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Review

The origin and distribution of human lice in the world

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ABSTRACT

Two genera of lice parasitize humans: *Pthirus* and *Pediculus*. The latter is of significant public health importance and comprises two ecotypes: the body louse and the head louse. These ecotypes are morphologically and genetically notably similar; the body louse is responsible for three infectious diseases: Louse-borne epidemic typhus, relapsing fever, and trench fever. Mitochondrial DNA studies have shown that there are three obviously divergent clades of head lice (A, B and C), and only one clade of body lice is shared with head lice (clade A). Each clade has a unique geographic distribution. Lice have been parasitizing humans for millions of years and likely dispersed throughout the World with the human migrations out of Africa, so they can be good markers for studying human evolution. Here, we present an overview of the origin of human lice and their role in vector pathogenic bacteria that caused epidemics, and we review the association between lice clades and human migrations.

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1. Introduction

Lice are obligate ectoparasites, and each host species carries its own type of louse. Parasite speciation frequently occurs at approx-

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imately the same time as the "cospeciation process" in the host species (Veracx and Raoult, 2012). In 2010, an investigation of sucking louse relationships using 18S, EF-1α and COI genes (Light et al., 2010) estimated the diversification of sucking lice (*Anoplura*) to be approximately 77 million years old, a time period coinciding with the emergence of the placental mammal superorders. There are conflicts between host and parasite phylogenies: these conflicts can be the result of events that intervene in the distribution of parasites on hosts, the speciation of the parasite independent of the host and colonization failure (Page RDM, 2003). Lice have been recorded in most parts of the world, including in mummies in the New World and most likely achieved worldwide distribution because of their human host, which indicates that the species share a long record of co-evolution (Ascunce et al., 2013). Because of this long association with humans, lice have become a model for the study of cophylogenetic relationships between hosts and parasites (Demastes et al., 2012), although the origins of parasitism in lice remain inadequately understood.

2. Taxonomic status of human head and body lice, morphology, biology and ecology

Lice are small, wingless insects that cannot live independently from their host (Weiss, 2009). The order of *Phthiraptera* is divided into two major groups: *Anoplura* (the hematophagous sucking lice of placental mammals) and *Mallophaga* (the chewing or biting lice of birds, marsupials and placental mammals) (Lance, 2008). Although the *Mallophaga* group is likely paraphyletic, it is widely agreed that sucking and chewing lice originated from a common nonparasitic ancestral group closely related to the order Psocoptera (book lice and bark lice). These two groups are suggested to have diverged in the late Jurassic or early Cretaceous Period, 100–150 million years ago (MYA) (Lance, 2008). Humans can be infested with two types of lice (Anoplura): *Pediculus* on the head and/or body and *Pthirus* (in the pubic area) (Busvine, 1948). Both are blood sucking, but the status of the former is controversial. Since the late 1970s, lice found on the head versus lice found on the body have been considered separate species (*Pediculus capitis* and *Pediculus humanus*, respectively) or subspecies of *P. humanus* (Busvine, 1945; Maunder, 1983), despite the fact that, under experimental conditions, the species can interbreed and produce fertile offspring (Bacot, 1916; Buxton, 1940; Maunder, 1983; Nuttal, 1919). Different sizes of head and body lice found on the same individuals led to the conclusion of separate subspecies (Busvine, 1978). There are no consistent points of distinctive morphology (Ferris, 1953), and identification relies only on the location on the human body surface (Maunder, 1983). The current view is that lice found on the human body and clothing (and close personal effects, e.g., bedding) have evolved specific behavioral and physiological adaptations (particularly egg laying in cloth) that are not shared by lice living on the scalp (head lice) (Busvine, 1978). There are obvious differences between typical specimens of lice taken from the head and from the body: the average size of head lice is smaller than the average size of body lice, the antennae of the forms show considerable differences in proportions, and the lateral indentations between the segments of the abdomen are more pronounced in typical head lice than in body lice (Busvine, 1948). There are differences in the form and chaetotaxy of the genitalia of the two races, but Ferris discusses these details and dismisses them as inconstant (Busvine, 1948). Head lice are generally darker than body lice, but this difference is highly variable and, as Ewing stated, the difference is largely dependent on the environment (background coloration) (Ewing, 1926). This hypothesis was refuted because this color difference is not constant as we can find gray body lice in Africa (e.g., body lice in Ethiopia) (Fig. 1) (Veracx et al., 2012a,b). Other differences occur in the natural behavior of lice, such as a higher mortality in body lice than in head lice (Busvine, 1948), starvation that can last longer in body lice, causing higher survival rates when rearing them on artificial blood feeding system (Nuttal, 1919), a smaller number of eggs in head lice than in body lice (Bacot, 1916), and a higher percentage of hatching in body lice then in head lice (Busvine, 1948; Veracx and Raoult, 2012).

