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Preparedness for potential PED emergence in France: modelling PEDV spread in a densely populated pig area

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Since april 2013, a devastating epizooty of porcine epidemic diarrhea (PED) has been striking USA and Canada that were previously free from this disease. More than 6400 herds have been affected with up to 90-95% mortality in suckling piglets. PED was first described in England in 1971 and is characterized by watery diarrhea and vomiting. In Europe, DEP has been described since the 1970's till the end of the 1990's except few cases reported in Italy in 2006. The epidemic character of the PED outbreak in Italy as well as some seroprevalence studies carried out in different countries suggest that the immunity of the population in Europe against PEDV is low.

The objective of the study was to assess the transmission dynamics of PEDV whenever introduced in a pig densely populated area in Europe using a modelling approach. Brittany, one of the main pig production area in France, was selected for application. A spatio-temporal individual-based, stochastic model has been built to simulate the spread of the virus in the area. Data reporting geographical location of the herds, herd type and size, annual movements of animals between herds and frequency of contacts with vehicles and other mechanic vectors have been used for parameterization. The model was developed using the North American Animal Disease Spread Model (Harvey et al., 2007). The model structure is based on a compartmental SEIRS structure with state transitions governed by parameters derived from the literature (Martelli et al., 2008; Pensaert and De Bouck, 1978) and reports from the current epizooty in the USA and Canada (Poulin and Klopfenstein, 2013). The impact of several combinations of control measures was also assessed (limitation of animal movements in a high risk zone around the identified case, increase biosecurity). Simulation results showed that a massive epizooty could be expected with similar characteristics in terms of estimated R0 than the observed epizooty in the US. The most efficient control measures were the limitation of pig movements combined with a detection delay shorter than 10 days and increased biosecurity. Whenever animal movements were not suppressed and only biosecurity was enhanced, the epidemics could potentially turn to an enzootic form.

Novel H1N2 swine influenza reassortant strain in Swedish pig population derived from the pandemic H1N1/2009 and avian like H1N2 viruses

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Swine influenza viruses (SIV), influenza type A virus, remains an important concern for animal and human health. Once considered a seasonal infection in pigs with rapid onset and recovery, SIV is now considered as one of the most economically significant pathogens causing morbidity and sometimes mortality in pig populations, particularly when infection occurs alongside with other respiratory pathogens. In pig population several subtypes (H1N1, H1N2, H3N2, H1N1pnd09 & avian-like H1N2) are currently circulating worldwide.

The Avian like H1N2 subtype influenza A virus was exclusively prevalent among the swine population in Sweden since 2009. In May 2013 a clinical outbreak was reported on a pig farm located in southern part of the Sweden. The clinical signs observed were coughing, dyspnoea, fever, anorexia and depression with roughly estimated 20% morbidity. Presence of H1-pdm09 infection was detected and confirmed by detail laboratory analysis as part of the diagnostic investigation.

The molecular characterization and phylogenetic analysis of the newly isolated viruses revealed that they possess pandemic H1N1 lineage HA and internal genes. However the NA gene was closely related to H1N2 SIV strains, previously isolated in Swedish pig population. Here we report the molecular and biological characteristics of the novel H1N2 viruses.

Long-term immunity against Schmallenberg virus in sheep after natural infection

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Schmallenberg virus (SBV), a novel *Orthobunyavirus* from the family *Bunyaviridae* and the Simbu serogroup, was discovered in autumn 2011 in Germany and rapidly spread among European countries. First clinical evidences of SBV transplacental infection were detected during the lambing period of January 2012 among still- or newborn lambs of the Ovine Research Center (ORC) belonging to the University of Namur and located in the southern part of Belgium. SBV emerged on 6th September and the period of virus circulation was estimated to extend to 26th October 2011 leading to rapid infection of the entire sheep flock (n=400) with 98.8% SBV seroprevalence in February 2012. The objective of this study was to determine anti-SBV antibody titers over years in adult ewes that experienced SBV outbreak in 2011.

