

AS CEPAS DE FILOVIRUS, O MEIO AMBIENTE E OS MORCEGOS DENTRO E FORA DA AFRICA

(Filovirus strains, the environment conditions and the Bats in and out of Africa)

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RESUMO: Marburgvirus (MARV) e Ebolavirus (EBOV) pertencem à família Filoviridae. A infecção por MARV e EBOV pode causar uma devastadora febre hemorrágica em primatas. Os surtos de EBOV ocorreram nas florestas úmidas da África Central e Ocidental e MARV nas zonas mais secas e mais abertas da África Central e Oriental, também presentes no Sudeste Asiático e nas Filipinas. Nesta revisão, um paralelo da fauna de morcegos e condições climáticas em trópicos africanos onde a maioria dos focos de Filovírus ocorreu e as condições de ambientais brasileiros foram consideradas. Os morcegos de frutas da família Pteropodidae (Megachiroptera) que foram considerados um dos possíveis reservatórios dos vírus não estão representados na fauna brasileira. Do mesmo modo, não há representantes de *Miniopterus schreibersii* que foram associados ao vírus Lloviu e nenhum outro membro da subfamília Miniopterinae (família Vespertilionidae). Portanto, a infecção por suínos Ebolavirus do subtipo Reston (RESTV) e a possibilidade desses animais serem reservatórios naturais de vírus devem ser um alerta sobre a importância de medidas preventivas para evitar a entrada deste vírus no país.

Palavras-chave: marburgvirus (MARV); ebolavirus (EBOV); histórico e alerta

ABSTRACT: Marburgvirus (MARV) and Ebolavirus (EBOV) belong to Filoviridae family. The MARV and EBOV infection can cause a devastating hemorrhagic fever in primates. The EBOV outbreaks occurred in the humid rain forests of central and western Africa and MARV in the drier and more open areas of central and eastern Africa also present in Southeast Asia and in the Philippines. In this review, a parallel of the bat fauna and climate conditions in Afrotropics, where the most Filovirus outbreaks occurred, and Brazilian environments conditions were considered. The fruit bat from Pteropodidae family (Megabats) which were considered one of the possible reservoir species for the virus, are not represented in Brazilian fauna. In the similar way, there not representative of *Miniopterus schreibersii* bats which were associated with Lloviu virus neither other member of sub family Miniopterinae (Vespertilionidae family). Therefore, the swine infection of Ebolavirus sub type Reston (RESTV) and the possibility of pigs as natural virus reservoirs should be an alert about the importance of preventive measures to avoid the entrance of this virus in country.

Key Words: marburgvirus (MARV); ebolavirus (EBOV); an historic and alert

INTRODUCTION

The Marburgvirus (MARV) and the Ebolavirus (EBOV) infection can cause a devastating hemorrhagic fever (HF) in primates (Gonzalez; Pourrut, 2007; Weingartl et al, 2012). In 1967, the MARV was first isolated in the Germany cities of Marburg and Frankfurt and in Belgrade (former Yugoslavia), in professionals that worked in laboratory and manipulated tissues and blood of a monkey *Cercopithecus aethiops* imported from Uganda. In total, 31 people were infected, seven of which died (CARROLL *et al.* 2013). In 1976, the EBOV in turn was isolated in two outbreaks occurred almost simultaneously in two African countries: Sudan and Zaire, present Democratic Republic of the Congo (DRC). In these outbreaks 550 cases and 430 deaths related to the EBOV were registered (BEER *et al.*, 1999). After 18 years, the MARV and the EBOV reappeared in distinct outbreaks of HF in the African continent. The MARV aggressive outbreak occurred in Angola in 2004 resulted in the high infant mortality rate (TOWNER *et al.*, 2006). In 2014 many HF outbreaks related to EBOV in West African countries, especially Guinea, Sierra Leone and Liberia with more than 11.000 deaths caused a commotion and panic in worldwide

(<http://www.cdc.gov/vhf/ebola/outbreaks/2014-west-africa>).

