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Sarcoptes scabiei mites in humans are distributed into three genetically distinct clades

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Abstract

Scabies is an ectoparasitic infestation caused by the mite *Sarcoptes scabiei.* Currently, *S. scabiei* is taxonomically divided into different varieties on the basis of host origin. Genetics-based research on scabies has been conducted, but the data on genetic diversity of populations of this mite in humans in Europe are lacking. We evaluated the genetic diversity of populations of *S. scabiei.* A large series of mites obtained from humans in France and the data of mites from various hosts and geographical areas retrieved from GenBank were included to investigate whether mites are divided into distinct populations. The study of *cytochrome c oxidase subunit 1* gene polymorphisms were found to be best suited for phylogenetic analysis. *S. scabiei* mites were distributed into three genetically distinct clades, with most mites clustering in clades B and C. The F_{st} value and the Nm value calculated for mites included in clades B and C indicated a strong population structure and a very low gene flow between mites of those clades. The results of the present study not only support the rejection of the hypothesis of panmixia for *S. scabiei* in humans but also suggest that mites belonging to different clades are genetically isolated. Moreover, the results suggest that the subdivision of *S. scabies* in varieties according to animal or human hosts is not warranted. In conclusion, *S. scabiei* mites in humans do not constitute a homogeneous population. Further investigations are now required to assess whether different clinical forms of scabies are associated with particular haplotypes or clades.

Keywords: *Cytochrome c oxidase subunit 1* gene, phylogenetic analysis, population genetic substructuring, *Sarcoptes scabiei*, scabies
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Introduction

Scabies, or mange as it is called in animals, is an ectoparasitic contagious infestation caused by the mite *Sarcoptes scabiei* [1–5]. This neglected and emerging/reemerging disease is a significant public health problem worldwide and is the most common skin disease in developing countries [6,7]. The

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worldwide prevalence in humans has been estimated to be >100 million cases in 2010 [8]. Sarcoptic mange is also an important veterinary disease, leading to significant morbidity and mortality in wild and domestic animals. It affects 104 species of mammalian hosts worldwide, including companion, livestock and wild animals, and it is an emerging problem in many countries [5,6,9]. The most commonly described clinical manifestations, among several possible different forms, are ordinary scabies (or classical scabies), nodular forms and crusted scabies, the latter being extremely contagious. Crusted scabies is usually associated with host factors such as older age and immunosuppressed conditions, but it is not known whether parasite factors may intervene [1].

The mite completes its entire life cycle on its host. Only female mites burrow into the skin. The generation time lasts about 3 weeks [3]. Most patients harbour a limited number of mites, usually 5 to 15 females on the whole body. In case of crusted scabies, the number can reach hundreds, thousands or even millions. Crusted scabies cases evolve during many months or years. Superinfection of ordinary or crusted scabies cases may in theory lead to infections with genetically distinct parasites, though this eventuality is probably rare [4].

For many years, host-associated populations of *S. scabiei* have been taxonomically divided into morphologically indistinguishable varieties [5,10,11]. The monospecificity of these host-specific varieties is still controversial, and current studies are investigating whether they belong to different species. Previous studies suggested that *Sarcoptes* is not a single panmictic population and that genetic subdivisions may occur according to the host and/or geographical location [12,13]. The historical proposed hypothesis of the "high degree of host specificity and low degree of cross-infectivity of *S. scabiei*" [10] has been challenged by some studies. Cross-infectivity was observed experimentally on some occasions [6,14]. Natural apparent cross-infectivity has been previously demonstrated during emerging epizootics in sympatric wild animal host populations [15–17]. Transmission of scabies mites between other species and humans can occur, but it usually results in a short and self-limiting infection [18–22].

Relatively little genetics-based research on *S. scabiei* has been conducted. This is primarily because of difficulties in obtaining suitable skin samples from hosts and in extracting adequate amounts of genetic material from individual mites [23]. In particular, data about the genetic diversity of *S. scabiei* in humans in Europe are lacking.

