Factors that precipitated human plague in Zambia from 1914 to 2014-An overview for a century (100 years)

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Summary

Plague is a zoonotic and re-emerging disease caused by Yersinia pestis. The disease has caused a devastating effect on the entire world since the time of Justinian plague in the sixth century. Data were searched, screened and compiled from various sources on time, place and magnitude of human plague occurrences in Zambia. It was revealed that plague disease outbreak was first reported in Zambia in early 20th century and human infections had been reported in three zones since 1914. During the first half of the century, the number of human deaths steadily decreased from 93 in 1917 to 1 in 1947. Another outbreak was reported in 1956 after which it remained quiescent until 1987. More human infections were reported between 1990 and 2008. Factors which contributed to plague outbreaks and spread in Zambia included heavy rains, which was usually followed by a large increase in rodent and flea populations, socio-cultural human behaviour and life-style practiced by communities. Such practices were optimal for occurrence and rapid spread of plague including polygamy, hunting, overcrowding in houses, and belief in witchcraft, consequently seeking treatment from traditional healers, and man-rodent contact which was intensified by hunting practice. Despite smaller numbers of cases reported, there is much public health concern about the current plague situation in the country and hence, the disease shouldn't be neglected. Improving surveillance strategies for the disease and intensifying public health education are therefore desirable in order to sensitise the communities and consequently minimise transmission of the disease.

Keywords: Plague, Yersinia pestis, Review, Overview, Zambia.

Introduction

Plague is one of the most important reemerging zoonotic diseases in the history of public health in the world and can remain quiescent for more than thirty to forty years. The disease is caused by a gram negative, non-motile forming and non-spore coccobacillus bacterium known as Yersinia pestis and has caused devastating effects in the entire world. The causative agent of plague disease was discovered by a young Swiss physician, Alexandre Yersin, in Hong Kong during the beginning of the third plague pandemic in 1894. The bacterium infects warm blooded animals, especially rodents, which are the usual host reservoirs while human beings and other mammals are occasionally affected. The bacterium is normally transmitted from one host to another through the bite of an infective flea such as *Xenopsylla* cheopis (Andrianaivoarimanana et al., 2013; Perry & Fetherston, 1997). It can also be transmitted through direct contact with infected materials and droplets especially in pneumonic infections (Wang et al., 2011; Oyston & Williamson, 2011). Occasionally, it can also be transmitted through contaminated soil as the bacterium can persist in the former for a substantial period of time (Boegler et al., 2012; Ayyadurai et al., 2008).

Historical perspectives of human plague

Cases of human plague have been known from time immemorial. The first authentically recorded outbreak of human plague occurred among the Philistines in 1320 BC, (Holy Bible I Samuel: 5 and 6) and was characterized by the appearance of tumours in their secret parts (Freemon, 2005). In the last two millennia, three plague pandemics occurred and the disease spread widely affecting a large number of countries in most continents. The first pandemic known as Justinian plague occurred in the

sixth century (542- 546 AD) and affected Asia, Africa and Europe, where it claimed nearly 100 million victims (Wagner et al., 2014). The second pandemic referred to as Black Death occurred in the fourteenth century (1347-1351) and caused about 50 million deaths, of which half of them were from Asia and Africa and the other half was from Europe, where a quarter of the population succumbed. This pandemic was the beginning of a number of outbreaks of the disease which ravaged Europe and Africa in subsequent centuries (Harbeck et al., 2013; Bos et al., 2011). The third pandemic, the Oriental plague, which is characterised bv sporadic generally outbreaks of the disease and has persisted to date, began in Canton and Hong Kong (China) in 1894 and spread rapidly throughout the world, probably due to infected rats such as Rattus rattus, carried aboard swifter steamships that replaced slow moving sailing vessels in merchant fleets. Within 10 years (1894–1903) the disease was reported to have affected 77 ports in five continents (Dennis et al., 1999). It had also been revealed that rats contracted plague through infective fleas and the classic rat flea Xenopsylla cheopis was the most common vector of Yersinia pestis (Butler 2013; Ben Ari et al., 2011).

Historically, plague existed as a natural infection of wild rodents in Central Asiatic plateau long before its appearance in human beings (Sussman 2011; Msangi 1975). The commensal rats of the genus *Rattus* migrated throughout the world from their original home in Asia and became numerous after development of towns, good transport network and emerging of densely populated urban and rural areas. In their endeavour of fetching food and shelter, salvatic rodents penetrated urban environments and consequently facilitated transfer of plague organisms to murine rodent population, thus creating a secondary focus of the disease which persisted alongside with its natural foci (Lin et al., 2012).