Serum was collected yearly in the same 10 adult ewes at the end of winter in 2012, 2013 and 2014. Serum neutralization tests (SNT) were performed on serum samples with 2-fold serial dilutions. Results of SNT were expressed as the log2 transformed dilution that neutralizes 50% of the challenge virus (ED50) and were considered positive if log2 ED50 was > 3.49.

At first sampling (2012), all ewes were SBV seropositive with a median (min – max) log2 ED50 anti-SBV antibody titer of 8.22 (6.48 – 9.97). One year later (2013), these animals remained 100% seropositive with a median (min-max) log2 ED50 anti-SBV antibody titer of 8.47 (6.48 – 10.96). Six ewes presented an increase of evaluated antibody titer between 2012 and 2013 while only 3 showed a slight decrease. In 2014, median (min-max) log2 ED50 anti-SBV antibody titer equaled 7.47 (6.47 – 7.97) and was significantly lower than those obtained in 2012 and 2013 ($p<0.05$). Seven animals presented a decrease of antibody titers between 2013 and 2014 but all sheep remained SBV seropositive.

Between mid-July and mid-October 2012, Claine et al. (2013) demonstrated important SBV re-emergence among 50 ewe lambs of the same sheep flock. The increase of anti-SBV antibody titer measured in six ewes between 2012 and 2013 could probably be explained by a booster effect. In 2014, all animals remained seropositive but a decrease of anti-SBV antibody titer was largely observed and can be related to the absence of SBV circulation in 2013. This study suggests evidences of neutralizing antibodies persistence against Schmallenberg virus at least two years after first natural infection and underlines the booster effect of SBV re-emergence on anti-SBV antibody titer in sheep. However, these preliminary results need to be confirmed on a larger scale.

Claine F, Coupeau D, Wiggers L, Muylkens B, Kirschvink N. Schmallenberg virus among female lambs, Belgium, 2012. Emerg Infect Dis 2013; 19: 1115-7.

Evaluation of the in utero infection caused by SBV on goats

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Schmallenberg virus (SBV) infections of pregnant ruminant dams can cause teratogenic effects including malformation of lambs, calves and goat kids with the arthrogryposis-hydranencephaly-syndrome (AHS) as the guiding symptom complex. Field observations suggest that goats are generally less prone to exhibit SBV induced fetopathologies than cows and ewes. However, at present it is still unknown whether this difference is linked to a different susceptibility of the three ruminant livestock species to SBV infections, or is rather due to different housing conditions or a preference of Culicoides biting midges for cattle and sheep. In the present study we have assessed the ability of SBV to elicit teratogenic effects in pregnant goats at two different times of gestation. A total of 30 goats were estrus synchronized using vaginal sponges and artificially inseminated with frozen semen from a single he-goat. Pregnancy was assessed by echography and confirmed for 14 out of 30 goats at day 42 of gestation. The 14 pregnant dams were distributed into 3 different groups: in group A, 5 dams were inoculated with 2 x 0.5 mL of infectious serum at day 28 of gestation; in group B, 5 dams were inoculated with infectious serum at day 42 of gestation; in the (mock control) group C, dams were (PBS)-inoculated at day 28 (2 animals) or 42 (2 animals) of gestation. At days 53 to 56 all animals were euthanized and necropsies were performed. All SBV infected dams (groups A/C) developed viremia between days 3 and 5 p.i. and SBV-specific seroconversion from day 14 p.i. However, no clinical signs of infection (including elevated body temperatures) could be observed for the SBV-inoculated animal groups. Upon necropsy, no gross lesions could be detected in the maternal carcasses from all groups. All 9 foetuses (1 x 1, 2 x 2, and 1 x 4 foetuses per dam) obtained from the 4 dams of the control group C showed a normal size and morphology. In contrast, among the 11 foetuses (1 x 1, 2 x 2, and 2 x 3 foetuses per dam) obtained from the 5 dams of group A (SBV-inoculation at day 28 of gestation), we found 3 foetuses from 2 different dams (2/2 and 1/3 foetuses, respectively) showing clear abnormalities with respect to size and morphology. In addition, we detected slight morphological alterations (haemorrhagic, glossy, and swollen aspect of foetuses) in 2 foetuses (2/2 foetuses) from an additional dam of group A. Similar alterations were observed for 2 foetuses (2/3 foetuses) from 1 of the 5 dams of group B (SBV-inoculation at day 42 of gestation). All the other of the 10 foetuses (1 x 1, 3 x 2, and 1 x 3 foetuses per dam) obtained from the 5 dams of group B showed a normal size and morphology. Further in-depth analyses, including histopathological evaluations and quantitative RT-PCR to assess the viral load in numerous maternal/fetal tissue samples, are underway and will be presented.

