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Human monkeypox *

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Between October 1970 and May 1971, six cases of human infection with monkeypox virus were identified in Liberia, Nigeria, and Sierra Leone. Four of the cases were confirmed by virus isolation and two were diagnosed on the basis of epidemiological and serological investigations. All the cases occurred in unvaccinated individuals.

Post-infection serological studies showed high haemagglutination-inhibition and neutralizing titres to pox group virus in four of the cases. Repeated challenge vaccination of all cases with potent smallpox vaccine resulted in equivocal reactions.

In all, 24 susceptible household contacts were exposed to the infected cases, but none developed disease. All the contacts subsequently responded to vaccination with a primary reaction, thus confirming their susceptibility and ruling out asymptomatic infection.

INTRODUCTION

Twenty West and Central African countries, assisted by the US Agency for International Development, have successfully completed a 5-year cooperative regional programme of smallpox eradication. A combined approach of mass vaccination, surveillance, and epidemic control has resulted in a decrease in reported smallpox cases from 10 896 in 1967 to zero in the 12-month period July 1970-June 1971 (Fig. 1). Intensive surveillance including epidemiological and laboratory investigation of reported cases of rash diseases has failed to detect a single case of smallpox in the 20 countries since May 1970. Surveillance systems in West and Central African countries have, however, identified 3 cases of a pustular disease, clinically indistinguishable from smallpox, from which a non-variola poxvirus, monkeypox virus, has been isolated. Three additional human monkeypox cases have been found during field investigation.

The World Health Organization is currently engaged in a worldwide programme to eradicate smallpox by 1975. This programme is based on the assumption that the interruption of transmission will bring about eradication of the disease in man. As a previous eradication programme, against yellow fever, had

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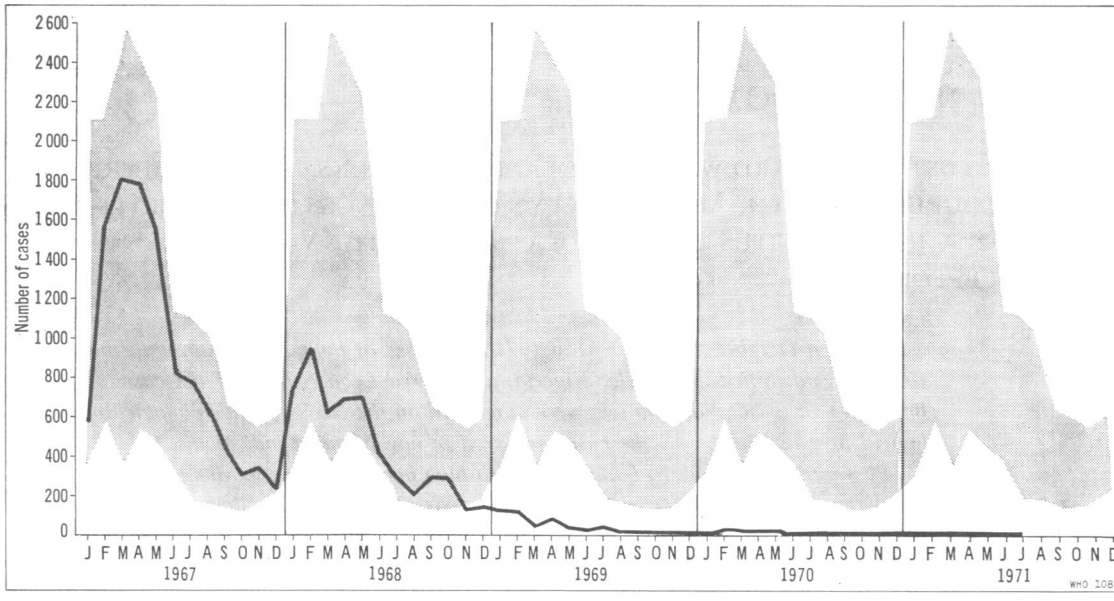

Fig. 1. Smallpox incidence in West and Central Africa, 1967–71. The shaded area represents the range between the highest and lowest incidence reported during the 5-year period 1962–66.

been severely hampered by the detection of non-human reservoirs of infection, WHO has carried out a search for evidence of non-human smallpox transmission, especially in primates. Although monkeys have been infected with and have transmitted variola virus experimentally in the laboratory, surveillance in smallpox endemic areas has failed to detect variola infection in natural primate populations (Hahon & McGavran, 1961; Noble & Rich, 1969).