Origin and distribution of Plague in Africa

In Africa, the disease has been reported in Northern, Eastern, Southern and Central Africa during the 19th and 20th centuries. In North Africa, plague outbreaks were reported in Libya and Algeria in 2009 and 2003, respectively where humans got infected after consuming goat and camel meat (Cabanel & Leclercq 2013; Bitam et al., 2010; Bertherat et al., 2007; Bin Saeed et al., 2005). In East Africa, countries affected were Kenya, Uganda and Tanzania and the latter experienced most recent and widely outbreaks (Kilonzo 1999; 1996) while in Uganda, the cases were first reported in 1876 (Roberts 1935). Many more outbreaks occurred in the country during the 20th century involving many cases and deaths (Borchert & Mach 2007), whereas 3,292 deaths were attributed to the disease in 1913 (Low & Newman 1920).

Plague was presumably introduced in Madagascar and Southern Africa by cargo ships carrying rice and other food materials from India in 1900 (Vogler et al., 2013; Isaacson 1986). Several countries, including Botswana, Namibia, Zimbabwe, Republic of South Africa and Mozambique experienced severe outbreaks of the disease during the 20th century (Neerinckx et al., 2008; Molefi 2001; Kumaresan et al., 1991). Similarly, the

island of Madagascar experienced severe outbreaks during the period and has been reporting such outbreaks to date. Indeed, the most recent outbreaks of the disease occurred in the island in 2014 and 2015 which involved 41 cases with 19 deaths (CF=46.3%) and 14 cases with 10 deaths (CF=71.4 %), respectively (WHO, 2015; Central 2014). In Africa. Malawi. Democratic Republic of Congo (DRC) and Zambia experienced plague outbreaks in different years. In DRC, such outbreaks occurred in the eastern part of the country bordering Rwanda, Burundi and Tanzania. In Zambia, formerly known as Northern Rhodesia, four plague outbreaks have been reported during the past 30 years. These occurred in North-western (McClean, 1995), Southern (Hang'ombe et al., 2012; Dennis et al., 1999) and Eastern provinces (Ngulube et al., 2006; Neerinckx et al., 2010).

The disease has caused a lot of concern and economic losses in African countries and the world at large. In many countries reemerging outbreaks of the disease have occurred after its disappearance of many years. These outbreaks include those reported in Botswana (1989), Madagascar (1991), Algeria (2003) and Tanzania (2007/2008), which recurred after being quiescent for 35 years, 60 years, 50 years and 30 years, respectively (Kumaresan et al., 1991; Boisier et al., 1997; Bertherat et al., 2007; Makundi et al., 2008).

The objective of this study was to determine factors that accelerated human plague outbreaks in Zambia since 1914 and advise the relevant authorities on the potential magnitude of the problem prior to developing appropriate preventive and control strategies for the disease.

Materials and Methods

A human plague database for Zambia between 1914 and 2014 was created. Several avenues were followed and various information sources were consulted. The table from the WHO Plague Manual (Dennis et al., 1999) gives information about the yearly numbers of cases per country and is limited between the period 1954 and 1997. in which Zambia is included (Table 1). Monthly incidences of plague in Northern Rhodesia (Barotseland), 1937-49 (Davis, 1953) was also checked. The WHO weekly epidemiological records (http://www.who.int/wer/en/) for 1951-2007 and **ProMED-mail** reports (http://www.promedmail.org/) for 1994-2007 were also consulted. The Zambian newspapers and Meteorological department were also checked to get some more information on rain patterns in relation to plague outbreaks. In addition, computerised searching international of literature databases was carried out, namely PubMed http://www.ncbi.nlm.nih.gov/PubMed/ and www.worldcat.org, for the public health and medical subjects in the Ministry of Health London and Web of Science (http://apps.isiknowledge.com/). Α systematic search strategy was employed using the following search terms: 'Africa' or the name of any African country (e.g. 'Northern Rhodesia or Zambia, Union of South Africa') and 'plague' (e.g. 'Yersinia pestis'). The search was refined to manuscripts that reported the occurrence of animal or human plague in Southern and

Central African countries. Original full texts were obtained for most studies and additionally, the WHO library and archives in Geneva, including non-published reports, were studied and incorporated in the database. This was supplemented with from information Web-accessible documents and other grey literature (mission reports, old distribution maps, newspapers etc.) from libraries and archives. The human plague database for Southern Africa comprises information on Zambia or Northern Rhodesia that had experienced at least human plague cases since 1914 were checked. Furthermore. also specific information regarding place, time and source of human plague was extracted and additional remarks were provided.