West Nile virus in a migrating Eurasian hobby in Liguria, Italy

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West Nile virus (WNV), a flavivirus and member of the Japanese encephalitis virus antigenic complex, has been recognized as a cause of human infection since 1934. Birds are the primary amplifying hosts, and the virus is maintained in an avian-mosquito cycle. In May 2013, an eurasian hobby, died in the rehabilitation centre for wildlife of Sanremo, Liguria region, Italy. It resulted infected with a lineage 1 strain of the West Nile virus. Samples of hearth, liver, kidneys and lung were stored in formalin and embedded in paraffin for immunohistochemistry (IHC) analysis. Tissue of the central nervous system were not processed as for the occurence of autolytic phenomena. Immunoreactivity was detected in the cardiac muscle fibers. However, host inflammatory response was not detected. So far, infectious virus has not been isolated on cell culture. However, whole genome sequencing of the involved WNV strain was attempted by next generation sequencing (NGS) using the Ion Torrent platform from the infected pool of organs of the dead hobby. The aliquots organs were placed together into one 2.0-ml cry tube and immediately frozen in liquid nitrogen. Mortar and pestle, previously sterilized, were used to pulverize about 0.1 mg of sample in liquid nitrogen; the sample was crushed until a fine powder was obtained. Total RNA was extracted from the fine powder following by TRIzol and used for library preparation. RNA was transcribed in presence of specific WNV lineage 1 reverse primers. Single strand cDNA was purified and double strand cDNA was prepared by using the second strand cDNA synthesis kit. ds cDNA was employed for enzymatic fragmentation and adapter ligation using the Ion Plus Fragment Library Kit. So far, only partial sequence of the genome of the involved strain has been recovered. As partially suggested by targeted sequencing of the NS5 gene, this strain clusters in the kenyan/western mediterranean clade, which comprises the majority of the western european WNV lineage 1 strains. Attempts to cover the whole genome of the strain are currently ongoing. Overall, this study highlights the importance of passive surveillance for WNV in wildlife. This case clearly shows the re-introduction of WNV lineage 1 in the Italian territory. Indeed, during 2013, only WNV of the lineage 2 has been detected or isolated from infected wild birds. Considering that WNV lineage 1, that infected and killed the hobby, has not been detected formerly and in the aftermath of this single episode in the sourrounding areas, the introduction of a novel WNV does not imply as a rule the establishment of an enzootic cycle as previously demonstrated.

First determination of Lumpy skin disease (LSD) in Turkey

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Lumpy skin disease (LSD)is a pox infection of cattle characterized by fever, nodules onthe skin, mucous membrane and internal organs. LSD is caused by a double stranded DNA virus in the genus Capripoxvirus of the family Poxviridae, which is antigenically closely related to sheep and goat pox viruses.

LSD was first described in 1929 in Zambia from where the disease spread to other southern African countries by the 1940s. To date, Lumpy Skin Disease spread all Africa continent and only four countries (Libya, Algeria, Morocco and Tunisia) declareted to be free from LSD. In 1989, LSD spread out Africa continent that Isreal reported first case. Nowadays, has more recently caused outbreaks in Jordan, Lebanon, Paletsine and Iraq. Turkey has not reported LSD before september 2013.

