# INVASIVE RATS AND BUBONIC PLAGUE IN NORTHWEST UGANDA

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***Abstract:*** Major introductions of roof rats (Rattus rattus) likely occurred in the ports of East Africa during the Third Plague Pandemic in the late 1800's. Transport via trains, boats, and trucking likely introduced this species to inland areas of East Africa, ultimately including Northwest Uganda. Historic plague outbreaks occurred during the early part of the 20th century and continue to cause a human disease burden in the West Nile region of NW Uganda via the bubonic, septicemic, and pneumonic forms. Four field sites in this area were trapped to determine the rodent species composition in commensal and peridomestic areas of villages and associated flea burdens of the rodents. Rattus rattus were the most prevalent rodent trapped in commensal areas followed by the Nile rat (Arvicanthis niloticus). The most common peridomestic species of rodent was the Nile rat. Other peridomestic rodent species captured included, Mastomys natalensis, Lophuromys flavopunctatus, L. sikapusi, Gerbil (Tatera spp.), Lemniscomys spp., and 4 unknown species. Flea burdens on commensal R. rattus averaged 1.7±1.2 fleas per animal and on all peridomestic rodents, average 2.0±0.7 per animal. Additionally, commensal areas were sampled to determine free-living flea populations. Burrow swabbing indicated an average 0.19±0.12 fleas/burrow. Lighted flea traps averaged 1.3±0.6 fleas per household and dark flea traps averaged 0.5±0.4 fleas per household.

***Key Words:*** flea vector, invasive species, plague, *Rattus rattus*, rodents, Uganda, *Yersinia pestis*.

Managing Vertebrate Invasive Species: Proceedings of an International Symposium (G. W. Witmer, W. C. Pitt, K. A. Fagerstone, Eds). USDA/APHIS/WS, National Wildlife Research Center, Fort Collins, CO. 2007.

## INTRODUCTION

As described by researchers in the Democratic Republic of the Congo (DRC), "Plague is a disease of rats, a variegated gathering of rodents with different degrees of tolerance and sensitiveness to *Yersinia pestis*, living in frail equilibrium" (translation from Dutch, Janssens and Pattyn 1994). More than any of the 1,814 (order Rodentia, Nowak 1991) known rodents in the world, *Rattus norvegicus*, and especially *Rattus rattus*, have repeatedly been the tipping point in this "frail equilibrium" of the enzootic cycle of bubonic plague. The recent historic and current yearly incidence of plague in Northwest Uganda is most likely influenced by the introduction of the non-native *R. rattus* to this area.

Although a simplified explanation, three biovars of plague (*Y. pestis*) caused the three great plague pandemics. The Justinian plague of the sixth century was caused by biovar, *antiqua*, the 16th century Black Death by *medievalis* and the Third/Modern pandemic by the *orientalis* biovar. The Justinian plague occurred between 542-546 in Asia, Africa and Europe and affected 100 million people (Zietz and Dunkelberg 2004). This pandemic likely originated in East Africa, traveled north along the Nile or via oceanic shipping routes from East Africa to Egypt, then Palestine and throughout the Mediterranean region. This pandemic was likely spread by flea-borne transmission of the disease and was the source of epidemics in the following two centuries (Politzer 1954, Gage 1998, WHO 2000, Zietz and Dunkelberg 2004). Keyes (1999) theorizes that a great climatic upheaval occurred during this period causing unusually cool and wet conditions in the Mediterranean region. These climatic events are thought to have allowed environmental conditions that favored the transfer and expansion of fleas among many rodent hosts and led to the spread of the disease.

The second pandemic, known as the "Black Death," occurred in the fourteenth century and caused 50 million deaths, half in Europe and half in Africa and Asia. This plague pandemic likely originated in Central Asia among marmots and

traveled along the Silk Road to Europe (McEvedy 1988). Unlike the first pandemic, this pandemic was driven not only by flea-borne transmission of the disease, but also by pneumonic transfer of the disease among humans. This pandemic was also the source of subsequent smaller plague outbreaks in Europe and Africa in the following centuries, often with equal ferocity (Politzer 1954, Gage 1998). The last large pandemic, called the "Modern Pandemic" began in Hong Kong in 1894 and spread rapidly throughout the world by rats carried on merchant steamships. Within 10 years, plague had entered 77 ports on six continents: 31 in Asia, 12 in Europe, 8 in Africa, 4 in North America, 15 in South America and 7 in Australia. Epidemics resulted on every affected continent and caused over 12 million deaths, most in India. Scientific study during this last pandemic resulted in the identification of the causative agent, *Y. pestis*, by Alexander Yersin in 1894 in Hong Kong and also identified fleas as the primary vector of plague, as well as the role of commensal rats in the outbreaks of human plague (Politzer 1954, Gage 1998, Tikhomirov 1999).