Results

From historical and epidemiological perspectives search, three different zones harbour plague endemic foci in Zambia. These are:- Eastern zone, which includes Luangwa valley, where outbreaks occurred in Tembwe village in Chama district, Mukomba village in Lundazi district and Chief Nyanje in Sinda district; the Southern zone, which includes Kabulamwanda and surrounding villages in Namwala district and Western zone which includes Chitokoloki in Balovale (Zambezi district) in Zambezi plain (Fig 1, Table 2).

Eastern zone

Luangwa and Lundazi outbreaks

According to available information, the first reported plague outbreak in Zambia (then Northern Rhodesia) was recorded in this area in 1917, and it involved 96 cases and 93 deaths (Case Fatality (CF) =96.8%). The outbreak occurred at Tembwe virizi village (11°20'S, 32°57'E) in the North of the Luangwa valley in Chama district (Fig 1). This was followed by another outbreak in 1918, which reportedly involved 59 cases and 57 deaths (CF=96.6%). Of these cases, 33 (55.5%) and 26 (44.1%) were females and males, respectively. Concurrently, during the plague outbreak, there was epizootic among local house rats (Rattus rattus) and increase in flea population, which was characterised by unusual mortality of the animals (Davis et al., 1960; Low & Newman, 1920). The outbreak was preceded by heavy rainfall in the area which flooded the rodent burrows and probably forced them to look for alternative dry habitants such as residential houses and grain storage barns in the villages. According to Low and Newman, (1920), the factors which facilitated the spread of the disease included socio-cultural practices such as polygamy, under which a man got married to many wives in different villages and hence, upon visiting his wives in affected homes, he could easily move with infective fleas and consequently transmit the disease to occupants in other homes. Furthermore, the culture and practice of catching, transporting, preparing and consuming rodents most likely facilitated transmission and spread of the disease in this area where 6 villages were affected (Low & Newman 1920).

In 1956, the disease re-emerged at the same focus and involved 36 cases and 5 deaths, respectively (CF=13.8%). Prior to the outbreak, houses and grain storage barns

(*Nkhokwe*) were heavily infested with rats and fleas and many rodents were found dead. This probably facilitated the transfer of infected fleas to human beings and subsequently transmitted plague pathogens to human population. According to Davies *et al.* (1960) most of the patients in the outbreak were infants and children, a fact which probably attributable to the sleeping habits in the area, where fathers usually sleep alone on plank beds while mothers and children sleep on reed mats on the floor and become vulnerable to flea bites since such floors are environmentally suitable for breeding of such insects.

In the same year, there was another outbreak of the disease in Mukomba village, 16km South-East of Lundazi district after some villagers had noticed the increase in numbers of rodents and fleas in their houses and grain storage barns. The outbreak occurred in March, when a fourteen 14 years old girl died of the disease and a man with similar symptoms was also admitted at the Lundazi hospital of the same village in the following month and he was successfully treated with Oxytetracycline and Penicillin (Davis et al., 1960).

Sinda district

According to the information retrieved, plague outbreaks in Sinda district in Nyanje chiefdom started in January 2001 and most affected villages were those near the border with Mozambique and later on it spread to other parts of the Chiefdom. The outbreak was reported after some villagers noticed heavy rodent and flea infestations in houses after the heavy rainfalls which flooded streams and rodent burrows. The outbreak involved 850 recorded cases and 11 deaths (CF=1.3%) (Ngulube et al., 2006). During group discussions and in-depth interviews Ngulube et al. (2006) observed that beliefs about witchcraft were common in the area and hence, discouraged patients from seeking medical advice to modern clinic. The community attributed the disease outbreak to witchcraft and at the same time accused one man for killing his daughter and a newly crowned village chief for killing his subjects probably to strengthen his chieftainship. These cultural beliefs indirectly facilitated the spread of the disease as they encouraged communities to seek health services from the traditional healers rather than modern clinics. These observations were in consistency with the findings reported in India (Aujoulat et al., 2003). Another outbreak, which involved 213 reported cases and 2 confirmed deaths (CF=0.9%) occurred at the same place in January 2007 and mostly affected villages were lying further away from the border (Ministry of Health 2007; Neerinckx et al., 2010). In addition to socio- cultural factors, spread of the disease was also facilitated by the movement of people from infected areas to uninfected zones. As for the case of Luangwa and Lundazi areas, movement of people in Sinda district also involved in transportation of personal belongings, livestock, rodents and fleas, all of which were pre-requisites of spreading and dissemination of plague pathogens. Indeed, this was in consistence with the reported observation in China, where a domestic dog transmitted plague to the herdsman in Zhihaigou, Xinghai County when they moved from Qinghai-Tibet plateau, which was an established plague focus (Wang et al., 2011).