3. Human louse as a vector of human pathogens responsible for epidemics

Pediculosis is the cause of parasitism by the body louse (Chosidow, 2000). A louse-infested person can be infested by thousands of lice, each of which bites five times per day (Roux and Raoult, 1999). The louse injects the skin with biologically active proteins that include an anticoagulant and an anesthetic (Roux and Raoult, 1999). These antigens provoke an allergic reaction within 3–4 weeks after the bite, which can lead to pruritus (Raoult and Roux, 1999). Severe infestations of body lice, also

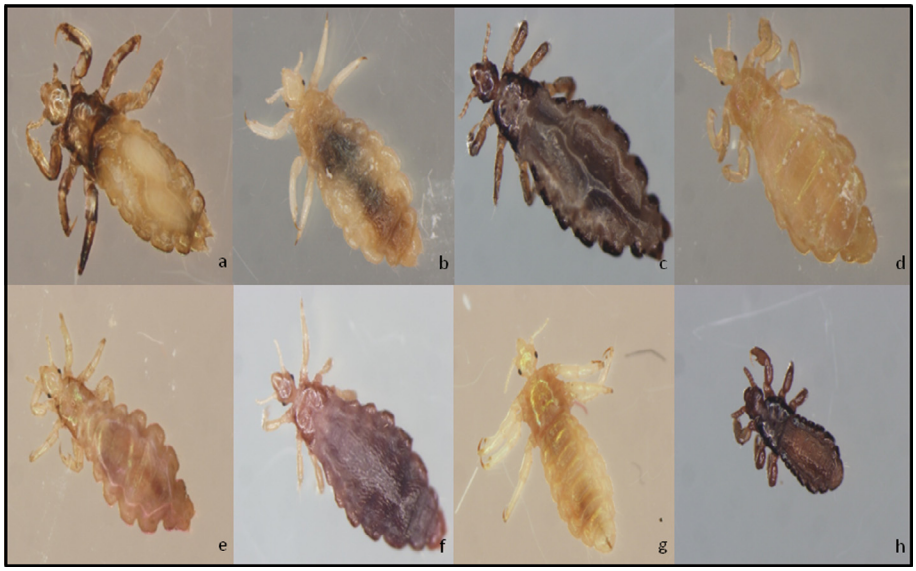

Fig. 1. Photographs of lice from worldwide regions; (a) HL from Japan; (b) HL from China; (c) HL from Nepal; (d) BL from Hungary; (e) HL from USA; (f) BL from USA; (g) BL from Ethiopia; (h) HL from Ethiopia. HL: head louse, BL: body louse.

known as vagabond's disease, are of concern in the homeless population (Fig. 2) because lice can walk for a number of meters to locate a host (Didier Raoult personal observation). Close body contact is strongly associated with louse transmission, and infestation occurs more frequently in crowded environments, such as homeless shelters, refugee camps, and jails, especially when hygienic standards are lacking (Brouqui, 2011).

