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Results of a two-year study of humoral immunity to influenza A and B viruses in children under the age of 14 years in Moscow and its suburbs*

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A serological survey of antibodies to influenza A(H1N1), A(H2N2), A(H3N2) and B viruses was done with sera collected in Moscow in October 1980 and November 1981 from 542 children under 14 years of age. The results of the study showed convincingly that influenza A(H2N2) viruses were not circulating in Moscow in 1980-81. Low titres found in the sera from four young children were due to cross-reactions which were eliminated from the sera by absorption with A/USSR/174/79(H3N2) virus. Low-level HI titres with A(H0N1) virus in 11 sera were not confirmed by single radial haemolysis (SRH).

Serological data showed that A(H3N2) viruses were the main cause of acute respiratory disease in children in July-September 1980 and July-September 1981. These illnesses occurred at the end of the influenza A(H3N2) epidemic of 1979-80 in the third quarter of 1980. The influenza A(H3N2) virus circulated in Moscow during December 1981 and January 1982, but influenza did not reach epidemic levels. A low proportion (10%) of children with antibodies to influenza B virus at titres of 1:40 or higher in 1980 indicated the possibility of an epidemic due to this virus in Moscow in 1980-81. Such an epidemic did occur in December 1980 and January 1981.

The last five years have been characterized by a complicated pattern in the circulation of influenza A viruses in the world. Since 1977, two subtypes of influenza A—(H3N2) and (H1N1)—have circulated simultaneously, which had not been observed previously (*1*, *2*). In addition, antigenic variants detected over this period did not replace each other, but older and more recent drifted viruses have continued to be isolated at the same time (*2*, *3*).

Another unusual finding was the isolation of H2N2 viruses from children in the spring of 1980 in Leningrad (*4*). One of the strains, A/Leningrad/549/80, was kindly given to us for use as an antigen in the present study.

With these facts in mind and with the aim of anticipating the likely influenza epidemic in the coming season, we studied in the autumn of 1980 and 1981 the pattern of humoral immunity to influenza viruses in children younger than 13 and 14 years, respectively, who could not have encountered the influenza A(H2N2) virus during its previous circulation (from 1957 to 1968). As children are often the first to be affected by influenza in epidemics and pandemics caused by new shift and drift variants of virus (*6*), we attempted to use comparative data on the level of humoral immunity to influenza A(H1N1), A(H3N2) and B viruses in children who had, or had not, an acute respiratory infection in the pre-epidemic period (July-October 1980 and 1981) to forecast the influenza virus for the next epidemic season.

MATERIALS AND METHODS

Sera from 5 healthy children were used to standardize the haemagglutination inhibition test (HI).

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Inhibitor-resistant antigens of influenza A(H1N1), A(H2N2), A(H3N2) and B viruses (*7*) were used and all the sera were treated before use with RDE (receptor-destroying enzyme) prepared by the Central Public Health Laboratory, Colindale, London. Sera were collected from children less than 13 years of age in 1980, and less than 14 years of age in 1981. In 1980, 225 sera were examined, of which 69 were from children aged between 1 and 3 years, 97 aged 4–7 years, and 59 aged 8–12 years. In 1981, sera from 317 children were examined including 70 from those aged 1 to 3 years, 140 aged 4–7 years, and 107 aged 8–13 years.

The previous history of the children in respect of influenza and other acute respiratory diseases (ARD) was obtained from health centre cards covering the period from July until October 1980 and from February until October 1981.

It was found that, in 1980, out of 199 children whose health centre cards were checked, 71 had had an acute respiratory illness in the pre-epidemic season. Out of 317 children examined in 1981, 68 had had influenza or ARD in the spring and 78 in the autumn of 1981.