In the last few decades, Africa reported the highest percentage of plague cases in the world and the largest number of human mortalities due to the disease (WHO 2004). During the period of 1980-1997, 19,349 cases (1781 deaths) were reported in Africa–representing 66.8% (75.8%) of the world's totals (WHO 2000). Most outbreaks, during this period, occurred in Eastern, Central and Southern Africa in the countries of Tanzania, South Africa, Madagascar, DRC, Zimbabwe, Mozambique, Malawi, Namibia, Kenya and Uganda (WHO 2000). Cases are rarely reported in northern Africa, but the disease recently reemerged in Algeria, 50 years after its last occurrence with the discovery of previously unknown natural foci (WHO 2004). On the island of Madagascar, during the 18-year period from 1980-1997, 5,986 cases with 493 deaths were recorded (Gratz 1999, Tikhomirov 1999).

## ARRIVAL OF PLAGUE TO WEST NILE REGION OF UGANDA

The plague foci in East/Central Africa are very ancient and were definitely present before British Occupation in the late 1800s (Barrett 1933). Although a small number of researchers theorized that plague evolved in Central Africa (Panagiotakopulu 2004), most authors think *Y. pestis* evolved from the Central Asiatic plateau (Guiyoule et al. 1994). Genetic analysis of biovars and ribotypes of *Y. pestis* agree with a Central Asiatic origin, as Asia has greater diversity of biotypes (Guiyoule et al. 1994). It is thought that biovar, *antiqua*, was introduced to Central Africa from Central Asia at some point before the sixth century, as there were reports of the introduction of plague into North Africa during the first century (Keyes 1999). Many rodent and their flea species throughout Central Asia, the Middle East and North Africa can harbor and transmit plague (Shenbrot and Krasnov 1999). Known as the Great Palaeartic Desert Belt, this desert area extends from the Atlantic coast of northwest Africa in the west to northeastern China. This is an area of high rodent and flea species richness (Shenbrot and Krasnov 1999). It is possible that these rodent complexes, including gerbils and jirds, may have transferred *Y. pestis* from Central Asia to North Africa and subsequently to Central Africa. Gerbils (represented across the entire belt) and jirds harbor numerous complexes of flea species and host switching of fleas among these rodents is common (Shenbrot and Krasnov 1999, Krasnov and Shenbrot 2002). Other authors think the disease may have been introduced from Egypt, Arabia and India by various medieval traders including slave and ivory caravans and/or via pilgrims to and from Mecca (Kilonzo 1976). These caravan routes extended from the Buganda Kingdom in Uganda where plague was or became endemic (Msangi 1975). Additionally, an epidemic of plague was known to have occurred in 1697 in Mombasa, Kenya, which held back a Portuguese attack against the Arabs for 33 months (Twigg 1978).

*Y. pestis*, biovar *orientalis*, was likely introduced into East Africa during the third plague pandemic, but currently appears to be restricted to East African ports (Guiyole et al. 1994). In the DRC, only biovar *antiqua* has been reported and this ancient biovar continues to be most prevalent inland in east/central Africa (Guiyole et al. 1994).

The earliest recorded accounts of plague in Uganda were by missionaries in 1877 (Orochi-Orach 2002). At the time, many indigenous ethnic groups were familiar with the disease and, in their traditional languages, had words for plague (Davis et al. 1968). The Buganda in Uganda called the disease "kampuli," in the Lendu language "zukpa" and in Swahili was called "tauni" (from the Arabic tā'ūn meaning the bubonic form of plague; Msangi 1975, Conrad 1982, Orochi-Orach 2002). In Uganda, between 1910-1946, over 61,000 people contracted the disease in the central districts of Buganda, Busoga, Mbale, Teso and Lango.

Records from the rest of the country were scarce (Hopkins 1949). Mortalities caused by plague during this period were high because treatment access in rural areas where the epidemics were confined was poor. The rodents responsible for the plague outbreaks in these areas were primarily *R. rattus, Mastomys natalensis* and *Arvicanthis niloticus.* The major rodent flea vectors responsible for the *Y. pestis* transmission was *X. bransilensis* and *X. cheopis* (Hopkins 1949, Politzer 1954).