The World Organization for Animal Health (OIE) categorizes LSD as a notifiable disease because of the substantial economic impact of an outbreak. LSD can cause a temporary reduction in milk production, temporary or permanent sterility in bulls, damage to hides and death due to secondary bacterial infections. Transmission is predominantly via arthropod vectors including hard ticks, biting flies and/or mosquitoes. Direct transmission is also possible through saliva, semen, milk, or contact directly with lesions of infected cattle, however the direct course is less efficient in the absence of insect vectors.

The aim of this study was to reveal first determination of Lumpy skin disease virus in cattle. The first LSD clinical suspected cattle were seen in Kahraman Maras and Batman Provinces on September 2013. After that 8 sample including, skin lesion, blood sample and nazal discharge were sent to LSD Reference Laboratory, Pendik Veterinary Control Institute, ın order to identification of the source virus that suspected LSD. Collected sample were found as positive LSD in 4 skin lesions, 3 blood sample and 1 nazal discharge sample by PCR test, according to OIE Manual (2010). The sequence result also confirmed that the circulating virus is LSD virus (The data not presented this poster).

In conclusion, the first entry of LSD into our Country was 2013. LSD outbreaks and epidemics were mainly associated with climate changes. These changes caused serious outbreaks and recently outbreaks highly affected in countries (Isreal, Egypt) that have been existing of disease. For this reason, implementation of control and prevention measure, vaccination and screening of LSD cases on field are very important to fight against LSD. Additionally, was concluded as main reason of entry of the disease in Turkey, uncontrolled animal movement and global climate change.

Migratory birds health status surveillance in Piedmont region (Northwestern Italy)

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Migratory birds can act as vectors in the geographic spread of zoonotic infections and are considered the natural reservoir of pathogens which cause extensive damage to poultry and potential threat for public health. In order to early detect the introduction of pathogens by migratory routes, in 2013 an active surveillance plan on migrating and wintering birds was enforced in Piedmont region (Northwestern Italy).

The most important wetlands of the region were selected, located in peri-urban and rural areas, in nature reserves and paddy fields; during the birds ringing sessions, sampling of cloacal and tracheal swabs were carried out on live birds belonging to long- and short-distance species. If re-captured, birds were sampled again in order to track changes in their health status. Also carcasses found in the same areas were subjected to investigations. Specimens where mainly collected during spring and fall migration.

On live animals, the investigated pathogens were Orthomyxovirus, Paramyxovirus, Flavivirus and chlamydial agents, while on found dead ones post-mortem examination was made to evaluate the cause of death.

The screening was performed with a real-time RT-PCR for the detection of the *M* gene of type A Influenza and Newcastle Disease viruses, and further a real-time PCR for the *23S* rRNA gene of the *Chlamidiaceae* family. In order to detect arboviruses, an end-point PCR protocol for the *NS5* gene, characteristic of *Flavivirus* genus, was applied. Avian influenza positive samples, were further characterized using *H5, H7, H9* real-time One Step RT-PCR assays. A real-time PCR for the *ompA* gene of *Chlamydia psittaci* and sequencing of the *NS5* fragment were performed for the characterization of the remaining etiological agents. Confirmatory tests were carried out by National Reference Centres. From all birds found dead the isolation of *Campylobacter* and *Salmonella spp*. was attempted.

From March 2013 to March 2014, 455 long- and short-distance migratory birds were sampled, coming from twelve different areas of the region. Among them, one great tit resulted positive for *C. psittaci,* four mallards for avian influenza (not H5, H7, H9), one mallard for the genotype 6 of type 1 Avian Paramyxovirus and seven mallards for an atipical chlamydial agent. The sampling design comprised areas rapresentative of the wetlands distribution in Piedmont region.

Although it is impossible to exclude the introduction of such infectious agents from other sites, the results collected during this study have provided a valuable and rapresentative description of the health status of the migratory avifauna hosted in Piedmont.

