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HUMAN MONKEYPOX 1970-1979

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ABSTRACT

With the eradication of smallpox throughout the world increasing attention has been given to human monkeypox. This disease, first described in central Africa in 1970, resembles smallpox clinically but differs epidemiologically. Forty-seven cases of human monkeypox have occurred since 1970 in five central and western African countries; Zaire has reported 38 cases. The evolution of the illness and the sequelae of monkeypox and severe smallpox are the same; monkeypox has a case fatality rate of 17%. Smallpox vaccination protects against monkeypox. All cases have occurred in the tropical rainforest. Children below 10 years of age comprise 83% of cases. Case clusters have been observed in certain zones within countries and within families. person spread has possibly occurred four times; the secondary attack rate among susceptible very close family members was 7.5% (3 cases/40 contacts) and among all susceptible contacts was 3.3% (4 cases/123 contacts). lower than occurs with smallpox which is between 25-40%. The low transmissibility, coupled with the low frequency of disease, indicates that monkeypox is not a public health problem; however, more data are needed.

While many animals near human monkeypox cases have orthopoxvirus antibodies, the natural reservoir and vector of monkeypox virus is unknown. Studies are in progress to identify the natural cycle of monkeypox virus and to define better the clinical and epidemiological features of this disease.

Introduction

The last case of endemic smallpox was reported in Somalia in October 1977. A World Health Organization Global Commission certified the global eradication of smallpox in December 1979 and this decision will be presented to the World Health Assembly in May 1980 for final approval. Accordingly, most countries have stopped vaccination and others are expected to stop soon. Since immunity levels in the population will soon decline rapidly, it is extremely important that diseases resembling smallpox be carefully evaluated. Such investigations are essential to provide continuing assurance to health officials and the public alike that smallpox has indeed been eradicated and that continuing vaccination is no longer necessary. They will help to define further those diseases which, although bearing a clinical resemblance to smallpox, may differ in important epidemiological features.

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The most important of these diseases is human monkeypox, which was first detected in the Basankusu district, Equateur region, Zaire (formerly the Democratic Republic of the Congo) in 1970, two years after the last case of smallpox had occurred in the area. 1,2 Some features of the first 21 human monkeypox cases have been reviewed. 1-5 It is the purpose of this report to describe the clinical and epidemiological features of all 47 cases of human monkeypox reported from 1970 through 1979.

Clinical features

There is a two to four day prodromal illness with fever and prostration. The eruption then begins. As with smallpox, the lesions develop more or less simultaneously and evolve together at the same rate through papules, vesicles and pustules before umbilicating, drying and desquamating. This process usually takes about two to four weeks, depending on the severity of the disease. The distribution of the rash is mainly peripheral. Severe eruptions can cover the entire body (Photos 1-6), including the palms and soles. Six of the 47 cases (13%) had a mild illness (less than 25 lesions with no incapacity, not usually requiring medical care), 19 (40%) had moderate disease (more than 25 lesions, necessitating stopping most physical activity, usually requiring medical care) and 22 (47%) had severe disease (more than 100 lesions, severely incapacitated, requiring medical care (Table 1).

Most skin lesions are about 0.5 cm in diameter but some up to 1 cm have been seen. Lesions have been noted on the mucous membranes, the tongue and genitalia (Photos 4, 6). Lymphadenopathy, especially in the neck (submandibular and cervical) and inguinal areas, was particularly prominent in 17 cases (Photos 4, 5).

Pitting scars develop, are most often on the face, and diminish with time. Secondary infection of the lesions is common and this may play a role in scarring. About half of the scars from lesions seen initially on the face and body were detectable one to four years after the acute illness. Desquamation of crusts leaves areas of hypopigmentation. Hyperpigmentation follows after a few months and diminishes with time (Photo 7). In some cases large shallow residual scars are seen. In one case a primary corneal lesion caused unilateral blindness (Photo 7). Another patient who was vaccinated several years previously developed only one lesion (Photo 9) further emphasizing that some cases can be exceedingly mild and may go unreported.

Only four of the 47 patients (9%) had a vaccination scar. These were persons, ages 35, 30, 24 and eight years, who were vaccinated more than five years previously.

Eight (17%) of the patients died from monkeypox during the acute illness. They were between seven months and seven years of age. None had been previously vaccinated. Three other patients died of other causes two months, four months and 15 months respectively after their illness.

Comparatively few laboratory tests have been done on these patients. This is because there are often major delays in reaching the patient after notification, difficulties in collecting scrapings from lesions or obtaining serum and problems in procuring follow-up samples.

