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Seroprevalence of parvovirus B19 in urban Chilean children and young adults, 1990 and 1996

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SUMMARY

An immunofluorescence test for detecting parvovirus B19 IgG was developed by infecting insect cells with recombinant baculovirus expressing the capsid protein VP1. The test was used to study the prevalence of antibodies in 725 healthy children and young adults living in Santiago, Chile. In total, 248 sera were taken in 1990 and 477 in 1996. The seroprevalence was low in children less than 5 years old (3% in 1990 and 21% in 1996). It rose during school age to a prevalence around 50%, reaching 60% in young adults. No differences were found between genders. There was a statistically significant higher seroprevalence in the low socio-economic status group in 1990 samples, but this was not observed in 1996. The higher prevalence observed in children less than 5 years of age in 1996 compared with 1990 could be explained by the occurrence of intervening epidemics of parvovirus B19 infection.

INTRODUCTION

The prevalence of antibodies to parvovirus B19 in adults is about 50% in the United States and Japan [1, 2] and 60–70% in England and Wales [3, 4]. In Brazil and Niger a higher prevalence has been found with more than 80% of the children parvovirus B19 antibody positive at 10 years of age [5, 6]. In some remote tribes in Brazil a very low prevalence has been reported [7]. The parvovirus B19 antibody prevalence in Chile is not known and our aim was to study it and examine variations over time and socio-economic status.

METHODS

Samples

A total of 250 sera were collected in 1990 and 480 in 1996 from 730 healthy people between the ages of 1 and 35 years, residing in the capital city, Santiago. A description of collection procedures and epidemiological data of the samples can be found elsewhere [8, 9]. Socio-economic status (SES) was determined by questions associated with income in the following areas: education, occupation, characteristics of the home, equipment in the home, water supply, sewage disposal, ratio of number of persons to number of beds in the home, and ratio of number of persons to number of bedrooms. Using this information, 399 subjects (136 from 1990 and 263 from 1996) were assigned to the low SES group and 331 (114 from 1990 and 217 from 1996), to the high SES group.

Parvovirus B19 IgG detection assay

Parvovirus B19 IgG was detected by an in-house immunofluorescence assay (IFA) using baculovirus expressed parvovirus B19 antigens [10]. It has previously been shown that the results of B19 IgG assays using baculovirus expressed parvovirus B19 antigens correlate closely with those of radioimmunoassay

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based on native parvovirus B19 antigen [11]. The IFA test was performed by fixing insect cells (*Spodoptera frugiperda*) infected with recombinant baculovirus expressing VP1 capsid protein [10] on slides. Serum dilutions (1 in 100) were dispensed onto the slides and incubated for 30 min at 37 °C in a moist chamber. The slides were rinsed with phosphate buffered saline (PBS) and then incubated with fluoresceine isothiocyanate (FITC) conjugated rabbit anti-human IgG for 30 min in a moist chamber. After washes with PBS, the slides were examined under a fluorescence microscope using ×400 magnification. Human sera characterized by radioimmunoassay were used as positive and negative controls. In IFA, characteristic fluorescent aggregates were seen with sera known to contain parvovirus B19-specific IgG. By testing the parvovirus B19 IgG International Standard it was determined that the test could detect at least 5 International Units per ml.

Statistical analysis

Data were analysed using the χ^2 test on each variable on its own and using logistic regression when assessing interactions and the adjusted effects of each variable. Exact confidence intervals on the percentages positive were also calculated.

RESULTS

1990 samples

Of the 250 samples, 248 could be accurately read by IFA. Two sera gave non-specific fluorescence that could not be interpreted. Of these 248 sera, 95 (38%, 95% CI 32–45%) were parvovirus B19 IgG positive. The antibody prevalence was analysed by age (Table 1) and socio-economic status (Fig. 1*a*). The low socio-economic status group showed higher seroprevalence at every age. This difference was statistically significant ($P = 0{\cdot}013$, and $P = 0{\cdot}017$ when adjusted for age). There was no interaction between age and socio-economic status ($P = 0{\cdot}44$), so the higher seroprevalence in those with low socio-economic status was consistent across the age groups.

