a particular time must also be considered.

The Australian *Ae. aegypti* populations tested possessed a barrier between disseminated infection and salivary gland infection and/or transmission. However, as salivary glands were not processed separately, we were unable to quantify the relative effect of each of these barriers. The demonstration of this restriction of transmission is contrary to the general belief that an *Ae. aegypti* with a disseminated infection will always transmit virus (Vazeille et al. 2003). The implication is that disseminated infection as determined by head squashes or by assaying heads or legs may not be a suitable indicator of transmission. Assessment of disseminated infection alone may overestimate transmission potential.

Although there are >100 islands in the Torres Strait, because of access limitations the *Ae. aegypti* colony was sourced from two islands separated by ≈200 km. It is unknown to what extent the vector competence parameters defined for the Torres Strait colony compare with those for wild-type populations throughout the region. Although genetic isolation by distance was shown for *Ae. aegypti* from Mexico, free gene flow occurred among *Ae. aegypti* populations separated by distances of up to 250 km (Gorrochotegui-Escalante et al. 2000). Correlations have also been noted between genetic distances and variation in vector competence phenotypes for DEN-2 (Bennett et al. 2002, García-Franco et al. 2002). Although there is potential for interisland movement of *Ae. aegypti* throughout the Torres Strait, analyses to determine the genetic diversity of mosquitoes from this region have not been conducted.

Although *Ae. aegypti* from the Torres Strait had a greater vector competence than those tested from the mainland, only 1 of the 10 outbreaks reported since 1981 have involved entry via this route. Most have involved direct introduction of dengue virus via viremic tourists arriving from Southeast Asia. The DEN-2 responsible for the 1996–97 epidemic in the Torres Strait and Cairns is thought to have been imported from Papua New Guinea to the Torres Strait, from where it was introduced to Cairns via a viremic traveler (Hanna et al. 1998). Although there is regular traffic from New Guinea across the Torres Strait, this is only a fraction of the 793,000 international visitors (Tourism Queensland 2002) and many residents returning to tropical northern Queensland from dengue-endemic regions, particularly Asia. Therefore, in spite of this increased receptivity, it is not surprising that most outbreaks have been initiated on the mainland, where, with these DEN-2 and DEN-4 strains at least, less competent *Ae. aegypti* occur. Because *Ae. aegypti* is an established global traveler, health authorities should ensure that the at present geographically isolated populations of *Ae. aegypti* in the Torres Strait are prevented from becoming established on the mainland. Increased susceptibility and ability of *Ae. aegypti* to transmit dengue viruses would contribute greatly to the periodicity and extent of dengue epidemics in northern Queensland.

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