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ORIGINAL CONTRIBUTION

Bacterial Infections in Humans and Nonhuman Primates from Africa: Expanding the Knowledge

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The close phylogenetic relationship between humans and other primates creates exceptionally high potential for pathogen exchange. The surveillance of pathogens in primates plays an important role in anticipating possible outbreaks. In this study, we conducted a molecular investigation of pathogenic bacteria in feces from African nonhuman primates (NHPs). We also investigated the pathogens shared by the human population and gorillas living in the same territory in the Republic of Congo. In total, 93% of NHPs (n=176) and 95% (n=38) of humans were found to carry at least one bacterium. Non-*pallidum Treponema* spp. (including *T. succinifaciens*, *T. berlinense*, and several potential new species) were recovered from stools of 70% of great apes, 88% of monkeys, and 79% of humans. Non-*tuberculosis Mycobacterium* spp. were also common in almost all NHP species as well as in humans. In addition, *Acinetobacter* spp., members of the primate gut microbiota, were mainly prevalent in human and gorilla. Pathogenic *Leptospira* spp. were highly present in humans (82%) and gorillas (66%) stool samples in Congo, but were absent in the other NHPs, therefore suggesting a possible gorillas-humans exchange. Particular attention will be necessary for enteropathogenic bacteria detected in humans such as *Helicobacter pylori*, *Salmonella* spp. (including *S. typhi/paratyphi*), *Staphyloccocus aureus*, and *Tropheryma whipplei*, some of which were also present in gorillas in the same territory (*S. aureus* and *T. whipplei*). This study enhances our knowledge of pathogenic bacteria that threaten African NHPs and humans by using a non-invasive sampling technique. Contact between humans and NHPs results in an exchange of pathogens. Ongoing surveillance, prevention, and treatment strategies alone will limit the spread of these infectious agents.

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Abbreviations: NHPs, Nonhuman primates; PCR, Polymerase chain reaction.

Keywords: Nonhuman primates, humans, infectious diseases, bacteria, Africa, surveillance, emergence

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INTRODUCTION

More than 60% of known infectious diseases have a zoonotic origin, the majority being caused by pathogens of wild origin [1]. The close phylogenetic relationship between humans and other primates creates high potential for pathogen exchange [2]. As a result, diseases emerge in humans as an unintended consequence of the hunting and butchering of the African great apes and other contacts. They were responsible for human Ebola outbreaks and the global AIDS pandemic [3,4], as well as the high mortality observed in wild chimpanzee populations (*Pan troglodytes*) associated with the anthropozoonotic transmission of human respiratory viruses [5].

In the tropics, in many rural areas, human population growth and changes in land use are leading to a growing overlap between humans and wild primates [6]. This is mainly the result of large-scale activities, such as extractive industries (ie, logging, mining); as well as small-scale interfaces, like subsistence use of natural resources, ecotourism, and research. These changes increase contact between people and non-human primates (NHPs), and they result in more intimate contact with wild primates [7-9]. By contrast, the increasing fragmentation of habitats is forcing primates to seek resources more widely, including through the active use of human-dominated systems (eg, crop raiding in agricultural fields and urban occupation) [10,11]. All these scenarios are likely to increase the risk of transmission of zoonoses [12,13]. Some NHPs that persist in anthropogenically modified landscapes, such as monkeys, are susceptible to many of the same pathogens as humans. As a result, these resilient species have the capacity to act as sentinels for ecosystem health and provide early warning of potential risks to human health [1].

Several bacteria have been reported to be transmitted by direct as well as indirect contacts from NHPs to humans [9]. Further, it had been demonstrated that proximity between wild primates and people can promote transmission of the common gastrointestinal bacterium *Escherichia coli*, as well as other pathogenic microorganisms, such as *Cryptosporidium* [7] and *Shigella*. Other studies stressed that direct contact between species is not mandatory for interspecific disease transmission (ie, *Shigella*, *Salmonella*, *E. coli*, etc.) [9]. Demonstration of human pathogens negatively impacting wild primates has sparked considerable debate regarding the costs and benefits to endangered primate populations of scientific research, ecotourism, and current conservation and management paradigms. Despite the disease-related risks, the consensus is that both research and tourism have contributed in overwhelmingly positive ways to primate conservation, enhancing their long-term survival by increasing their scientific and economic value. Nevertheless, such activities as well as overlap of humans and NHPs may have unintended consequences for the health and survival of wild primate populations.

The central hypothesis of this work is that key NHPs could carry pathogen agents for both humans and animals. In addition, human behaviors, wildlife behaviors, ecological conditions and landscape features increase the risks of interspecific disease transmission.

Thus, given the deadly epidemics and pandemics that have already occurred related to NHPs (HIV, malaria, Ebola, etc.), it is of utmost importance to study microorganisms common to both NHPs and humans. It is difficult to carry out such studies because, in most cases, we cannot capture them and collect the necessary samples. Using stool samples, it is possible to find not only enteric pathogens, but also blood and urinary pathogens (like filaria, Plasmodia, and *Leptospira*). Its collection is absolutely non-invasive. Here, we performed an extensive epidemiological survey for bacteria on NHP feces, using polymerase chain reaction (PCR) systems known for their specificities and sensitivities. Moreover, in a One Health context in Congo, we studied a human population sharing the same living area as gorillas in order to study the transmission of pathogens between species.

MATERIAL AND METHODS

Ethical Statement, Animals, and Study Area

In Senegal, in August 2016, 48 western chimpanzees (*Pan troglodytes verus*) feces were collected. The study was approved by the Senegalese Ministry of the Environment (Direction of the National Parks, No. 1302, 16 October 2015). The Direction des Eaux, Forêts, Chasses et Conservation des Sols of the Republic of Senegal gave authorization to collect and export fecal samples (No. 1914/DEF/DGF of 5 June 2016) in Collaboration with Jane Goodall Foundation (https://www.janegoodall.org/). Between 2017 and 2019, in the Republic of Congo, 38 gorilla (*Gorilla gorilla*) and 38 human feces were collected as part of a collaborative project carried out by the Government of the Republic of Congo and the Aspinall Foundation, which manages a protected area of 170,000 ha located about 140 km north of Brazzaville. In addition, the project was authorized by the Ministry of Health (No 208/MSP/CAB.15 of 20 August 2015) and the Forest Economy and Sustainable Development (No 94/MEFDD/CAB/DGACFAP-DTS of 24 August 2015) of the Republic of Congo. In this country, wildlife protected areas are not fenced and there is close interaction between apes and the communities. In some instances, they even share open-well water sources and/or gardens. This can lead to microorganism transmission between humans and NHPs. In the Republic of Djibouti, six fe-