Western zone

Barotseland (Zambezi plain) and Balovale

The first recorded outbreak of plague disease, in Zambezi plain (Barotseland province) occurred in January 1937 near Chitokoloki mission hospital. It was generally believed that an improved transport system facilitated the spread of plague disease from endemic foci in South Africa to plague free areas in Zambia through cargo transported by train (Molefi 2001). Motor transport and labour migration were most likely responsible for plague introduction into the area as people from the area were employed as casual workers in gold mines and farms. The 1937 outbreak involved 9 reported cases. More outbreaks were reported in the same area in February 1940, October 1942 and November 1942 and involved 4, 6 and 7 reported cases respectively (Davis 1953).

The focus experienced further outbreaks in December 1943 (5 cases), January 1944 (3 cases), December 1947 (1 case), January and February 1948 (5 and 2 cases, respectively) and February 1949 (2 cases) (Table 2) (Davis 1953; Molefi 2001). The Balovale area (currently Zambezi district) in the Zambezi plain experienced another outbreak of the disease in October 1954 after being quiescent for 17 years. This outbreak involved 9 and 5 reported cases deaths (CF=55.6%) respectively and (Worsfold, 1955). In this outbreak all the cases came from the same village probably. because they stayed in one house. The village headman reportedly died suddenly,

probably due to the fact that he was not taken to the hospital followed by other members of the family (Worsfold 1955). The disease disappeared for 40 years and resurfaced at the same area in 1994 when 10 cases and 3 deaths (CF=30%) were reported (McClean 1995). The outbreaks were reported after a heavy downfall and flooding of the Zambezi River, which compelled rodents and their fleas to evacuate their burrows and sought for alternative residences on dry land in the surrounding villages. Disease outbreaks in the region usually occurred between October and March during which period there were heavy rains and most rodent fleas, especially ubiquitous species tend to migrate from the field to murine hosts.

The reported outbreaks of the disease in the area, which had experienced the problem in the past and become quiescent for a long period of time, suggests that the disease can break out at an endemic area, anytime if and when favourable conditions prevail. In view of this fact, therefore, recurrence of another outbreak in the same area cannot be ruled out.

Southern zone

Namwala district

Namwala district in Kafue plain in Southern Province, experienced an outbreak of human plague for the first time between December 1996 and February 1997, and involved 316 reported cases of whom 26 (CF=8.2%) died. Since then, sporadic outbreaks with various magnitudes had been occurring in the area (Hang'ombe et al., 2012; Dennis et al., 1999). These include the 2007 outbreak, which involved over 60

reported cases and 4 deaths (CF=6.67%), and was associated with heavy rodent infestations in granaries and fields where Rattus rattus and *Gerbils* spp were respectively abundant (Hang'ombe et al., 2012). The disease broke out in the area during the wet season (November to March) which flooded the Kafue plains and rodent burrows and consequently forced them together with their fleas and seek refuge in settlements. The observations human revealed that rodent and flea population in the area were high and rodents died in big numbers in grain storage barns and shallow well (Hang'ombe et al., 2012). As in most other plague outbreaks in the country, communities in Namwala were reluctant to be attended to by health personnel as they associated the disease with witchcraft. Such reluctance and consequent delay or failure to administer appropriate treatment, facilitated spread of the disease. Furthermore, the lifestyle of residents such as polygamy and the habit of staying in the bush looking after livestock facilitated transfer of infective fleas from one area to another and exposed people to such infective fleas (Goel et al., 2014).