In 1958, Magnus identified a pox disease in *Cynomolgus* monkeys in Copenhagen (Magnus et al., 1959). The etiological agent, known as monkeypox, has since been identified in animal colonies in the USA (Sauer el al., 1960; Prier & Sauer, 1960; McConnell et al., 1964) the Netherlands (Peters, 1966), and France (Milhaud et al., 1969). The virus has also been isolated in tissue cultures prepared from monkey kidney (Gispen & Kapsenberg, 1968).

Prior to the recognition of monkeypox virus, seven episodes of pox disease in monkeys had been described (Arita & Henderson, 1968). In four outbreaks, a direct association with human smallpox had been reported, but in only one outbreak was a poxvirus isolated (Anderson, 1861; Schmidt, 1870; Bleyer, 1922; Gispen, 1949). As techniques for poxvirus differentiation were not available at the time of that isolation, and as the isolate has since been lost, it is not possible to prove conclusively whether it or the other reported outbreaks of pox disease in monkeys were due to smallpox, monkeypox, or to another virus.

In addition to the cases of human monkeypox discussed in this paper, one further case has been identified in Zaire (Ladnyi et al., 1972; Marennikova et al., 1972).

CLINICAL MANIFESTATIONS

In West Africa, four cases of human monkeypox infection have been diagnosed by virus isolation (cases 1, 4, 5, and 6). Two additional cases (cases 2 and 3) have been diagnosed on epidemiological and serological grounds. The clinical information on the six cases, none of which was fatal, is summarized in Table 1.

Case 1

A 4-year-old unvaccinated female (L.T.) from Boudua, Liberia, became ill on 10 September 1970, with a " severe cold " manifest by fever, sore throat, and malaise. On 13 September, she developed a generalized rash and was admitted the following day to the district hospital in Zwedru as a suspected case of smallpox. Examination on 18 September, the fifth

Table 1. Clinical cases of human monkeypox infection, Liberia, Nigeria, and Sierra Leone, 1970–71

Case	Country	Village	Age	Sex	Vaccination history	Date of rash	Prodrome in days	Severity of rash [a]	Duration of rash (days)
1	Liberia	Boudua	4	F	Negative	13 Sept. 70	3	++	24
2	Liberia	Boudua	4	M	Negative	12 Sept. 70	1	+	4
3	Liberia	Boudua	6	F	Negative	13 Sept. 70	2-3	+	4
4	Liberia	Tarr	9	M	Negative	2 Oct. 70	?	++	21
5	Sierra Leone	Aguebu	24	M	Negative	1 Dec. 70	3-4	+++	28
6	Nigeria	Aba	4	F	Negative	19 May 71	5	+++	26

[a] + Mild
++ Moderate
+++ Severe

day of rash, revealed an afebrile, uncomfortable child with a diffuse vesiculopustular rash of peripheral distribution. The lesions were discrete with rare coalescence, were deep-seated, and were present on the palms and on the soles of the feet. The collection of specimens was difficult because of the viscosity of the pustular fluid and the adhesiveness of the scabs. Fig. 2 shows the skin lesions as photographed on the eighth day of rash. Adjacent lesions were all in the same state; cropping was not observed. A follow-up examination on 13 December showed a healthy, active child with scattered hyperpigmented spots on the face and extremities. No pitted scars were seen.

Case 2

A 4-year-old unvaccinated male (I. G.) from Boudua, Liberia, developed a low-grade fever on 11 September 1970. The following evening a mild vesicular rash, consisting of approximately 10 scattered lesions, was noted. These lesions cleared in 3–5 days and left no scars or pigment changes.