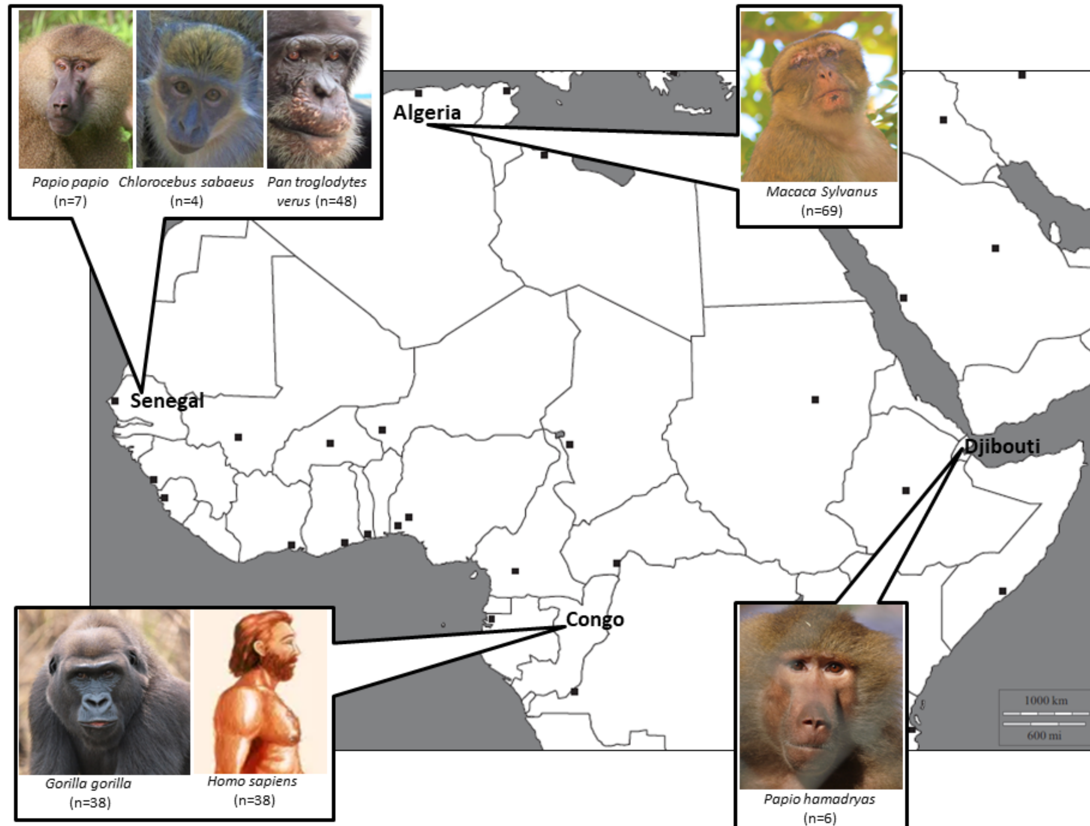

Figure 1. Map indicating the countries where fecal samples were collected. **In Senegal:** 48 fecal samples of western chimpanzees (*Pan troglodytes verus*) were collected from three sites located within the Dindefelo Community Natural Reserve in the Kédougou region. Site 1: three samples (12°22′57.1404″ N, 12°17′16.7172″ W), Site 2: seven samples (12°22′53.1732″N, 12°17′26.7936″W), and Site 3: 38 samples (12°22′47.7084″ N, 12°17′48.588″ W). Four green monkey (*Chlorocebus sabaeus*) and seven Guinea baboon (*Papio papio*) fecal samples were collected near the Niokolo forestry guardhouse of the NKNP (13°04′28.6″ N 12°43′18.2″ W). **In the Republic of Congo:** 28 gorilla (*Gorilla gorilla*) feces were collected from the Lésio-Louna (LLR) and South-West Léfini gorilla reserves (2°58′33.1″S 15°28′33.4″E), 10 from Odzala-Kokoua National Park (OKNP) (1.3206°"N, 14.8455°"E), 35 human feces from (1.3206°"N, 14.8455°"E) village of Mbomo, located within the OKNP, and three from eco-guards in the LLR. **In Djibouti:** six hamadryas (*Papio hamadryas*) feces. These baboons lived outside the village of Oueah, 38 km from the city of Djibouti (11°29′56.1″N 42°51′14.8″E). **In Algeria:** 69 Barbary macaques (*Macaca sylvanus*), including 30 samples collected from two sites, the Stream of Monkeys and the Gorges of la Chiffa in Blida Province, 50 km north of Algiers (36°23′42.9″N 2°45′53.6″E), and 39 samples from Cap Carbon (36°46′31.6″ N 5°06′11.2″ E) in the suburbs of Béjaïa, 250 km east of Algiers.

cal samples of hamadryas baboons (*Papio hamadryas*) were collected in 2017. This collection was carried out in partnership with the Center for Studies and Research of Djibouti. Finally, in Algeria, fecal samples were collected from 69 Barbary macaques (*Macaca sylvanus*), with the authorization of the management of the Chréa National Park (CNP). These primates were synanthropic and lived in close contact with the people who provided them with food (Figure 1).

The sampling was non-invasive and did not disturb any wild animal. For NHPs, feces were collected at sleeping sites, feeding sites, and places where the primates had been observed. Human stool samples were collected after obtaining the verbal consent of all the participants because of their low level of literacy.

All the humans in our study were apparently healthy. In addition, the fact that the NHP stools were not diarrheal may indicate that they were also in relatively good health. All collected samples were transported to the IHU Méditérranée Infection Laboratory, 13005 Marseille, France, for analysis. They had been identified and stored at either -20°C or -80°C.

DNA Extraction

Initially, 40 mg of stool were mixed with 360 µL of G2 lysis buffer from EZ1®DNA Tissue Kit (Qiagen,

Hiden, Germany). This was mechanically lysed with tungsten beads (Qiagen, Hiden, Germany) using Fast-Prep-24TM 5G Grinder for 40 sec. After 10 min of incubation at 100°C to allow for complete lysis, tubes were centrifuged at 10,000g for 1 min. Subsequently, 200µL of supernatant was enzymatically digested using 20µL of proteinase K (20mg/mL, Qiagen) and incubated overnight at 56°C. DNA was extracted from 200µL of sample using the EZ1®DNA Tissue Kit on BIOROBOT EZ1 (Qiagen, Hiden, Germany), according to the manufacturer's instructions. Elution was performed in 200µL volume, then aliquoted in individual tubes of pure extracted DNA, dilutions to 1:10 and to 1:100.

The extraction quality and the absence of PCR inhibitors were controlled using the universal eubacterial qPCR targeting the 16S rRNA bacterial genes [14] on pure DNA, dilutions to 1:10 and to 1:100. By comparison of the Ct values obtained, the dilution to 1:10 was chosen for the analysis. DNA tubes were stored at -20°C until use.

Molecular Screening for Bacteria by Real-time PCR Assays (qPCR)

The approach consists of screening for bacteria that may be pathogenic for humans and NHPs. We used single target real-time genus or species-specific qPCR assays. We targeted pathogens of medical interest, previously reported in NHPs. At least 12 qPCR assays targeting bacterial genera and 16 species-specific qPCRs, all known for their specificity and sensitivity, were used in multiparallel assays, as shown in Table S1 (Appendix A). For *Rickettsia* spp., a new qPCR was designed and validated targeting the 16S rRNA gene. qPCR was tested for its specificity using several laboratory-maintained colonies as well as DNA from arthropods, humans, monkeys, donkeys, horses, cattle, mice, and dogs as described previously [15].

Assays were carried out in 20 µl final volume containing 10µl of Master Mix Roche (Eurogentec), 0.5µl each primer per reaction at the concentration of 20µM, 0.5 µl UDG, 0.5µl of each probe at the concentration of 5µM and 5µl of the DNA template. The qPCR amplifications were performed in a CFX96 Real-Time system (Biorad Laboratories, Foster City, CA, USA). The thermal conditions included two hold steps at 50°C for 2 minutes, followed by 95°C for 15 minutes and 40 cycles of two steps each (95°C for 30 sec and 60°C for 30 sec). Each PCR plate contains 96 wells. Known microorganisms' DNAs or plasmids were used as positive controls and master mixtures as a negative control in each reaction.

Genetic Amplification by Standard PCR, Sequencing, and Phylogeny

In addition to species-specific qPCR screening, we continued identification by PCR/sequencing for *Treponema* spp., *Acinetobacter* spp., *Rickettsia* spp., *Mycobacterium* spp., and *Leptospira* spp. Positive samples in qPCR assays were subjected to standard PCRs targeting genes and using primer pairs summarized in Table S1 (Appendix A).

Genetic amplifications were carried out in 50µl volume consisting of 5µl of DNA template, 25µl of AmpliTaq Gold master mix, 18µl of ultra-purified water DNAse-RNAse free and 1µl of primers at 20µM of concentration. This was performed in a thermocycler (Applied Biosystem, Paris, France). Protocols of amplification were as follows: incubation step for 15 minutes at 95°C, 40 cycles (one minute at 95°C, 30 sec at the annealing temperature, an elongation step at 72°C), and a final extension step for 5 minutes at 72°C. Amplicons were visualized on 2% agarose gel and were then purified using NucleoFast 96 PCR plates (Macherey Nagel EURL, Hoerdt, France) as per the manufacturer's instructions. Amplicons were sequenced using the Big Dye Terminator Cycle Sequencing Kit (Perkin Elmer Applied Biosystems, Foster City, CA, USA) with an ABI automated sequencer (Applied Biosystems). Generated electropherograms were assembled and edited using ChromasPro software (ChromasPro 1.7, Technelysium Pty Ltd., Tewantin, Australia) and compared with those available in the GenBank database by NCBI BLAST (https://blast.ncbi.nlm.nih.gov/Blast.cgi). The fragments obtained were compared with each other and with the related fragments available in the GenBank database. The phylogenetic analyses were inferred using neighbor joining methods and tree reconstructions were performed using MEGA software version 7 (https://www.megasoftware.net/). Bootstrap analyses were conducted using 1000 replicates.

RESULTS

In this study, we were able to collect NHP stool samples from different African countries, including 59 samples from Senegal, 6 from Djibouti, and 69 from Algeria. In addition, in Republic of Congo, we collected 38 samples from humans and 38 from gorillas sharing the same territory. In total, 93% (160/172) of NHPs tested positive for at least one (min: 1; max: 6) of the bacterial species tested in their feces while 94.7% (36/38) of humans were positive for at least one (min: 1; max: 7) bacterium. Prevalence in NHPs and humans are summarized in Figure 2; Table 1.