Angola Cases Deaths	Year	1980	1981	1982	1983	1984	1985	1986	1987	1988	1989	1990	1991	1992	1993	1994	Total
		21	6	1	1	.	,	.	1	1	1	1	1	.	1	1	27
		4	1	1	1	1	1	1	1	1	1	1	1	1	1	1	4
Botswana Cases		.			1		.	1	1	1	103	70					173
Deaths						1				1	6	e					12
Kenya Cases		5										4					49
Deaths		2										~					10
Libya Cases				1		~			1	.		1					×
Deaths			1			1	1		1	1		1	1	1	1		
Madagascar Cases		11	44	38	24	39	85	29	23	93	170	226	137	198	147	126	1390
Deaths		5	13	19	10	18	18	9	4	19	41	55	30	26	23	15	302
Malawi Cases			1	,	,		ı	ı	,	ı		ı		ı		6	6
Deaths							1	1		1	1						
Mozambique Cases																216	216
Deaths																ω	e
South Africa Cases				19						1							19
Deaths				-						1							-
Uganda Cases			1	153		1	1	340	ı	1	.	1	1	1	167	1	660
Deaths			1	m	1	1	1	27	ı	1		1	1	1	18		48
Tanzania Cases		49	6	76	569	603	129	360	356	647	31	364	1293	16r	18r	444	4964r
Deaths		11	9	18	49	41	22	57	34	33	4	32	60	2r	ı	50	419r
Zaire Cases		ı	I	1	ı	I	I	I	474	369	1	I	289	390	636	82	2242
Deaths			1	0	ı	ı	ı	ı	160	86	0	ı	28	140	89	10	513
Zambia Cases		1	I	ı	ı	1	1	I	1	ı	1	1	I	I	I	10	11
Deaths			1	ı	,	1	1	1	-	ı		ı	1	1	1	m	4
Zimbabwe Cases			m	-	1	1	1	1	1	1		1	1	1	1	392	397
Deaths			5	0	ı	ı	-	1	ı	ı	 1	ı	ı	1	1	28	31
Total Cases		86	62	338	593	642	215	729	854	1109	305	704	1719	604r	968r	1279	10207
Deaths		22	22	41	59	59	41	90	199	138	54	98	118	168 r	130	109	1348

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S/N	Year	District	Location	No. of recorded cases	No. of recorded deaths	References
	1917		Tembwe	96	93	Low & Newman, 1920;
1			Virizi			Davis , et al., 1960
	1918	Chama	Tembwe	59	57	Low & Newman, 1920;
			Virizi			Davis , et al., 1960
	1937	Zambezi	Barotseland	09	09	Davis , 1953
		plain				
2		(Balovale)				
	1940		Barotseland	04	04	Davis, 1953
	1942		Barotseland	13	13	Davis, 1953
	1943		Barotseland	05	05	Davis, 1953
	1944		Barotseland	03	03	Davis , 1953
	1947		Barotseland	01	01	Davis, 1953
	1948		Barotseland	07	07	Davis, 1953
	1949		Barotseland	02	02	Davis , 1953
	1954		Chitokoloki	9	5	Davis , et al., 1960; Worsfold,
			(CMML			1955
			mission)			
3	1956	Chama	Tembwe	36	05	Davis , et al., 1960
4	1956	Lundazi	Mukomba	03	01	Davis , et al.,1960
5	1987	*	Zambia	1	01	Dennis , et al., 1999
6	1994	Zambezi	Chitokoloki	10	03	McClean, 1995
		plain				
7	1997	Namwala	Namwala	316	26	Dennis , et al., 1999
			district			
8	2001	Sinda	Nyanje	850	11	Ngulube, et al., 2006
9	2007	Sinda	Nyanje	213	02	MOH , 2007;
			• •			Neerinckx , et al., 2010
10	2007	Namwala	Namwala	68	04	Hang'ombe, et al., 2012
			Total	1705	252	-*

 Table 2. Recorded human plague outbreaks in Zambia from 1914-2014

* Location not available

Discussion

Socio-cultural human behaviour

The finding of this study suggests that behaviour socio-cultural largely contributed to the spread of plague disease in Zambia as observed in most villages that experienced an outbreak of the disease. The type of behaviour included polygamy in which the husband move from one house to another in the same or different villages and in so doing he could move with infected fleas. Additionally, the life-style of most residents in the villages such as sleeping habits was observed to have an important impact on the spread of the disease in most outbreak areas. Furthermore, witchcraft reportedly contributed to the spread of the disease in these areas as most people believed that the disease was caused by magic, hence most patients were reluctant to visit medical facilities for appropriate and prompt treatment a fact which probably led to the death of many patients as observed in Sinda district and Chitokoloki (Ngulube et al., 2006; Worsford 1955).