Fig. 2. Human monkeypox: eighth day of rash in 4-year-old female, Boudua, Liberia.

Case 3

A 6-year-old unvaccinated female (M.T.) from Boudua, Liberia, had a mild illness consisting of a 2–3-day febrile prodrome followed by a 3–5-day episode of rash. The rash consisted of 10 scattered vesicular lesions.

Case 4

A 9-year-old unvaccinated male (G.G.) from Tarr, Liberia, developed a rash on 2 October 1970, and was isolated on the family farm. When examined on 10 October, the ninth day of rash, the boy had a generalized vesiculopustular rash with all lesions appearing in the same stage. The lesions were firm, deep-seated, and measured approximately 0.75 cm in diameter. The lesions on the face and lower extremities had been broken by local treatment. The rash had a peripheral distribution with greatest involvement of the face, arms, and legs. Scattered lesions were present on the back, abdomen, and buttocks. Lesions were noted on the palms and on the soles of the feet. The patient had a warm tender mass in the right submaxillary area, which produced a generalized swelling of the right side of the face. A pustular lesion was present in the right cornea. The submaxillary mass, diagnosed as a bacterial abscess, was treated with penicillin. A follow-up examination on 26 October, the 25th day of rash, showed areas of depigmentation, a right-corneal scar, a subsiding submaxillary mass, and an active child. The areas of depigmentation were again noted on 20 November. Reexamination on 16 December revealed a 4-mm right-corneal scar.

Case 5

A 24-year-old single, unvaccinated male (B.K.) from Aguebu, Sierra Leone, became ill with severe headache, fever, stiff neck, and cough on 26 November 1970. Four days later he developed a generalized rash and was seen at the Moyamba Hospital, where the lesions were described as "pustular" and the diagnosis of smallpox was made. Examination on the tenth day of rash revealed a moderately ill man with a generalized pustular rash. Approximately half of the scabs had desquamated by 17 December, the 22nd day of rash. Reexamination of the patient on 1 January 1971, the 37th day of rash, showed residual hypopigmented lesions 3–12 mm in diameter, surrounded by 1–2 mm areolae of hyperpigmentation. Three to four shallow pits were noted on the face.

Case 6

A 4-year-old unvaccinated girl from Ihie Umuduru, Aba, Nigeria, developed fever, malaise, headache, sweating, and severe prostration on 14 April 1971. Five days later she developed a maculopapular rash that appeared initially on the distal extremities. On 13 April, the fourth day of rash, the child was isolated at Aba General Hospital with a clinical diagnosis of smallpox. Examination of the patient on 18 April, the eighth day of rash, showed a weak, feverish, lethargic, and toxic child who was unable to stand. There was a generalized pustular rash with lesions 3–5 mm in diameter. These pustules were firm, deep, discrete, and were not tender. The rash involved all areas of the body including the palms and the soles of the feet.

EPIDEMIOLOGY

Cases 1, 5, and 6 were identified by the communicable disease surveillance systems in Liberia, Sierra Leone, and Nigeria, respectively (Fig. 3). As each case occurred in an area that had been free of known smallpox transmission for at least one year, immediate investigation and control procedures were initiated on verification of the clinical diagnosis; these included: (1) the collection of specimens for laboratory diagnosis, (2) the identification and vaccination of susceptible contacts, (3) an active search for unreported cases, (4) an estimation of village smallpox immunity by scar survey, and (5) the vaccination of the susceptible population. Case 5 occurred in an area vaccinated in 1969. The other cases occurred in areas scheduled for vaccination during 1971.

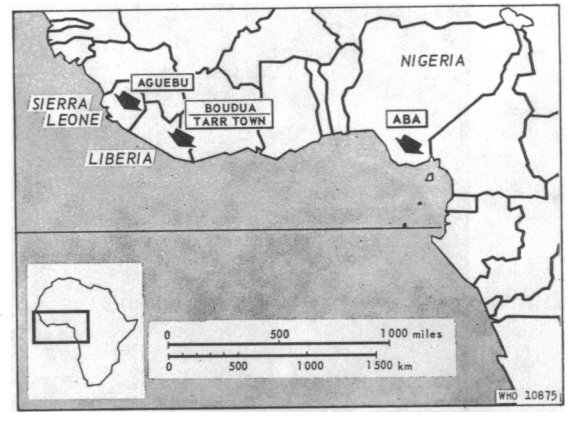

Fig. 3. Location of human monkeypox cases in Africa.