1996 samples

Of the 480 samples, 477 could be accurately read by IFA. Of these 477 sera, 212 (44%, 95% CI 40–44%) were parvovirus B19 IgG positive. Table 1 shows the prevalence by age and Figure 1 by socio-economic status. There were no significant differences between the high and low socio-economic status in 1996 ($P = 0{\cdot}46$, and $P = 0{\cdot}42$ when adjusted for age). The age-specific seroprevalence, however, differed significantly by year ($P = 0{\cdot}012$). This was mainly due to the higher seroprevalence in children less than 6 years old in 1996 compared to 1990 (21% *vs.* 3·2%).

Table 1. *Prevalence of parvovirus B*19 *IgG antibodies by age, 1990 and 1996*

	1990		1996	
Age group (years)	No. tested	No. (%) positive	No. tested	No. (%) positive
1–5	62	20 (3·2)	82	17 (20·7)
6–10	44	20 (45·4)	90	43 (47·7)
11–20	78	34 (43·6)	176	83 (47·1)
> 20	64	38 (59·3)	129	68 (52·7)
Total	248	94 (37·9)	477	211 (44·2)

DISCUSSION

This serological survey is to our knowledge the first performed for parvovirus B19 on sera from Chile and also the first to have evaluated the impact of the socio-economic status on the seroprevalence of parvovirus B19 infection. Our results show that parvovirus B19 is common in Chile and support the view that parvovirus B19 virus has a worldwide distribution. Analysis of age-specific seroprevalence showed a similar pattern to that described in USA [1], England [3, 4] and Japan [2] with a low seroprevalence in infants and pre-school children, a rise during school-age and a rate of around 50–60% in young adults. The different seroprevalence in children less than 5 years old in 1990 compared to that in 1996 may be explained by the 3–4 year epidemic cycle of parvovirus B19 infection, as observed in Europe [12]. However, more evidence needs to be gathered to establish the epidemic cycle in Chile. It is possible to hypothesize that few young children in Chile in 1990 had been exposed to any outbreak of parvovirus B19 infection, but that more had been by 1996.

The seroprevalence rates in urban Santiago, Chile were lower at every age group in both years studied than in another Latin-American city, Rio de Janeiro [5]. Other seroprevalence studies in Chile have shown two epidemiological patterns of several infectious agents, such as CMV, *Toxoplasma gondii* and Epstein Barr virus: the high socio-economic groups shows a