The level of humoral immunity to influenza A(H1N1), A(H3N2) and B viruses was evaluated by a calculation of the percentage of the patients who had antibody titres 1:20 or 1:40 and higher and by a calculation of the geometric mean titres. A comparison of the geometric mean antibody titres was done by the Student *t*-test. Confirmation of the specificity of antibodies against A/Singapore/1/57 (H2N2), found in the sera of 4 children aged 4–5 years, was done by the absorption of these sera by A/USSR/174/79(H3N2) virus. A volume of concentrated and purified virus was added to the sera. After 30 minutes at 37 °C the sera were freed from the virus-antibody complex and surplus virus by centrifugation at about 100 000 *g* for one hour, and then titrated by haemagglutination inhibition (HI) simultaneously with an unabsorbed control.

Forty-two sera with the highest titres of antibodies to A(H3N2), including all 5 positive in HI to the virus A/Singapore/1/57(H2N2), were tested by neuraminidase inhibition (NI) for antibodies to neuraminidase by the method described by Aymard-Henry et al. (*8*). In NI, the fresh allantoic virus-containing fluids of the following antigens were used: A/turkey/Massachusetts/65(Hav6N2), the neuraminidase of which was identical to the neuraminidase of the strain A/Singapore/1/57, the recombinants A/equi/Prague/1/56-Hong Kong/1/68(Heq1N2) and A/equi/Prague/1/56-Texas/1/77(Heq1N2).

The specificity of antibodies to the virus A/Shklaver/49(H0N1), according to the 1971 nomenclature of influenza A viruses (*5*), found in low titres in the sera of 14 children examined in 1981 was investigated by comparing with the single radial haemolysis (SRH) reaction. All sera were titrated by SRH using sheep erythrocytes treated with the viruses A/PR/8/34(H0N1) (nomenclature of 1971) or A/Khabarovsk/1/77(H1N1) according to the method of Tischer (*9*). Homologous immune rat sera were used as virus control and a normal rat serum and normal allantoic fluid as negative controls.

RESULTS

The results of HI tests on sera from 542 children are presented in Table 1. In 1980 and 1981 different strains were used with the intention of evaluating the immunity of the child population in 1981 to the latest variants of viruses A/Brazil/11/79(H1N1), A/Bangkok/1/79(H3N2), A/Bangkok/2/79(H3N2) and B/Singapore/222/79 that are at present circulating among the population. The highest geometric mean antibody titres (4.32–4.42 $\log_2$) were found against A/Texas/1/77(H3N2) and the related variant Bangkok/1/79. More than half of the examined children had antihaemagglutinins to these viruses at titres of 1:20 or higher. At the same time, almost half of all the children and 45.5% of 1–3-year-old children had antibody titres at 1:20 or more to influenza virus A/Hong Kong/1/68 with geometric mean antibody titres of 4.05–3.96 $\log_2$. It should be noted that antibody titres to the above-mentioned early strains of influenza A(H3N2) virus were almost at the same level in the autumn of 1980 and 1981. The geometric mean titre to the rarely encountered A/Bangkok/2/79 was only half that of other variants. In children examined in 1981, far more had antibodies to A/Brazil/11/79 than had children in 1980 to the strain A/USSR/90/77.

Antibodies to influenza B virus were more frequently found to the variant B/Singapore/222/79 in 1981 ($P < 0.01$) than to the strain B/Hong Kong/5/72 in 1980.

Antibodies to A/Leningrad/549/80(H2N2) in 1980 and to the strain A/Iksha/1/57(H2N2) in 1981 were not found. Antibodies to A/Singapore/1/57 at 1:20 and 1:40 were found in the sera of 5 children (Table 2), one of whom was 12 years old and the others 4–5 years old. In the sera of all these children, the highest antibody titres were to influenza A(H3N2) virus — Bangkok/1/79 and Texas/1/77. Absorption of these sera with concentrated and purified virus A/USSR/174/79(H3N2) led to the removal of antibodies to the virus A/Texas/1/77 and simultaneously the antibodies to the virus A/Singapore/1/57 (H2N2). In these 4 sera, antineuraminidase activity to the neuraminidase of the strain A/Texas/1/77 (H3N2) was 2–3 times higher than to the neuraminidase of the virus A/turkey/Massachusetts/65