Positive rates for all of *Treponema* (non-*pallidum*) (79.1%), *Mycobacterium* (non-*tuberculosis*) (38.4%), pathogenic *Leptospira* spp. (26.7%), and *Acinetobacter* spp. (9.9%) were high in NHPs as well as in humans, 78.9%, 31.6%, 81.6%, and 73.7% in humans respectively. Bacteria such as *Rickettsia* (4.7%), *Mycoplasma*

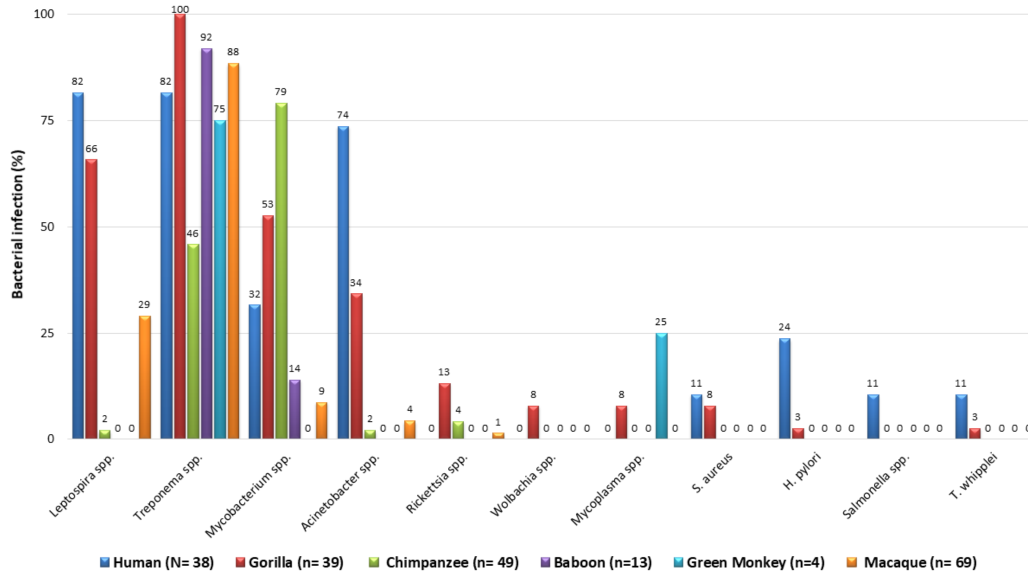

Figure 2. Infections detected in African NHPs and humans.

(2.3%), and *Wolbachia* spp. (1.7%) were found only in NHPs. By contrast, *H. pylori* (23.7%), *S. aureus* (10.5%), and *T. whipplei* (10.5%) were more prevalent in humans than in NHPs, for which the prevalence rates were 1.7%, 0.6%, 0.6% respectively. *Salmonella* spp. was detected only in humans, 10.5% (4/38), including two samples (5.2%) positive for *S. typhi/paratyphi.*

No significant differences were observed between great apes (n=86) and monkeys (n=86) for pathogens (great apes %, monkeys %; Z test p-value) such as *Leptospira* spp. (30.2%, 23.3%; 0.388), *Rickettsia* spp. (8.1%, 1.2%; 0.066), *Wolbachia* spp. (3.5%, 0.0%; 0.240), *Mycoplasma* spp. (3.5%, 1.2%; 0.612), *S. aureus* (3.5%, 0.0%; 0.240), *H. pylori* (1.2%, 0.0%; 1.000), *T. whipplei* (1.2%, 0.0%; 1.000). By contrast, significant differences were observed for *Treponema* spp. (69.8%, 88.4%; 0.004), *Mycobacterium* spp. (67.4%, 9.3%; <0.0001) and *Acinetobacter* spp. (16.3%, 3.5%; 0.009). In the Republic of the Congo, there were no differences in bacteria carriage between gorillas (n=38) and humans (n=38) who cohabited the same area as gorillas (gorillas%, humans%; p-value) for *Leptospira* spp. (65.8%, 81.6%; 0.186), *Mycobacterium* spp. (52.6%, 31.6%; 0.096), *Rickettsia* spp. (13.2%, 0.0%; 0.055), *Wolbachia* spp. and *Mycoplasma* spp. (7.9%, 0.0%; 0.229), *S. aureus* (7.9%, 10.5; 1.000), *Salmonella* spp. (0.0%, 10.5%; 1.113), and *T. whipplei* (2.6%, 10.5%; 0.349). Significant differences were observed between gorillas and humans for *Treponema* spp. (100.0%, 78.9%; 0.005), *Acinetobacter* spp. (34.2%, 73.7%; 0.010), *H. pylori* (2.6%, 23.7%; 0.012).

All the other bacteria, also listed in the Table S1 (Appendix A), that were searched for using the PCR method, were ultimately not found.

Furthermore, we performed PCR/sequencing for positive samples in qPCR for all of *Mycobacterium* spp., *Leptospira* spp. (pathogenic), *Acinetobacter* spp., *Treponema* spp., and *Rickettsia* spp. Sequencing did not succeed for *Mycobacterium* and *Leptospira* spp. despite obtaining specific bands using standard PCR, and superposed peaks were obtained for the same nucleotide which made it impossible to conclude about sequences. Two good quality sequences of 700 bp length of *gltA* gene of *Rickettsia* were obtained, one from an Algerian macaque and the other from a gorilla from Republic of the Congo. The sequences were identical in each other and were 99% identical to *R. africae* isolate QtHyaegR9 (MN306555) detected from a spur-thighed tortoise (*Testudo graeca*) sold in a Qatar live animal market, and isolate Egy-RickHm-Raas detected on an adult male *Hyalomma marginatum* (camel tick) in Egypt (KX819298) (Figure 3).

We obtained 24 sequences of 322-365 bp length for *rpoB* gene of *Acinetobacter* spp. including three sequences obtained from macaques, five others obtained from gorillas, and 16 humans. One sequence obtained from gorilla feces (G9) showed 98% identity with *A. baumannii* strain MS14413 (CP054302). Two human genotypes (Mbo033, Mbo064, Ibou02) and one gorilla genotype (G06B) were almost identical and they highlighted 95%-99.5% of identity to *A. berezinae* strain YMC79 (JF302886). Two other human isolates (Mbo047 and Mbo057) were closely similar and exhibited 92% and 93% similarity with *A. berezinae.* They constitute a potential new species. Three

Table 1. Prevalence of bacterial infections detected on feces from different African NHP species as well as humans.

Species	Origin	n=	*Leptospira* spp.		*Treponema* spp.		*Mycobacterium* spp.		*Acinetobacter* spp.		*Rickettsia* spp.		*Wolbachia* spp.*		*Mycoplasma* spp.		*S. aureus*		*H. pylori*		*Salmonella* spp.		*T. whipplei*	
			Pos	**%**	**Pos**	**%**	**Pos**	**%**	**Pos**	**%**	**Pos**	**%**	**Pos**	**%**	**Pos**	**%**	**Pos**	**%**	**Pos**	**%**	**Pos**	**%**	**Pos**	**%**
Gorilla	Congo	38	25	65,8	38	100,0	20	52,6	13	34,2	5	13,2	3	7,9	3	7,9	3	7,9	1	2,6	0	0	1	2,6
Chim-panzee	Senegal	48	1	2,1	22	45,8	38	79,2	1	2,1	2	4,2	0	0,0	0	0,0	0	0,0	0	0,0	0	0	0	0,0
Guinea baboon		7	0	0,0	6	85,7	2	28,6	0	0,0	0	0,0	0	0,0	0	0,0	0	0,0	0	0,0	0	0	0	0,0
Hama-dryas baboon	Djibouti	6	0	0,0	6	100,0	0	0,0	0	0,0	0	0,0	0	0,0	0	0,0	0	0,0	0	0,0	0	0	0	0,0
Green Monkey	Senegal	4	0	0,0	3	75,0	0	0,0	0	0,0	0	0,0	0	0,0	1	25,0	0	0,0	0	0,0	0	0	0	0,0
Macaque	Algeria	69	20	29,0	61	88,4	6	8,7	3	4,3	1	1,4	0	0,0	0	0,0	0	0,0	0	0,0	0	0	0	0,0
Great Apes	-	86	26	30,2	60	69,8	58	67,4	14	16,3	7	8,1	3	3,5	3	3,5	3	3,5	1	1,2	0	0	1	1,2
Monkeys	-	86	20	23,3	76	88,4	8	9,3	3	3,5	1	1,2	0	0,0	1	1,2	0	0,0	0	0,0	0	0	0	0,0
Total NHP	-	172	46	26,7	136	79,1	66	38,4	17	9,9	8	4,7	3	1,7	4	2,3	3	1,7	1	0,6	0	0	1	0,6
Human	Congo	38	31	81,6	30	78,9	12	31,6	28	73,7	0	0,0	0	0,0	0	0,0	4	10,5	9	23,7	**4**	**10,5**	4	10,5