Surveillance of wild birds infections in Liguria, Italy

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The Liguria region in Northwestern Italy, facing the Ligurian sea, represents a risk area for the introduction of zoonotic infections such as Avian Influenza, Newcastle disease (ND), West Nile disease and avian chlamydiosis, being located along flyways used by migrating waterfowl to cross the Western Palearctic. During spring and autumn migration, birds stop to feed and rest by Italian coasts, before undertaking the flight to Northern Europe or Africa according the flyways. Herein, we present the results of the passive surveillance on wild birds found dead, ill or injured in Liguria region, in collaboration with the Veterinary authority of the Imperia province, from January 2011 onwards.
During the study a total of 188 wild birds, belonging to fifteen avian Orders, were collected and underwent autoptsy examination. Tissue samples (CNS, lung, kidney, heart, liver, spleen and intestine) were tested for Influenza type A and Newcastle disease virus by real-time RT-PCR assays targeting the matrix genes and for avian chlamydiosis by real-time PCR a 16S rRNA real-time PCR assay. In order to monitor arboviral infections eventually harboured by wild birds, all specimens were checked also with an end point PCR protocol specific for a tract of the NS5 gene of the *Flavivirus* Genus.
Positivities for a not yet classified species of *Chlamydiaceae* have been found in two yellow-legged gulls and one flamingo, for *C. psittaci* in a turtle dove and another yellow-legged gull was found infected with H13N2 strain of Influenza A virus.
Low pathogenic avian Influenza viruses are seasonally common in many Charadriformes species, while, to our knowledge, these are the first findings of the new chlamydial agent in yellow-legged gull.
In May 2013, an Eurasian hobby, dead at the rehabilitation centre of Sanremo and showing chachexy and discrasic edemas at the autopsy examination, resulted infected at the kidney and CNS level with a Lineage 1 strain of the West Nile virus. By the phylogeographic analyses, this strain clustered in the Kenyan/western mediterranean clade, which comprises the majority of western European sequences. In particular the sequence is located in the subclade A', together with other West Nile sequences from Italy and Morocco. This evidence may suggest a re-introduction of the virus in the Italian territory from Northern Africa, considering the migratory habits of the Eurasian hobby, which is used to stop along the Italian west coasts for feeding, during its northward migration from Africa in spring.

Emergence of Encephalomyocarditis virus in a French Zoo

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Encephalomyocarditis virus (EMCV) belongs to the Cardiovirus genus within the Picornaviridae family. This positive-sens single stranded RNA virus has been recognized worldwide as a pathogen infecting a wide range of domestic and wild animal species. It can induce myocarditis, reproductive failures, diabetes or nervous disorders. Sudden death is often the first indication of infection and is associated to acute fatal myocarditis. Rodents are considered to be the natural hosts and thought to be the primary reservoir and disseminators of the virus. Several cases of fatal EMCV infections involving a variety of exotic mammals have been described worldwide in zoos. EMCV infection was also implicated in the deaths of elephants in Florida in 1976 and in South Africa in 1993. Here, we report the first case of African elephant death due to EMCV infection in a French zoo.

In November 2013, one month after her transfer from a German to a French zoo, a female African elephant was found dead without any previous clinical signs. Histological diagnostic highlighted lesions which could be correlated with EMCV infection.

Real-time RT-PCR analysis targeting EMCV 3D protein coding sequence was performed on RNA extracted from myocardium, kidney and liver and was found positive. These results were confirmed by amplification and sequencing of other regions within the EMCV genome (L, VP1, 2A, 3D). In parallel, EMC virus was isolated on cell culture (BHK-21) from the samples and confirmed by real-time RT-PCR carried out on RNA extracted from infected cells. Virus neutralization test performed on the elephant sera was however negative.