The MARV and the EBOV belong to the Filoviridae family in the Mononegavirales order because harboring a non-segmented RNA genome with negative sense. They have distinct characteristics in the serologic, biochemical and genetic profile (BEER *et al.* 1999; OLIVAL; HAYMAN, 2014). The Filoviridae genomic RNA contain seven genes which code for nucleoprotein (NP), P protein (VP35), matrix protein (VP40), glycoprotein (GP), second nucleoprotein (VP30), protein associated to the envelope (VP24) e a RNA – RNA dependent Polymerase (L) distributed in the following arrangement 3’-

NP- VP35- VP40-GP-VP30-VP24-L-5’ (IKEGAMI *et al.*, 2001). The EBOV GP gene is found in two forms sharing identical NH 2 terminal sequences: secreted glycoprotein (sGP) and transmembrane glycoprotein GP (LEE, SAPHIRE, 2009). The MARV genus harbors the strains *Marburgvirus marburgvirus* which was the former *Lake Victoria marburgvirus* or *Musoke virus* (Kenya, 1980) and the *Ravn virus* (1987). The EBOV were subdivided into five subtypes based on the significant differences in antigenicity and in the genome nucleotides sequence: Zaire (EBOZ), Sudan (EBOS), Reston (RESTV) and the Tai Forest virus – Ivory Coast (TAFV) and the Bundibugyo (TOWNER *et al.*, 2008).

The main mode of transmission between human and non-human primates was the direct and/or indirect contact with secretions, excretions, tissues and especially blood from infected people and animals (BEER *et al.*, 1999). After investigation in several wild animals that could harbor the virus in nature, the fruit bat (Pteropodidae) and the insectivorous bats were considered a possible reservoir species for the virus (TANIGUCHI *et al.*, 2011).

In this study, a parallel of the bat fauna and climate conditions in Afrotropics, where the most Filovirus outbreaks occurred, and Brazilian environments conditions showing the differences in the Brazilian bats fauna. The Filovirus described out Africa were described. The swine infection with Ebolavirus subtype Reston (RESTV) and the possibility of pigs as natural reservoirs of the virus were also considered.

Filovirus outbreaks: the environment conditions and the Bats

The EBOV outbreaks occurred in the humid rain forests of central and western Africa and MARV in the drier and more open areas of central and eastern Africa also present in Southeast Asia and in the Philippines (PETERSON *et al.*, 2004). The larger part of the humid rain forests is located in or around the tropic of equator.

This kind of biome composed vegetation and animal species which interacts in specific ecologic environment marked by high temperature and intense rainfall. Besides Africa and Asia, the tropical rainforests climate expands through Central America and South America including larger geographic area of Brazil: Amazon forest. Since the Brazilian continental territory expands beyond from the humid rain forest to others biomes with different climate conditions as humid or drier as Cerrado, Mata Atlântica, Caatinga, Pampa and Pantanal. Those varied biomes consist of several kinds of forests expanding over the country providing the habitat for several animal species, including the bats, which could be susceptible to infection with Filovirus.

The bats are mammals belonging to the order Chiroptera. This order is divided into two sub-orders the Microchiroptera or Microbat and Megachiroptera or Megabat. At least nine families of the Microchiroptera are found in Brazilian forests and caves in contrast, the Megachiroptera are not described in any Brazilian forest environment (REIS *et al.*, 2007).

The Megachiroptera has one only family Pteropodidae also called fruit bats with 166 species which are distributed through the Europe, tropical region of Africa, India, Southeast of Asia and Australia (REIS *et al.*, 2007). They are large bats when compared to insectivorous bats, they do not navigate by echolocation and are herbivores and feed on fruit, nectar or pollen (REIS *et al.*, 2007). Until now, the Filovirus have been identified either antibodies detection or isolation in several species of fruit bats suggesting the role of this family as reservoirs and vectors for EBOV and MARV (LAMINGER; PRINZ, 2010).