In bolt: Number include 2 samples positive by *S. typhi/paratyphi* qPCR. *: where also positive by *Anaplasmataceae* qPCR

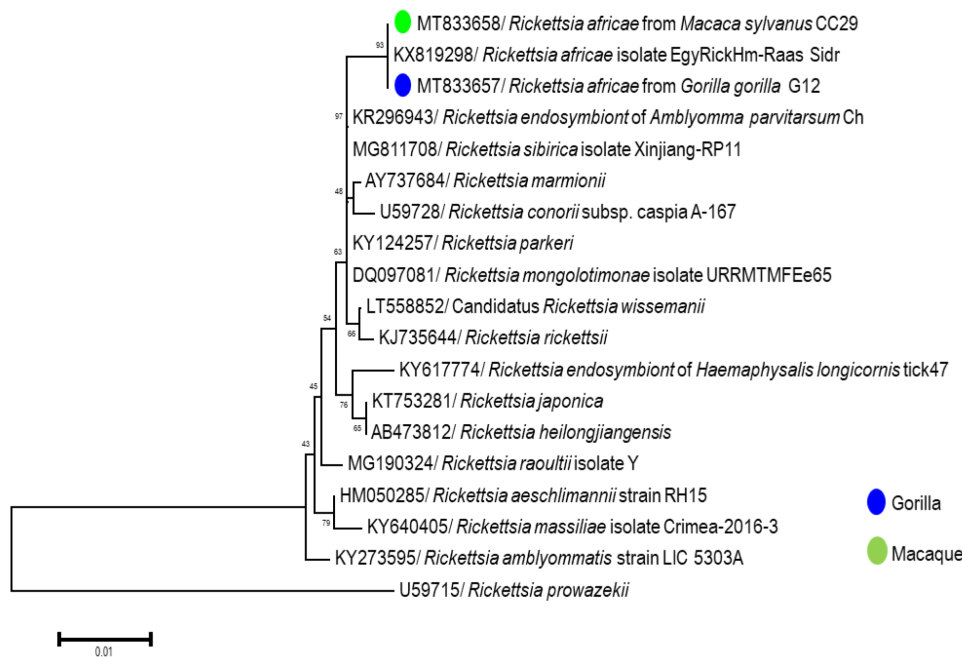

Figure 3. Phylogenetic tree for *Rickettsia* spp. in detected in African NHPs. The evolutionary history, based on 700 bp *gltA* partial gene, was inferred using the Neighbor-Joining method. The optimal tree with the sum of branch length = 0.10688518 is shown. The confidence probability (multiplied by 100) that the interior branch length is greater than 0, as estimated using the bootstrap test (1000 replicates is shown next to the branches). The tree is drawn to scale, with branch lengths in the same units as those of the evolutionary distances used to infer the phylogenetic tree. The evolutionary distances were computed using the Tamura-Nei method and are in the units of the number of base substitutions per site. The differences in the composition bias among sequences were considered in evolutionary comparisons. The analysis involved 19 nucleotide sequences. All positions containing gaps and missing data were discarded. There were a total of 682 positions in the final dataset. Evolutionary analyses were conducted in MEGA7.

other sequences obtained from humans constitute potential new species. One of them (Mbo056) showed 88% of similarity with *A. berezinae,* while the two others had 86% (Mbo058) and 93% (Mbo054) of identity with *A. genomosp.* (EU477133 and KT997528). Sequence Mbo003 from a human constituted a separate branch (Figure 4) and showed 92% similarity with *A. tandoii* strain LUH 13385 (KU961639). In addition, two identical isolates (Mbo040, Mbo054) from human stools created a separate branch and showed 91% identity to *A. bohemicus* strain ANC4315 (KJ124827). Another sequence (Mbo028) showed quasi-identity (98.5%) with *Acinetobacter sp.* strain WCHAc060041 (MH190065) found in wastewater in China. It also showed 90% similarity with the official *A. defluvii* strain WCHA30 (KY435935). Mbo036 and Ibou001, two almost similar genotypes from humans, were close (93% of identity) to the species *A. venetianus*. Two others, one (Mbo062) showed 91% similarity to *A. nosocomialis* (KX444511), the other one (Mbo001) was 93.5% identical to *A. seifertii* (KJ956464). Three sequences from gorilla feces and one other from a macaque constituted a separate clade (G05, G05A, G06, and CC37). They were almost similar and were 92-93% identical to *A. wuhouensis* strain WCHAc060049 (MK518338). Finally, two sequences from macaques (GC05 and CC10) close to *Acinetobacter* sp. 'isolate 30Bi' (FJ157977) were detected in a dog (Figure 4; Table S2 (Appendix A).

We succeeded in obtaining at least 52 sequences of 760-870 bp of *Treponema* spp. 23S gene on NHP feces including six from chimpanzees (accession numbers: MT257111-MT257117), 13 sequences from gorillas (MT257084, MT257085, MT257088-MT257098), one from a green monkey (MT257101), five from Guinea baboons (MT257102-MT257106), four from hamadryas (MT257107-MT257110), and 23 others from Barbary macaque samples (MT257228-MT257250) (Figure 5). In addition, four good quality sequences were obtained from feces of humans from the Mbomo locality (MT257099, MT257100, MT257086, MT257087) (Figure 6). None of the identified *Treponema* species have been reported as clearly pathogenic. A specific DNA search for *T. pallidum* spp. gave negative results.

In human samples, we obtained two sequences (MT257099, MT257086) that were perfectly identical to

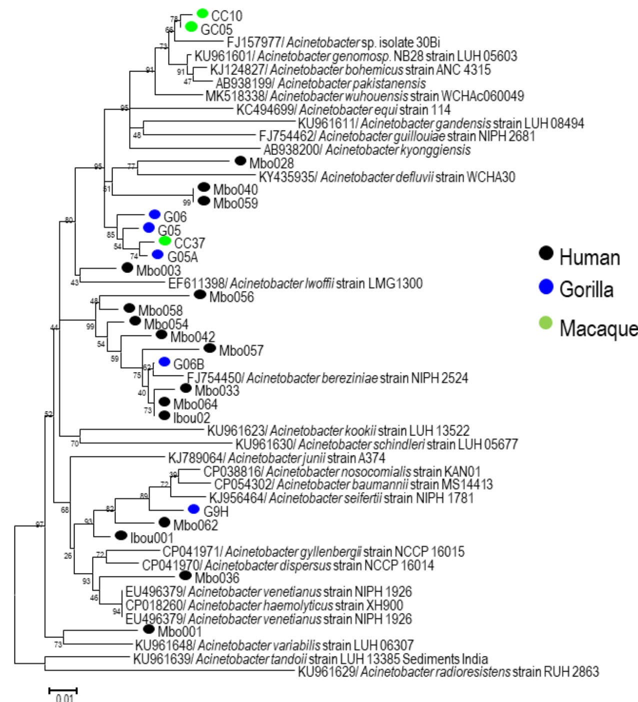

Figure 4. Phylogenetic tree for *Acinetobacter* spp. detected in African human and NHP feces. The evolutionary history based on *rpoB* partial gene was inferred using the Neighbor-Joining method. The optimal tree with the sum of branch length = 1.17635033 is shown. The confidence probability (multiplied by 100) of the interior branch length is greater than 0, as estimated using the bootstrap test (1000 replicates) is shown next to the branches. The tree is drawn to scale, with branch lengths in the same units as those of the evolutionary distances used to infer the phylogenetic tree. The evolutionary distances were computed using the Tamura-Nei method and are in the units of the number of base substitutions per site. The differences in the composition bias among sequences were considered in evolutionary comparisons. The analysis involved 50 nucleotide sequences. All positions containing gaps and missing data were discarded. There were a total of 260 positions in the final dataset. Evolutionary analyses were conducted in MEGA7.