Table 1. Comparison of antihaemagglutinin levels to various strains of influenza A and B viruses in the sera of children less than 14 years of age in the autumn of 1980 (225 children) and in the autumn of 1981 (317 children)

Antibody level indicated by:	Virus strain and year of investigation												
	A(H0N1)[a]		A(H1N1)		A(H2N2)		A(H3N2)					B	
	A/PR/8/34 1980	A/Shkla-ver/49 1981	A/USSR/ 90/77 1980	A/Brazil/ 11/79 1981	A/Singa-pore 1/57 1980	A/Iksha/ 1/57 1981	A/Hong Kong/1/68 1980	A/Hong Kong/1/68 1981	A/Texas/ 1/77 1980	A/Bang-kok/1/79 1981	A/Bang-kok/2/79 1981	B/Hong Kong/5/72 1980	B/Singa-pore/ 222/79 1981
(1) Percentage of children with titre of 1:20 or higher	3.1	3.5	19.9	55.2	2.2	0.31	45.0	48.9	56.7	64.0	39.7	7.3	25.9
(2) Percentage of children with titre of 1:40 or higher	2.1	1.9	11.3	45.1	1.8	0	36.8	35.3	45.9	42.6	21.8	5.2	14.2
(3) Geometric mean titre[b] in $\log_2$	2.43	2.44	4.94	4.24	2.44	2.34	4.05	3.96	4.42	4.32	3.52	2.62	3.1

[a] According to the nomenclature of influenza A viruses of 1971 (*5*).

[b] In calculation of geometric mean titres, all negative sera were considered to have a titre of 1:5.

Table 2. Titres of antihaemagglutinin and antineuraminidase activity to influenza A and B viruses in 1980, by HI and NI testing of sera with antibodies to A(H2N2) from 5 children aged ⩽ 12 years

Serum number	Child's age (years)	Influenza virus strains								
		A/Leningrad/ 549/80(H2N2)	A/Singapore/ 1/57/(H2N2)		A/Hong Kong/ 1/68(H3N2)		A/Texas/ 1/77(H3N2)		A/USSR/ 90/77(H1N1)	B/Hong Kong/ 5/72
		HI	HI	NI	HI	NI	HI	NI	HI	HI
72	12	0	40[a]	0	80	0	80	80	20	160
195	4	0	20	20[a]	40	0	0	80	0	0
227	5	0	40	40	160	60	40	160	20	10
228	5	0	40	20	320	60	80	80	80	40
230	4	0	40	40	160	0	20	160	20	10

[a] Reciprocal of serum dilution.

Table 3. Percentage of sera from children showing antihaemagglutinin and antineuraminidase antibodies to specific strains of antigen

Strains	Antigen	Percentage of sera with antibody titre of:		
		< 1:20	1:20 1:40	1:80 1:160
A/Singapore/1/57	H2	91	9	0
A/turkey/Massachusetts/65	N2	88	12	0
A Hong Kong/1/68	H3	16	30	54
A/equi/Prague/1/56-Hong Kong/1/68	N2	56	31	13
A/Texas/1/77	H3	3	46	51
A/equi/Prague/1/56-Texas/1/77	N2	17	10	73

(Hav6N2). The results of the tests of antineuraminidase activity of 42 sera from children in comparison with the results of their titration in HI are presented in Table 3. The almost complete absence of antibodies to the neuraminidase of the strain A/turkey/Massachusetts/65, the neuraminidase closest to that of A/Singapore/1/57, together with the presence of antibodies to the neuraminidase of the virus A/Texas/1/77 at titres of 1:20 or higher in 83% of tested sera, is striking. Only 44% of these sera contained antibodies in such titres to the neuraminidase of the virus A/Hong Kong/1/68.