Table 2 gives virological and serological test results. For the 47 patients, monkeypox virus isolation confirmed the diagnosis in 30 among 40 patients from whom skin scrapings were taken. Of the remaining 17 patients, seven had poxvirus particles seen on electron microscopic (EM) examination of skin samples, but no virus could be isolated. Ten others had serological as well as clinical and epidemiological evidence for monkeypox infection; skin specimens were not collected from seven of these patients and another specimen was not suitable for testing.

The virus could be isolated from specimens taken up to 18 days after onset of the rash. A specimen from one patient (case 19) which was EM negative, was positive for monkeypox virus upon culture. The precipitation-in-gel test was less helpful than other virological methods.

Serological tests confirmed a previous orthopoxvirus infection in 34 of 35 patients tested (Table 2). The fluorescent antibody $test^6$ and radioimmune assay examination 7 identified monkeypox antibodies in sera from 19 of the cases. One of these patients was negative by virological tests and skin samples could not be collected from four others.

In an attempt to detect specific immunological defects, IgG and IgM were quantitated for six patients, using a radio-immunoassay technique. All monkeypox cases had elevated IgG and some were markedly increased; three cases had increased IgM. This may only reflect the consequences of parasitic, bacterial and viral infections to which these patients are exposed.

Epidemiology

Where

Cases have been detected in five countries: Zaire (38), Liberia (four), Nigeria (three), Ivory Coast (one) and Sierra Leone (one). One recent case, number 39, originating in Nigeria, was detected in Benin. All 47 cases have occurred in the tropical rainforest of western and central Africa, between 8° south and 8° north latitude, where hunting of wild animals for food is common (Figures 1, 2). All but two of the cases have lived in villages of about 200 to 1000 persons. One of these two cases (case 18) ate meat originating from the tropical rainforest before disease onset and the other (case 47) is under investigation.

When

The cases have occurred sporadically since August 1970. Six cases were reported in 1970, three in 1971, five in 1972, three in 1973, one in 1974, two in 1975, three in 1976, six in 1977, 12 in 1978 and six in 1979.

Cases occurred most often in the dry season although they are reported throughout the year (Figure 3). Twenty of the 38 cases in Zaire had onset of rash between January and March.

Who

Children were affected more frequently than adults. The mean age of patients was eight years (range seven months to 35 years); the median was four years. Thirty-nine of the 47 cases (83%) were below 10 years; 25 (55%) were below five years and five were below one year. Twenty-six patients were male and 21 were female. However, among patients older than 15 years, five of seven were women.

Clustering of cases

Clustering of patients in countries, in localities within countries and within families has been observed (Figures 1, 2). Three of four cases in Liberia (2, 3, 4) lived in one village as did two of the three patients in Nigeria.

In Zaire, the north-western Equateur region, one of nine regions, has reported 21 of 38 cases (55%). Within this region 13 of the 21 cases (62%) have been reported from the Bumba zone, one of 21 zones in the Equateur region. Kasai Oriental region has reported nine cases; eight came from the Kolé zone, one of 12 zones in this region. Seven were detected in Bandundu region and four of these were in the Popokabaka zone.

There have been five instances in which presumed co-primary cases occurred in the same family (cases 2-3, 11-12, 26-27, 32-33, 37-38). Case number four lived next door to one of these families (cases 2, 3). The interval between onset of illness was less than 24 hours for one case, one day for two cases, two days for one case, three days for one case and five days for one case; the latter patient (case 38) developed a rash seven days after the co-primary, case 37.

Person-to-person spread

In four families, the onset of rash of second cases occurred nine, 12, 15 and 17 days respectively after the first case (Table 3). These cases may have been infected from a common source or secondary transmission may have occurred. Three of the four index patients had severe disease and the fourth had moderate disease. The disease in secondary cases was milder than the primary case in two instances and in two episodes the secondary cases were of comparable severity. All of the index and secondary cases were unvaccinated. No cases of possible tertiary spread were found.

If it is assumed that all four cases represented person-to-person spread of monkeypox, the potential for transmission may be assessed by relating the number of cases to the total of susceptibles (those without a vaccination scar) among family and other close contacts. In this setting the contacts had a vaccination scar rate of 70% or more.

Within immediate family members (parents, siblings, children or spouse), the secondary attack rate among susceptibles was 7.5% (3/40) (Table 4); susceptible siblings of monkeypox cases had a 10% attack rate (2/20). Among all other persons having known face-to-face contacts with patients, including more distant relatives, the secondary attack rate among susceptibles was 1.2% (1/83). When all known susceptible contacts are included the secondary attack rate is 3.3% (4/123). This secondary attack rate is low compared to smallpox which is about 25-40%. 8,9

Surveillance for monkeypox

Human

In 1975, special vaccination scar and facial pockmark surveys were conducted among populations living near where human monkeypox cases had occurred during the previous four to five years in the Ivory Coast, Nigeria and Sierra Leone. Immunity levels were comparatively low in younger age-groups. Fifty-seven per cent. of 2125 children 0-4 years, and 29% of 8047 school-age children had no vaccination scars, indicating their probable susceptibility to monkeypox infection. In those areas, there was no evidence of other cases of monkeypox (or smallpox) as determined by surveys of facial pockmarks or by records at health units in the area.