From 1889-1954, the main plague foci in Uganda were localized in following areas: the Ituri forest, around Lake Albert, Burungu Island in Lake Victoria, Rubaga hill near Kampala and the Kyaggwe areas in the Districts of Mukono, Masaka, Rakai, Pallisa, Tororo, and Busia. These districts eventually became quiescent except for 2 villages in the Toro District (Ogen-Odoi 1999, Davis et al. 1968). Currently, the active plague foci are in the West Nile region of northwest Uganda. Plague outbreaks have occurred in Okoro County of the Nebbi District and Vurra County of the Arua District and cases of plague are often reported at the Nyapea Mission Hospital in the Nebbi District (Ogen-Odoi, personal communication). Major reported epidemics of human plague have been recorded numerous times during the last 20 years. Most of the cases have occurred in the Nebbi District of Northwestern Uganda where the disease has been endemic for over 40 years. Outbreaks occurred in 1982 (153 reported cases, 3 deaths), 1986 (340 reported cases, 27 deaths), 1993 (167 reported cases, 18 deaths), 2000 (202 reported cases, 50 deaths) and 2001 (319 reported cases, 42 deaths; Kilonzo 1999, WHO 2003). Although not fully understood in the West Nile region, the incidence of human plague cases in other areas has been observed to increase due to deforestation, the result of military operations, civil unrest, deterioration of sanitary conditions and the displacement of populations (Akiev 1982, WHO 2004). In other areas of Africa, times of civil unrest can cause a breakdown in most health services and reporting (Kilonzo 1999).

The West Nile region in northwest Uganda shares its plague focus with the Democratic Republic of the Congo (DRC) to the east. In the DRC, plague mainly exists in two foci situated in the extreme northeast of the country near the border of Uganda. Named the Lake Albert and Lake Edward foci, the Lake Albert foci likely formed an extension into the West Nile area of Uganda. Cases of plague in the Lake Albert region were first officially recognized in 1928 and prior to the 1950s *Mastomys coucha* [*natalensis*] was the primary rodent found in human habitations (98%). Additionally, *Arvicanthis abyssinicus* [*niloticus*] was also present. There were no early reports of *R. rattus* in this area (Politzer 1954). Unlike the Lake Albert focus, rodents collected in the Lake Edward focus were *R. rattus* (52%), *M. coucha* [*natalensis*] (32%) and *A. abyssinicus* [*niloticus*] (14%; Politzer 1954). This indicates that the introduction of *R. rattus* to the region seemed to displace the position of *M. coucha* [*natalensis*] in the huts. Both *X. cheopis* and *X. bransiliensis* were considered the early vectors of plague in the Lake Albert region and primarily *X. bransiliensis* in the Lake Edward focus (Politzer 1954).

In recent times, most of the plague cases in the West Nile Region were reported from the southern Nebbi District. Beginning in 1998, plague cases began to be reported from the northern Arua District (Orochi-Orach 2002). A US Centers for Disease Control and Prevention (CDC) analysis of local health records indicated that there were 1,610 cases of plague form 1989-2003 in Northwestern Uganda but less than 1% of cases were laboratory confirmed. Plague spread from 23 local parishes to 30 and averaged 298 cases/year during this period. The overall mortality rate was 25.9% with higher mortality from the pneumonic (52.8%) and septicemic (40.5%) forms compared to the bubonic (18.3%). Only 1/3 of cases were admitted into a health facility. Eighty-seven percent of the cases were bubonic, 6.2% pneumonic and 5.3% septicemic. There was female predominance (52.7%) with an average age of 19.0 years (Staples et al. 2004).

Plague cases in the West Nile region are known to be highly underreported (R. Enscore, CDC DVBID, personal communication). Likewise, the WHO concedes that officially reported data on plague do not adequately reflect the true incidence of plague, represents only a portion of the actual number of cases and may not even represent the known, active foci in the world. Global statistics on plague are incomplete because of the reluctance to officially notify plague cases as well as inadequate surveillance and reporting (WHO 2004). In Uganda, cultural and religious beliefs are known to affect the reporting of plague (Orochi-Orach 2002). When a death occurs among the Alur tribe in Northwest Uganda it is often thought to be a result of a bad omen or due to witchcraft. This belief is augmented if inexplicable deaths occur repeatedly as in the case of an epidemic of plague. When rats are observed to have died in a village or

a home, they are often believed to have been "dropped" by a witch-doctor in the village. Often, the belief is that the deaths occur from an "ill spirit" "sweeping the village" in reprisal of an unpaid debt or other reasons (Orochi-Orach 2002).

The peak months of plague in the West Nile region are September through December, corresponding to the rainy season. Although the Nebbi district in the West Nile has been surveyed for the last 13 years, the epizoology of plague continues to be poorly understood (Laudisoit et al. 2007). A possible enzootic cycle in the West Nile region of Uganda is described in Figure 1 (based on: Politzer 1954, Gage 1998, Gratz 1999).