In the sera with a reaction to A(H0N1), as shown in Table 4, antibody to A/Brazil/11/79(H1N1) was also present. For the determination of the specificity of antibodies to the strain A/Shklaver/49(H0N1), all the 14 positive sera obtained in 1981 were examined by SRH with the viruses A/PR/8/34 and A/Khabarovsk/1/77(H1N1). As shown in Table 4, antibodies to Khabarovsk/1/77 were found by SRH in most of these sera, including the sera with titres of 1:20 and 1:40. At the same time, none gave a zone of haemolysis with the virus A/PR/8/34; a specific immune rat serum, however, gave a zone of haemolysis 6–7 mm in diameter.

Table 5 compares the level of antibodies to influenza A and B viruses in the sera of children who had been ill with ARD in the third quarter of 1980 and children who had no record of illness. It also compares the level of antibodies in children who had been ill with ARD in the spring or in the third quarter of 1981 and the children who had not had ARD from February until October 1981.

None of the sera of these five groups of children had antibodies at titres of 1:20 and higher to the virus A(H2N2) except for one 12-year old child from the group with ARD in 1980 who had a titre of 1:40 to A/Singapore/1/57. In the group of children who were not ill with ARD in 1980, one child had antibody to the same virus at a titre of 1:20. Neither the children who were ill nor those who were not ill with ARD in 1981 had any antibodies to A/Shklaver/49(H0N1). Of the children examined in 1980 who were ill, a slightly higher, and statistically significant ($t = 3.05$) proportion had antibody to B/Hong Kong/5/72 compared with those who were not ill with ARD in the

Table 4. Results of HI and SRH testing of sera with antihaemagglutinins to A(H0N1) antigen from 14 children aged ⩽ 14 years

			Antigens used			
			A/PR/8/34(H0N1)		A/Khabarovsk/1/77(H1N1)	
Test number	Serum number	Child's age (years)	HI test[a]	SRH (zone in mm)	HI test[a]	SRH (zone in mm)
1	12	12	40	no	80	10
2	13	10	20	no	80	6.5
3	17	6	10	no	20	9
4	19	3	20	no	40	no
5	20	13	40	no	80	8
6	21	3	40	no	160	not tested
7	23	4	40	no	80	11
8	92	5	20	no	40	11
9	129	6	10	no	0	no
10	191	14	10	no	0	no
11	203	5	20	no	160	11
12	223	3	40	no	80	4
13	275	10	10	no	40	10
14	285	2	10	no	10	no
15	Control immune serum	–	160	6	640	12

[a] Reciprocal of serum dilution.

third quarter of 1980. Higher titres were found in the sera of these two groups of children to A/USSR/90/77(H1N1); they were also statistically significantly different from one another ($t = 2.78$). The highest titres of antihaemagglutinins (5.5 and 3.9 $\log_2$) in both groups of children examined in 1980 were found to the influenza A/Texas/1/77(H3N2) virus, the prevalent virus in the epidemic of 1979–80 in the USSR. The difference in the level of antibodies in the sera of the two groups, both to A/Texas/1/77 and to A/Hong Kong/1/68, was statistically significant at the level of probability $P < 0.001$. The results on the three groups of children studied in 1981 showed higher geometric mean titres to the last variants of A(H1N1) and to influenza B. There was a high and statistically significant difference in the level of antibodies to A(H1N1) and A(H3N2) viruses in children who had been ill with ARD in the spring or in the third quarter of 1981, as compared with children who had not been ill. Moreover, this difference to A(H1N1) was more significant in the group of children who had been ill with ARD in the spring and, in the case of the difference to virus A (H3N2), in children who had been ill with ARD in the third quarter of 1981. However, differences in the level of antibodies in both groups of children who had suffered from ARD were not statistically significant ($t = 1.7$ for A(H1N1) and 1.4 for the virus A(H3N2)). The level of antibodies to the influenza B/Singapore/222/79 virus in the group of children who had been ill with ARD in the spring of 1981 was not statistically significantly higher than in children who had not been ill ($t = 1.76$) and essentially differed from that in children who had been ill with ARD in the third quarter of 1981 ($t = 3.6$).

