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The age-specific prevalence of human parvovirus immunity in Victoria, Australia compared with other parts of the world

H. A. KELLY[1]*, D. SIEBERT[2], R. HAMMOND[1], J. LEYDON[1], P. KIELY[3] AND W. MASKILL[1]

[1] *Victorian Infectious Diseases Reference Laboratory, Melbourne, Victoria*
[2] *Division of Microbiology, Queensland Health Pathology Services, Royal Brisbane Hospital, Queensland*
[3] *Australian Red Cross Blood Service – Victoria, Melbourne, Victoria*

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SUMMARY

The age-specific immunity to human parvovirus infection was estimated in Victoria, Australia using prospectively collected samples from the Royal Children's Hospital, the Royal Women's Hospital and the Australian Red Cross Blood Service and from sera stored at the Victorian Infectious Diseases Reference Laboratory (VIDRL). All testing was performed at VIDRL using a commercial enzyme-linked immunosorbent assay (Biotrin). Of the 824 sera tested, 28% of those drawn from people aged 0–9 years contained protective antibodies to human parvovirus. This rose to 51% in the next decade of life. There was then a slow rise to about 78% immunity over 50 years of age. An analysis of all requests for parvovirus serology at VIDRL from 1992 to 1998 suggested that parvovirus tended to occur in 4-year cycles, with 2 epidemic years followed by 2 endemic years. A review of published reports of parvovirus immunity suggested that parvovirus infection may be more common, with a correspondingly higher proportion of the community immune, in temperate as opposed to tropical countries.

INTRODUCTION

Human parvovirus B19 is the cause of the childhood disease erythema infectiosum, also known as slapped cheek or fifth disease [1]. The virus is spread by the respiratory route and infects red cell precursors [2]. Amongst adults parvovirus B19 may cause prolonged anaemia in immuno-compromised persons [3], transient anaemia amongst otherwise healthy adults [4], aplastic crises in infected persons with an underlying blood disorder [5] and arthritis or arthralgia, principally amongst infected adult females [6]. In addition parvovirus can cause hydrops foetalis and foetal death if a pregnant woman becomes infected, with the risk being greatest in the first two trimesters of pregnancy [7].

* Author for correspondence: Dr H. Kelly, Victorian Infectious Diseases Reference Laboratory, Locked Bag 815 Carlton South, Victoria, Australia 3032.

Estimates of population susceptibility to human parvovirus are useful in assessing the risk to pregnant women. Although it is generally thought that about half the young adult population is immune [8], there are no Australian data on parvovirus immunity. This study provides an estimate of age-specific immunity to infection with parvovirus in a sample of healthy people drawn from the Australian population and compares that with reports of immunity amongst various samples of people from other countries. In addition the pattern of laboratory diagnosis of acute parvovirus infection is used to demonstrate the epidemic pattern of parvovirus infection over 7 years in Victoria.

METHODS

Four samples of convenience were selected to represent a sample of 824 residents of Melbourne,

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Table 1. *Samples for the estimation of human parvovirus immunity in Victoria*

Institution	Composition of sample	Sample size		Age range (years)
		Male	Female	
Royal Children's Hospital	Children admitted for elective surgery	108	49	0–16
Australian Red Cross Blood Service, Victoria	Healthy adult blood donors	218	129	16–64
Royal Women's Hospital	Pregnant women attending ante-natal clinic	0	156	17–42
Victorian Infectious Disease Reference Laboratory	Stored sera from patients tested for autologous blood transfusion	92	72	51–94
Combined sources		418	406	0–94

Victoria (Table 1). Ethical approval was obtained for the collection and analysis of each serum sample from the Ethics Committees of the four institutions responsible for patients or donors from whom the sera were drawn. All sera were tested for IgG antibodies to the parvovirus capsid antigen VP2 using a commercial enzyme-linked immunosorbent assay (Biotrin, Dublin, Ireland) at the Victorian Infectious Diseases Reference Laboratory (VIDRL). The χ^2 distribution was used to test for differences in the proportion of positive sera by source of subject, age group and sex.

All requests for parvovirus serology between the years 1992–8 at VIDRL were reviewed and, after deletion of duplicates, the pattern of IgM positive specimens was used to describe the epidemic pattern of parvovirus infection.