Man-rodent contact

The search revealed that in all areas where plague broke out residents were in constant contact with rodents and their fleas when hunting the animals for food. As such people were exposed to flea bites thus contributing to outbreaks and the spread of the disease. They tend to hunt and transport or dispose of dead rodents in a manner which facilitates infective fleas jumping from dead rodents to human beings or any other domestic animals. The tendency of fleas to jump from dead rodents to alternative host most likely occurred in all the foci where dead rodents were found in granaries, fields, borehole and shallow water wells all of which were

environmentally conducive for fleas to attack any animal which may pass nearby (Low & Newman 1920; Davis et al., 1960).

Climate

The findings in this study further suggest that heavy rains were important contributing factor to outbreak and spread of plague in Zambia, indeed, most outbreaks usually occurred between October and March in all foci. In Zambia ambient temperatures for breeding occurred during the rainy season and are suitable for breeding of flea vectors as such most epizootic and human disease are favoured by sufficient populations of fleas and susceptible rodents (Perry & Fetherston 1997). Likewise, heavy rainfall floods rodent burrows and consequently force occupants together with their flea ecto-parasites to migrate to human habitations. The observation in is consistency with findings elsewhere, that plague outbreak occurs during rainy seasons. Ben Ari et al. (2011), observed that abundance of rodent fleas was affected by suitable temperature, rainfall and relative humidity and that warm, moist weather was most suitable and associated with probable high flea index, as such weather has a direct effect on the survival. development. behaviour and reproduction of the insects (Ben Ari et al., 2011).

Conclusion

On the basis of the foregoing information, it is justifiably conclusive that plague disease in Zambia usually occurs during rainy seasons between October and March, and that outbreaks are generally associated with increased rodent and flea populations, which are the natural reservoirs and vectors, respectively. This is consistent with the observation of Boisier et al. (1997) in Madagascar, where human plague occurred during the same period of time (Boisier et al., 1997). Other factors which facilitated the disease outbreaks and spread, included socio-cultural human behaviour such as beliefs in witchcraft, life-style of residents such as polygamy, overcrowding in single house and manrodent contacts due to practice of hunting and eating in such a rodent-ridden environment. Furthermore, the pattern of plague in Zambia demonstrates the typical re-emerging nature of the disease and its ability to break out after many years of quiescence if and when favourable conditions prevail as observed in the Luangwa valley, where the first and second outbreaks occurred in 1917 and 1956. respectively. Chitokoloki in Zambezi plain, where the first, second and third outbreaks occurred in 1937, 1954 and 1994 respectively. In Namwala, the first outbreak occurred in 1997 and the second occurred in 2007 while in Nyanje in Sinda, the first and second outbreaks occurred in 2001 and 2007 respectively. However, it is important to note that reported statistics on plague may be inadequate due to the communication problems, inadequate surveillance and reporting activities, lack of adequate laboratory facilities and inadequate trained health personnel for carrying out confirmatory diagnosis of the disease.

In view of the cumulative information reviewed in this paper and established facts about the nature of plague globally, improving surveillance services in established potential foci in Zambia is recommended in order to get a clear picture on endemicity of the disease in the country. Data acquired from such activities may serve as a basis for studying the epidemiology and ecology of the disease, which may help in early detection and subsequently develop appropriate preventive and control measures and consequently reduce morbidity and mortality rates of the disease.

Conflicts of interest: None declared.

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References

- Andrianaivoarimanana V., Katharina K., Elissa N., Duplantier J.-M., Carniel E., Rajerison M. and Jambou R. (2013).
 Understanding the Persistence of Plague Foci in Madagascar. *PLoS Neglected Tropical Diseases Journal*, 7 (11).
- Aujoulat I., Johnson C., Zinsou C., Guédénon A. and Portaels F. (2003).
 Psychosocial aspects of health seeking behaviours of patients with Buruli ulcer in Southern Benin. *Tropical Medicine & International Health*, 8 (8), pp.750–759.
- Ayyadurai S., Houhamdi L., Lepidi H., Nappez C., Raoult D. and Drancourt M. (2008). Long-term persistence of

virulent *Yersinia pestis* in soil. *Microbiology*, 154, pp.2865–71.