From 1975 to 1977 additional facial pockmark surveys were conducted in western and central Africa to detect possible cases of smallpox and thus evidence of continuing transmission. Over 6 500 000 children of school age and younger were seen; 1 825 380 were of preschool age. None had facial pockmarks suggestive of smallpox or monkeypox other than for cases known previously. 10

Data from smallpox vaccination scar surveys done in 1978 and 1979 in villages where cases occurred and in surrounding villages are shown in Table 5. Less than one half of the children from 0-4 were vaccinated in villages where cases occurred and about 60% of this age-group was found vaccinated in nearby villages. The area in Nigeria where case number 39 occurred had substantially less vaccination coverage than in Zaire. In some areas of the Bumba zone over 90% of children less than 15 have smallpox vaccination scars as do virtually all adults, due to repeated vaccination associated with investigation of monkeypox cases.

During the past 10 years, 1652 specimens have been collected from patients with febrile eruptive disease in western and central Africa to confirm or rule out smallpox or monkeypox infection (Table 6). Some specimens were collected in preparation for visits of International Commissions to certify freedom from smallpox. Zaire has submitted 74% of these specimens and has reported 81% of the cases.

Since 1971 Zaire has had 14 surveillance teams in the field responsible for promoting surveillance, including distribution of specimen collection kits to health units. This accounts in part for the relatively large number of specimens taken. A widely publicized reward of about US\$ 40 is given to persons who report a confirmed monkeypox case. All but two of the monkeypox cases were detected and reported by the fixed health units. Two cases were found in Bumba zone by active surveillance in villages near known foci of monkeypox.

Within Zaire there has been variation in specimen collection activities in the regions (Table 7). The Equateur and Haut-Zaire regions have together submitted over 52% of the specimens. However, 21 of the 38 cases occurred in Equateur and none in Haut-Zaire. The ratio of monkeypox cases to specimens collected is highest in Kasai Oriental (one in eight). Roughly 40% of the African tropical rainforest is in Zaire. No cases have been found outside of rainforest areas in Zaire or elsewhere.

Animal

Although cases and outbreaks of monkeypox infections have occurred among non-human primates and other animals in laboratories and zoos in Europe and North America, no such cases have been detected in nature. Thus the source of human monkeypox infections is still unknown. 11 Epidemiological studies have suggested monkeys and/or rodents as a possible source but, until recently, only a small number of specimens for viral culture have been obtained. Earlier serological studies showed a low prevalence of orthopoxvirus neutralizing antibodies in mammals captured in western and central Africa. In one survey, 10 of 372 sera were positive; 3 seven were from non-human primates (four chimpanzees from Sierra Leone, two monkeys from the Ivory Coast, and one monkey from Liberia). Another sero survey failed to detect significant antibody in over 2000 sera taken from Asian and African non-human primates, although none of these animals is known to come from areas near human monkeypox cases. 12 However, recent surveys conducted in areas where human monkeypox cases have occurred have shown a 23% (50/215) prevalence of poxvirus neutralizing antibodies in non-human primates.4,13 A study done in Zaire near human monkeypox cases showed that 11 of 55 monkeys had neutralizing antibody. 14 Antibodies have also been found in rodents, other large mammals and birds in forest areas of the Ivory Coast4 and in rodents in Zaire. 15 it has not been possible until recently to determine whether these antibodies had developed in response to monkeypox virus infection or to infection caused by any other orthopox virus species which might have infected mammals and birds. Recent work has shown that monkeys found near human monkeypox cases have monkeypox specific antibody. 6,13 These and further refined tests will serve as valuable tools during current epidemiological studies underway in Zaire and others which are planned. Special ecological studies began in the Equateur region of Zaire in June 1979 and these results will be reported separately.

Although attempts to isolate monkeypox virus from animals captured near human monkeypox cases have failed, four whitepox virus strains have been identified in organs of animals captured in the wild near such cases. These "wild whitepox" strains came from kidney tissue of one chimpanzee (Pan troglodytes), one forest dwelling monkey (Cercopithecus ascanius) and two rodents (Heliosciurius rufobrachium, the sun squirrel; Mastomys natalensis, the multimammate rat) captured in Zaire. 16,17 Prior to these isolations two strains of whitepox virus had been previously isolated from routine Cynomolgus kidney cell cultures. 18,19 Whitepox virus cannot be distinguished from variola virus by current biological and chemical methods and, thus, these isolations are of special interest. These findings, however, must be interpreted with caution since it is not possible to conclusively rule out that these isolates might represent laboratory cross-contamination rather than primary isolations. Moreover, existing epidemiological evidence indicates that the virus causing

smallpox is not present in this area because (1) no human infections with a variola-like virus have occurred in Zaire during more than eight years since the last smallpox case occurred, and (2) it can be presumed that the surveillance system is sensitive enough to detect such cases, as many monkeypox cases have been found.