Two independent searches of Medline were made to find published reports of population estimates of parvovirus immunity, using the search criteria 'parvovirus and epidemiology' and 'parvovirus immunity'. Since population immunity is more likely to be of interest to national readers, no language restriction was used and the search was conducted back to 1983 when the association between human parvovirus and erythema infectiosum was first established [1]. This yielded more than 360 papers. Only the English abstract was accessed for papers published in another language. References to other studies of parvovirus population immunity in the English language papers and other studies known to the authors were also included in the review. In addition Dr Bernard Cohen of the Public Health Laboratory Services in the United Kingdom provided references which had not been found from other sources.

RESULTS

Age-specific immunity and epidemic pattern of parvovirus in Australia

The prevalence of immunity to human parvovirus by source of subject, sex and 10-year age group is shown in Table 2. Where subjects sampled from different sources could be compared by age group, there were no significant differences in the prevalence of immunity to parvovirus. For instance, the prevalence of immunity was similar in the 20- to 29-year-old female blood donors and pregnant women of the same age (52 *vs.* 67%, $P = 0{\cdot}14$) and amongst the 50- to 59-year-old healthy blood donors and people of the same age tested for autologous blood transfusion (75 *vs.* 77%, $P = 0{\cdot}89$) (Table 2). There was no difference in the prevalence of immunity by sex at any age group. By the age of 20 years, half of the people in this sample were immune to parvovirus infection. Seroconversion continued to occur up to the age of 50 years when the level of immunity appeared to plateau at approximately 78%.

Between 1992 and 1998, VIDRL tested 8399 sera for the presence of IgM antibodies to human parvovirus. The total number of positive sera was 682 (8·1%), ranging from 32 in 1994 (4·4% of tests for that year) and 29 in 1995 (4·3% of tests) to 206 in 1992 (19·5% of tests) and 189 in 1997 (10·6% of tests). When parvovirus was more prevalent in the com-

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Table 2. *Age-specific immunity to human parvovirus by source of subject, age group and sex*

Age group (years)	Source of subjects*	Males	Percent immune	Females	Percent immune	All subjects	Percent immune
0–9	RCH	86	27%	32	31%	118	28%
10–19	RCH	22	55%	17	47%	39	51%
	RWH	0		12	42%	12	42%
	VBB	13	54%	20	55%	33	55%
	All sources	35	54%	49	49%	84	51%
20–29	RWH	0		83	67%	83	67%
	VBB	39	51%	44	52%	83	52%
	Both sources	39	51%	127	62%	166	60%
30–39	RWH	0		58	57%	58	57%
	VBB	61	59%	23	70%	84	62%
	Both sources	61	59%	81	60%	142	60%
40–49	RWH	0		3	100%	3	100%
	VBB	61	75%	24	58%	85	71%
	Both sources	61	75%	27	63%	88	72%
50–59	VBB	34	76%	12	75%	46	75%
	VIDRL	16	75%	15	80%	31	77%
	Both sources	50	76%	27	78%	77	77%
60–69	VBB	10	100%	6	50%	16	81%
	VIDRL	30	73%	31	84%	61	79%
	Both sources	40	80%	37	78%	77	79%
70+	VIDRL	46	74%	26	28%	72	77%

* RCH, Royal Children's Hospital; RWH, Royal Women's Hospital; VBB, Victorian Blood Bank; VIDRL, Victorian Infectious Diseases Reference Laboratory.