In this study, we evaluated the genetic diversity of populations of *S. scabiei,* including a large series of mites obtained in humans in France and the data of mites from various hosts and geographical areas retrieved from GenBank to investigate whether mites are divided into distinct populations.

Methods

Ethical clearance

This study was reviewed and approved by the Comité de Protection des Personnes (institutional review board) of the ethics committee CPP-Ile-de-France X (approval 2012/10/23), and informed consent was obtained from all patients.

Collection of *S. scabiei* mites

Mites were prospectively collected in patients consulting at Avicenne Hospital, Bobigny, and at Henri Mondor Hospital, Créteil, two cities located in the suburbs of Paris, between January 2013 and March 2014. Mites from dog and pig were kindly provided by the parasitology department of the Veterinary College of Alfort, Maisons-Alfort, France. All samples used in this study were independent; only one patient was included in the analysis when there were multiple cases within a family or at the scale of a larger localized epidemic.

DNA extraction and gene amplification

In total, genomic DNA from 62 mites (60 humans, one pig and one dog) was individually extracted with the NucleoSpin Tissue kit (NucleoSpin, Macherey-Nagel, Germany) [24,25]. A part of the *cox1* gene was amplified. PCR was carried out in 50 µL, and reaction mixture contained 1× PCR buffer, 2.5 mM $MgCl_2$, 1 mM of dNTPs, 1.25 U DNA polymerase AmpliTaq Gold (Applied Biosystems, Courtaboeuf, France) and 0.25 µM of primer (NavF: 5′-TGATTTTTTGGTCACCCAGAAG-3′; NavR: 5′-TACAGCTCCTATAGATAAAAC-3′) [26]. Amplifications conditions were as follows: an initial denaturation step at 94°C for 5 minutes, followed by 35 cycles of denaturing at 94°C for 30 seconds, annealing at 51°C for 30 seconds, and extending at 72°C for 40 seconds and 5 minutes of final extension at 72°C.

Sequence and phylogenetic analyses

The PCR-amplified products of 400 bp were purified and directly sequenced. The *Otodectes cynotis cox1* sequence (KF891933) was retrieved from GenBank and used as the outgroup to perform phylogenetic analysis. Multiple sequence alignments of nucleotide sequences in this study and sequences available from GenBank (n = 62) were generated using MAFFT 6.951. The data set was analysed with maximum likelihood (ML) and Bayesian inference (BI). ML were performed using MEGA5 and RAxML-HPC 7.0.4 in CIPRES portal under a General Time-Reversible (GTR + G) model. Support of internal branches was evaluated by nonparametric bootstrapping with 500 replicates. BI analysis was performed with MrBayes 3.2.1, conducting two simultaneous runs with four parallel Markov chains (one cold

TABLE 1. ***Sarcoptes scabiei cox1*** **haplotypes from isolates collected in suburb of Paris, France (*n* = 62), and sequences available in GenBank (*n* = 62)**