T. succinifaciens DSM 2489 (CP002631). The two others seem to be new species, one of them (MT257100) was 83% identical to *T. succinifaciens* DSM 2489, while the second (MT257087) displayed 87% similarity with *T. pedis* str. T A4 (CP004120). Three gorilla genotypes, four genotypes from hamadryas, three others from baboons, one from a green monkey and one from a Barbary macaque were almost similar and close to *T. succinifaciens* official strains to which they highlighted >99% of similarity. Three other similar gorilla genotypes constituted a separate cluster, they had 83.5% of resemblance with *T. pectinovorum* strain Marseille-1-CSURP6641 (UOUI01000011) and 82.5% with *T. brennaborense* strain DD5/3 (NR076878). One other gorilla (MT257091) sequence split off separately, it was 81.5% identical to *T. brennaborense*. Three sequences from gorilla feces, two from baboons, and 16 from macaques were almost similar and exhibited most similarity 97-99.5% to *T. berlinense* strain ATCC BAA-909 (FUXC01000026) recovered from Swine feces in Berlin. A distinct isolate from gorilla (MT257084) shared 86% of similarity with *T. berlinense.* Four chimpanzee sequences constituted a new species and showed a 93.5% identity with *T. berlinense* strain ATCC BAA-909. Two other chimpanzee sequences (MT257115, MT257116) were almost similar to each other and showed 88-89% similarity to *T. berlinense* strain ATCC BAA-909, for which another gorilla isolate (MT257093) showed 84.5% identity and one other isolate showed 95% of similarity (MT257095).

DISCUSSION

This study identified pathogenic bacteria in human and NHP fecal samples from Africa. The technique used, qPCR/sequencing in feces, presented no ethical requirements. PCR was used in the diagnosis of gastrointestinal infections [16,17]. Using large specificity qPCR, we detected pathogenic bacteria, such as *Leptospira* spp. and nonpathogenic bacteria, such as *Treponema* spp., including several potential new species.

We found many *Treponema* spp. nonpathogenic despite the existence of the syphilis in NHPs. Most of

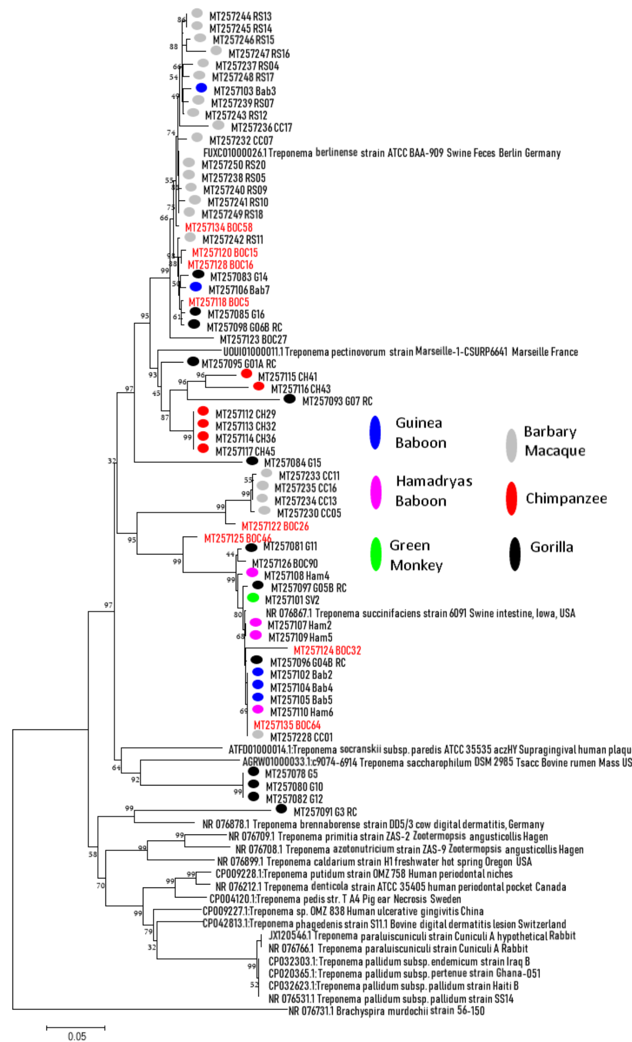

Figure 5. Phylogenetic tree for *Treponema* spp. detected in NHP feces. The evolutionary history based on 23S rRNA gene was inferred using the Neighbor-Joining method. The optimal tree with the sum of branch length = 2,23995197 is shown. The confidence probability (multiplied by 100) of the interior branch length is greater than 0, as estimated using the bootstrap test (1000 replicates) is shown next to the branches. The tree is drawn to scale, with branch lengths in the same units as those of the evolutionary distances used to infer the phylogenetic tree. The evolutionary distances were computed using the Tamura-Nei method and are in the units of the number of base substitutions per site. The differences in the composition bias among sequences were considered in evolutionary comparisons. The analysis involved 81 nucleotide sequences. All positions containing gaps and missing data were discarded. There were a total of 594 positions in the final dataset. Evolutionary analyses were conducted in MEGA7.

the *Treponema* spp. detected (non-pathogenic) such as *T. succinifasciens* are part of the normal flora of primates, and all species in this study shed treponemes in their feces. The presence of Spirochaetes has been reported in the gut microbiota of NHPs [18]. High prevalence rates were observed in all primate species of *Treponema* spp. (not *pallidum*), *T. succinifasciens*, *T. berlinense*, and at least six potential new species. *T. succinifasciens* and *T. berlinense* were highly prevalent species in the microbial genomes from NHPs gut metagenome [19]. It had been reported that rural individuals were enriched with Spirochaetes, especially *Treponema succinifasciens* and *T. berlinense* being the most prevalent species identified. This may be due to the rare use of antibiotics [20]. Special attention should be drawn to endemic *Treponema pallidum* infection with genital stigmata in NHPs from Guinea, Senegal, and Tanzania. Many NHPs in Africa were found to suffer from treponematoses [21-24]. During fieldwork in Senegal, an epizootic of venereal disease was directly observed in green monkeys *(Chlorocebus sabaeus)* due

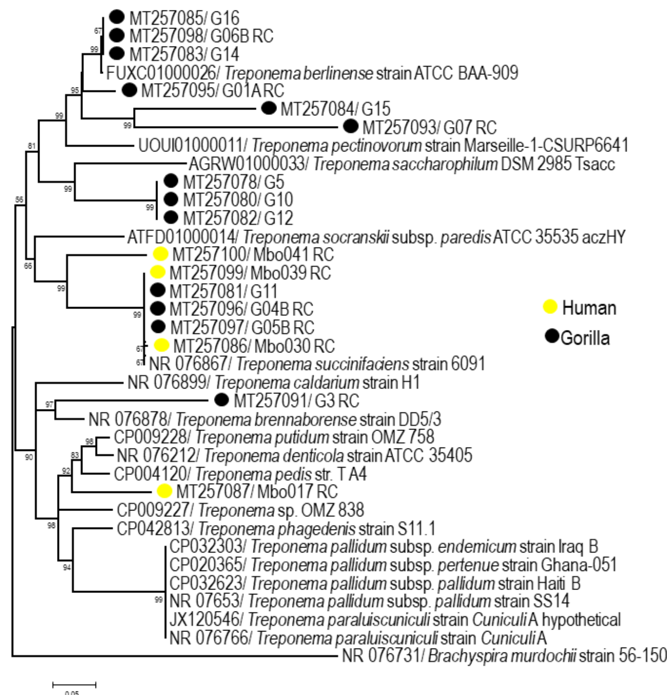

Figure 6. Phylogenetic tree of *Treponema* spp. detected in gorilla and human feces living in the same area in Mbomo, Republic of Congo. The evolutionary history based on 23S rRNA partial gene was inferred using the Neighbor-Joining method. The optimal tree with the sum of branch length = 2,51751187 is shown. The confidence probability (multiplied by 100) that the interior branch length is greater than 0, as estimated using the bootstrap test (1000 replicates) is shown next to the branches. The tree is drawn to scale, with branch lengths in the same units as those of the evolutionary distances used to infer the phylogenetic tree. The evolutionary distances were computed using the Tamura-Nei method and are in the units of the number of base substitutions per site. The differences in the composition bias among sequences were considered in evolutionary comparisons. The analysis involved 36 nucleotide sequences. All positions containing gaps and missing data were eliminated. There were a total of 361 positions in the final dataset. Evolutionary analyses were conducted in MEGA7.

to infection with *T. pallidum* subsp. *pertenue,* then an epizootic was observed in Senegal which spread to baboons one year later [25].