Discussion

Most cases of human monkeypox have a characteristic clinical appearance, with a two day prodrome and typical smallpox-like rash evolving over two to four weeks. Severe tymphadenopathy is more prominent among patients with monkeypox than those with smallpox. Six of 47 cases (13%) have been mild or very atypical, suggesting the possibility that unidentified cases may have occurred, especially in areas where surveillance is poor.

While clinical features cannot readily distinguish between smallpox and monkeypox, the epidemiological features are quite distinct. Human monkeypox is a sporadic infrequent disease detected in small villages in the tropical rainforest of central and western Africa. Only four episodes of possible secondary spread of human monkeypox have been recorded. The 7.5% inter-human transmission rate of monkeypox to susceptible close family members is much less than that for smallpox. This very limited avidity of monkeypox for humans indicates the virus is probably a zoonoses. In addition to these important epidemiological differences, monkeypox and variola virus, both orthopoxviruses, have distinct biological and genetic differences. 20 , 21

The occurrence of human monkeypox in small villages in the moist rainforest of west and central Africa gives only general clues as to the source of this infection. The disease appears to be more frequent in the dry season, similar to that seen for smallpox. Whether this relates to a possible respiratory mode of transmission, as occurs with smallpox, is unknown. The geographical distribution may represent, in part, an artefact and may be a function of the sensitivity of surveillance and the number of specimens sent for analysis.

Most people in these areas have multiple contacts with a variety of wild animals. All cases have had contact with primates and almost all have had contacts with two or more other animals. Young, unvaccinated children and adult females seem to be at special risk; the former, perhaps, because they play with the carcasses of animals after the hunters return to the village and the latter because they are responsible for dressing and cooking the animals and may inoculate themselves during this procedure.

The incubation period of naturally transmitted human monkeypox is unknown, although experiments with monkeys infected by the parenteral route have shown an incubation period of 7 to 14 days resembling that of smallpox. Based on evidence from the few cases who had only one or two animal contacts in the month preceding infection, the incubation time in humans may be about 12 days, similar to that observed in smallpox. Possible "secondary" cases were generally milder and atypical, if indeed they represented inter-human spread rather than primary infection from a common source. When the level of vaccination immunity decreases in Zaire and elsewhere in west and central Africa, a possible increase in cases of human monkeypox may occur, thus providing further information.

Although the natural source of human cases is still obscure, epidemiological and serological surveys suggest that certain animals (forest-dwelling monkeys, squirrel, porcupine or pangolin) may be involved in the natural cycle of transmission. Field studies will focus initially on these animals. Concurrently, serological techniques are being developed which will permit precise measurement of past infection with monkeypox and other orthopoxviruses. This will greatly aid epidemiological investigations of human and animal populations.

The finding of whitepox virus requires that, as for monkeypox, surveillance be maintained and specimens promptly collected and tested if monkeypox/smallpox-like disease occurs in the tropical rainforest. Laboratory analysis of these and similar strains is necessary for continued confirmation that there is no animal reservoir of various virus and to monitor orthopoxviruses that might menace humans in the future.

Postscript

Monkeypox case 48: A 3 year old unvaccinated girl from the forest area of southern Cameroon developed a severe rash on 14 September 1979. Specimens were collected on 30 September. Due to special problems, monkeypox virus was not isolated from these specimens until January 1980. In February 1980 a complete field investigation was done by a joint team from the Cameroon Ministry of Health, the Organisation de Coordination pour la Lutte contre les Endémies en Afrique Centrale (OCEAC) and the World Health Organization. The team confirmed the diagnosis. No other human cases were detected. The child had contact with a dead squirrel about two weeks before the illness began. This is the first case of monkeypox reported from the Cameroon.