DISCUSSION

An analysis of the antihaemagglutinin titres in the sera of 542 children during the autumn of 1980 and 1981 shows that viruses similar to A/Singapore/1/57(H2N2) were not in circulation in Moscow. Four sera with low (up to 1:40) titres to A/Singapore/1/57 virus were probably non-specific or cross-reactive, since in all sera the antibodies to strain A/Singapore/1/57 were present in a lower titre (Table 2) than to the viruses A(H3N2) and were completely removed by absorption with the virus A/USSR/174/79(H3N2).

The possibility that A(H0N1) or A(H1N1) 1933–49 viruses have circulated recently is not confirmed by

Table 5. Influence of acute respiratory diseases (ARD) on the antibody level to influenza A and B viruses in the sera of children examined in 1980 and 1981

		Strains to which antihaemagglutinins were revealed[a]																			
		A/Shklaver/ 49(H0N1)		A/USSR 90/77(H1N1)			A/ Singa- pore (H2N2)	A/Hong Kong/ 1/68(H3N2)			A/Texas/ 1/77(H3N2)			A/Bangkok/ 2/79(H3N2)			B/Hong Kong/ 5/72		
No. of ill/well children with dates	Date of serologi- cal exam- ination	GMT[b] in log_2	% of children with titre of 1 : 40	GMT in log_2	% of children with titre of 1 : 40	t	GMT in log_2	GMT in log_2	% of children with titre of 1 : 40	t	GMT in log_2	% of children with titre of 1 : 40	t	GMT in log_2	% of children with titre of 1 : 40	t	GMT in log_2	% of children with titre of 1 : 40	t
71 with ARD (July-Sept. 1980)	Oct. 1980			3.3	18.3	2.8	2.4	4.6	46.4	3.8	5.5	69.0	6.8				2.9	9.8	3.1
128 healthy (July-Sept. 1980)	Oct. 1980			2.7	7.8		2.3	3.5	25.0		3.9	32.8					2.4	0.8	
68 with ARD (Spring, 1981)	Nov. 1981	2.43	1.47	5.2	73.5	7.4	2.4	4.5	52.9	5.7	4.6	51.5	3.0	3.8	32.4	3.3	3.2	14.8	1.8
78 with ARD (July–Oct. 1981)	Nov. 1981	2.4	0	4.9	60.3	5.1	2.3	5.1	64.1	8.0	4.9	60.5	4.6	3.9	32.1	3.3	3.5	20.9	3.6
171 healthy (Feb.–Oct. 1981)	Nov. 1981	2.5	2.9	3.5	26.9		2.3	3.2	15.2		4.0	30.4		3.3	12.9		3.0	7.6	

[a] In 1981, instead of the strains A/USSR/90/70, A/Singapore/1/57, A/Texas/1/77 and B/Hong Kong/5/72, antigens of the strains A/Brazil/11/79, A/Iksha/1/57, A/Bangkok/1/79 and B/Singapore/222/80, respectively, were used.

[b] GMT = geometric mean titre.

our results. First, all sera positive for A(H0N1) contained at the same time antibodies in higher or equal titres to currently circulating variants of A(H1N1) 1977, and none of 14 sera with HI antibodies to A(H0N1) produced haemolysis with A/PR/8/34 in SRH, whereas a control immune serum produced haemolysis 6–7 mm in diameter. Most of the sera with HI titres of 1:20 and 1:40 to A/Brazil/11/79 gave zones of haemolysis with the virus A/Khabarovsk/77 in SRH, which confirms the specific nature of antihaemagglutinin to the virus A/Brazil/11/79 and suggests that low HI titres with the virus A/Shklaver/49 were cross-reactive or non-specific.