We agree with the conclusion drawn by Manara et al. 2019 [19], the overlap (20% of microbial candidate species in NHPs also found in the human microbiome) occurs mainly between NHPs and non-Westernized human populations and NHPs living in captivity, suggesting that host lifestyle plays a significant role comparable to host speciation in shaping the primate intestinal microbiome. Several NHP-specific species are phylogenetically related to human-associated microbes, such as *Treponema*, and could be the consequence of host-dependent evolutionary trajectories. Gut *Treponema* have been found in NHPs and all rural peoples studied to date, suggesting that they are lost symbionts in urban-industrialized societies [26].

Potentially new species of *Acinetobacter* were detected in humans, gorillas, and macaques. In humans, *Acinetobacter* is an organism of questionable pathogenicity as an infectious agent of importance to hospitals worldwide—it easily infects wounds [27]. The organism has the ability to accumulate diverse mechanisms of resistance, leading to the emergence of strains that are resistant to all commercially available antibiotics [28]. *Acinetobacter* is one of the main genera detected in primate gut microbiota. They might play a significant role in breaking down plant exudates [29]. Very high diversity was observed in humans, gorillas, and macaques.

Tuberculosis is rare in wild NHPs, but animals carrying *M. tuberculosis* could infect humans. Conversely, humans are the source of most NHP infections. *M. tuberculosis* and *M. bovis* can be acquired from infected humans or ruminants [30]. In this study, *M. tuberculosis* has not been detected and great apes were found to be more carriers of *Mycobacterium* spp. than monkeys and humans. Natural infections with *M. leprae* was reported in chimpanzees and sooty mangabeys (*Cercocebusatys*) [31]. Recently, different strains of *M. leprae* have been isolated from NHPs, including chimpanzees, sooty mangabeys, and cynomolgus macaques [32]. *Mycobacterium orygis* was isolated from captured rhesus monkeys [33]. It will be necessary to isolate the mycobacteria detected

in this study to investigate their species diversity and identify their roles.

In addition, high prevalence for pathogenic *Leptospira* spp. has been observed in both humans and gorillas, which remains difficult to understand. All humans are apparently healthy and gorilla stools do not reflect any sign of diseases. Unfortunately, we were not able to genotype these *Leptospira* and the sequences obtained were unclear and suggested possible co-infection by more than one species. NHPs could be sensitive to *Leptospira* infection, an outbreak of severe leptospirosis was reported in capuchin (*Cebus*) monkeys [34]. The presence of *Leptospira* in the feces of wild NHPs could be due to environmental contamination because the samples were collected from the soil, which is not the case in humans, leading to the weakness of this hypothesis. Inappropriate breeding of NHPs could create new reservoirs and transmission routes for *Leptospira*, threatening conservation efforts and public health. Furthermore, the extent of *Leptospira* transmission between humans and NHPs remains unknown.

Surprisingly, *Rickettsia* and *Wolbachia* had been detected in great apes and monkeys in the present study. *Wolbachia* is assumed to be *Wolbachia* eaten together with their insect hosts by monkeys. For *Rickettsia*, it is *R. africae*, a pathogenic species and widespread in the continent, but very common in ticks. It is either *R. africae* eaten together with their tick hosts or a natural infection of NHPs (very unlikely).

Enteric bacteria, such as *Salmonella* spp., including *S. typhi/paratyphi,* were detected in humans only, and pathogenic *H. pylori*, *T. whipplei*, and *S. aureus* were detected in both humans and gorillas sharing the same living area, and humans were found to be more infected. *H. pylori* and *T. whipplei* are *a priori* anthroponotics and their origin remains unknown. These results suggest a transmission of pathogens from humans to gorillas. The threat is a direct function of the pathogens' mode of transmission and their ability to survive in aerosols, soil, water, food, or feces [9]. In Uganda, it was observed that the number of gorillas carrying human gut *Salmonella* or *Campylobacter* had doubled in 4 years, and *Shigella* was isolated for the first time in this group of apes, probably because of ecotourism [35]. In addition, the fecal-oral transmission of *Shigella flexneri* and *S. sonnei,* enteropathogenic *Escherichia coli, Salmonella enteritidis, S. typhimurium, Campylobacter fetus, C. jejuni, Helicobacter pylori,* and many other infections are common in NHPs [36]. For *T. whipplei,* two important but as yet unresolved issues are the natural habitat and the route of infection. Analysis of stool samples by PCR has detected *T. whipplei* DNA in patients with Whipple disease. Recovery of *T. whipplei* from culture of stool from a patient with Whipple disease has highlighted the presence of viable bacteria, suggesting that the disease could be linked to fecal-oral transmission [37]. Asymptomatic carriage in stool was found in humans (ranging from a prevalence of 4% in the control group, to 12% among a subgroup of sewer workers), but not in monkeys and apes [37]. Thus, *T. whipplei* identified here in the gorilla most likely originates from humans.

Also, the samples were negative for the other bacteria, including the sexually transmitted pathogens in the current study (*Chlamydia* spp., *T. pallidum*, *N. gonorrhea*). Obtaining samples from different origins would also broaden the range of pathogens not found in stools and provide a clearer diagnostic vision in NHPs. It would therefore be interesting, in further studies, to use other types of excrement and biological fluids from these animals, especially for those in contact with humans, in order to assess the potential risks of interspecies transmission.

The prevalence of extra-intestinal infections might be underestimated in this study. Despite this, this method (search for pathogens in the feces) nevertheless made it possible to discover the origins of *Plasmodium* and HIV.

The study presents certain limits that were required by the difficulties encountered in the field and the study does not represent all African countries. The sample size of humans in Congo and some NHP species (such as hamadryas) are not important. This required larger investigations in the other regions with larger sample size and other NHP species. Unfortunately, we did not characterize all the detected microorganisms, such *Leptospira, Treponema,* and *Mycobacterium* spp., nor isolated them. If it was performed, this could give more information on their transmission and pathogenesis. Feces are not the ideal sampling method to search for pathogens, such as blood or sexually transmitted pathogens.

This work has contributed to the expansion of knowledge on NHP threatening bacteria in Africa by using a molecular stool technique. In addition, we have also shown that humans and gorillas in the same ecosystems share pathogens, indicating a real interspecies transmission. Constant monitoring is highly recommended to prevent any overflow of these pathogens. This effort entails a combination of epidemiology, molecular ecology, behavioral ecology, social and clinical survey, and spatially explicit modeling. The end outcomes are achievable plans to protect the health of humans and wildlife, while ensuring the sustainability of the ecosystems in which they live. In addition to better understanding the role of human-induced habitat change on pathogen dynamics, this work allows for the early detection of emerging pathogens that may pose a threat to global health and/or wildlife conservation.

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Appendix A: Table S1. Oligonucleotide sequences of primers and probe used for qPCRs and conventional PCRs in this study

Bacteria	Target gene	Primer name	Sequence (5'- 3')	Source
Acinetobacter spp.	*rpoB*	Acineto-rpoBF	TACTCATATACCGAAAAGAAACGG	Bouvresse et al. 2011
		Acineto-rpoBR	GGYTTACCAAGRCTATACTCAAC	
		AcinetorpoB	6-FAM-CGCGAAGATATCGGTCTSCAAGC	
Acinetobacter baumannii	*OmpA/mtB*	Forward	TCAACATCACAATCTTTAGTAGCTGA	Ly TDA et al. 2019
		Reward	CGCTCTTGCCAGCATAAAGA	
		Probe	6FAM-AAGTCGCCAAGAAACCTTGA	
Acinetobacter spp.*	*rpoB* (Zone 1)	F	TAYCGYAAAGAYTTGAAAGAAG	La Scola B et al. 2006
		R	CMACACCYTTGTTMCCRTGA	
Anaplasmataceae spp.	*23S*	TtAna_F	TGACAGCGTACCTTTTGCAT	Dahmani et al. 2015
		TtAna_R	GTAACAGGTTCGGTCCTCCA	
		TtAna_P	6FAM- GGATTAGACCCGAAACCAAG	
Wolbachia spp.	*16S*	all.Wol.16S.301-F	TGGAACTGAGATACGGTCCAG	Laidoudi et al. 2020
		all.Wol.16S.347-P	6FAM-AATATTGGACAATGGGCGAA	
		all.Wol.16S.478-R	GCACGGAGTTAGCCAGGACT	
Bartonella spp.	*ITS*	Barto_ITS3_F	GATGCCGGGGAAGGTTTTC	Mourembou et al. 2015
		Barto_ITS3_R	GCCTGGGAGGACTTGAACCT	
		Barto_ITS3_P	6FAM- GCGCGCGCTTGATAAGCGTG	
Borrelia spp.	*16S*	Bor16S3F	AGC CTT TAA AGC TTC GCT TGT AG	Parola et al. 2011
		Bor16S3R	GCC TCC CGT AGG AGT CTG G	
		Bor16S3P	6FAM-CCG GCC TGA GAG GGT GAA CGG	
Coxiella burnetii	*IS1111A*	CB_IS1111_0706F	CAAGAAACGTATCGCTGTGGC	Mediannikov et al. 2010
		CB_IS1111_0706R	CACAGAGCCACCGTATGAATC	
		CB_IS1111_0706P	6FAM- CCGAGTTCGAAACAATGAGGGCTG	
Helicobacter pylori	*23S rRNA*	HPY_F	AGGTTAAGAGGATGCGTCAGTC	Ménard et al. 2002
		HPY_R	CGCATGATATTCCCATTAGCAGT	
		HPY_P	6FAM-TGGGAGCTGTCTCAACCAGAGATTCAGTG	