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REFERENCES

- Ladnyj, I. D., Ziegler, P. & Kima, A. (1972) A human infection caused by monkeypox virus in Basankusu Territory, Democratic Republic of the Congo, <u>Bull. Wld</u> <u>Hlth Org.</u>, <u>46</u>, 593-597
- Marennikova, S. S. et al. (1972) Isolation and properties of the causal agent of a new variola-like disease (monkeypox) in man, <u>Bull-Wld Hlth Org.</u>, 46, 599-611
- 3. Foster, S. O. et al. (1972) Human Monkeypox, Bull. Wld Hlth Org., 46, 569-576
- 4. Breman, J. G. et al. (1977) Human poxvirus disease after smallpox eradication, Amer. J. trop. Med. Hyg., 26, 273-281
- Arita, I. & Henderson, D. A. (1976) Monkeypox and whitepox viruses in West and Central Africa, <u>Bull. Wld Hith Org.</u>, <u>53</u>, 347-353
- Gispen, R., Brand-Saathof, B. & Hekker, A. C. (1976) Monkeypox specific antibodies
 in human and simian sera for the Ivory Coast and Nigeria, <u>Bull. Wld Hlth Org.</u>, <u>53</u>,
 355-360
- 7. Walls, H. H., Ziegler, D. W. & Nakano, J. H. (1979) A study of the specificity of sequential antisera to variola and monkeypox viruses by radioimmunoassay, <u>Bull. Wld Hlth Org.</u> (In press)

- Rao, A. R. et al. (1968) Epidemiological studies in smallpox. A study of intra-familial transmission in a series of 254 infected families, <u>Indian J. med. Res.</u>, <u>56</u>, 1826-1854
- 9. Foster, S. W. & Smith, E. A. (1970) The epidemiology of smallpox in Nigeria, <u>J. Nigeria</u>

 <u>Med. Assoc.</u>, <u>7</u>, 41-45
- 10. Breman, J. C. (sic) (1978) Bilan et enseignements de la campagne d'éradication mondiale de la variole, Med. Mal. Infect., 11, 550-558
- 11. Arita, I. & Henderson, D. A. (1968) Smallpox and monkeypox in non-human primates,
 Bull. Wld Hith Org., 39, 277-283
- 12. Arita, I. et al. (1972) Outbreaks of monkeypox and serological surveys in non-human primates, <u>Bull. Wld Hlth Org.</u>, 46, 625-631
- 13. Breman, J. G., Bernadou, J. & Nakano, J. H. (1977) Poxvirus in West African non-human primates: serological survey results, <u>Bull. Wld Hith Org.</u>, <u>55</u>, 605-612
- 14. Marennikova, S. S. et al. (1975) The results of examinations of wildlife monkeys for the presence of antismallpox antibody and viruses of the smallpox group, Vop. Virus, 3, 321-326 (In Russian)
- 15. Marennikova, S. S. (1979) Field and experimental studies of poxvirus infections in rodents, Bull. Wld Hlth Org., 57, 461-464
- Marennikova, S. S. et al. (1972) Poxviruses isolated from clinically ill and asymptomatically infected monkeys and chimpanzee, <u>Bull. Wld Hith Org.</u>, <u>46</u>, 613-620
- 17. Marennikova, S. S., Shelukhina, E. M. & Shenkman, L. S. (1976) "White-wild" (variola-like) poxvirus strains from rodents in Equatorial Africa, <u>Acta Virol.</u>, <u>20</u>, 80-82
- Marennikova, S. S., Gervich, E. B. & Shelukhina, E. M. (1971) Identification of virus indistinguishable from variola virus among monkeypox virus strains, <u>Vop. Virus</u>, <u>4</u>, 470-473 (In Russian)
- 19. Gispen, R. & Brand-Saathof, B. (1972) "White" poxvirus strains from monkeys, <u>Bull. Wld</u>
 Hlth Org., 46, 585-592
- 20. Fenner, F. (1977) The eradication of smallpox, Prog. med. virol., 23, 1-21
- 21. Mackett, M. & Archard, L. C. (1979) Conservation and variation in orthopoxvirus genome structure, J. Gen. Virol. (In press)
- 22. Cho, C. T. & Wenner, H. A. (1973) Monkeypox Virus, Bact. Rev., 37, 1-18

TABLE 1. HUMAN MONKEYPOX CASES IN WEST AND CENTRAL AFRICA, 1970-1979

* Severity: 1 = Mild (less than 25 lesions with no incapacity, not usually requiring medical care)
2 - Moderate (more than 25 lesions, necessitating stopping most physical activity, usually requiring medical care but not always hospitalized)
3 = Severe (more than 100 lesions, severely incapacitated, requiring medical care)

(B) = Bumba zone

TABLE 2. VIROLOGICAL AND SEROLOGICAL EXAMINATIONS DONE ON MONKEYPOX CASES

		Ví	rological te	sata		Se	rologi	cal test	8	
Case No.	Days after rash	EM	Virus isolation	Precipitation in gel	Days After rash			antibodi il of tit		Monkeypox specific
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^{*} Specimen in formol.
** Elisa titre 256.

