Respiratory syncytial virus infection and prevalence of subgroups A and B in Hawaii

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Respiratory syncytial virus was isolated from hospitalized children in Hawaii in each month of the year during the period January 1987 to August 1989. Subgroup A and subgroup B strains cocirculated, with subgroup A predominating. There was an alternating early-season and late-season peak incidence cycle as reported elsewhere.

In temperate regions, epidemics of respiratory syncytial virus (RSV) infection occur in late fall, winter, and early spring (10, 15, 22); the virus is seldom isolated during the summer. Epidemics tend to follow a pattern of peak incidence of infection alternating between early and late in the winter season in successive years (10). Both RSV subgroups A and B may circulate during the same RSV season with subgroup A predominating, but the relative proportions vary, and subgroup B may occur more frequently in some years (1–4, 8–12, 14, 18–21). There is evidence that infection with subgroup A provides some protection against reinfection with homologous viruses but not against heterologous (subgroup B) viruses (7, 13, 14, 21). Thus, studies on the prevalence of RSV subgroups A and B in different communities may help to determine whether community immunity to strains of one subgroup leads to the predominance of strains of the second subgroup (20).

In tropical and semitropical regions, epidemics of RSV may occur during the summer months if these coincide with the rainy season (17, 18). Epidemics of RSV in Hong Kong, which is at the same latitude as Hawaii, tend to be correlated with rainfall, relative humidity, and temperature (18). Although Hawaii is in the tropical zone, it has a semitropical climate with two seasons recognized by ancient Hawaiians and modern climatologists alike: the high-sun period of warm temperatures and steady trade winds, and a cooler period when trade winds are less frequent and rainfall is more common (6).

In order to determine the seasonal incidence of RSV infections in Hawaii, we attempted to isolate RSV from respiratory secretions of 522 hospitalized children 1 week to 17 years of age over a period of 32 successive months (January 1987 to August 1989). The presence of RSV in HEP-2 cell cultures inoculated with clinical material was determined by the appearance of a characteristic cytopathic effect within 14 days and confirmed by indirect fluorescein-labelled-antibody test using bovine anti-RSV immunoglobulin G (Burroughs-Wellcome, Research Triangle Park, N.C.). Stocks of virus isolates were stored at −70°C until serotypes could be determined. Respiratory secretions were also tested for presence of RSV antigen by using a commercial kit (Pathfinder; Kallestad, Chaska, Minn.) according to the manufacturer’s directions.

Serotype determination of RSV isolates was done in 96-well tissue culture plates seeded with HEP-2 cells. The LONG strain (subgroup A) and the 18537 strain (subgroup B) were included in each plate as positive controls. Noninfected HEP-2 cells served as negative controls. The plates were incubated at 37°C in 5% CO₂ until an approximately 50% cytopathic effect was present in all infected wells, and the virus strains were serotyped by enzyme immunoassay using three monoclonal antibodies (MAb) obtained from Larry J. Anderson (Centers for Disease Control, Atlanta, Ga.): a panspecific anti-RSV (anti-strain A2) MAb, an anti-RSV LONG strain MAb, and an anti-RSV 18537 strain MAb.

Approximately 18% of specimens from 522 patients were positive by either culture, enzyme immunoassay or both. The two methods had a concordance of 91%. The virus was isolated in cell cultures from 21 specimens negative by enzyme immunoassay, and RSV antigen was detected in 24 that were negative by culture.

Over the 32 months of this study, RSV was isolated from at least one patient in every month of the year. Although the incidence of infection was greater in the winter wet season (October to April), as in temperate climates, the virus also circulated throughout the summer dry season (May to September) (Fig. 1). The peak incidence of RSV infection tended to alternate with an early and a late peak in successive years. In 1987, the peak incidence was in mid-February (late) and was followed by a peak in mid-January 1988 (early) and then by a peak in early February in 1989 (late) (Fig. 1). This pattern is consistent with that previously reported for temperate regions (10).

The mean age of hospitalized patients infected with RSV was 12 months (median, 4 months). This is similar to the reported mean age of 9.5 months for respiratory disease

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patients with an RSV isolate in Washington, D.C. (5). Bronchiolitis due to RSV occurs most commonly at 2 months of age (16), but we did not specifically note the types of clinical illness in our patients.

Subgroup A and B strains cocirculated in the susceptible population except during the summer periods of lowest incidence. In the summer of 1987, only subgroup B strains were isolated; in the summer of 1988, only subgroup A was isolated. Subgroup B strains predominated in the first half of 1987, but subgroup A strains predominated throughout most of 1988 and 1989 (Fig. 2). The prevalence of subgroup B strains decreased from 1987 to 1989. There was no significant difference between the average ages of infants infected with subgroup A RSV and infants infected with subgroup B RSV (P = 0.26, Student’s two-tailed t test). Similar results were obtained by Monto and Ohmit (11) in Michigan. There was no indication that one serotype caused more-severe infection than the other.

The presence of RSV in Hawaii on a year-round basis may be related to the tourist industry as well as to geographical location and climate. Each month, approximately 300,000 visitors from the West and 200,000 visitors from the East arrive in Hawaii (data from the Hawaii Visitors Bureau), potentially providing a constant source of RSV to the local community. For example, during the summer in Hawaii, visitors from Australia and New Zealand will have left in winter, at the height of their RSV season. Others come from tropical or semitropical Asia during their rainy season, which correlates with their RSV epidemic season. Introductions from outside this small island state may also influence the seasonal distribution of RSV subgroups A and B. However, no data specifically addressing this question were obtained in this study.

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REFERENCES