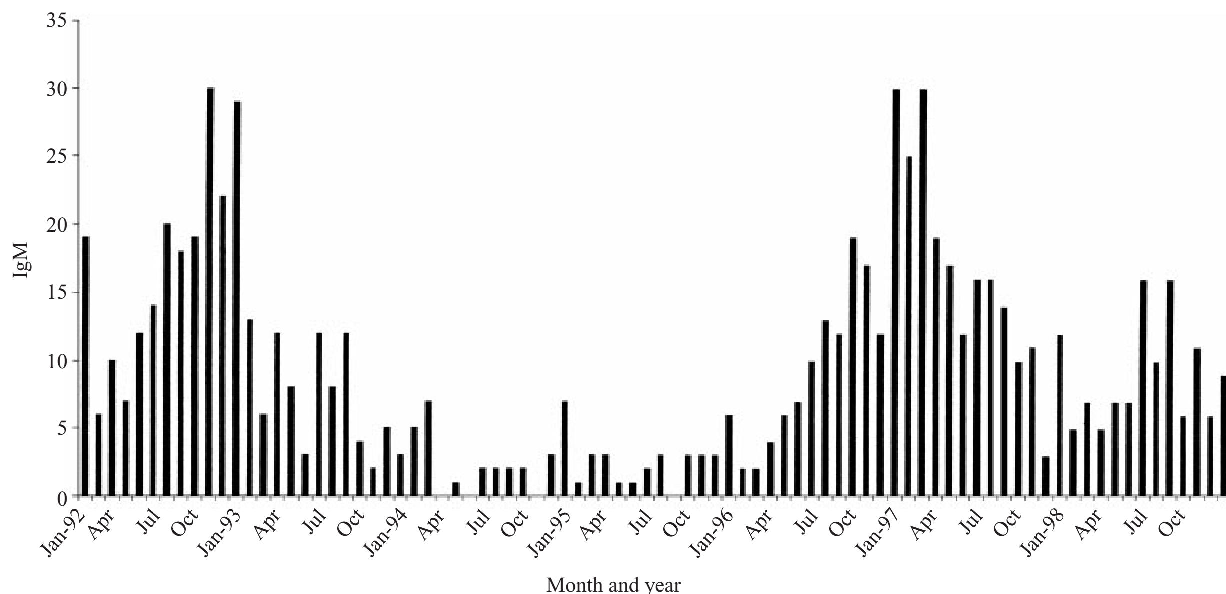

Fig. 1. The pattern of human parvovirus infection as defined by detection of parvovirus IgM from diagnostic specimens at the Victorian Infectious Diseases Reference Laboratory, 1992–8.

munity, the number of requests for testing was higher, as was the proportion of positive results. Figure 1 demonstrates the pattern of positive IgM tests by quarter between 1992 and 1998. The epidemic pattern of human parvovirus infection in Victoria appears to be described by a 4-year cycle, consisting of 2 higher frequency (epidemic) years followed by 2 lower frequency (endemic) years. The most common age group for the testing and detection of human parvovirus IgM varied each year from 5 to 9 years to

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20–34 years. Many of the 20–34 years age group were pregnant women.

Immunity to parvovirus in other parts of the world

Table 3 compares results from this study with those from studies in other countries. It appears that between 20 and 60% of women of child bearing age will be immune to parvovirus infection, depending on where in the world they live.

DISCUSSION

Most published studies have used samples of convenience to estimate the immunity to parvovirus in various populations and we have adopted the same approach. However, unlike studies of pregnant women or patients, the samples in this study were chosen to give an unbiased estimate of immunity over a wide age range and are likely to represent population immunity. Sera of patients from the Royal Children's Hospital were collected prospectively in 1997 at the end of an epidemic year, while those from the Royal Women's Hospital and the Red Cross Blood Service were collected the following year when there was a decrease in parvovirus prevalence. The sera stored at the Victorian Infectious Diseases Reference Laboratory had been collected over a number of years.

Because human parvovirus is not a notifiable disease in Victoria, we cannot test the assumption that IgM positive results from a reference laboratory are representative of the pattern of acute infection in the community. However rubella is a notifiable disease and we have compared the pattern of IgG and IgM positive samples from VIDRL with notifications of rubella to the Department of Human Services, Victoria between 1992 and 1998 (Fig. 2). The pattern of IgM positive samples at VIDRL has the same distribution over time as the pattern of notifications of rubella, supporting the similar assumption we have made for parvovirus.

Collection time is unlikely to account for differences in the seroprevalence of human parvovirus antibodies in various countries. In endemic years, reliable estimates of a seroconversion rate of 1·5% have been estimated, at least for pregnant women [9, 10], while in epidemic years or during local epidemics, seroconversion rates have been estimated to range from 13 to 19% [9, 11, 12]. Although parvovirus occurs in outbreaks, its periodicity has been less well described. This study and data from England and Wales [13] suggest that parvovirus has a 4-year cycle with 2 epidemic and 2 endemic years. Seroprevalence data collected and reported in 5- or 10-year age groups will therefore represent people who have been exposed in both epidemic and endemic years and the collection time should not therefore influence the estimate of population immunity.