		nucleotide position																																						
	N° of haplotype	3	5	6	7	8	9	14	16	17	32	41	50	53	59	60	71	80	86	95	96	104	116	131	134	137	140	143	147	155	185	191	197	198	200	224	227	236	239	254
CLADE C	H1	G	G	A	T	A	A	T	A	T	A	A	A	T	T	T	T	G	T	T	T	T	T	T	T	A	A	T	G	A	C	C	A	G	A	T	A	A	A	C
	H2		A																			C											G							
	H3		A											C								C											G							
	H4																	A														T	G							
	H5		A																														G							
	H6		A																						C							T	G							
	H7																															T	G							
	H8																															T	G							
	H9						G				G																					T	G							
	H10																															T	G							
	H11								T																							T	G							
	H12																															T	G				T			
	H13																							A							T	T	G							
	H14																															T	G							
	H15																												A			T	G	A						
	H16																											C					G							
	H17																																G							
	H18																	A														T	G							
	H19										G							A		C	C											T	G							
	H20																																G							
	H21																																G							
	H22																	A														T	G							
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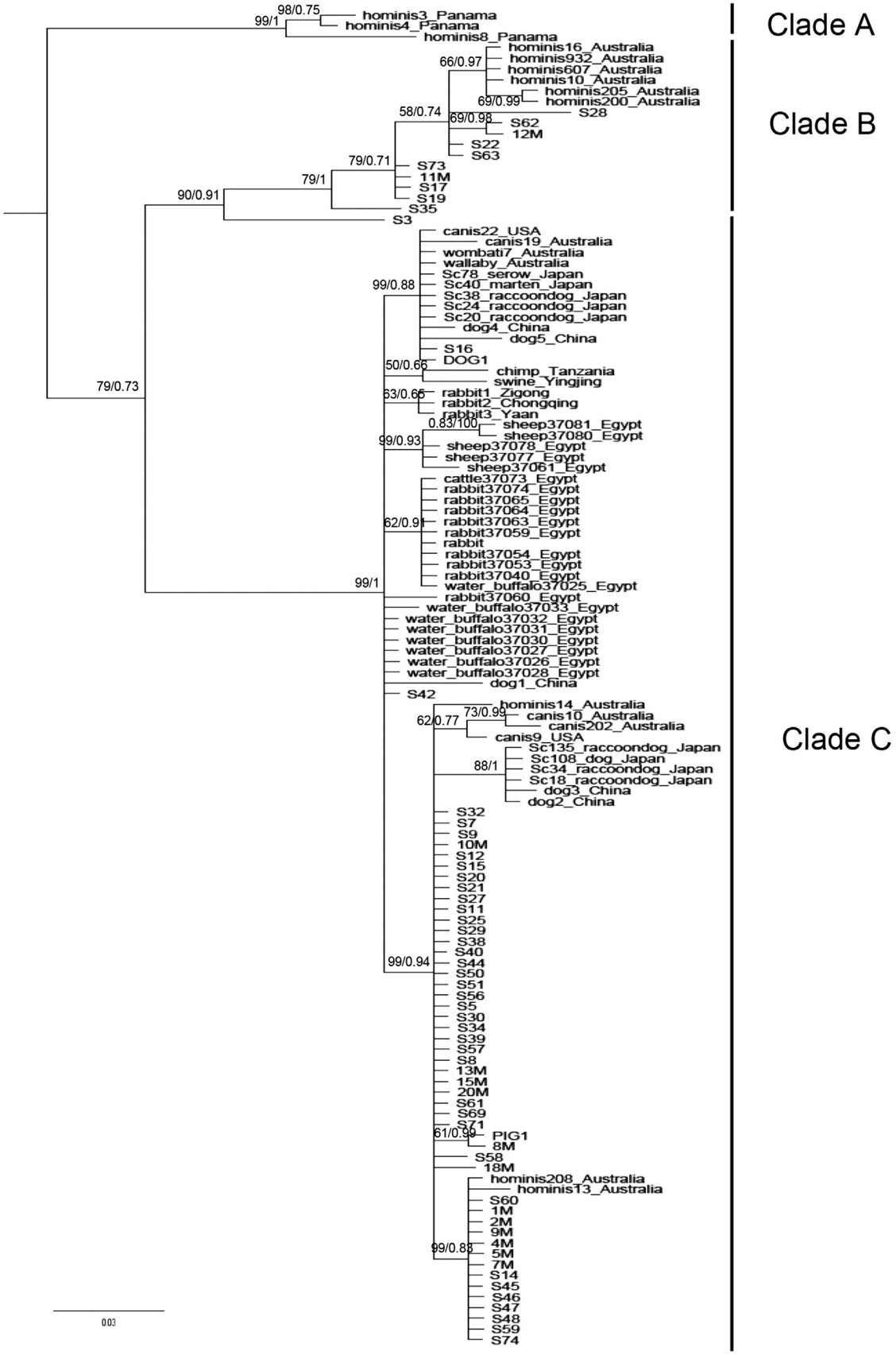
	H24																																							
	H25																	A															G							
	H26																																G							
	H27												T																				G							
	H28																														T	T	G							
CLADE A	H29											G	T		A			A	C							T					T	T	G		G	A				T
	H30											G	T		A			A	C							T					T	T	G		G	A				T
	H31		A									G	T		A		C	A	C							T					T	T	G		G	A		G		T
CLADE B	H32									C			T							C			C			T					T	T						G		T
	H33									C			T							C			C			T					T	T						G	G	T
	H34		A	G	A	T				C			T							C			C			T					T	T	G					G		T
	H35									C			T							C			C			T	G				T	T						G		T
	H36									C			T							C			C			T					T	T						G		T
	H37	T						C					T										C			T		C			T	T							G	T
	H38	T								C			T			C				C			C			T				G	T	T						G		T
	H39									C			T			C				C			C			T					T	T						G		T