The high percentage (69%) of children with antibodies to the virus A/Texas/1/77, in the titre 1:40 and higher, who had been ill with ARD in the third quarter of 1980 and the statistically significant difference from the titres in the sera of children who had not been ill with ARD suggest that A(H3N2) was the main cause of their illnesses. This was apparently the final stage of the epidemic A(H3N2) 1979–80. A higher percentage (up to 73%) of sera had antibodies to the neuraminidase of A/Texas/1/77, the virus which caused the influenza epidemic of 1979–80. As shown in Table 3, the sera contained more antineuraminidase antibodies to the strain A/Texas/1/77 than to the strain A/Hong Kong/1/68, which suggests that although both neuraminidases belong to subtype N2, they are rather far apart antigenically and the high titres of antibodies to neuraminidase of A/Texas/1/77 may result from a new infection. The neuraminidases of the two subtypes, A/Singapore/1/57 and A/Hong Kong/1/68, have a close antigenic relationship (*10*, *11*). Perhaps this might explain the presence of antibodies in low titres to the neuraminidase of the virus A/Singapore/1/57 in a small number of sera. The presence of the statistically significant differences of the antibody level to A(H3N2) in sera of the children who had been ill with ARD in the spring and in the third quarter of 1981 and the titres in sera of the children who had not been ill with ARD (Table 5) supports the idea of continuing active circulation of these viruses among children in the period 1981–82 in the absence of an obvious influenza A(H3N2) epidemic in Moscow.

The low proportion of children with antibodies at titres 1:40 and higher to influenza B and A(H1N1) viruses in 1980 indicated the possibility of an influenza outbreak among Moscow children, as there had been no influenza B epidemics for 3 years. The higher geometric mean titre of antibodies to this virus in children who had been ill with ARD in the third quarter of 1980 suggest that some of their illnesses were caused by influenza B virus. Later, in November–December 1980 in Moscow, an influenza B epidemic occurred first among children and then among adults. The epidemic was followed by a considerable rise of antibody titres to influenza B virus in the sera of children examined in 1981. Statistically significant higher levels of antibodies to B/Singapore/222/79 in children who had been ill with ARD in the autumn of 1981, compared with the children who had not been ill with ARD, testifies to the continuation of influenza B virus circulation among children up to the autumn of 1981. The development of an epidemic of A(H1N1) in that period was less probable because of the higher level of antibodies, especially in adult sera (*12*), although from the higher level of antibodies in the children who had been ill in the third quarter of 1980, it seems that some of the ARD was caused by A(H1N1). This led to a small outbreak after the influenza B epidemic in March–April 1981 due to this A(H1N1) virus. For this reason the geometric mean titre of antibodies to virus A/Brazil/11/79 was the highest in the group of children who had been ill with ARD in spring 1981 (Table 5). From the analysis of data, it was anticipated that the epidemic of 1980–81 would be caused by virus A(H3N2). However, it was actually caused by influenza B virus and the spring outbreak by virus A(H1N1). The circulation of A(H3N2) in the winter of 1981–82, as evidenced by the data of serological diagnosis (*13*), did not reach an epidemic level.

Thus, for making a forecast, it is always necessary to take into account several factors: (1) the etiology of the disease; (2) the rise of new drift-variants of influenza viruses; (3) the level and dynamics of population immunity in different age groups; (4) the season of the previous outbreak with the same etiology, etc. Taking into consideration these principal indices, we successfully predicted the influenza B epidemic in 1980.

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RÉSUMÉ

RÉSULTATS D'UNE ÉTUDE DE DEUX ANS SUR L'IMMUNITÉ HUMORALE AUX VIRUS DE LA GRIPPE A ET B CHEZ DES ENFANTS DE MOINS DE 14 ANS À MOSCOU ET DANS LA BANLIEUE

Depuis 1977, deux sous-types de la grippe A, les sous-types A(H1N1) et A(H3N2), circulent activement parmi la population du monde entier. On a également observé le virus porcin A(H1N1) chez l'homme aux Etats-Unis d'Amérique et des virus analogues à la souche A/Singapore/1/57(H2N2) à Léningrad, en 1980.