^{... =} No specimen; FA = Fluorescent antibody; RIA = Radioizmunoassay antibody; NT (virological) or blank space (serological) = not rested.

TABLE 3
PAIRS OF CASES OF HUMAN MONKEYPOX REPRESENTING POSSIBLE SECONDARY SPREAD

									Lab	Laboratory Results	y Res	ults			
4		**************************************	4 0 0	Date	Vacc.			Virological	al			Se	Serological	ca1	
ຕຸ ໝໍ		X D C	Kelation	onser rash	scar	Severity*	Ž,	Virus	Precipi-	Days after	Ort (rec	hopoz iproc	Days Orthopox antibodies after (reciprocal of titre)	odies :itre)	Monkeypox
							17.1	isolation	in gel	rash onset	H	CF	Neut	RIA	specific antibody
4		Ć.,	i	9.4.71	1	m	+	+	+	1 518	:	:	70	;	FA+
24		[Eq.	Mother of case 7	18.4.71	ı	, - -1	1	ı	:	1.509	:	:	178	:	FA+
ς.			-	10.1.73	-	2	+	+	+	1 491	80	20	20 1 200	230	RIÅ+
Ŋ		F	Sister of case 15	22.1.73	Doubt	62	+	•	l	1.482	80	20	049	007	RIA+
2		Ы	1	11.9.78	ı	3	+	+	+		;	:	:	:	
۱۹		×	Cousin of case 35	28.9.78	•	33	:	:	•	58	20	10	:	5 900	RIA+
ćΩ		Σ	•	5.2.79	ı	£)	+	necropsy	psy		;	:	:	:	-
6	6/12	M	Brother of case 43	20.2.79	•	2	;		•	** 70	32	:	:	:	:
l							Į.		***************************************		1	1	4		

.. Not done

FA = Fluorescent antibody; RIA = Radioimmunoassay antibody

*Severity: 1= mild (less than 25 lesions with no incapacity, not usually requiring medical care)

2= moderate (more than 25 lesions, necessitating stopping most physical activity, usually requiring medical care but not always hospitalization)

3 severe (more than 100 lesions, severely incapacitated, requiring medical care)

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TABLE 4

VACCINATION SCAR RATES OF CONTACTS OF HUMAN MONKEYPOX CASES AND SECONDARY TRANSMISSION RATES

Total contacts Without vaccination scar Secondary monkeypox cases	No. Attack rate among susceptibles (%)	* 171 40 23 3 7.5	276 83 30 1 1.2	447 123 28 4 3.3
Contact type To		Immediate family*	Other**	TOTAL

*Parents, siblings, children or spouse, living in same house during illness.

**All other persons having contact with patient during illness, including more distant relatives, but not necessarily living in same house.

VACCINATION SCAR SURVEYS DONE NEAR HUMAN MONKEYPOX CASES, 1978-1979 TABLE 5.

<u> </u>			1													1
		% vacc.	***	95	46	93	6	****	65	90	66	92	99	96		92
80 41	15+	No.	**	1 500	2 255	396	513	*	569	5 291	581	1 987	87	2 288	ı	15 467
g villag	4	% vacc.	****	76	93	95	83	水水水水水	55	92	* 58	95	95	89		91
Surrounding villages	5-14	No.	*	1 126	1 363	2 296	385	*	231	3 883	362	1 537	155	2 420	t	13 758
JS.	4	% vacc.	***	81	58	69	34	***	45	99	50	52	58	55		09
	9- 0	No.	**	627	1 212	278	256	*	144	2 602	197	766	106	1 796	ı	8 215
	+	% vacc.	100	*	*	98	94	83	63	89	66	96	100	97	100	77
urred	15+	No.	5 9			43	119	246	1 897	219	43	131	24	421	340	3 551
where case occurred	14	% vacc.	93	*	3 ¢	100	83	95	53	78	84	91	100	98	83	77
1	5-14	No.	28			21	71	332	485	148	20	107	Ó	226	196	1 640
Village	4-	% vacc.	88	*	- x	50	7	56	36	51	49	37	89	97	46	44
	9-0	No.	25			42	57	112	205	118	ניט	70	25	207	161	1 025
	Case No.		30	31	32, 33	34	35, 36**	37, 38	39***	07	41	42	43	44, 45**	46	Total

* Combined with data from surrounding villages.

** Secondary case.

*** Survey done in Omifounfoun, Oyo State, Nigeria; all others done in Zaire.

**** Age breakdown not available; 2558 persons seen, 93% vaccinated.

***** As case 40 was discovered while investigating cases 37 and 38, the age breakdown is included in the villages visited for case 40.