		nucleotide position																																	
	N° of haplotype	256	257	263	269	275	277	278	283	284	287	288	293	296	299	301	302	308	311	314	326	329	336	344	347	353	362	364	367	374	377	387	389	390	395	n=
CLADE C	H1	C	C	G	A	T	G	C	T	G	T	C	T	A	G	G	A	T	G	T	T	C	T	T	A	C	C	A	T	T	C	G	T	T	T	15
	H2																																			1
	H3																																			1
	H4		T																									G	C							1
	H5																																			1
	H6		T																												T					1
	H7		T																					C		T					T					1
	H8		T																A																	3
	H9		T																																C	2
	H10		T																																C	2
	H11		T																																C	1
	H12		T	A																																1
	H13		T																																	5
	H14		T			C																														11
	H15		T				A						C	C																						1
	H16		T															C			C															1
	H17		T															C			C															5
	H18		T															C																		1
	H19		T													A																				1
	H20																																			30
	H21																																			2
	H22		T																																	10
	H23		T																																	2
	H24																			C																1
	H25	T																												C						1
	H26																																			1
	H27																																			1
	H28		T																																	1
CLADE A	H29		T	A				T	C	A		T			A			C	A			T			G		T				T		A	C		1
	H30		T	A				T		A		T			A			C	A			T			G		T				T		A	C		1
	H31		T	A				T		A		T			A		G	C	A			T			G		T				T	A	A			1
CLADE B	H32		T	A	G			T		A	C	T			A				A							T	T						A			4
	H33		T	A	G			T		A	C	T			A				A							T	T						A			2
	H34		T	A	G			T		A	C	T			A				A							T	T									1
	H35		T	A	G			T		A	C	T			A				A							T	T									2
	H36		T	A	G			T		A	C	T			A				A							T	T									2
	H37			A				T		A	C	T			A				A				C			T	T									1
	H38		T	A				T		A		T			A				A							T	T									1
	H39		T	A	G			T		A		T			A				A							T	T									4



and three heated) for one million generations, sampling every 1000 generations and discarding the first 25% of samples as burn-in. The potential scale reduction factor approached 1.0, and average of split frequencies under 0.01 were used for examining convergence. All trees were visualized using FigTree (http://tree.bio.ed.ac.uk/software/figtree). The number of polymorphic and parsimony-informative sites, haplotype and nucleotide diversity were computed with DnaSP5 and Arlequin 3.5. To visualize the relationship between haplotypes, a median joining haplotype network of *cox1* sequence was constructed using Network 4.6 according to geographical origin and host.

Results

The sequence of the mitochondrial *cytochrome c oxidase subunit 1* gene, *cox1*, was obtained in mites from 60 humans (age range, 1–77 years), one pig and one dog. Other sequences corresponding to 62 mites from various hosts and geographical areas were retrieved from GenBank (Supplementary Table S1). The multiple alignments of a part of *cox1* gene including 396 bp revealed 73 polymorphic sites (18.4%), including 27 singleton variable sites (two variants) and 46 parsimony-informative sites (45 two variants and one three variant) (Table 1). All of the successfully sequenced samples were assigned to only one haplotype. In all, 39 haplotypes were observed among mites collected from 13 different hosts, including humans, and eight geographical areas. Twenty haplotypes were observed among mites collected in humans: of these, (a) 10 were sequenced in the present study and deposited in GenBank (accession nos. KR058184–KR058193), haplotypes H20, H26, H27, H34–H39 and H21, with the latter being shared with mites collected from animals; (b) 10 were previously described, H1, H24, H25, H29–H33, H22 and H23; the two latter are shared with mites collected from animals [12,27]. Nineteen haplotypes, H2–H19 and H28, were observed among mites collected from animals only.