Leptospira spp. (*L.interrogans, L.borgpetersenii, L.kirscheri, L.santarosai, L.fainei, L.weilii, L. inadai, L. noguchi, L. alexenderi, L. genomospecies-1/3, L. interrogans icterohaemorragia*)	*16S*	Lepto 171F	CCCGCGTCCGATTAG	Smythe et al. 2002
		Lepto 258R	TCCATTGTGGCCGRA/GACAC	
		Lepto P	6-FAM- CTCACCAAGGCGACGATCGGTAGC	
Leptospira spp.*	*adk*	F	GGGCTGGAAAAGGTACACAA	Ahmed N et al. 2006
		R	ACGCAAGCTCCTTTTGAATC	
	icdA	F	GGGACGAGATGACCAGGAT	
		R	TTTTTTGAGATCCGCAGCTTT	
	LipL41	F	TAGGAAATTGCGCAGCTACA	
		R	GCATCGAGAGGAATTAACATCA	
	RrS2	F	CATGCAAGTCAAGCGGAGTA	
		R	AGTTGAGCCCGCAGTTTTC	
	LipL32	F	ATCTCCGTTGCACTCTTTGC	
		R	ACCATCATCATCATCGTCCA	
Rickettsia spp.	*16S*	Rick-16S-F	TAATGGCCTACCAAGCCAAC	This study
		Rick-16S-R	GTCACCCACTCGGTATTGCT	
		Rick-16S-P	6FAM- ATATTGGACAATGGGCGAAA	
Rickettsia felis	*bioB*	Rfelis_0527_F	ATGTTCGGGCTTCCGGTATG	Mourembou et al. 2015
		Rfelis_0527_R	CCGATTCAGCAGGTTCTTCAA	
		Rfelis_0527_P	6FAM- GCTGCGGCGGTATTTTAGGAATGGG	
Rickettsia spp.*	*GltA*- PCR1	CS2D	ATGACCAATGAAAATAATAAT	Mediannikov et al. 2004
		CSEndR	CTTATACTCTCTATGTACA	
	GltA- PCR2 (Nested)	409D	CCTATGGCTATTATGCTTGC	
		1258R	ATTGCAAAAAGTACAGTGAACA	
Salmonella spp.	*invA*	invA_F	TCTGTTTACCGGGCATACCA	Mourembou et al. 2015
		invA_R	CACCGTGGTCCAGTTTATCG	
		invA_P	6FAM- CCAGAGAAAATCGGGCCGCG	

	sipC	sipC_F	GTCAGGCGTCGTAAAAGCTG	Mourembou et al. 2015
		sipC_R	ACGTCGACTGGTGGTACTGG	
		sipC P	6FAM- CTCCAGGCGCGAACAGCTGG	
Salmonella typhi	*hypothetical protein*	Styphi_put_F	TCTCATGCTGCGACCTCAAA	
		Styphi_put_R	TTCATCCTGGTCCGGTGTCT	
		Styphi_put_P	6FAM- GCTTTTTGTGAAGCAACGCTGGCA	
Salmonella typhi/paratyphi	*narG*	Styphi_narG_F	GCGCCACATCTTCATCAGAC	
		Styphi_narG_R	CCCGTCCTGATATGCCAAAC	
		Styphi_narG_P	6FAM- AGTAACTTGCCCCGCGCGGG	
Staphylococcus aureus	*NucA*	Saur_NucA_F2	GTTGTGGATGGTGATACATTTATTGC	Mourembou et al. 2015
		Saur_NucA_R2	CCAAATGGTTGTACAGGCGTATTC	
		Saur_NucA_P2	6FAM- AGGCTTATAGGGGTTGATACGCCAGAAACGG	
	Amidohydrolase	Saur_Amido_F	CCTCGACAGGTAACGCATCA	
		Saur_Amido_R	AAACTCCTATCGGCCGCAAT	
		Saur_Amido_P	6FAM- TGCAATGGTAGGTCCTGTGCCCA	
Clostridium difficile	*TcdA*	Cdiff_tcdA_F	GGTAATAATTCAAAAGCGGCT	Luna RA et al. 2011
		Cdiff_tcdA_R	AGCATCCGTATTAGCAGGTG	
		Cdiff_tcdA_P	6FAM- AGCCTAATACAGCTATGGGTGCGAAG	
	TcdB	Cdiff_tcdB_F	GAAAGTCCAAGTTTACGCTCAAT	
		Cdiff_tcdB_R	GCTGCACCTAAACTTACACCA	
		Cdiff_tcdB_P	VIC- ACAGATGCAGCCAAAGTTGTTGAATT	
Mycobacterium spp.	*ITS*	Mycob_ITS_F	GGGTGGGGTGTGGTGTTTGA	Bruijnesteijn Van Coppenraet ES et al. 2004
		Mycob_ITS_R	CAAGGCATCCACCATGCGC	
		Mycob_ITS_P	6FAM- TGGATAGTGGTTGCGAGCATC	
M. tuberculosis	*ITS*	Mtub_ITS_P	6FAM- GCTAGCCGGCAGCGTATCCAT	
Mycobacterium spp.	*rpoB*	MycoF	GGCAAGGTCACCCCGAAGGG	Adékambi T et al. 2003
		MycoR	AGCGGCTGCTGGGTGATCATC	
Mycoplasma spp.*	*ITS1*	Mycop_ITS_F	GGGAGCTGGTAATACCCAAAGT	Bittar F et al. 2015
		Mycop_ITS_R	CCATCCCCACGTTCTCGTAG	
		Mycop_ITS_P	6FAM-GCCTAAGGTAGGACTGGTGACTGGGG	

Mycoplasma hominis	*16S*	Mhom_16S_F	GCTGTTATAAGGGAAGAACATTTGC	Bittar F et al. 2015
		Mhom_16S_R	GGCACATAGTTAGCCATCGC	
		Mhom_16S_P	6FAM- AAATGATTGCAGACTGACGGTACCTTGTCAG	
Mycoplasma genitalium	*fusA*	Mgen_fusA_F	CTGGGAAAACCACCACATCA	Fenollar F et al. 2006
		Mgen_fusA_R	CACTGAAGTGGCTGCAGAGG	
		Mgen_fusA_P	6FAM- TGGTGAATCAGTGATGGACTGGATGGA	
Gardnerella vaginalis	*Cpn60*	Gvaginalis_F	CGCATCTGCTAAGGATGTTG	Fenollar F et al. 2006
		Gvaginalis_R	CAGCAATCTTTTCGCCAACT	
		Gvaginalis_P	VIC- TGCAACTATTTCTGCAGCAGATCCT	
Atopobium vaginae	*16S*	Atop_F	CCCTATCCGCTCCTGATACC	Fenollar F et al. 2006
		Atop_R	CCAAATATCTGCGCATTTCA	
		Atop_P	VIC- GCAGGCTTGAGTCTGGTAGGGGA	
Neisseria gonorrhoae	*Hypothetical protein*	Ngono_2F	CATCAGCGCAATCCCTATGA	Hopkins M et al. 2010
		Ngono_2R	TGGTTGTCCTCAACCGTGTC	
		Ngono_2 P	6FAM- CCACGGCGCTTCCTGATGGG	
	porA	Pap-Tm-F	CAGCATTCAATTTGTTCCGAGTC	
		Pap-Tm-R	GAACTGGTTTCATCTGATTACTTTCCA	
		Pap-Tm-P	6FAM- CGCCTATACGCCTGCTACTTTCACGC	
Treponema spp.	*23S*	forw2570	CGCCGTCACTTAACGGAYA	This study
		rev2660	GATGCGATGAGCCGACAT	
		sonde2620	6FAM- CCCCAAGAGTTCACATCGAC	
Treponema pallidum	*polA*	SyphT_F1	GTCGAGACTGAAAAGGAGTGCA	Edouard S et al. 2017
		SyphT_R1	GTGAGCGTCTCATCATTCCAAAG	
		SyphT_P1	6FAM- TGCTGTGCAGGATCCGGCATATGTCC	
	flaA	Tpal_flaA_F	GCGGTTGCACAGTGGGAG	Salazare JC et al. 2007
		Tpal_flaA_R	CAGCATGGGCGACAGGAT	
		Tpal_flaA_P	6FAM- TTGTGCTGAATTCTTCCGCGCG	
Treponema spp.*	*23S*	F1- 3274	GGGAGTGAGACTGCGIGCG	Medkour et al. submitted
		F2- 3592	GAGAATGCAGGYATAAGTA	
		R1- 3671	CCGRCTTACCCTGRGSAGAT	