Weather, food presence (including periods of harvest), natural rodent mortalities (epizootic) and excessive human fleabites influence this enzootic cycle. Throughout Africa, excessive rodent population outbreaks often occur after periods of excessive rainfall, especially after periods of drought (Fiedler 1994, Leirs et al. 1996), which promotes excessive growth of vegetation, decreased competition, increased reproduction rates, and increased cover. It is believed that epidemics in some areas of Africa follow an unusually prolonged drought in the area, which may force many field rodents to seek food within human dwellings, thus passing infection to peridomestic and commensal

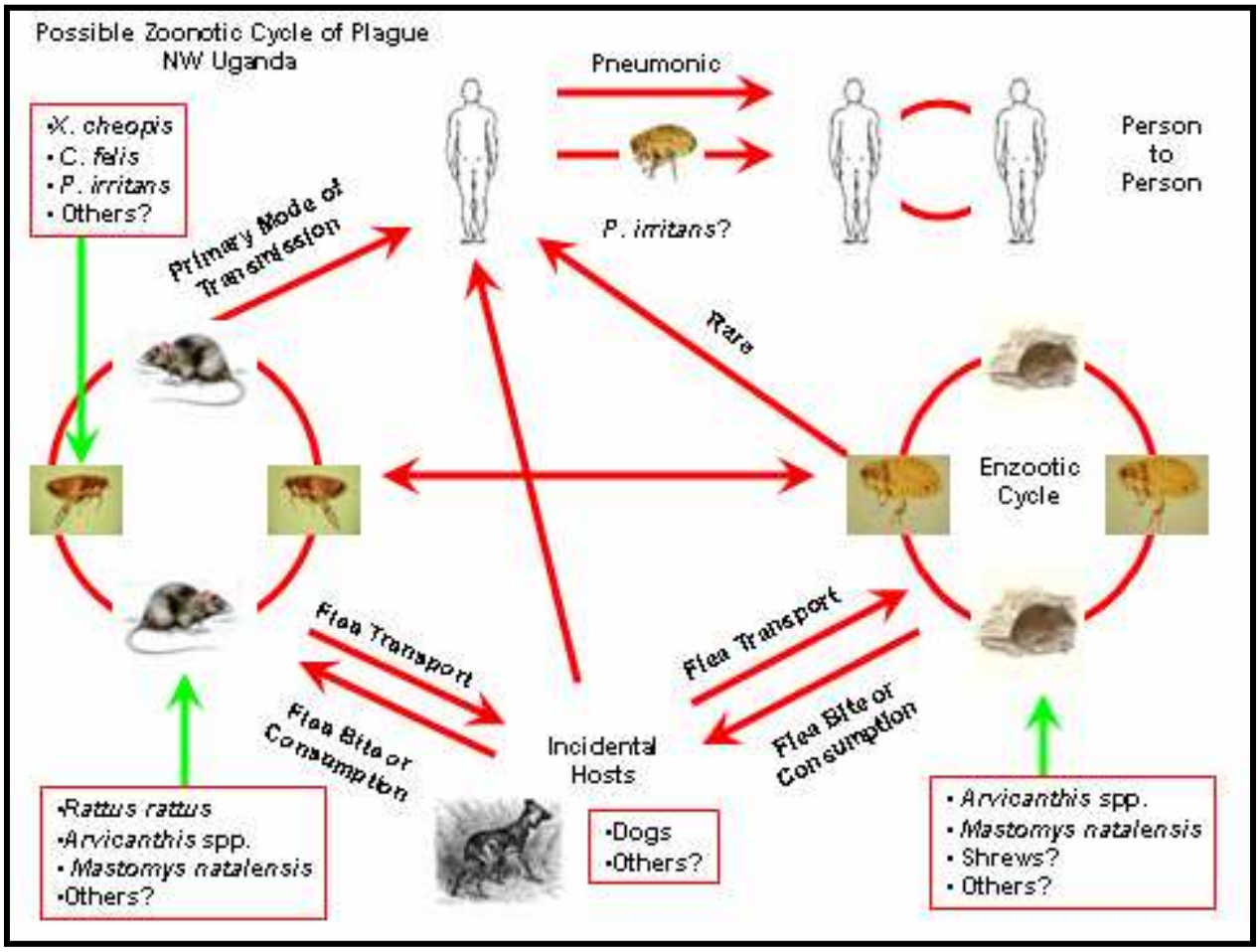


**Figure 1.** Proposed enzootic cycle of plague in the West Nile region of Uganda.

rodents, domestic animals and humans. Observations in this region indicate that nearly all-human epidemics are preceded by moderate to massive epizootics in rodents and excessive fleabites to humans (Ogen-Odoi, personal communication). These observations are yet to be studied in detail.

Additionally, the enzootic cycle is affected by cultural activities. The Alur ethnic group in the West Nile region historically stored harvested grain in granaries outside of the structures used for living and sleeping. Due to increased theft of grain and animals, food and often livestock are largely stored in the same structure where people sleep. Likewise, when a person dies in the Alur culture, people gather and sleep in the home of the deceased for 3-4 days. This activity is based on belief that the deceased spirit lingers and, if ignored by surviving relatives and friends, may later negatively affect crop harvest or cause ill to the survivors (Orachi-Orach 2002). These activities may increase the risk of contracting plague.