The phylogenetic trees based on ML and BI analyses showed similar topologies with few differences in node support values. Clades A (n = 3) and B (n = 17) harboured only mites collected from humans (Fig. 1). Clade C included mites from animals (n = 52) and humans (n = 52). Mites from animals were collected from 12 animal species: rabbit (*Oryctolagus cuniculus*) (n = 13), dog (*Canis familiaris*) (n = 12), water buffalo (*Bubalus bubalis*) (n = 8), raccoon dog (*Nyctereutes procyonoides*) (n = 6), sheep (*Ovis aries*) (n = 5), pig (*Sus scrofa domesticus*) (n = 2), chimpanzee (*Pan troglodytes*) (n = 1), cattle (*Bos taurus*) (n = 1), wombat (*Wombatus ursinus*) (n = 1), wallaby (*Macropus agilis*) (n = 1), Japanese serow (*Capricornis crispus*) (n = 1) and Japanese marten (*Martes melampus*) (n = 1). Both phylogenetic and network analyses showed that *S. scabiei* mites were distributed into three genetically distinct clades and that each clade formed a well-supported monophyletic group (Fig. 2).

Mites belonging to clade A were obtained in Panama (Fig. 2). The geographic distribution of clade B was restricted to Australia and France. Mites belonging to clade C were more largely distributed on five continents. Mites of clades B and C were sympatric in France and in Australia.

Estimates of genetic diversity for the 124 sequences and within clade B and clade C were high (Table 2). The F_{st} value, a measure of genetic population differentiation, calculated for clade B and clade C was 0.844, indicating very strong population structure. The population gene flow value, Nm, was 0.09, reflecting very low gene flow estimates between mites of clades B and C. Considering mites collected only from humans in clades B (n = 17) and C (n = 52), the F_{st} value was 0.890, indicating very strong population structure, and the Nm value was 0.06, reflecting very low gene flow estimates between mites of those subpopulations. Within clade C, for mites obtained from animals (n = 52) and mites obtained from humans (n = 52), the F_{st} value was 0.435, indicating strong population structure; the Nm value was 0.65, reflecting relatively low gene flow. Considering mites collected from animals (n = 52) and all mites collected from humans (n = 72), the F_{st} value was 0.228 and the Nm value was 1.69. For mites collected in Australia (n = 14) and in France (n = 62) the F_{st} was 0.165 and the Nm value was 2.52, reflecting substantial gene flow.

Discussion

The most striking observation of the present study was that *S. scabiei* mites in humans were mainly distributed into three genetically distinct clades. Our results are consistent with two previous analyses using *cox1* as a genetic marker but not including human samples of European origin [12,28]. We did

FIG. 1. Phylogenetic relationship among *Sarcoptes scabiei* from humans and animals based on the *cox1* nucleotide sequences. Tree was rooted with *Otodectes cynotis* (KF891933). Maximum likelihood (ML) and Bayesian inference (BI) gave similar topology. Bootstrap percentages of ML (left of slash) and posterior probabilities of BI analysis (right of slash) are indicated at nodes. All nodes supported with bootstrap and posterior probabilities >50% and 0.5 are shown. PIG1, DOG1 and samples including "M" or "S" and one or two numbers in their names were collected in France. Accession numbers of other sequences are provided in Supplementary Table S1.