Case 1 lived in Boudua Town, a remote village of 27 people located in the tropical rain forest of north-eastern Liberia. Investigation of the contacts of the index case demonstrated the simultaneous occurrence of vesicular disease in two playmates, case 2 and case 3. As cases 1, 2, and 3 developed rashes at the same time, it is probable that infection resulted from a common exposure. As case 2 had been out of the village on 20–28 August, 16–24 days prior to the onset of rash, exposure must have occurred during the 16-day period from 29 August to 13 September. Except for the death of case 1's great-grandmother from a " stomach illness " on 9 September, no exposure to a sick person, either resident or visitor, could be recalled by any of the residents of Boudua Town.

At the time of the illnesses in cases 1, 2, and 3, 9 of the 23 residents of Boudua Town were classified as susceptible to poxvirus infection by the absence of a history of, or scarring from, smallpox disease or smallpox vaccination. None of the susceptible persons developed disease and all responded with a primary type of major reaction when vaccinated 2–4 weeks after exposure.

When poxvirus particles had been identified with an electron microscope and the agar gel test on specimens submitted from case 1 gave a positive result, a search for undetected smallpox transmission was launched. Liberian National Public Health Service personnel, assisted by local government officials, conducted a village-to-village active surveillance–assessment–vaccination programme in the northern two-thirds of Grand Gedeh County. This county has a population of 63 000 people living in remote villages in an area of about 18 000 km^2. Less than 10% of the population live in places accessible by motor road; the remainder of the population is reached over bush paths at distances up to 64 km. Active surveillance was directed towards the detection of smallpox cases that had occurred in the previous 12 months. Of 19 025 people examined by the surveillance agents, 15 suspect cases of pox disease were identified. Follow-up investigation of these cases identified varicella or other non-pox diagnosis in all cases except case 4.

Case 4 lived in Tarr Town in adjoining Niabo Clan and developed his rash on 2 October, 2½ weeks after the first three cases. Although the two infected towns lie approximately 19 km apart, contact between the two groups is limited because of administrative, linguistic, and traditional differences. Despite intensive interrogation, no direct or indirect contact between the infected cases or other people from the towns could be established. Similar surveillance, investigation, and vaccination programmes were carried out in Sierra Leone and Nigeria where direct inspection of all individuals living within an 8-km radius of the infected compound failed to identify additional cases of pox disease.

Table 2. Susceptibility of household and village contacts of monkeypox cases, Liberia, Nigeria, and Sierra Leone, 1970–71

Case	Household			Village contacts		
	No.	No. susceptible	Percentage susceptible	No.	No. susceptible	Percentage susceptible
1	23[a]	6	26	21	8	38
2	2	1	50	21	8	38
3	5	0	0	18	9	50
4	7	2	28	129	62	48
5	6	3	50	23	17	74
6	29	12	29	344	153	44
total	72	24	29	556	257	45

[a] Includes Zwedru compound and hospital room contacts.

The pox-susceptibility status of household and village contacts of the six cases, as estimated by scar surveys, is summarized in Table 2. None of these persons experienced a smallpox-like infection and thus no human-to-human transmission of infection could be demonstrated.

DIAGNOSIS

Virus was isolated from 4 of the 6 cases (Table 3). The characterization of the isolates as monkeypox virus is described in a paper by Lourie et al. (1972). Sera were collected from four of the six patients (Table 4).