However the variation in seroprevalence may reflect the assay used to estimate parvovirus IgG. Commercial assays which use a peptide that is likely to identify early IgG (for instance, Ferring Diagnostica Parvoscan, Sweden) are less suitable for seroprevalence studies than assays which use recombinant proteins (for instance, Biotrin, Dublin, Ireland; Dako, Glostrup, Denmark; and MRL Diagnostics, California, USA). In house comparison of these four commercial kits at VIDRL has shown that sensitivity ranged between 82 and 100% and specificity ranged between 57 and 100%. Other laboratories have also found problems with some of the commercial assays available. Parvoscan has been shown to be a poor indicator of population immunity with IgG levels quickly falling to undetectable levels after infection [14] and the MRL assay has been shown to be unsuitable for use with serum specimens that have been heat inactivated, in addition to having poor detection of low concentrations of IgG [15].

The age-specific immunity in England and Wales [16] is similar to the Victorian data and can be explained by exposure to school aged children. Parvovirus is primarily a disease of childhood and, in a community outbreak, the highest attack rate is amongst children of school age [17]. It appears that a proportion of non-immune adults exposed to this age group continue to be infected, with the majority of infections being asymptomatic [5]. Population immunity will plateau when adult exposure to school-aged children is uncommon. This interpretation is supported by the recent Danish study which demonstrated that the highest risk of parvovirus infection is associated with having school-aged children in the house and that the risk increases with the number of children [9].

Many studies of the epidemiology of parvovirus have concentrated on women because of the risk to the foetus. A higher prevalence of parvovirus immunity amongst women might be expected because of their domestic and occupational exposure, but this is not the case in this study and another study of blood donors [18]. On the other hand there are published reports of a higher apparent immunity amongst women [10, 19, 20].

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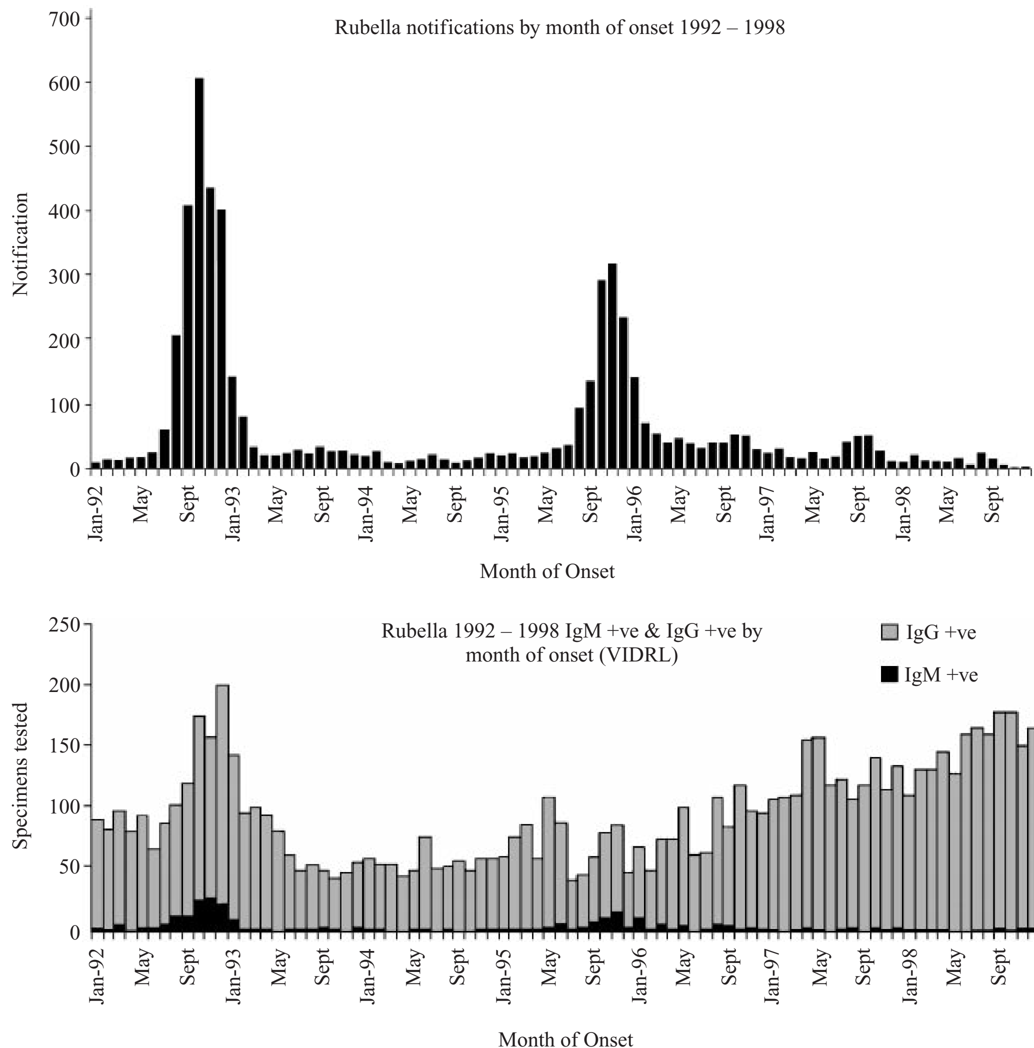