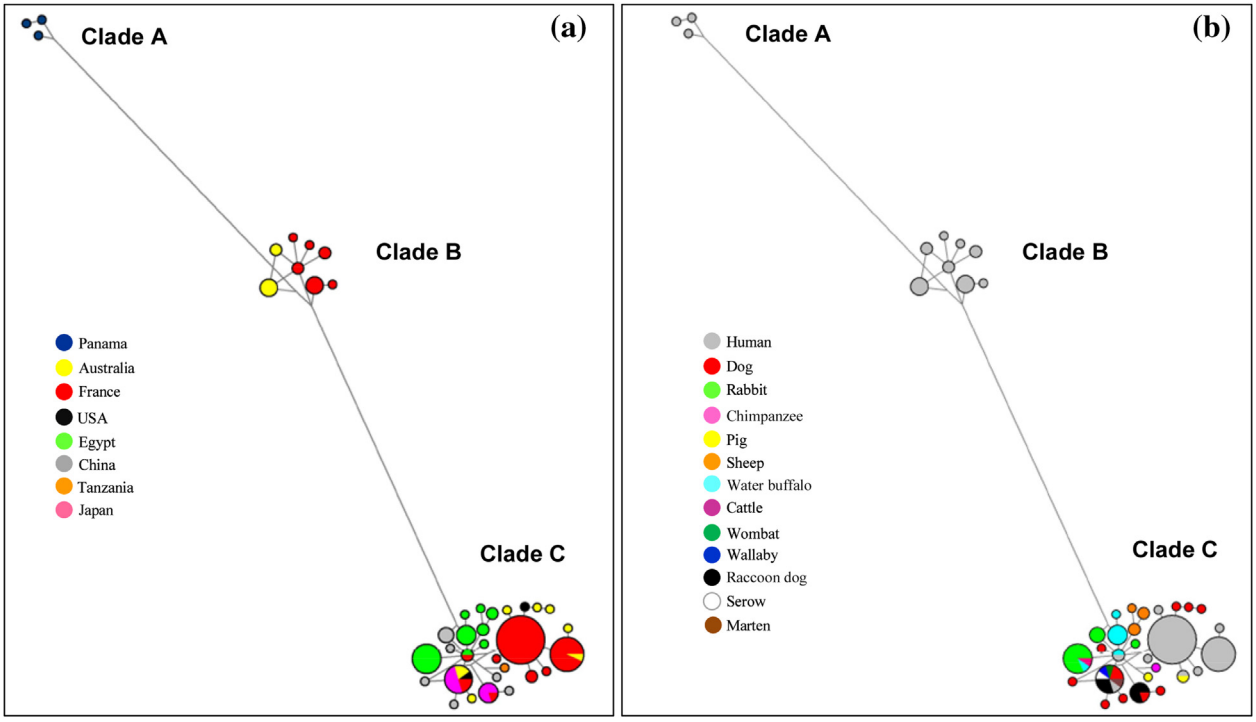


FIG. 2. Haplotype map for part of *cox1* gene of *Sarcoptes scabiei* collected from humans and animals in different geographical locations. Size of circles is proportional to haplotype frequency. (a) Haplotype network according to geographical origin and (b) host, inferred under median joining as implemented in Network 4.6 software.

not include the samples collected in Chinese humans (n = 5) of the study by Zhao *et al.* [28] in our analysis because the size of the *cox1* fragment they used, 317 bp, was shorter than in our study.

Clades A and B clustered only mites collected from humans. More samples collected from various hosts and geographical areas are required to assess whether clades A or/and B may include mites from animals. Clade A was represented only by three samples collected in Panama with no information relative to the human host. Two other mites collected in the New World, from dogs in the United States, clustered in clade C.

The F_{st} value and the Nm value calculated for all mites included in clades B and C indicated a strong population structure and a low gene flow between mites of those clades, reflecting a clear genetic population differentiation. These data not only support the rejection of the hypothesis of panmixia for *S. scabiei* in humans but also suggest that mites belonging to different clades are genetically isolated even if they were collected in the same area.