In order to elucidate the history of EMCV infection, viral neutralization tests were performed on sera collected between 2004 and 2014 from individuals in French and German zoo (n=13). Antibodies against EMCV were only detected in 2 sera sampled from the same elephant: in France in June 2013 and in Germany in December 2013. Investigations of the origin of this EMCV infection were extended in the French zoo on rodents. Among 23 rodents sampled in the French zoo, 9 were found positive by real-time RT-PCR, and EMCV was isolated on cell culture from 3 of them. In addition, high level of antibodies against EMCV was detected in 1 rodent serum (1/3 analyzed). Those primary results suggest that EMCV infection started in the French zoo and that rodents are probably the source of this elephant infection. To confirm this hypothesis it will be interesting to compare genomic sequences obtained from isolated virus from elephants and rodents.

These results show for the first time the circulation of EMCV in a French zoo and isolation of the virus. They confirm the susceptibility of African elephant to EMCV and the risk of this virus for zoo animals. Detection of infected rat stress a need for prevention by rodent control to avoid spread of the disease.

Detection of Rabbit Haemorrhagic Disease Virus 2-like Variant in Great Britain.

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Rabbit haemorrhagic disease virus (RHDV) a *lagovirus*, family *Caliciviridae* causes an acute, fulminating and generally fatal disease in the European rabbit (*Oryctolagus cuniculus*). RHDV was first identified in China in 1984. In 1986 it was observed in Italy, after which it became widespread across Europe with transmission being linked to movement of rabbit products as well as live animals. Following its emergence, RHDV greatly effected rabbit populations and was responsible for significant economic losses for the rabbit industry. Subsequently, the availability of efficacious commercial vaccines that afforded complete protection against circulating RHDV strains enabled its control.

In summer 2010 a RHDV variant, deciphered on basis of the major capsid protein (VP60) sequence and designated RHDV-2, was detected in France, following which it spread across mainland Europe. Since August 2013 we have received an increased number of samples submitted for the investigation of rabbit haemorrhagic disease virus (RHDV). Analysis of a portion of the VP60 and subsequent sequencing and phylogenetics revealed incursion of a RHDV-2 like variant. The RHDV-2 like variant was noted in both wild and domestic populations. Retrospective VP60 PCR and sequencing analysis on samples dating back to 2006 which were positive for RHDV via electron microscopy or haemagglutination assay, showed incursion of the RHDV-2 like variant since 2010. Prior to 2013 there had been limited interest with regards to RHDV in Great Britain; the occurrence in domestic populations may have prompted the increased submissions in 2013. These data highlight a greater need for vigilance particularly as there is concern regarding efficacy of existing RHDV vaccines against RHDV-2.

Discovery of a New Member of the Zoonotic *Pteropine Orthoreovirus* Species Isolated from Fruit Bats Imported in Italy from Indonesia

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Bats are reservoir of zoonotic viruses. In particular, fruit bats or flying foxes which belong to the genus *Pteropus* and are widely spread in Australia, Indonesia and Malaysia, may act as natural reservoir of Hendra and Nipah viruses. Analogously, from flying foxes the Nelson Bay, a fusogenic Orthoreovirus, was first isolated in Australia in 1968. The first evidence of the zoonotic potential of the Orthoreoviruses of bats was in 2007 when a man suffered from fever and respiratory symptoms after coming in contact with a flying fox. The responsible virus was called Melaka virus. Aftermath, other viral isolates such as Kampar, Miyazaki-Bali/2007 genetically and antigenically related to the prototype Nelson Bay virus have been isolated from patients with respiratory symptoms. All viruses have been shown to have modest human-to-human transmission. Together with Pulau virus, isolated from a flying fox in 2007, they form the recently proposed *Pteropine Orthorevirus* species. In the present study, the entire genome of the strain Indonesia/2010 isolated from fecal and salivary samples of flying foxes imported from Indonesia, has been accomplished by NGS. The samples were received at the laboratories of the IZSAM in order to exclude the presence of Hendra and Nipah viruses. Indonesia/2010 was successfully propagated *in vitro* on a wide variety of cells, including insect cells. Genome constellation was shown to be composed by ten segments of dsRNA. Indonesia/2010 is genetically related (90-95 % nt similarity) in each segment with the other viruses of the *Pteropine Orthoreovirus* species, thus it is potentially zoonotic. This study highlights the importance of flying foxes as vectors of potentially zoonotic viruses and the biological hazard that lies in the import of animals from geographical areas that are ecologically diverse from Europe.