Fig. 2. Notifications of rubella to the Department of Human Services, Victoria compared with detection of rubella IgM at the Victorian Infectious Diseases Reference Laboratory, 1992–8.

Differences in population immunity between countries are unlikely to be explained by collection during epidemic or endemic years, but may be sensitive to the assay used. Parvovirus appears to be ubiquitous and to have a similar age-specific immunity in many countries, with at least half of pregnant women having evidence of previous parvovirus infection. However countries such as Taiwan [20], Hong Kong [21], Singapore [22] and South Africa [23] appear to have a different pattern of infection, resulting in population immunity among women aged 20–45 years of approximately 25–35%. A similar difference is seen for the epidemiology of chickenpox with lower seroprevalence estimates in tropical as compared with temperate countries [24]. It therefore appears that we cannot assume that approximately 50% of women of child-bearing age will be immune to parvovirus independent of country of residence. However a lower prevalence of immunity also implies a lower incidence of disease and this has been demonstrated in Japan in different epidemic cycles [25]. The risk of parvovirus infection during pregnancy is therefore also likely to depend on country of residence. In Australia this risk should be of the same order as that in other countries with a 4-year epidemic cycle and a prevalence of immunity amongst pregnant women of 50–60%.

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Table 3. *Estimated prevalence of immunity to human parvovirus infection in various countries*

Country	Sample size and source	Prevalence of parvovirus immunity	Assay used (See text for details of commercial assays)	Reference and year of publication
Australia	824 children, pregnant women and blood donors		Commercial enzyme-linked immunosorbent assay (Biotrin)	This study
	0–19 years	38%		
	20–39	60%		
	40+ years	76%		
Belgium	441 randomly selected blood donors	74%	Commercial enzyme-linked immunosorbent assay (Dako)	Letaief et al., 1997 [18]
Brazil	542 inhabitants of Belem, Para	43%	In-house antibody capture radioimmune assay	de Freitas et al., 1990 [26]
	461 members of 3 Brazilian tribes	5–11%		
	Serum samples from Rio de Janeiro		In-house antibody capture radioimmune assay and counter immunoelectrophoresis	Nascimento et al., 1990 [27]
	0–4 years	35%		
	11–15 years	80%		
	50+ years	90%		
Chile	92 blood donors	10%	Commercial enzyme-linked immunosorbent assay (no details)	Mata Rebon et al., 1998* [28]
Czech Republic	562 subjects		Not stated	Sodja et al., 1995* [29]
	0–4 years	10%		
	School age	27–36%		
	15+ years	53–58%		
Denmark	30, 946 pregnant women	65%	Commercial enzyme-linked immunosorbent assay (Dako)	Valeur-Jensen et al., 1999 [9]
England and Wales	1422 patients from an influenza survey		In-house antibody capture radioimmune assay	Cohen and Buckley, 1988 [16]
	0–10 years	27%		
	11–20 years	56%		
	21–40 years	54%		
	41+ years	80%		
Germany	138 medical students	34%	In-house enzyme-linked immunosorbent assay using viral antigen	Schwarz et al., 1992 [30]
	26 nurses	65%		
	197 pregnant women	24%		
	786 patients and 692 blood donors	38%	In-house enzyme-linked immunosorbent assay using viral antigen	Schwarz, Roogendorf & Deinhardt, 1987* [31]
	3289 routine patients	24%	In-house enzyme-linked immunosorbent assay using viral antigen	Schwarz et al., 1990* [32]
	586 healthy people		In-house indirect immunofluorescence assay using recombinant antigen	Eis-Hubinger et al., 1998 [33]
	20–25 years	63%		
	26–30 years	77%		
	31–45 years	71%		
	60+ years	78%		
Greece	308 healthy females	58%	Commercial enzyme-linked immunosorbent assay (Dako)	Kyriazopoulou et al., 1997 [34]
Holland	203 children and blood donors		Commercial enzyme-linked immunosorbent assay (Biotrin)	Mauser-Bunschoten et al., 1998 [35]
	0–20 years	31%		
	21–40 years	67%		
	41+ years	82%		
Hong Kong	276 patients with possible parvovirus infection	20%	Commercial immunofluorescence or enzyme-linked immunosorbent assay (Biotrin)	Lim, Wong & Lau, 1997 [21]
Japan	612 blood donors		In-house antibody capture	Tsujimura et al.,