TABLE 6. NUMBER OF SPECIMENS RECEIVED BY WHO, GENEVA FROM SELECTED COUNTRIES IN SUB-SAHARAN AFRICA 1970-1979

Country	1970	1971	1972	1973	1974	1975	1976	1977	1978	1979
Angola*	ı	1		,	ı		ı	1	66	23
Benin*	ı	1	ı	1	1	+	يسر	ı	1(1)	40
Burundi	κ'n	ı	Ŋ	7	E)	-	ı	ı	1	,
Cameroon*	ı	ı	ı	1	,	1	ŧ	ı	ı	1
Central African Republic*	ı	ı	ı	,	1	,	ı	,	ı	m
Congo*	ı	ı	ن	ı	ı	ı	1	7	7	i
Gambia*	,	,	ı	ı	ı	,		,	ı	1
Ghana∻	r	ı	ı	ı	ı	-	ı	ı	,	'
Ivory Coast*	r	**(1)	ı	ı	ı	φ		J	,	ı
Liberia*	(4)	,	1	ı	ſ	6	ı	ı	1	ı
Nigerla*	1	**(2)	1	ı	ı	4	e'n	r	,4	t
Rwanda*	10	ı	1	73	ı	t	ı	c.j	ı	,
Senegal*	ı	4	i	ı	•	ı	1	1	ı	ı
Sierra Leone*	**(1)		ì	ı	1	ζ.	e.	H	1	н
Uganda	r	ı	ı	•	ı	Н	1	t	119	1
Zaire*	23(1)	67	140(5)	79(3)	63(1)	207 (2)	125(3)	181(6)	120(11)	213(5)
Zambia	ſ	ľ	ť	t	ī	2	ı	50	50	r
Total	36(6)	67 (3)	148(5)	(6) 58	(1) 99	239(2)	136(3)	238(6)	391(12)	246(5)

* Ras tropical rainforest.

** Specimens sent directly to WHO Collaborating Centre.

() Monkeypox case.

SPECIMENS RECEIVED IN ZAIRE BY REGION, 1971-1979

Region	Population est. 1974 (000)	% of pop.	Medircal	Surveill- ance units	1971	1972	1973	1974	1975	1976	1977	1978	1979	Total (1971-1979)	% monkey- pox cases	% specimens collected	Ratio monkeypox/ specimens collected
Bandundu	3 000	11.8	976	2	11	21(1)	F-	~	14(1)	4(3)	15(1)	38(2)	9	123 (7)	1.8	10.1	1/18
Bas Zaire	1 700	6.7	236		14	4	r.	m	7	æ	18	2	,	50	ì	4.1	
Equateur	2 700	10.6	405	2	8/17	47(3)	49(3)	9(1)	33	77(2)	58(3)	(5)99	144 (4)	458/1/(21)**	55	36.6	1/22
Haut-Zaire	3 600	14.1	618	2	r-	91	6	Γ-	104	21	5 2	-	60	202	ı	16.6	•
Kasai Occidental	2 800	11.0	268	-	72/64	15	۰.	01		2	2	en .	-ਰ	<i>(2)</i> *6	t	7.4	ı
Kasai Oriental	2 100	8.3	386	-	11	6(1)	2	2	6(1)	2	16(1)	18(5)	13(1)	76 (9)	54	6.2	1/8
Kinshasa	2 400	9.6	120		21	+- -	ν	۴-	11	2.7	13	ν'n	,	97	ı	7.9	1
Kiw	3 900	15.4	645	7	20	0,	е	œρ	φ	'n	14(1)	7	4	73 (1)	m	6.0	1/13
Shaba	3 200	12.6	164	2	27	12	6	œ	<u>س</u>	1	4	1	ব	54	,	5.2	, •
TOTAL	25 400	100.0	100.0 4 148	14	168/3/ 138(5)		(6)68	53(1)	189(2)	108(3)	164(6)	164(6) 144(11)	184(5)	184(5) 1.237/3/(38)	100	100.1	1/34
-					1			1		1							

*
Source: Ministry of Public Health, Zaire; discrepancies noted with WHO records due to administrative factors such as time specimens received, multiple specimens received from some patients, etc.

** Includes one monkeypox case detected in 1970; that case not included in ratio monkeypox cases/specimens collected.

 $\star\star\star$ 23 specimens taken in 1970 and not included because breakdown by region not available.

() Monkeypox.

🖊 Variola.