Conjunctivitis Due to Laboratory Infection of Chlamydophila Abortus

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Introduction

Conjunctivitis is a well-recognized disease caused by *Chlamydia trachomatis* in humans. A few cases of conjunctivitis due to other chlamydiae infection, such as *Chlamydophila psittaci* and *Chlamydophila felis*, have also been reported, suggesting that non-trachomatis conjunctivitis may be frequent [1, 2]. Here we report a laboratory acquired case of a researcher with conjunctivitis during the periods of his engagement in research on *Chlamydophila abortus*. From the conjunctival scrapings, a *Chlamydophila abortus* was isolated.

Materials and Methods

Conjunctival scrapings were obtained from a 31-year-old male laboratory researcher who got conjunctivitis when engaged in research on *Cp. abortus*. As has been alerted to the experience of contact with *Cp. abortus*, he received a topical treatment with doxycycline. He recovered after a week course of treatment.

Chlamydiae isolation attempts were performed using blind passages on monolayer cell cultures.Initial chlamydial culture was subjected to antibody and chemical staining. DAPI was used to visualize DNA. The mouse anti-MOMP monoclonal antibodies (Santa Cruz) were used to visualize chlamydial organisms. *Cp. abortus* species-specific PCR-based RFLP analysis was performed as described previously [3].

Results

Cultures from the eye swabs revealed chlamydial inclusions when stained with the *Chlamydophila abortus* MOMP-specific antibody but no inclusions were stained with the C. trachomatis-specific anti-MOMP antibody (Fig. 1A). The isolate was identified by analysis of the helicase genes clone 8 and *omp*2 PCR-RFLP patterns, which are indistinguishable from that of the corresponding *Chlamydophila abortus* strain SX5 kept in our laboratory (Fig. 1B).

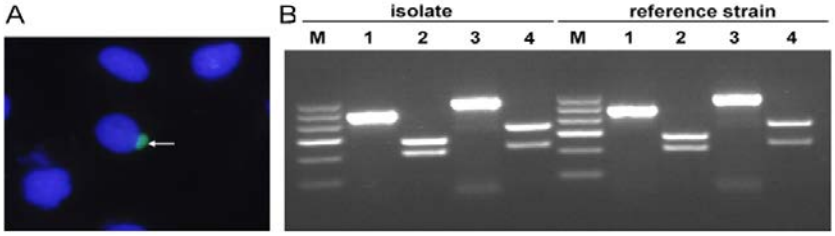

Figure 1. Identification of chlamydial isolate from eye scrapings. A. Determination of the chlamydial cultures by immunofluoresence. B. RFLP patterns of the chlamydial isolate. Lane M: 100 bp WM marker; Lane 1: helicase genes clone 8 PCR products; Lane 2: *Alu* I digestion of clone 8 PCR products; Lane 3: *omp*2 PCR products; Lane 4: *Alu* I digestion *omp*2 products.

Discussion

Conjunctivitis caused by non-trachomatis infection may be missed unless other chlamydial species are specifically suspected. The reported cases of conjunctivitis due to *Chlamydophila psittaci* and *Chlamydophila felis* make us aware of the possibility of *Chlamydophila abortus* as a cause of the human eye disease. In this case, a non-trachomatis chlamydia was cultured from the eye of the patient and identified as *Chlamydophila abortus*, suggesting that this conjunctivitis case is probably acquired from laboratory infection. *Chlamydophila abortus*, a zoonotic agent, infection in livestock is common throughout the world; however, epidemiological or pathological data of human infection is not available, which may limit the immediate access to proper medical treatment when infected. Further research in this filed needs to be emphasized.

References

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