- Ben Ari T., Simon N., Gage K.L., Kreppel K., Laudisoit A., Leirs H. and Stenseth N.C. (2011). Plague and Climate: Scales Matter. *PLoS Pathogen*, 7 (9), pp.1-6.
- Bertherat E, Bekhoucha S., Chougrani S., Razik F., Duchemin JB., Houti L., Deharib L., Fayolle C., Makrerougrass
 B., Dali-Yahia R., Bellal R., Belhabri L., Chaieb A., Tikhomirov E. and Carniel E. (2007). Plague reappearance in Algeria after 50 years, 2003. *Emerging Infectious Diseases Journal*, 13 (10), pp.1459–62.
- Bin Saeed A., Al-Hamdan N. and Fontaine R. E. (2005). Plague from eating raw camel liver. *Emerging Infectious Diseases Journal*, 11 (9), pp.1456–7.
- Bitam I., Ayyadurai S., Kernif T., Chetta M., Boulaghman N., Raoult D. and Drancourt M. (2010). New ruaral focus of Plague, Algeria. *Emerging Infectious Diseases journal*, 16 (10), pp.1639– 1640.
- Boegler A.K, Christine G.B., John M.A., Katherine M., Jennifer H.L., Jeannine P.M., Kenneth G.L. and Eisen R.J. (2012). Evaluation of the infectiousness to mice of soil contaminated with Yersinia pestis-infected blood. *Vector Borne and Zoonotic Diseases*, 12 (11), pp. 948–52.
- Boisier P., Rasolomaharo M., Ranaivoson G., Rasoamanana B., Rakoto L., Andrianirina Z., Andriamahefazafy B. and Chanteau S. (1997). Urban epidemic of bubonic plague in Majunga, Madagascar: epidemiological aspects. *Tropical Medicine & International Health*, 2 (5), pp.422–427.
- Borchert J. and Mach J. (2007). Invasive rats and bubonic plague in Northwest

Uganda. *Managing Vertebrate Invasive Species*, 3, pp.283–293.

- Bos K.I., Schuenemann V.J., Golding G.B., Burbano H., Waglechner N., Coombes B.K., McPhee J.B., Witte S.N.D., Meyer M., Schmedes S., Wood J., Earn D.J.D., Herring D.A., Bauer, P., Poinar H.N. and Krause J. (2011). A draft genome of *Yersinia pestis* from victims of the Black Death. *Nature*, 478 (7370), pp.506–10.
- Butler T. (1983). Plague and other Yersinia infections. Springer Science and Business Media., Plenum Press, New York
- Butler T. (2013). Plague gives surprises in the first decade of the 21st century in the United States and worldwide. *American Journal of Tropical Medicine and Hygiene*, 89 (4), 788–793.
- Cabanel N. and Leclercq A. (2013). Plague outbreak in Libya, 2009, unrelated to plague in Algeria. *Emerging Infectious*, 19, pp. 230–236.
- Davis D.H. (1953). Plague in Africa from 1935 to 1949; a survey of wild rodents in African territories. *Bulletin of the World Health Organization*, 9, pp.665– 700.
- Davis DH., Fisher BW. and Goldring F. (1960). The Luangwa Valley plague outbreaks and their significance in relation to Savannah plague in Central Africa. *Bulletin of the World Health Organization*, 23, pp. 405–8.
- Dennis D.T., Gage K.L., Gratz N., Poland J.D. and Tikhomirov E. (1999). Plague manual: Epidemiology, distribution, surveillance and control. *Geneva*.
- Freemon F. (2005). Bubonic plague in the Book of Samuel. *Journal of the Royal Society of Medicine*, 98 (9), pp.436.
- Goel S., Kaur H., Gupta A.K., Chauhan U. and Singh A. (2014). Socio-

epidemiological determinants of 2002 plague outbreak in Himachal Pradesh, India: a qualitative study. *BMC public Health*, 14 (1), pp. 325.

- Hang'ombe B.M., Nakamura I., Samui K.
 L., Kaile D., Mweene A.S., Kilonzo
 B.S., Hirofumi S., Chihiro S. and
 Brendan W.W. (2012). Evidence of *Yersinia pestis* DNA from fleas in an
 endemic plague area of Zambia. *BMC Research Notes*, 5 (1), pp.72.
- Harbeck M., Seifert L., Hänsch S., Wagner D.M., Birdsell D., Parise K.L., Wiechmann I., Grupe G., Thomas A., Keim P., Zöller L., Bramanti B., Riehm J.M. and Scholz H.C. (2013). *Yersinia pestis* DNA from skeletal remains from the 6(th) century AD reveals insights into Justinianic Plague. *PLoS Pathogen*, 9 (5), pp.1-8
- Isaacson M. (1986). Plague and cholera surveillance in southern Africa. *Southern Africa Medicine Journal*, pp.43–46.
- Kilonzo BS. (1996). Plague in Africa: Epidemiology and Control. *Africa Health*, 19, pp.14–15.
- Kilonzo B. (1999). Plague epidemiology and control in Eastern and Southern Africa during the period 1978 to 1997. *Central African Journal of Medicine*, 35, pp.70–76.
- Kumaresan JA., Grova J.B., Mmatli P.K. and Magany E.D. (1991). Plague in Botswana. *Central African Journal of Medicine*, 37, pp.271–272.
- Lin X., Guo W., Wang W. and Zou Y. (2012). Migration of Norway rats resulted in the worldwide distribution of *Seoul hantavirus* today. *Journal of Virology*, 86 (2), pp.972–981.
- Low R.B. and Newman S.G. (1920). The progress and diffusion of plague, cholera and yellow fever throughout the