SEARCH FOR NON-HUMAN SOURCE OF INFECTION

With the identification of monkeypox virus, investigation was directed towards the identification of an animal source for the human infections. All the cases had a limited exposure to domestic animals, including dogs, cats, poultry, sheep, goats, and pigs. Residential or farm contact with household rodents and bats was also possible. Cases 1–5 lived in the tropical rain forest where hunting of game was common. Monkeys

Table 3. Pox virus identification in human monkeypox cases, Nigeria, Liberia, and Sierra Leone, 1970–71 *

Case	Vesicular fluid		Crusts		
	EM	CAM	EM	AG	CAM
1	+	+	+	+	+
2	NT	NT	NT	NT	NT
3	NT	NT	NT	NT	NT
4	+	+	–	–	–
5	+	+	+	+	+
6	NT	NT	+	+	+

* EM = electron microscope examination for pox virus; AG – agar gel; CAM = pox virus isolation at 72 h on chorioallantoic membrane of embryonated eggs; + = positive; – = negative; NT = not tested.

and duiker were the most frequent game animals and served as the main sources of animal protein. No evidence of definite exposure to sick animals, wild or domestic, could be obtained in any of the cases. Cases 4 and 5 occasionally consumed freshly killed monkeys for food. During the field investigation cases 1, 2, and 3 were observed to play with internal organs removed from recently killed monkeys. However, no evidence of definite monkey contact could be established for any of the 5 cases in the 3 weeks prior to the onset of rash disease. Although monkeys have occasionally been observed near the village of case 6, monkeys are not eaten there and monkey exposure was highly unlikely.

Serological surveys for poxvirus antibody have been undertaken as a means of estimating the prevalence of pox infection. Studies carried out by the World Health Organization on over 2 000 monkey sera collected from Chad, from Malaysia, and from primate laboratories have failed to identify serological evidence of poxvirus infection (Arita et al., 1972). As part of the current investigation, serological specimens were obtained from selected species of monkeys collected in the Boudua and Tarr areas of Liberia (Table 5). Five animals had low level HAI titres to pox virus and one animal, a *Cercopithicus buttikoteri* from Tarr Town, had a neutralizing titre of 1 : 30 to vaccinia virus in LLcMK2 cells. The specificity of these low titres has not been determined. Sera have also been collected from cooperating laboratories in 6 countries (Table 6).

Table 4. Serological data on monkeypox cases, Liberia and Sierra Leone, 1970

Case	No. of days between onset of rash and specimen	Vaccinia				Herpes (CF)	Varicella (CF)
		HAI	CF	AG	Neut.		
1	90	64	8	+		<8	<8
2	13	64	<8	–	500	<8	<8
3	12	16	<8	–	128	<8	<8
4	8	128	16	+	1 024	16	<8
5	14	160	16	+	450	<8	<8

HAI = haemagglutination inhibition; CF = complement fixation; AG = agar gel; Neut. = neutralization.

Table 5. Poxvirus haemagglutination inhibition and neutralizing antibody titres of 18 monkey sera collected from Grand Gedeh County, Liberia

Species	No.	HAI titre		Neut. titre
		<8	8	1 : 30
Cerpithecus buttikoferi	5	3	2	1
Cercopithecus cambelli cambelli	5	5		
Procolubus badius badius	1	1		
Procolubus verus	1	1		
Colubus polykomos polykomos	1	1		
Cercopithecus diana diana	2	1	1	
Cercocebus torguatus atys	1		1	
Cercopithecus nictitans stampfli	1		1	
Perodictius potto potto	1	1		

DISCUSSION

Human monkeypox had not been recognized prior to 1970. At the time of initiation of the West African regional smallpox eradication programme in 1966, vesicular diseases were diagnosed on clinical criteria and an estimated 5% of smallpox cases were officially reported. During 1967 and 1968, regional efforts were directed towards the identification and control of smallpox outbreaks. A total of 40 pox isolates collected during this period have been tested for monkeypox and all have been reconfirmed as variola. Since 1969, every reported case of suspect pox disease has received the thorough epidemiological and laboratory investigation necessary to identify a low fre-

Table 6. Pox virus haemagglutination titre from selected African mammals

Source	Animal	No. of specimens	No. of positives	HAI titre
Ivory Coast [a]	monkeys	38	2	1 : 5 1 · 40
Chad [b]	mammals	189	3	1 : 5 1 : 40 1 : 80
Senegal [c]	rodents	54	0	
Sierra Leone [d]	chimpanzees	16	4	1 : 5 (3) 1 : 10 (1)
Liberia [e]	chimpanzee	2	0	
Nigeria [f]	patas monkeys	55	0	