Afin d'essayer de prévoir quelle serait la situation au cours des prochaines saisons de recrudescence grippale, nous avons effectué une enquête sérologique, pendant l'automne de 1980 et de 1981, sur des enfants de moins de 13 et 14 ans dont la plupart n'avaient pu être en contact avec le virus A(H2N2) au cours de sa précédente période de circulation (1957-69).

Pour déterminer la spécificité des anticorps anti-A/Singapore/1/57(H2N2) trouvés dans certains sérums, on a pratiqué une épreuve d'absorption croisée avec le virus de la grippe A(H3N2) ainsi qu'une épreuve d'inhibition de la neuraminidase (IN). Les résultats positifs obtenus en inhibition de l'hémagglutination (IH) avec le virus A(H0N1), en 1981, ont été comparés à ceux d'une hémolyse radiale simple (HRS).

L'étude des résultats de l'épreuve IH sur 542 sérums confirmé que des virus analogues à A/Singapore/1/77 (H2N2) n'ont pas circulé dans la population de Moscou en 1980-81.

Les anticorps IH anti-A/Singapore/1/57 qu'on a observés à un faible titre dans 4 sérums étaient apparemment des anticorps donnant des réactions croisées puisqu'ils ont été complètement éliminés par absorption avec le virus A/USSR/174/79(H3N2).

La totalité des 11 sérums trouvés positifs en IH, à des titres de 1/20 ou 1/40, vis-à-vis du virus A/Shklaver/49 (H0N1) ont été titrés en HRS avec les virus A/PR/8/34 et A/Khabarovsk/1/77(H1N1). Aucune hémolyse n'a été obtenue avec le virus A/PR/8/34, tandis que les résultats de l'épreuve ont été positifs avec la souche A/Khabarovsk/1/77(H1N1). Il semble donc que les faibles titres IH observés avec le virus A/Shklaver/49 correspondent à des réactions croisées et que les virus A(H1N1) de la période 1933-49 ne soient actuellement pas en circulation chez les enfants de Moscou et de la banlieue.

Une analyse comparative a été effectuée pendant deux ans sur les taux d'anticorps dirigés contre les virus de la grippe A et B dans les groupes de sérums uniques provenant d'enfants ayant ou non été victimes d'une maladie respiratoire aiguë au cours des saisons pré-épidémiques de 1980 et 1981. Cette comparaison a montré indirectement que la morbidité était principalement le fait du virus grippal A(H3N2) et, dans une moindre mesure, des virus A(H1N1) et B, spécialement en 1980.

Compte tenu de l'écart des titres sériques (moyenne géométrique) chez les enfants, on aurait pu prévoir que l'épidémie de 1980-81 serait provoquée par le virus A(H3N2). Pour prévoir les épidémies de grippe, il est nécessaire de tenir compte d'un certain nombre de facteurs, à savoir: 1) l'étiologie de la maladie, 2) l'apparition de nouveaux variants de virus grippal résultant d'un glissement (ou dérive) antigénique, 3) le degré et la dynamique de l'immunité collective au sein des différents groupes d'âge, 4) la saison de la poussée épidémique antérieure de même étiologie, etc.

Le faible pourcentage de sérums d'enfants présentant un titre d'anticorps égal ou supérieur à 1/40 vis-à-vis des virus de la grippe B et du sous-type A(H1N1) en 1980 pouvait laisser prévoir une épidémie de grippe B étant donné qu'il n'y en avait pas eu depuis trois ans. Par la suite, en décembre 1980, une épidémie de grippe B s'est effectivement déclarée dans la population de Moscou.

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