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Country	Population	Seroprevalence	Assay	Reference
	16–25 years	44%	enzyme-linked immunosorbent assay using viral antigen	1995 [36]
	26–40 years	45%		
	41–55 years	71%		
	56+ years	90%		
	900 healthy individuals		In-house enzyme-linked immunosorbent assay using recombinant antigen	Matsunaga et al., 1995 [37]
	0–4 years	10%		
	5–9 years	54%		
	10–14 years	59%		
	15–19 years	46%		
	20–29 years	38%		
	30–39 years	48%		
	40–49 years	64%		
	50+ years	76%		
Kuwait	218 children less than 16 years old	17%	Commercial enzyme-linked immunosorbent assay (Dako)	Alsaeid et al., 1996 [38]
Mauritius & Rodriguez Islands	577 sera		In-house antibody capture enzyme-linked immunosorbent assay using viral antigen	Schwarz et al., 1989* [39]
	Sao Tome and Principe	51%		
	Malawi	58%		
	Mauritius	55%		
	Rodriguez Island	2%		
Norway	49 household contacts of patients with haemophilia	49%	In-house antibody capture radioimmune assay using viral antigen	Rollag et al., 1991 [40]
	45 blood donors	42%		
Portugal	435 healthy people	66%	Not stated	Araujo, Koch & Araujo, 1995 [41]
Saudi Arabia	517 healthy people aged 2–40 years	19%	Commercial enzyme-linked immunosorbent assay (Ferring Diagnostica)	al-Frayh et al., 1993 [42]
Singapore	600 healthy individuals		In-house enzyme-linked immunosorbent assay using viral antigen	Matsunaga et al., 1994 [22]
	0–4 years	0%		
	5–14 years	4%		
	15–19 years	8%		
	20–24 years	10%		
	25–34 years	28%		
	35+ years	65%		
South Africa	1967 pregnant women	25%	Commercial enzyme-linked immunosorbent assay (Mecconti, Hamburg, Germany)	Schoub et al., 1993 [23]
Spain	136 blood donors	65%	In-house enzyme-linked immunosorbent assay	Munoz et al., 1998* [43]
Sweden	457 pregnant women	81%	Two in-house enzyme-linked immunosorbent assays, one using a viral peptide, the other using a recombinant antigen	Skjoldebrand-Sparre et al., 1996 [44]
Taiwan	862 randomly selected healthy people	33%	Commercial enzyme-linked immunosorbent assay (Denka Seiken, Tokyo, Japan)	Lin et al., 1999 [20]
Tunisia	378 randomly selected blood donors	65%	Commercial enzyme-linked immunosorbent assay (Dako)	Letaief et al., 1997 [18]
United States	322 teachers and day care workers	58%	In-house antibody capture enzyme-linked immunosorbent assay	Gillespie et al., 1990 [11]
	2730 employees at 135 schools and 751 hospital employees	60%	In-house antibody capture enzyme-linked immunosorbent assay	Adler et al., 1993 [10]

* Abstract only in English.

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