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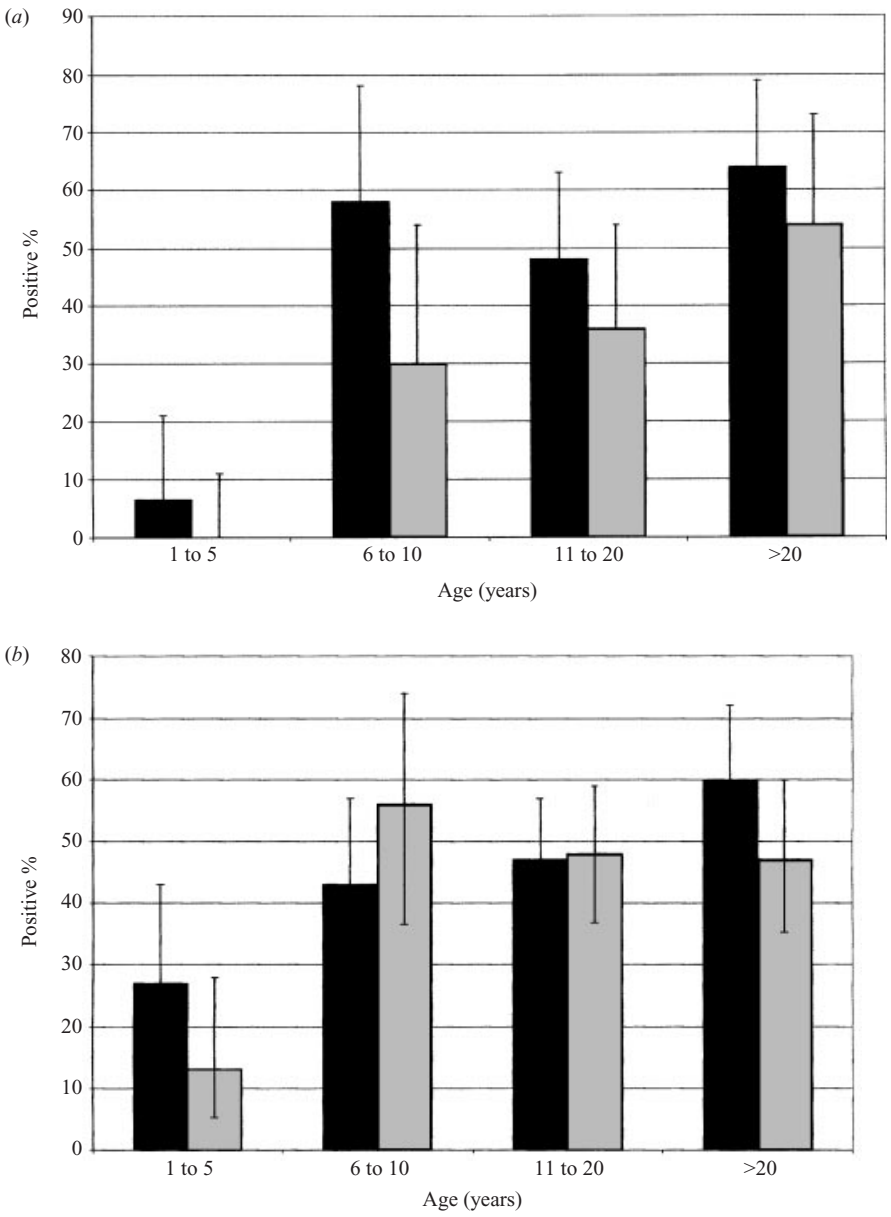

Fig. 1. (*a*) Prevalence of parvovirus B19 specific IgG in Chilean population by age and socioeconomic status (SES) in 1990. Error bars give 95% confidence intervals. (*b*) Prevalence of parvovirus B19 specific IgG in Chilean population by age and socioeconomic status (SES) in 1996. Error bars give 95% confidence intervals. ■, Low SES; ▨, high SES.

pattern closer to those of developed countries, and the low socio-economic status a pattern similar to those of underdeveloped countries [13, 14]. Moreover, statistically significant differences in the parvovirus B19 seroprevalence rates between the low and the high socio-economic status groups were shown in 1990. It is possible to speculate that crowding associated with low income facilitates the spread of parvovirus B19 infection. Thus, a densely populated city, such as Rio de Janeiro, shows high seroprevalence rates whereas remote tribes in Brazil show very low prevalence of parvovirus B19 antibodies [7]. The more homogeneous prevalence in the different socio-economic groups in 1996 could be explained by an improvement in the living conditions in Chile over the 6-year period [15] or, probably more likely, by higher infection rates in the population resulting from a widespread parvovirus B19 epidemic.

In this Chilean population, 50–60% of the young adults had antibodies to parvovirus B19. Therefore, around a half of women of childbearing age are susceptible to parvovirus B19 infection with the known risk to the fetus. Maternal infection with parvovirus B19 is estimated to occur in approximately

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1% of pregnant women in the United States and in England and Wales [4, 16], and fetal deaths follows in 9% of these cases if infection occurs in early pregnancy [17]. With such figures and considering the Chilean birth rates it is possible to estimate around 300 fetal deaths due to parvovirus B19 infection in Chile each year and many underdiagnosed cases of foetal hydrops. An appropriate knowledge of the morbidity and fetal mortality due to parvovirus B19 infection in every country is desirable, so specific therapeutic and preventive strategies can be applied. Further studies need to be carried out to establish the epidemic cycle in Chile and to estimate the impact parvovirus B19 infection has on fetuses.

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