## ARRIVAL OF *RATTUS RATTUS* TO THE WEST NILE REGION

Species of the genus *Rattus* have likely been responsible for more human suffering than any other group of vertebrates via their role in transmission of disease and destructive impact on food crops (Alpin et al. 2003). The dispersal of *Rattus* spp. from its native homeland is remarkable in that it occurred in the presence of pre-existing, diverse rodent fauna (Alpin et al. 2003). The presence of numerous, established competitors would normally represent a significant impediment to the establishment of an invasive species, but *Rattus* spp. have not only managed to gain a foothold in diverse habitats throughout the world, but are thriving, including areas in Northwest Uganda. *R. norvegicus* is known to displace *R. rattus*. In general, displacements of *R. rattus* by *R. norvegicus* usually occur in ports and urban sites, but are much less common in the rural areas of developing countries where the *R. rattus* is encountered more frequently (Gage 1998). Such is the case in the West Nile region of Uganda. Originating in India or Burma, *R. rattus* tends to have the advantage in warmer climates while *R. norvegicus* appears to have the advantage in cooler climates (Avery 1985). The first appearances of *R. rattus* in Eastern and Southern Africa seem to have predated the Third Plague Pandemic. Iron Age sites in Zambia have recorded the presence of *R. rattus* around 1500 and 1600 AD, the northern Transval (currently South Africa) around 1000 A.D. and Natal (currently South Africa) in the eighth century (Davis and Fagan 1962, Plug et al. 1979, Voight and Von den Driesch 1984).

Although *R. rattus* may have been present in prior times, major introductions of the rodent to East Africa seemed to have occurred in the late 1800s (Fieldler 1988) from the East. It was proposed that the construction of the railway from the Eastern port of Mombasa, Kenya to Kisumu, Kenya and ultimately into Uganda led to an importation of plague (*Y. pestis* biovar *orientalis*) and *Rattus* spp. from docking ships (Twigg 1978). During the period of 1903-1908 most of the ports in Lake Victoria became involved in plague epidemics, which continued to spread inland (Politzer 1954). Prior to the arrival of *R. rattus*, previous outbreaks of plague must have occurred due to other species of rodents maintaining the enzootic and epizootic cycles (Politzer 1954). During this period prior to the publication of Politzer's book (1954), *M. natalensis* was the most common commensal rodent found in areas of Uganda not invaded by the *R. rattus*. In East Africa, *M. natalensis* was displaced by invading *R. rattus* and augmented the frail equilibrium of plague in these areas (Janssens and Pattyn 1994). *R. rattus* was not observed in the West Nile region as late as 1938 (Anonymous 1939).

Alternatively, the invasion of *R. rattus* into the West Nile region may have arrived from the west. Rats were observed in Northeast DRC in 1909 in what is now the city of Kisingani, and one report suggested the presence of this species slightly East of this region (AMNH 1909). *R. rattus* was observed from the Lake Albert plague foci in 1948 and was in the Lake Edward focus since 1936 (Davis 1953). The West Nile region of Uganda is comprised of ethnic group diversity distinct from the rest of Uganda. These ethnic groups (Nubi, Alur, Lugbara and others) retain close links with related groups over the borders with Sudan and DRC and, in many aspects, their ties to these groups are closer than their relationship with southern Ugandans (Leopold 2006). The West Nile region was historically maintained as a "closed area" from which outsiders were excluded, cash crops discouraged, and was a labor reserve that supplied workers for plantations in the southern part of Uganda (Leopold 2006). Although Uganda became a British protectorate in 1886, the West Nile region did not fall under this protectorate until 1914. Previously, it had been occupied by Belgian

troops from 1892 until King Leopold's death in 1909, then briefly incorporated into Sudan until 1914 (Leopold 2006). It is possible that the isolation of this region prevented rats from arriving until later in the 20th century (possibly from the East) and/or movement of trans-border populations led to the arrival of plague and rats to the West Nile region from the plague focus in the DRC to the west.

Overall, the major rodents responsible for plague outbreaks in Africa are thought to be *R. rattus*, *Mastomys* spp., *Arvicanthis* spp., and *Tatera* spp. *M. natalensis* and *A. niloticus* appear to be relatively resistant to infection and are, therefore, considered enzootic hosts (Politzer and Meyer 1961). *A. niloticus* ranges from Eastern to Western Africa and throughout the Nile valley to Egypt (Taylor 1984). The rodent is mainly herbivorous feeding on grasses and, unlike the majority of rodents, is diurnal. It is not normally a pest of stored grains, but will occasionally enter grass huts and grain stores (Fieldler 1988).