The genetic differentiation, within clade C, between mites from humans and mites from animals appeared substantial, but some gene flow, albeit limited, does occur between these mites. Another finding of the study was that mites from dogs appeared to be not genetically different from mites from humans on the basis of *cox1* genotyping. This result is consistent with a previous study using a part of *12S rRNA* for genotyping and including a smaller number of isolates [29]. Additional analyses of our samples with 12S rRNA and 16S rRNA genotyping

TABLE 2. Estimates of genetic diversity of *Sarcoptes scabiei* mites

Characteristic	No. of sequences	No. of haplotypes	Haplotype diversity (SD)	Nucleotide diversity (π) (SD)
All mites	124	39	0.91 (0.016)	0.021 (0.0025)
Mites clade B	17	8	0.89 (0.046)	0.0103 (0.0028)
Mites clade C	104	28	0.87 (0.02)	0.007 (0.00045)
Mites collected in humans, clade C	52	9	0.59 (0.057)	0.0021 (0.0004)
Mites collected in humans, all clades	72	20	0.78 (0.041)	0.0267 (0.0034)
Mites collected in animals, clade C	52	22	0.91 (0.023)	0.0082 (0.0007)
Mites collected in Australia	14	9	0.9 1 (0.059)	0.035 (0.0031)
Mites collected in France	62	13	0.717 (0.050)	0.0183 (0.0035)

showed convergent subclustering of mites in clades B and C (data not shown). We could not incorporate in these analyses 12S rRNA and 16S rRNA sequences from mites of other studies because such data are lacking in GenBank.

In northern Australia, Walton *et al.* [30], using microsatellites markers, claimed that gene flow between scabies mite populations on humans and dogs was extremely rare. However, their study included only seven human noncrusted cases. When the same team studied the relationship of *S. scabiei* mites collected from dogs and humans by using *cox1*, they found that mites from dogs and humans clustered in the same group. Using the microsatellite approach on the same samples, the three mites from humans were then assigned to another group including only mites collected in humans. Considering the low number of cases and the inconsistency of results between approaches, the conclusions drawn from those studies appear questionable. In a recent study, Zhao *et al.* [28], using *cox1* for phylogenetic analysis, reported that mites from dogs in China, Australia and the United States clustered with mites collected from Australian people. Those authors concluded that humans could be infected with mites from dogs. Our results on this point are in agreement with those authors.

Currently, *S. scabiei* is taxonomically divided into different varieties based on host origin [10]. According to our results and other recent studies [12,28], such varieties are no longer warranted. A generally accepted hypothesis suggests that humans and protohumans were the initial source of animal scabies; dogs and other domestic animals were then infested and were themselves a source for other species of wildlife [5]. One can imagine that infection of animals from humans or protohumans resulted from the adaptation to animal hosts of mites from clade C and not from clade B (provided that no animal host will be found in the future for mites of clade B). Given the gene flow observed between mites collected on humans and animals, scabies may be considered as a zoonosis, even if scabies acquired from animals is usually clinically moderate and transient in humans [5]. Our results and that of other recent studies suggest that we ought to reevaluate the level of transmission between humans and animals and between domestic and wild animals [16,31].

Clade B harboured mites collected only in Australia and France. More samples from various geographical areas are required to assess whether clade B may include mites of other geographical origin. Clade C harboured also, but not exclusively, mites from Australia and France. Those data are consistent with the hypothesis that Australian mite populations result from a European introduction of *S. scabiei* by immigrating individuals and/or their companion animals, as suggested by previous studies [29,32]. Moreover, our data strongly suggest a double event for the introduction of the two clades, B and C, in Australia.

In conclusion, *S. scabiei* mites in humans do not constitute a homogeneous population. By using the methodology implemented here, each isolate may be genotyped and assigned into one of the distinct clades. Sarcoptes population genetic substructuring may have major consequences in terms of pathophysiology and epidemiology. Further investigations are now required to assess whether different clinical forms of scabies are associated with particular haplotypes or clades. *Sarcoptes* population genetic substructuring ought to be considered in the emergence and spread of acaricide resistance, in *Sarcoptes* vaccine development and in diagnostic test development and implementation.

Transparency declaration

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Appendix A. Supplementary data

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.cmi.2015.08.002.

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