The MARV was associated with outbreaks in persons who visited caves or worked in mines situated particularly in central African tropical rain forest (TOWNER *et al.*, 2009). For example, in 1980 and 1987, two tourists became ill after visited to the Kitum Cave famous “elephant

caves” at Mount Elgon, in Kenya, they are infected with the MARV. Swanepoel *et al.* (2007), studied the fauna of Goroubwama mine in Durba village in RDC, found MARV nucleic acid in tissues of Egyptian fruit bats (*Rousettus aegyptiacus*) and antibody in the insectivorous African bats (*Rhinolophus eloquens* and *Miniopterus inflatus*) but, failed to isolate virus. The bats roosted in Goroubwama mine were associated to MARV outbreak occurred from 1998 to 2000 which was described as repeated occurrences of infections associated with multiple genetic lineages of virus circulated during the outbreak (TOWNER *et al.*, 2009).

In the Gabon, nucleotides sequences of MARV were identified in *Rousettus aegyptiacus* surveyed in 2005 and 2006 as well, from bats from caves in northern of country in 2009 and 2010, which suggested the enzootic character of MARV infection in Gabon (MAGANGA *et al.*, 2011). In the Uganda, in 2007, the MARV nucleotides sequences and virus-specific antibodies were detected in 5.1% and 2.4%, respectively, of 611 *Rousettus aegyptiacus* roosted in Kitaka Cave during an outbreak of HF occurred among lead and gold miners. Those MARV outbreaks suggested the *Rousettus aegyptiacus* from the Pteropodidae family could be a natural reservoir of MARV consequently a risk for animals and human which interacted directed with blood, secretion and excretions of this bat (TOWNER *et al.*, 2009; MAGANGA *et al.*, 2011).

In turn, Pourrut *et al.* (2007) demonstrated nucleotides sequences of ZEBOV and immunoglobulin G (IgG) virus-specific in *Hypsignathus monstrosus*, *Epomops franqueti* and *Myonycteris torquata* collected during 2003–2006 in Gabon and the RDC. Bats naturally or experimentally infected with ZEBOV or MARV are healthy and shed virus in their feces for up to 3 weeks (SWANEPOEL *et al.*, 1996; TOWNER *et al.*, 2006).

Although, the survey of antibodies and virus nucleotides sequences detected in bats have been suggested the Filovirus as a

virus from Africa, several investigations have demonstrated the presence of Filovirus in bats from others continents. Taniguchi *et al.* (2011) surveyed the prevalence of specific-antibody against RESTV in bats from the Philippines during 2008 – 2009, the survey figured out positive in the fruit bat *Rousettus amplexicaudatus*. Those bats were trapped in Diliman forest and Quezon forest around 30km and 60km, respectively from the monkey facility and the Bulacan farm where REBOV infections in monkeys and swine, were detected. Yuan *et al.* (2012) survey the antibodies against EBOV in Chinese bat populations applied an enzyme linked immunosorbent assay (ELISA) with a recombinant nucleocapsid protein as antigen. The authors demonstrated the presence of antibodies to Ebolavirus in 32 of 843 bat sera samples and 10 of 16 were further confirmed by western blot analysis. HE *et al.* (2015) found Filovirus RNA sequence in a fruit bats *Rousettus leschenaultia* captured in Yunnan Province, China. Olival *et al.* (2013) surveyed bats from Bangladesh being 141 *Rousettus leschenaultia*, 75 *Cynopterus* spp, 59 *Megaderma lyra*, and 1 *Macroglossus sobrinus* during April 2010–March 2011 from the Faridpur, Rajbari, Lalmonirhat, and Comilla Districts from Bangladesh. The authors found that five (3.5%) bats from 276 tested were positive for antibodies against Ebola Zaire and Reston viruses. Although no virus was detected by PCR, this survey suggests that EBOV can be widely distributed throughout Asia.