The major rodent-flea vectors responsible for *Y. pestis* transmission are *X. bransilensis* and *X. cheopis* (Politzer 1954, Gratz 1999). In northern Africa, rodent species of the genus *Gerbillus* and *R. rattus* are the primary reservoirs in the region with *X. cheopis, X. ramesis* and *X. nubica* being the principal flea vectors (Gratz 1999). The introduced (from South America) human flea, *Pulex irritans*, is also a likely transmitter of plague in some areas of Africa including the Lushoto region of northeast Tanzania (Gratz 1999, Laudisoit et al. 2007). The role of the European cat flea, *Ctenocephalides felis strongylus*, in the transmission of plague in East Africa is questionable. Historically, the cat flea was observed to be the most common flea found on humans and parasitizes dogs, cats, rodents, sheep and goats (Hopkins 1947). The cat flea (*P. irritans*) does not undergo "blocking" (the rapid proliferation of *Y. pestis* in the proventriculus, as with *X. cheopis*), which is thought to increase the vector capacity of *Y. pestis* transmission in fleas. Eisen et al. (2006) recently showed that *Oropsylla montana*, a flea of prairie dogs (*Cynomys* spp.) in the United States could transmit the bacteria in an "unblocked" state. *O. montana* does not, or rarely, becomes blocked, but was shown to be capable of transmitting infection for 4 days ("early phase transmission") and may remain infectious for up to 8 weeks. The mechanism for early phase transmission is still unknown, but may be from the regurgitation of previously consumed infectious bloodmeals. Early phase transmission in *C. felis* and *P. irritans* (Verjitski 1908 described in Eisen et al. 2006), was shown, but never effectively evaluated as important in the enzootic cycle of plague. These findings could cause a major re-thinking of the epizoology of historic and current plague outbreaks in the world.

The oriental rat flea, *X. cheopis*, seems to have made two separate invasions of East Africa, one from the north down the Nile Valley, independent of *R. rattus* and one from the East coast along with the arrival of *R. rattus* and transported inland with the rats (Hopkins 1947). The flea was reported in Ugandan areas absent of *R. rattus* (Hopkins 1947) and some authors suggest that the oriental rat flea, *X. cheopis*, coevolved with the Nile rat, *A. niloticus*, in northern Africa (Lewis 1967, Panagiotakopulu 2004). Many fleas including *X. cheopis* and *Ctenocephalides* spp. fleas are capable of growing inside indigenous huts. Politzer (1954) noted that in areas where grain is stored and hence the dust of grain, combined with constant temperature, little ventilation and relatively constant humidity young *X. cheopis* were very successful at breeding and developing.

Risk of exposure to infectious rat fleas are especially high in plague affected rural areas of certain developing countries that have large rat populations as a result of poverty and poor sanitation and food storage practices. Epidemics of bubonic plague are most likely to occur among residents of such areas when plague epizootics ("ratfalls") result in high mortality among rats and cause their fleas to seek other hosts, such as humans (Gage 1998). In West Nile communities, many individuals observed mortality of *R. rattus*, as a pre-cursor to the risk of human plague cases (Genesis Laboratories, unpublished data).

## MATERIALS AND METHODS

This study was performed in cooperation with the Ugandan Viral Research Institute, Ugandan Ministry of Health Plague Research Laboratory in Arua, Uganda. Four field sites were chosen in the Arua district, Vurra Sub-county, Opia and Ayanvu parishes. Each site was typical of a single indigenous homestead typical to the area and contained between 7-20 structures and was bordered by peridomestic area that included crops and habitat typical to the area. Sites were chosen through discussion with the homestead residents and based on negative reports of (1) recent die-offs of rodents, and (2) human plague cases in 2005.

Each homestead was sampled for both rodent and flea populations. Rodents were collected using live traps. If the structures were large (e.g., sleeping, storage or cooking areas), 3 Tomahawk traps (48.3 x 17.1 x 17.1 cm, Tomahawk, WI) were placed in and around each structure (2 inside and 1 outside). If the structure was small (e.g., latrine or washing areas), 2 Tomahawk traps were placed in and around each structure (1 inside and 1 outside). The peridomestic areas surrounding each village were sampled for rodents as well. The estimated center of the village was determined. Using a compass, Tomahawk traps were set on the north, south, east and west compass lines, starting at the edge of the peridomestic area, every ten meters for 100 m into the surrounding habitat. Each line therefore contained up to 11 traps (point 0 – point 10). In the event that trap lines encountered neighboring villages, large roads or habitat that prevented the continuation of the trap-line, they were shortened. Rodent trapping was performed for 3 nights before and after the rodent baiting period. Traps were checked twice a day (am and pm). When captured, each rodent was sedated using Halothane (Halocarbon Laboratories, River Edge, New Jersey, US). Fleas were collected from each rodent by combing them over a white plastic pan with a commercially available flea comb. The number of fleas was counted and recorded. The body weight of each rodent was recorded as well as measurements of body, tail and hind-foot and ear in some cases.