FIG. 1 DISTRIBUTION OF TROPICAL RAIN FOREST AND 47 HUMAN MONKEYPOX CASES 1970 - 1979

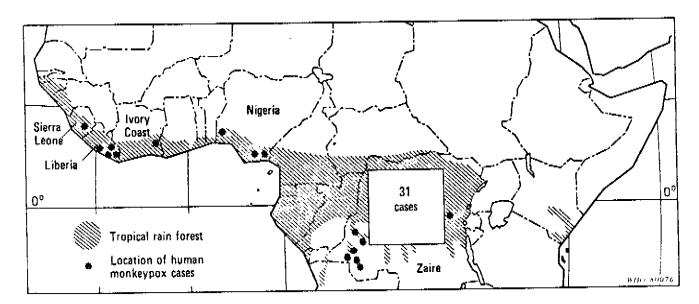


FIG. 2 DISTRIBUTION OF 31 HUMAN MONKEYPOX CASES IN NORTH-WESTERN AND CENTRAL ZAIRE, 1970 – 1979

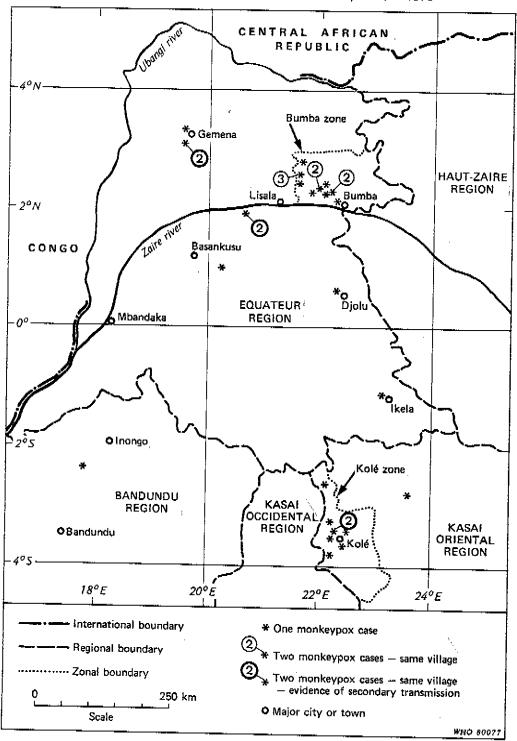
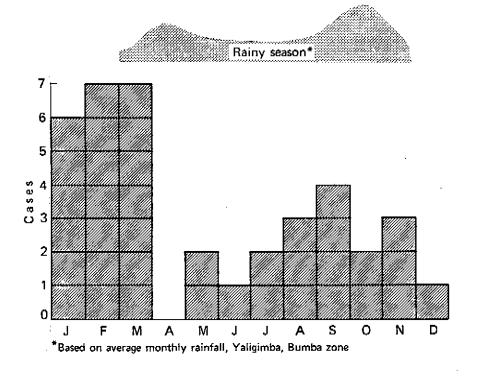
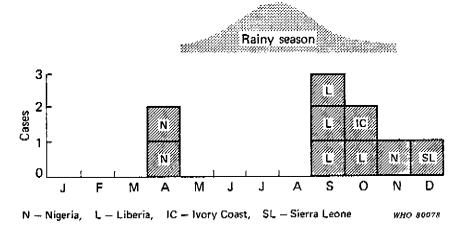


FIG. 3 SEASONAL DISTRIBUTION OF HUMAN MONKEYPOX CASES, 1970 – 1979

ZAIRE (38 cases)



WEST AFRICA (9 cases)



HUMAN MONKEYPOX 1970-1979





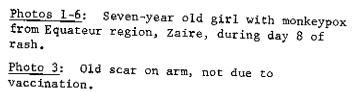






Photo 4: Heavy concentration of lesions on the hands, inguinal lymphadenopathy and pustules on genitalia.



 $\underline{\text{Photo 5}}\colon$ Swollen lower face and neck due to cervical and sub-mandibular and lymphadenopathy.



Photo 7: Same patient 16 months after initial illness: hyperpigmentation of lesions with shallow, pitting scars most prominent over bridge of nose.



Photo 6: Lesions on lips, tongue and eyelid

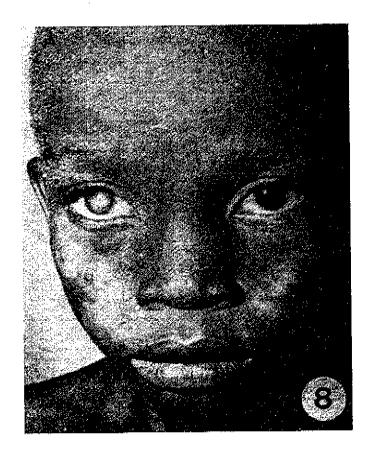


Photo 8: Eight year old boy with unilateral blindness following primary lesion on cornea; monkeypox occurred one year previously.



Photo 9: 0.5 mm pockmark in centre of upper lip in 35-year-old woman who had disease three months previously; this was the only lesion on patient who had been vaccinated more than 10 years before. Monkeypox virus was grown from the scab taken during the acute illness.