[a] Institut d'Hygiène, Abidjan.
[b] Veterinarian Institute, Farcho.
[c] Institut Pasteur, Dakar.
[d] Ministry of Health, Freetown.
[e] Imported into USA.
[f] West African Council for Medical Research, Lagos.

quency non-variola pox disease such as monkeypox. Although human infection with monkeypox could be a new occurrence, it is most probable that the developments in surveillance of the smallpox eradication programme have brought about the recognition of human monkeypox.

Between October 1970 and May 1971, six cases of human infection with monkeypox virus were identified in three West African countries, Liberia, Nigeria, and Sierra Leone. Four of the cases were confirmed by virus isolation and two were diagnosed on the basis of epidemiological and serological investigations. All cases occurred in unvaccinated individuals. Post-infection serological studies showed high HAI and neutralizing titres to pox group virus in cases 2–5. Repeated challenge vaccination of all cases with potent smallpox vaccine resulted in equivocal reactions. This lack of a response to post-infection vaccination is consistent with laboratory studies that show cross-protection between vaccinia, variola, and monkeypox viruses (Gispen et al., 1967).

In all, 24 susceptible household contacts were exposed to the infected cases, but none developed disease. All the contacts subsequently responded to vaccination with a primary reaction, thus confirming their susceptibility and ruling out asymptomatic infection. If monkeypox in West Africa is as infective as smallpox, which has an infection rate of 37 cases per 100 susceptible household exposures, 9 cases of monkeypox would have been expected among the 24 contacts (Foster & Smith, 1970). This difference between the number of expected and observed cases is highly significant and indicates that monkeypox has a much lower rate of human-to-human transmission than smallpox, possibly zero.

Human monkeypox appears to be the result of chance infection of man with an animal virus. The natural reservoir, pattern of transmission, and route of infectivity remain unknown. Animals infected with monkeypox virus develop high antibody titres that persist for months and probably years (Wenner et al., 1968; Gispen, personal communication [1]). Antibody studies on sera collected from animals in Liberia (Table 5) and West Africa (Table 6) have shown low-level HI titres in a small number of animals. Although these titres are much lower than those seen in infected animals and may be due to nonspecific inhibiting factors further studies, including neutralization tests, are required. Surveys of the mammalian population in Liberia and Nigeria are being carried out at present to identify the animal reservoir of infection.

Animal infection with variola virus has been postulated as a possible threat to the achievement of worldwide smallpox eradication. Since monkeypox is transmitted by man to only a slight extent if at all, it cannot be considered a direct threat to smallpox eradication. However, the identification of a clinical syndrome indistinguishable clinically and serologically from smallpox points to the importance of obtaining a viral diagnosis from all suspected cases of smallpox in countries in which eradication has been, or is about to be, achieved. Diagnosis of monkeypox as smallpox could falsely invalidate methods and programmes used in the achievement of eradication. The progress of smallpox eradication in West Africa, which has given great impetus to the worldwide eradication programme, could have been jeopardized if the etiology of the cases reported in this paper had not been identified. Misdiagnosis of smallpox as monkeypox could lead to failures in uncovering undetected transmission and to a failure of eradication. Thus, the discovery of human monkeypox emphasizes the need for surveillance and full epidemiological and laboratory investigation of all cases of pox disease in the worldwide eradication programme.

[1] Gispen, personal communication, cited by Arita et al. (1972).

RÉSUMÉ

MONKEYPOX CHEZ L'HOMME

D'octobre 1970 à mai 1971, durant les opérations de surveillance succédant à la campagne d'éradication de la variole menée avec succès dans 20 pays d'Afrique occidentale et centrale, on a découvert 6 cas d'infection humaine par le virus du monkeypox au Libéria, au Nigéria et en Sierra Leone; 4 ont été identifiés par isolement du virus et 2 après une enquête épidémiologique et sérologique.