However, Wacharapluesadee *et al* (2015) studied wildlife during active surveillance for zoonotic pathogens conducted a cross-sectional study for EBOV infection in bats and macaques in Thailand surveyed 699 bats species being 532 from the Pteropodidae family from this number 500 *Pteropus lylei* bats tested by Elisa to antibody against EBOV were collected from 10 roosting sites during March–June 2014. The nucleotides sequences of virus were investigate by PCR in remaining bats samples belonged to the following families of bats including 32 from Pteropodidae,

Hipposideridae, Megadermatidae, Vespertilionidae, Rhinolophidae and Emballonuridae. They did find neither antibody nor virus RNA sequence in all animal or specimens tested.

Filovirus strains out of Africa

From Spain: Lloviu virus

A new genus of Filoviridae family was published in 2011, concerned an ebolavirus-like filovirus, the Lloviu virus (LLOV) identified in 2002. The virus nucleotide sequences were detected in extracts from lung, liver, rectal swabs or spleen of five dead insectivorous bats from Cueva del Lloviu localized in Asturias, Spain (NEGREDO *et al*, 2011). The mortality dies-off in colonies of Schreiber's bats *Miniopterus schreibersii* from Vespertilionidae family. This bat family least four geographically discrete lineages distributed in Oceania, southern Europe, Southern Africa, and Southeast Asia. This finding was also highly significant since it was the first discovery of an *Ebolavirus* outside of Africa or Asia. The LLOV did not infect the humans which manipulated more the 200.000 samples.

From China: Bt-DH04

He *et al.* (2015) published a likely a novel bat-borne filovirus named Bt-DH04 was detected in apparently healthy *Rousettus leschenaultia* fruit bats (Pterotidae family) were captured in Yunnan Province, China, in 2013. The authors affirmed based in the phylogenetic analysis the Bt-DH04 strain is at basal position and intermediate between EBOV and MARV. It is divergent from all known filoviruses, with F1 sharing the highest nucleotide identities (46%–49%) to members of the genus *Ebolavirus*, followed by 44% to LLOV and <40% to MARV.

From Philippines: Ebola Reston

A new subtype of the Ebolavirus was described in 1989; Ebolavirus subtype Reston (RESTV), thus named because it was discovered in the city of Reston, Virginia in

the United States. The RESTV was identified co-infecting with the simian hemorrhagic fever virus - SHFV of the family Arteriviridae, *Cynomolgus* monkeys imported from the Philippines islands. This was the first time an Ebolavirus was identified in non-African monkeys and outside of Africa (MIRANDA; MIRANDA, 2011). Although the RESTV was described infecting monkeys from Philippines, the geographic origin and distribution of RESTV is controversy. But, the ecologic conditions found in the Philippines are similar to African rainforest where EBOV occurred. It can suggest the potential of these areas in harboring the host which could maintain the virus in nature (PETERSON *et al.*, 2004). In 2008, the RESTV was identified in co-infection with porcine respiratory and reproductive syndrome virus - PRRSV of the family Arteriviridae in swine herds from Philippine islands and in China (MIRANDA; MIRANDA, 2011; PAN *et al.*, 2014). RESTV review was expanding due the importance of this virus for swine producers and for the risk of swine to be natural reservoirs for an Ebolavirus for animals and human.

Identification of the Reston Ebolavirus in cynomolgus monkeys

In October 1989, an outbreak of hemorrhagic fever affected cynomolgus monkeys *Macaca fascicularis* at the quarantine headquarters of the American company Hazleton Research Products in the city of Reston in the state of Virginia, USA. According to Jahrling *et al.* (1990), 100 cynomolgus monkeys were taken in quarantine to the F shed where there were already 500 cynomolgus, all of non-African origin. In one month some animals died and others became ill and were euthanized. Necropsy findings as splenomegaly, enlarged kidneys and hemorrhagic organs and a great increase in the lactic dehydrogenase rate in serum suggested a fatal viral disease to the simians: the simian hemorrhagic fever (SHF, Arteriviridae). The laboratorial diagnosis confirmed the SHF