A maximum of 10 larger structures in each village were sampled for fleas. Free-living flea populations in structures were sampled using 3 different methods. The first method sampled free-living fleas within dwellings and employed (1) a modified Kilonzo type flea trap (based on Orachi-Orach 2002), which suspended a flashlight over a metal pan containing water (for collection of photosensitive fleas), and (2) an additional metal pan, containing water, placed in each structure to capture non-photosensitive fleas. Village residents were instructed to turn the flashlights on at night and to leave on throughout the night. Flea traps were used to collect traps for two nights. Fresh batteries were used for each night of collection. Secondly, each home was also evaluated for the presence of rodent burrows both inside and outside the structure. If rodent burrows were present, each burrow was sampled by a "burrow swabbing" technique. A ~15 x 15 cm piece of white flannel, attached to a plumbers snake was inserted into each burrow. The flannel was shaken vigorously as it was removed. Lastly, if clothing or bedding was present in the structures chosen for flea population evaluation, samples of clothing and bedding were removed and placed on a white sheet. Clothing was slowly unraveled and observed for the presence of fleas. Fleas were collected and saved for later evaluation.

The number of rodents collected from each site was totaled, and rodent flea index was defined as the total number of fleas collected from rodents divided by the number of rodents collected. Similarly, household and burrow swab flea indices were calculated by the number of fleas collected for each index divided by the number of traps placed in huts or burrow swabbed.

## RESULTS

On the 4 study locations, 37 commensal area dwellings were evaluated. A total of 94 rodents were collected, nearly equal amounts in both commensal and peridomestic areas (Table 1). Forty-five (47.8%) were captured in the commensal

**Table 1.** Species of rodents collected from commensal and peridomestic areas of all field sites.

| **Commensal (n=45) (Number Captured, % of Captures)** | **Peridomestic (n=49) (Number Captured, % of Captures)** |
|---|---|
| *Rattus rattus* (40, 88.9%) | *Arvicanthis niloticus* (28, 57.1%) |
| *Arvicanthis niloticus* (4, 8.9%) | *Mastomys natalensis* (6, 12.2%) |
| Shrew (species?) (1, 2.2%) | *Rattus rattus* (3, 6.1%) |
| | *Lophuromys flavopunctatus* (3, 6.1%) |
| | *Lophuromys sikapusi* (2, 4.1%) |
| | Gerbil? (Tatera?) (2, 4.1%) |
| | *Lemniscomys* spp. (1, 2.0%) |
| | Unknown species (4, 8.2%) |

representing three species of rodents, while 49 (52.2%) were captured in peridomestic areas, representing at least 8 different species of rodents. Of the rodents captured in commensal areas, the most commonly collected was the invasive *R. rattus* (88.9% of captures; Table 1). Nile rats, *A. niloticus*, comprised 4.1% of captures in commensal areas. In peridomestic areas, the most common rodent collected was *A. niloticus* comprising 57.1% of captures followed by *M. natalensis*, which comprised 12.2% of captures. Of the remaining rodents collected in peridomestic areas, two species represented capture rates of approximately 6% each and the remaining identified rodents <5% each (including *R. rattus*). Of the collected rodents, 8.2% were not identified in the field. Voucher specimens of these rodents were submitted to the Denver Museum of Nature and Science (Denver, Colorado, US). At the time of writing, positive identification had not been determined.

Rodents collected in commensal and peridomestic areas yielded flea indices of 2.3±1.3 and 2.0±0.7, respectively (Table 2). Considering only *R. rattus*, animals collected in commensal and peridomestic areas yielded identical flea indices of 1.7±1.2. Collections of free-living household fleas using traps yielded flea indices of 1.3±0.6 (light trap) and 0.5±0.4 (dark trap). A total of 66 burrows were swabbed for the collection of burrow-dwelling fleas. Fleas collected from swabbing yielded a flea index of 0.19±0.12 (Table 2).

## DISCUSSION

These data suggest that the most common rodent inhabiting these commensal areas are invasive *R. rattus*. No *M. natalensis* were captured in commensal areas supporting the findings of other authors, which indicate that *M. natalensis* is displaced by *R. rattus*. Less than 5% of peridomestic captures were *R. rattus*. The associated flea indices of both peridomestic and commensal rodents was not substantially great. Regarding household flea index obtained from the use of light and dark traps, Orochi-Orach (2002) reports that a household flea index 1.5 represents the need for urgent action to prevent an epidemic of plague. The average household flea index of lighted traps in our study was greater than 1.5 indicating an increased risk of transmission of disease to village residents.