world, 1914-1917. *HM Stationery Office*.

- Makundi R.H., Massawe A.W., Mulungu L.S., Katakweba A., Mbise T.J. and Mgode G. (2008). Potential mammalian reservoirs in a bubonic plague outbreak focus in Mbulu District, Northern Tanzania, in 2007. *Mammalia Journal*, 72 (3), pp.253–257.
- McClean K.L. (1995). Plague in Northwestern Zambia. *Clinical Infectious Diseases*, 21, pp.650–652.
- Molefi R.K.K. (2001). Of rats, fleas and peoples: towards a history of bubonic plague in southern Africa, 1890-1950. *Botswana Journal of African Studies*, 15(2), pp.259–267.
- Msangi A. (1975). The surveillance of rodent populations in East Africa in relation to plague endemicity. *University of Dar es Salaam Library Journal*, 1, pp.8–20.
- Neerinckx S., Bertherat E. and Leirs H. (2010). Human plague occurrences in Africa: an overview from 1877 to 2008. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 104 (2), pp. 97–103.
- Neerinckx S.B., Peterson A.T., Gulinck H., Deckers J. and Leirs H. (2008). Geographic distribution and ecological niche of plague in sub-Saharan Africa. *International Journal of Health Geographics*, 7 (1), pp.54.
- Ngulube T.J., Mwanza K., Njobvu C.A. and Muula A.S. (2006). Knowledge, attitudes and public health response towards plague in Petauke. *Tropical Doctor*, 36, pp.223–225.
- Oyston P. and Williamson D. (2011). Plague: Infections of Companion Animals and Opportunities for Intervention. *Animals Journal*, 1 (4), pp.242–255

- Perry R.D. and Fetherston J.D. (1997). Yersinia pestis-etiologic agent of plague. *Clinical Microbiology Reviews.*, 10 (1), pp.35–66.
- Roberts J. (1935). The endemicity of plague in East Africa. *East African Medical Journal*, 12, pp.200–219.
- Sussman G. (2011). Was the black death in India and China? *Bulletin of the History of Medicine*, 85 (3), pp.319–355.
- Vogler A.J., Chan F., Nottingham R., Andersen G., Drees K., Beckstrom-Sternberg S.M., Wagner D.M., Chanteau S. and Keim P. (2013). A decade of plague in Mahajanga, Madagascar: insights into the global maritime spread of pandemic plague. *MBio Journal*, 4 (1), pp.1–12.
- Wagner D.M., Klunk J., Harbeck M., Devault A., Waglechner N., Sahl J. W., Enk J., Birdsell D.N., Kuch M., Lumibao C., Poinar D., Pearson T., Fourment M., Golding B., Riehm JM., Earn D.J.D., Dewitte S., Rouillard J., Grupe G., Wiechmann I., Bliska JB., Keim P.S., Scholz H.C., Holmes E.C. and Poinar H. (2014). *Yersinia pestis* and the Plague of Justinian 541–543 AD: a genomic analysis. *Lancet Infectious Disease*, (13), pp.1–9.
- Wang H., Cui Y., Wang Z., Wang X., Guo Z., Yan Y., Li C., Cui B., Xiao X., Yang Y., Qi Z., Wang G., Wei B., Yu S., He D., Chen H., Chen G., Song Y. and Yang R. (2011). A dog-associated primary pneumonic plague in Qinghai Province, China. *Clinical Infectious Diseases*, 52 (2), pp.185–90.
- WHO (2014). Plague in Madagascar.Global Alert Response. DiseaseOutbreak News, pp. 1. Geneva.
- WHO. (2015). Plague in Madagascar. Global Alert Response-Disease Outbreak News, p. 1. Geneva.

Worsfold J.T. (1955). An outbreak of plague at Chitokoloki, Balovale. *Central African Journal of Medicine*, 1 (3), pp.116–117.