virus (SHFV) infection in the animals. At the same time, the presence of the Filovirus was observed in tissues through an electron microscopy. Following studies identified in the co-infection a virus from the Ebolavirus genus denominated Reston (RESTV) and the previously collected samples were studied again. The isolation of the Ebolavirus in the F samples confirmed the infection. The RESTV was pathogenic to the infected monkeys; however, this viral strain did not cause disease in human beings proven infected by the virus due to the presence of antibodies against RESTV (ROLLIN *et al.*, 1999). In 1990 and 1992, the RESTV was diagnosed in primates from the Philippine islands in the city of Alicia in the state of Texas, USA and in the city of Siena, in Italy, in SHF outbreaks. In 1996, a new outbreak of the RESTV in USA was described in monkeys imported from the Philippines, once again in the city of Alicia in Texas (MIRANDA *et al.*, 2002). The tracking of the RESTV infection in all the outbreaks pointed to the same breeding of monkeys at 40km from Manila.

The spreading mechanism by which RESTV either remained or was re-introduced into the nursery was not cleared. However, inside the headquarters and between the American quarantine rooms the aerogenous spread of the virus was suggested. The experimental infection only with the RESTV of the cynomolgus caused a similar disease of humans and monkeys infected with the subtypes EBOZ and EBOS (JAHRLING *et al.*, 1996; MIRANDA *et al.*, 2002; MARSH *et al.*, 2011). The main pathological events diagnosed were anorexia, nasal discharge, petechial hemorrhages on the face and severe subcutaneous hemorrhage and splenomegaly. Nonetheless, viral particles were observed in the interstitial alveolar cells and in the alveoli suggesting potential to the generation of infectious aerosols (JAHRLING *et al.*, 1996).

Reston Ebolavirus in swine

In 2008, an acute disease in swine herds causing breathing problems and abortion in multiple outbreaks in the Philippine Islands led the country to ask for American help for the diagnosis of the disease that was suspected to be Porcine respiratory and reproductive syndrome (PRRS). This disease is caused by PRRS virus (PRRSV) which is also a member of the family Arteriviridae, i.e., close relative of the SHFV (NORMILE, 2009; DIETZE *et al.*, 2011). The PRRSV was diagnosed and by the sequencing of the NSP2 gene it showed homology with the isolated from the Chinese strain of atypical PRRSV. However, the inoculation of macerated lymph nodes of animals killed by PRRSV, in Vero cells triggered cytopathic effect. The Vero cells are resistant to the PRRSV infection and could not suffer cytopathic effect. A new molecular investigation led to the discovery of the genes L of the Reston Ebolavirus. Immediately the samples were taken to the safety level four laboratory and the specific exams were done and proved the animals were also infected with Reston Ebolavirus. It is interesting to add that in some samples were identified genome of porcine Circovirus type 2 (PCV-2) and Porcine Teschovirus-1 (PTV-1) (BARRETTE *et al.*, 2009). The virus isolated from dead swine of the Philippines swine herds was experimentally inoculated in healthy swine of five weeks old. The inoculated animals were infected but didn't show clinical signs of the disease. However, replication of the virus in the internal organs of the animals and in the nasopharyngeal alveolar tissues was registered (MARSH *et al.*, 2011).

In Shanghai, China, Pan and contributors (2014) demonstrated that 2.92% (4/137) of samples of spleen of swine that died with clinical signs of the atypical PRRS in the period from February to September 2011 were positive and harbored sequences of the Reston Ebolavirus. All the Reston positive swine were less than eight weeks old and were co-infected with PRRSV.

DISCUSSION

The prevention of a virus infectious disease is fundamental to avoid the entrance the virus in the country but, if it is not completely possible at least, prepare the country and its sanitary institutions for the better and more efficient attention to the people or animal infected eventually, as well explained by De La Torre *et al.* (2014). The risk analysis as the first barrier to prevent the introduction of the infection in the country, the second barrier represented by passenger baggage control coming from risk areas, should be strengthened (FREITAS, LYRA, 2015). When the virus infection in human or animals can develop to HF outbreak with high level of morbidity and mortality, the prevention and surveillance became a critical factor (DE LA TORRE *et al.*, 2014; Beer *et al.*, 1999). The Filovirus infection as a zoonotic factor can development in an human since asymptomatic infection as RESTV infection to epidemic outbreaks as Ebola Zaire with death rates of more than 80%

(<http://www.cdc.gov/vhf/ebola/outbreaks/2014-west-africa>).