In regards to control, the basic objective of plague prevention is to reduce the likelihood of humans being bitten by infected fleas. Control of rodents in the village setting is often complicated by the constant exposure of commensal rodents to peridomestic wild rodents. Even when rodents are effectively controlled in a village setting, wild rodents can often invade the village areas. Invasion of rodents often occurs after cropping and harvesting procedures and inhabitants should be warned of potential plague risk during these periods. Rodent control around a dwelling is important during this time, but more practically, storing food in rodent-proof containers and destruction of rodent harborage areas are good

**Table 2.** Mean Flea Index in Uganda.

| Collection | Mean Flea Index ± Standard Deviation |
|---|---|
| Commensal rodents[1] | 2.3±1.3 |
| Peridomestic rodents[2] | 2.0±0.7 |
| Commensal *R. rattus*[1] | 1.7±1.2 |
| All *R. rattus*[1, 2] | 1.7±1.2 |
| Household trap[3] | 1.3±0.6 (light trap); 0.5±0.4 (dark trap) |
| Commensal burrow swab[4] | 0.19±0.12 |

[1] Four field sites, n=37 dwellings evaluated.
[2] Starting at edge of commensal area.
[3] Total number of fleas captured / number of trap-nights.
[4] Total number of fleas captured / number of burrows swabbed.

ways to prevent invasion. Storing grain in the ceilings of dwellings will allow a food source for rodents and bring them into close contact with humans. Unkempt dwellings with an accumulation of solid food wastes promote rodent breeding and the distance between the human dwelling and the refuse pile was a risk factor for rat infestation (Olaseha et al. 1994, Makudi et al. 1999). Conversion of land into agriculture increases the habitat for sylvatic rodents and potential of interaction with peridomestic rodents (Makudi et al. 1999). Clearing brush from around a dwelling can create a hindrance to immigrating rodents. Other preventative measures include not sleeping on the floor and occasionally pouring boiling water on the floor of dwelling for sanitation. Fleas typically are associated with dirty or dirt floors, and this method could be especially useful during epidemic periods to kill fleas in dwellings. The use of cats to control plague vectors is questionable. Cats are helpful at controlling rodents, but should be avoided if sick because of potential spread of pneumonic plague (Kilonzo 1999). In Zimbabwe, epidemiological analysis after a human plague outbreak indicated that the presence of a sick cat was a risk factor in contracting the disease (Manungo et al. 1998).

Plague control targeting rodents elsewhere in Africa has focused on dusting of human dwellings and rodent burrows with powdered insecticide, rodent trapping, and education in Namibia (Shangula 1998), DDT powder followed by anticoagulant bait in Botswana (Kumaresan et al. 1991), trapping in Tanzania, Nigeria, and Madagascar, bounty schemes and rodenticide use in Tanzania as well as the burning of dwellings while villagers killed escaping rodents with clubs (Kilonzo 1994, Kilonzo 1999). Recently in Madagascar, deltamethrin and diazinon dust were evaluated to control fleas on rats (primarily *R. rattus*). Dust was placed at burrow openings. Flea suppression was obtained during the first month, but each dusting was unsuccessful at maintaining residual control of fleas on the rodent host (Rotovonjato and Duchemin 2001). Often in Africa, plague control is performed reactively, not proactively (Govere and Dürrheim 1999), often due to lack of resources for surveillance. In Tanzania, governmental financial constraints on plague control activities in some years are believed to have led to the outbreaks of plague in following years (Kilonzo and Komba 1993).

Regardless of the method used to control rodents and fleas, it is imperative that flea control be performed before, or concurrently with, rodent control practices. Lethally controlling rodents without first controlling fleas could create an increased risk of plague to humans as many fleas are released into the environment after widespread rodent deaths occur (Gage et al. 1995). An example of this occurred in Tanzania in which an epidemic of human plague was reported to be preceded by rodent control operations indiscriminately using rodenticides, thereby causing fleas to be released into the environment and, subsequently, parasitizing humans (Kilonzo 1994). Likewise, a case control study in Peru investigating the risk factors of bubonic plague found a strong association with indiscriminate rat extermination (Odds Ratio, OR=71.4). Other risk factors determined were: the tendency to sleep on the floor (OR=16), the presence of rubbish inside and outside of the dwelling (OR=8.89), the death of guinea pigs, the increase of flea and rodent populations, and the storage of food inside the dwelling (Angulo et al. 1997).

Regardless of how and when *R. rattus* arrived in the West Nile region of Uganda, the presence of the invasive rodent appears to now be critical in the epizootic cycle of plague. The rodent arrived into an area that had an already established, or soon to be established, a foci of plague, likely displaced the resident commensal rodent populations and took its place near humans, as it has done efficiently throughout the world. Unfortunately, its presence has been detrimental to the people of Northwest Uganda causing increased disease burden of plague. Additional research is needed to clearly understand the zoonotic cycle of plague in this region as well as methods to prevent and control the disease.

## ACKNOWLEDGMENTS

Our gratitude is expressed to the staff of the UVRI Plague Laboratory in Arua, The Ugandan Ministry of Health, Richard Poché, J. Joshua Bruening, Larisa Polykova, Russell Enscore and Kenneth Gage at the CDC DVBID, field technicians Crystal Campbell and Katie Borchert, and especially, the residents and homeowners in the villages used for our field sites.

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