Tous les cas sont survenus chez des sujets non vaccinés contre la variole. Chez 4 malades gravement atteints, les prodromes et les manifestations cliniques de l'affection étaient indiscernables de ceux de la variole. Les épreuves courantes de laboratoire — examen au microscope électronique, précipitation en milieu gélifié, ensemencement sur membrane chorio-allantoïde d'embryon de poulet — ont permis d'isoler 4 souches de poxvirus à partir de matériel (sérosité de vésicules, croûtes) recueilli chez les patients. Des tests complémentaires ont été nécessaires pour les identifier comme étant des virus du monkeypox.

L'examen sérologique pratiqué chez 4 convalescents a décelé des titres élevés d'anticorps inhibant l'hémagglutination et neutralisants pour le groupe des poxvirus. Chez tous les malades, la vaccination antivariolique pratiquée à plusieurs reprises a donné des réactions équivoques.

L'enquête épidémiologique a révélé que 3 des cas étaient survenus dans le même village, et résultaient probablement d'une exposition commune à l'infection. Aucune atteinte de monkeypox n'a été observée parmi un total de 24 contacts; tous ont été vaccinés avec succès contre la variole, ce qui atteste leur réceptivité et exclut l'éventualité d'une infection asymptomatique.

Le monkeypox chez l'homme semble résulter d'une infection fortuite par un virus animal dont on ne connaît ni le réservoir ni le mode de transmission. Lors d'enquêtes sérologiques sur des primates et autres mammifères africains, on a trouvé des titres d'anticorps très inférieurs à ceux que suscite l'infection expérimentale par le virus du monkeypox.

Les implications de la découverte de cas humains de monkeypox au regard des programmes d'éradication de la variole sont examinées.

REFERENCES

Anderson, I. (1861) *Study of fever*, London, J. Churchill, p. 180

Arita, I. et al. (1972) *Bull. Wld Hlth Org.*, **46**, 625

Arita, I. & Henderson, D. A. (1968) *Bull. Wld Hlth Org.* **39**, 277-283

Bleyer, J. C. (1922) *Munch. med. Wschr.*, **69**, 1009

Foster, S. O. & Smith, E. A. (1970) *J. Nigeria med. Ass.*, **7**, 41-45

Gispen, R. (1949) *Ned. T. Geneesk.*, **93**, 3687

Gispen, R. & Kapsenberg, S. G. (1968) *Versl. Volksgezondh., 1967*, pp. 140-144

Gispen, R. et al. (1967) *Arch. ges. Virusforsch.*, **21**, 205-216

Hahon, N. & McGavran, M. H. (1961) *J. inf. Dis.*, **109**, 294-298

Ladnyj, I. D., Ziegler, P. & Kima, E. (1972) *Bull. Wld Hlth Org.*, **46**, 593

Lourie, B. et al. (1972) *Bull. Wld Hlth Org.*, **46**, 633

McConnell, S. S. et al. (1964) *Amer. J. vet. Res.*, **25**, 192

Magnus, P. von et al. (1959) *Acta. path. microbiol. scand.*, **46**, 156-176

Marennikova, S. S., Šeluhina, E. M., Ma'ceva, N. N. & Ladnyj, I. D. (1972) *Bull. Wld Hlth Org.*, **46**, 613

Milhaud, C. et al. (1969) *Exper. anim.*, **2**, 121-135

Noble, J. & Rich, J. A. (1969) *Bull. Wld Hlth Org.*, **40**, 279-286

Peters, J. C. (1966) *T. Diergeneesk.*, **91**, 387-391

Prier, J. E. & Sauer, R. M. (1960) *Ann. N.Y. Acad. Sci.*, **85**, 951-959

Sauer, R. M. et al. (1960) *Amer. J. vet. Res.*, **21**, 377-380

Schmidt, M. (1870) *Zoologische Klinik Die Krankheiten der Affen*, Berlin, A. Hirschwald, p. 97

Wenner, H. A. et al. (1968) *Amer. J. Epid.*, **87**, 551-566