The knowledge of the possible virus reservoirs and its environments conditions allowed to realized and minimized the risk factors. The most of Ebolavirus epidemic outbreaks occurred in countries localized in geographic regions in African and Asian rain forest with hot and humid climate (PETERSON *et al.*, 2004; GONZALEZ *et al.*, 2007). This biome can be described in Brazil and others countries from Central and South America. Peterson *et al.* (2006) described the geographic potential for Filovirus outbreaks, using an ecologic niche modeling. The survey of more than 1000 animals species as possible reservoirs of the virus pointed of to the bats especially the fruit bats, Megabats as natural reservoir of Filovirus (SWANEPOEL *et al.*, 2007; TOWNER *et al.*, 2009; LAMINGER; PRINZ, 2010; LEROY *et al.*, 2005; TANIGUCHI *et al.*, 2011; OLIVAL *et al.*, 2013; HE *et al.*, 2015). Gonzales *et al.*

(2007) explained the importance of bat species as potential reservoir species of EBOV since when bats eat fruits drops the remains of fruit on the ground and if the bats shed virus in saliva the transmission to dwelling mammals will be possible.

The Lloviu virus the infection in die-off bats *Miniopterus schreibersii* appeared accidental but, pathologic for this bats can suggested the tropism of Filovirus for bats, (NEGREDO et al, 2011). *Miniopterus schreibersii* bats belonged the sub family Miniopterinae, family Vespertilionidae which is one family with greater diversity and geographical distribution among Chiroptera distributed throughout the southern Palearctic, Ethiopic, Oriental, and Australian regions also present in the southern half from Iberia to the Caucasus (REIS et al, 2007). However, the subfamily Miniopterinae is not represented in Brazilian environments. In the Brazilian bat fauna, the Vespertilionidae family has only the subfamilies Vespertilioninae e Myotinae (REIS et al, 2007). Although, the habit of several terrestrial dwelling mammals as rodents, agouti, hare, foxes, maned-wolf, hedgehog and others, is to feed on partially bat bitted fruits (Biota frugivora) which is one of ways to Filovirus infection can be acquired (REIS et al, 2006).

The RESTV ability to infect swine may turn in the most important risk factor for Brazilian surveillance of swine rearing. The identification of the RESTV Ebolavirus in monkeys that presented acute simian hemorrhagic fever (SHF) in Reston unity in Virginia, USA, initially occurred by observation of viral particles by electron microscopy which referred to the long tubular shape typical of the Filovirus (JAHRLING et al., 1990). Then, these samples was assayed by ELISA (Enzymatic linked immunosorbent assay) capture and it was confirmed the presence of the Ebolavirus. Before the Ebolavirus was detected, every effort was to demonstrate the presence of the SHFV, the workers who manipulated the monkeys infected samples did not care about the RESTV although, they

were infected with the virus did not develop clinical sign disease. The infection was demonstrated by the production of antibodies IgG against the RESTV (JAHRLING, 1990).

In swine, the infection with RESTV was diagnosed unexpectedly, because primarily the investigation was led toward the PRRSV (DIETZE et al., 2011). The occurrence of the cytopathic effect in Vero cells induced by swine samples PRRVS positive led to a new investigation that allowed the molecular detection of nucleotide sequences RESTV, the virus CVS-II (with immunosuppressive potential) and the PTV-1 or Teschovirus-A (Picornaviridae) were also detected (BARRETE et al., 2009). Highlighting that the atypical PRRS was defined by the high virulence and pathogenicity power of the virus (Highly Virulent PRRS virus or HVPRRV). And, since 2006, the HVPRRV was spreading through the swine herds of China, Vietnam, Philippines and Thailand (BARRETE et al, 2009). In 2010, the HVPRRV was identified in outbreaks in the Lao People's Democratic Republic and Cambodia while caused an unprecedented epidemic in Thailand (DIETZE et al., 2011).