		R2- 4163	GGTGTCASCMCCTATACGTCYCAT	
Tropheryma whipplei	*WiSP family protein (whi 2)*	T_whi2_F	TTGTGTATTTGGTATTAGATGAAACAG	Fenollar et al. 2010
		T_whi2_R	CCCTACAATATGAAACAGCCTTTG	
		T_whi2_P	6FAM- GGGATAGAGCAGGAGGTGTCTGTCTGG	
	WiSP family protein (whi 3)	T_whi3_F	TTGTGTATTTGGTATTAGATGAAACAG	
		T_whi3_R	CCCTACAATATGAAACAGCCTTTG	
		T_whi3_P	6FAM-GGGATAGAGCAGGAGGTGTCTGTCTGG	
Coxiella burnetii	*IS1111A*	CB_IS1111_0706F	CAAGAAACGTATCGCTGTGGC	Sokhna C et al. 2013
		CB_IS1111_0706R	CACAGAGCCACCGTATGAATC	
		CB_IS1111_0706P	6FAM-CCGAGTTCGAAACAATGAGGGCTG	
Vibrio cholerae	*toxR*	toxR-F1	CCGAATAACCACCCTGATCTTT	Greig et al., 2018
		toxR-R1	ACCTGTGGCAATGACTTCTATC	
		toxR-Pr	6FAM -ACTGGCTACCGTCAATCGAACTGT	

*Primers for standard PCR

Table references

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Table S2. Table summarizes blast results for *Acinetobacter* spp. detected on African humans, gorillas and macaques.

Sample ID	Species	Length	Description	Max Score	Total Score	Query Cover	E value	Per. Ident	Accession
CC10	*Macca sylvanus*	356 bp	Acinetobacter sp. 'isolate 30Bi' RNA polymerase subunit B (rpoB) gene, partial cds	599	599	100%	3,00E-167	96.91%	FJ157977.1
			Acinetobacter bohemicus strain ANC 4315 RNA polymerase subunit B (rpoB) gene, partial cds	540	540	99%	2,00E-149	94.07%	KJ124827.1
GC05		357 bp	Acinetobacter sp. 'isolate 30Bi' RNA polymerase subunit B (rpoB) gene, partial cds	582	582	100%	3,00E-162	96.08%	FJ157977.1
			Acinetobacter wuhouensis strain WCHAc060049 DNA-directed RNA polymerase subunit beta (rpoB) gene, partial cds	555	555	100%	7,00E-154	94.68%	MK518338.1
			Acinetobacter bohemicus strain ANC 4315 RNA polymerase subunit B (rpoB) gene, partial cds	551	551	99%	9,00E-153	94.65%	KJ124827.1
CC37		365 bp	Acinetobacter equi strain 114, complete genome	536	536	100%	3,00E-148	93.15%	CP012808.1
			Acinetobacter wuhouensis strain WCHAc060049 DNA-directed RNA polymerase subunit beta (rpoB) gene, partial cds	531	531	98%	1,00E-146	93.31%	MK518338.1
G05A	*Gorilla gorilla*	326 bp	Acinetobacter schindleri strain HZE30-1 chromosome, complete genome	449	449	96%	3,00E-122	92.65%	CP044483.1
			Acinetobacter wuhouensis strain WCHAc060049 DNA-directed RNA polymerase subunit beta (rpoB) gene, partial cds	464	464	99%	1,00E-126	92.59%	MK518338.1
G9H		354 bp	Acinetobacter baumannii strain MS14413 chromosome, complete genome	616	616	100%	3,00E-172	98.02%	CP054302.1
G05A		346 bp	Acinetobacter wuhouensis strain WCHAc060049 DNA-directed RNA polymerase subunit beta (rpoB) gene, partial cds	501	501	99%	9,00E-138	92.75%	MK518338.1
G06		346 bp	Acinetobacter wuhouensis strain WCHAc060049 DNA-directed RNA polymerase subunit beta (rpoB) gene, partial cds	529	529	100%	4,00E-146	94.24%	MK518338.1
G06B		347 bp	Acinetobacter bereziniae strain NIPH 2524 RNA polymerase subunit B (rpoB) gene, partial cds	614	614	100%	1,00E-171	98.56%	FJ754450.2
Mbo001	*Homo sapiens*	339 bp	Acinetobacter seifertii strain NIPH 1781 RNA polymerase subunit B (rpoB) gene, partial cds	510	510	100%	2,00E-140	93.53%	KJ956464.1
Mbo003		328 bp	Acinetobacter tandoii strain LUH 13385 RNA polymerase subunit B (rpoB) gene, partial cds	470	470	99%	2,00E-128	92.42%	KU961639.1
Mbo028		322 bp	Acinetobacter sp. strain WCHAc060041 DNA-directed RNA polymerase subunit beta (rpoB) gene, complete cds	568	568	100%	8,00E-158	98.45%	MH190065.1
			Acinetobacter defluvii strain WCHA30 chromosome, complete genome	412	412	100%	4,00E-111	89.75%	CP029397.2
Mbo033		348 bp	Acinetobacter berezinae strain YMC79 RNA polymerase subunit B (rpoB) gene, partial cds	630	630	99%	1,00E-176	99.42%	JF302886.1
Mbo036		348 bp	Acinetobacter venetianus strain NIPH 1926 RNA polymerase subunit B (rpoB) gene, partial cds	573	573	99%	2,00E-159	96.53%	EU496379.2

Mbo040	348 bp	Acinetobacter bohemicus strain ANC 4315 RNA polymerase subunit B (rpoB) gene, partial cds	472	472	100%	7,00E-129	91.09%	KJ124827.1
Mbo054	348 bp	Acinetobacter genomosp. 18 strain ANC 4109 RNA polymerase subunit B (rpoB) gene, partial cds	508	508	100%	6,00E-140	93.10%	KT997528.1
Mbo059	347 bp	Acinetobacter bohemicus strain ANC 4315 RNA polymerase subunit B (rpoB) gene, partial cds	459	459	100%	6,00E-125	90.49%	KJ124827.1
Mbo057	348 bp	Acinetobacter berezinae strain YMC79 RNA polymerase subunit B (rpoB) gene, partial cds	566	566	100%	3,00E-157	95.98%	JF302886.1
Mbo056	356 bp	Acinetobacter bereziniae strain RUH 2222 RNA polymerase beta subunit (rpoB) gene, partial cds	420	420	92%	3,00E-113	87.88%	EU445665.1
Mbo042	343 bp	Acinetobacter berezinae strain YMC79 RNA polymerase subunit B (rpoB) gene, partial cds	501	501	98%	9,00E-138	91.72%	JF302886.1
Mbo062	349 bp	Acinetobacter nosocomialis strain PHL41 RNA polymerase subunit beta (rpoB) gene, partial cds	496	496	99%	4,00E-136	91.19%	KX444511.1
Mbo058	354 bp	Acinetobacter genomosp. 15BJ strain NIPH 1866 RNA polymerase subunit B (rpoB) gene, partial cds	424	424	100%	2,00E-114	86.24%	EU477133.2
Mbo064	343 bp	Acinetobacter berezinae strain YMC79 RNA polymerase subunit B (rpoB) gene, partial cds	553	553	98%	3,00E-153	94.67%	JF302886.1
Ibou001	348 bp	Acinetobacter venetianus rpoB gene for DNA-directed RNA polymerase subunit beta, complete cds, strain: GTC 14626	540	540	99%	2,00E-149	94.80%	LC102698.1
Ibou002	348 bp	Acinetobacter berezinae strain YMC79 RNA polymerase subunit B (rpoB) gene, partial cds	632	632	100%	3,00E-177	99.43%	JF302886.1