In China, the RESTV genomic RNA identified during an atypical PRRSV outbreak occurred in Shanghai, 2008, showed a similarity of 96.1% - 98.9% when compared with the RESTV discovered in the Philippines Islands (Pan et al, 2014). Only one sequence analyzed was quite close to the swine RESTV from the Philippines. But, three sequences showed differences suggesting mutations from the genetic origin. This molecular aspect points to a polyphyletic evolution or the possibility to exist different ancestors rather than a single to all viruses (BARRETTE et al., 2009).

Morris (2009) suggested virus transmission from swine to humans was by based on government information from the Philippines about the infection of workers who had contact with infected animals: In January 23rd 2009, a person that had contact with an infected swine presented IgG

antibodies against the Ebola Reston virus. A week later, in January 30th, four more people presented antibodies against the Ebola Reston: two farm workers in Bulacan, one farm worker in Pangasinan and one refrigerator worker in Pangasinan, these people had direct contact with sick pigs. Pan *et al.*, 2014, consider that the the role of the pig as in incidental host or as part of the transmission cycle has yet to be determined.

The Ebola Reston was transmitted to human beings without causing clinical disease, however, the assertion that RESTV does not cause disease in human beings has been considered precipitated, because, not only the number of infected people was small, they were healthy individuals. The infection of other groupings of people of different ages and diverse immunological conditions such as pregnant women, children and people with impaired immune system and/or with inferior health conditions, was not analyzed (GIL *et al.*, 2009; MORRIS, 2009; MIRANDA; MIRANDA, 2011).

The co-infections detected on *Cynomolgus monkeys* (SHFV e RESTV) in Reston city, USA and the co-infections of swine (PRRSV and RESTV) on the Philippines and in China, have in common, the presence of the Ebolavirus and of the Arterivirus, since both SHFV and PRRSV belong to the same group. Pan *et al.* (2014) suggested that it was possible that the Ebola Reston co-infection had contributed to the severity of atypical PRRS that spread through Asia, since when the RESTV was inoculated in healthy swine did not cause clinical disease or hemorrhagic fever (DIETZE *et al.*, 2011). The Arterivirus such as PRRSV can interfere in the swine immune system and trigger a weak innate antiviral immune response by interfering on the metabolism of the Interferon type I (ZHOU *et al.*, 2012). However, the Ebolavirus infection disturbs the immune innate and adaptive response (BRAY; MAHANTY, 2003).

Although it can be early to define the role of pigs as virus vector or as incidentally-infected hosts, these information alerts for

prevention and care in the handling samples that arrive for diagnose and/or research of a virus or specific viral disease, because when a viral disease emerges or re-emerges presenting or aggravating a clinical picture the possibility of a viral genomic mutation that would lead to this new clinical picture must be considered. But one should not rule out the risk that co-infection with another virus or an immunosuppressive agent or factor favors the primary pathogen agent associated with the clinical picture.

CONCLUSIONS

The Ebolavirus infections in human and animals were reported in the most in African rain forest biomes. Although the similar climate conditions the bat fauna is not the same. The fruit bats (Pteropodidae) were involved as reservoirs of Ebolavirus and Marburgvirus. However, the Megabats are not represented in Brazilian fauna. In the similar way, there are not representative of *Miniopterus schreibersii* bats which were infected with Lloviu virus neither other member of sub family Miniopterinae, family Vespertilionidae. The RESTV was isolated in swine originated from the Philippines with an acute clinical sign of PRRS. The atypical PRRS outbreaks spread in China, Vietnam, Laos, and Thailand. Currently, antibodies against RESTV have been demonstrated among pigs from several Asian countries. In turn, the infection Ebolavirus sub type Reston (RESTV) in swine and the possibility of pigs as natural reservoirs of the virus should be an alert about the importance of preventive measures to avoid the entrance of this virus in country.

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