Pediculosis corporis is a major public health concern because it can transmit at least three intracellular pathogenic bacteria: *Rickettsia prowazekii* and *Bartonella quintana*, which belong to the α subgroup of Proteobacteria, and the spirochete *Borrelia recurrentis* (Woese, 1987). Each of these bacteria has another common characteristic, an unusually reduced genome compared to their closest relatives (Moliner et al., 2010; Ogata et al., 2001; Veracx and Raoult, 2012). The genome of *B. recurrentis* appears to be a degraded subset of that of the tick-borne relapsing fever causing agent *Borrelia duttonii* (Lescot et al., 2008). *B. quintana* was described as a genomic derivative of *Bartonella henselae*, which is transmitted by the cat flea and to humans by cat scratches or cat bites (Alsmark et al., 2004). The genome of *R. prowazekii* is reduced, with hundreds of degraded genes (Andersson et al., 1998; Ogata et al., 2001) (Fig. 3). The highly pathogenic bacteria should have more genes than other nonpathogenic bacteria (Georgiades and Raoult, 2010), but the opposite was demonstrated (Georgiades and Raoult, 2010). Bacteria evolve to adapt to environmental changes, and their genetic content frequently varies through gene gain and gene loss. When a bacterium becomes intracellular, this specialization reduces the likelihood of a gene exchange, so new characteristics are more difficult to acquire and the potential of the bacterium to adapt to a changing environment is reduced. In such a model, gene losses will outnumber gene gains, resulting in a genome size reduction that will lead to deregulation and a higher pathogenicity (Georgiades and Raoult, 2010; Merhej et al., 2009) (Fig. 3).

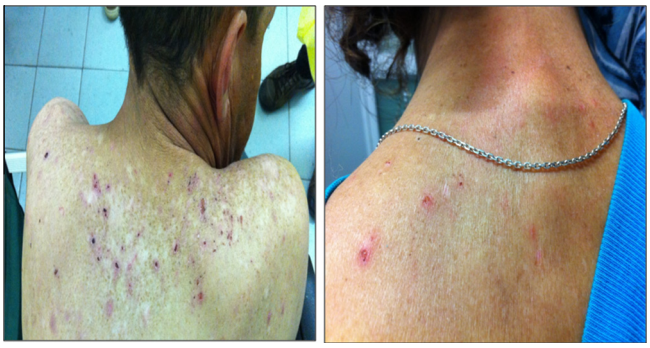

Fig. 2. Pruritic and scratching lesions in a homeless man and woman in Marseille.

4. Trench fever

Trench fever was first described during World War I, so named because the disease affected Allied and German troops crowded into trenches during World War I (Badiaga and Brouqui, 2012). The disease is caused by *B. quintana*, a Gram negative bacteria. The incidence of *B. quintana* dramatically fell after World War II. In the early 1990s, trench fever was recognized as a major reemerging disease in the poor living conditions of urban homeless populations in developed countries (Brouqui and Raoult, 2006). It was recovered in soldiers from Napoleon's army (Raoult et al., 2006; Roux and Raoult, 1999) and in the dental pulp of a person who died 4000 years ago (Drancourt et al., 2005).

5. Epidemic typhus

Epidemic typhus has caused more deaths than all of the wars in history (Zinsser, 1935); its transmission by the body louse was demonstrated by Charles Nicolle (Gross, 1996; Nicolle, 1910). It is caused by *R. prowazekii*, an obligate intracellular bacterium that also kills the lice within one week of infection (Andersson and Andersson, 2000).The origin of typhus is controversial; some consider it to be an old European disease that caused the Athens plague (Zinsser, 1935), while others believe that the reservoir is extra human and of American origin, as suggested by its presence in isolates from flying squirrels (Raoult and Roux, 1999). Epidemic typhus has been identified by molecular technique in the bodies of soldiers from the army of Napoleon (Raoult et al., 2006; Roux and Raoult, 1999), but the earliest human case was found on remains from 1712 in Douai, France (Nguyen-Hieu et al., 2010).

6. Relapsing fever

Epidemic relapsing fever is caused by the spirochete *B. recurrentis*, and humans are the sole reservoir (Cutler, 2006). Although the disease has disappeared in extensive regions of the world, it remains an important endemic disease in northeastern Africa (Mitiku and Mengistu, 2002). It was initially described in Ireland and was one of the first infectious lice disease identified by microscopy (Mackie, 1907). Relapsing fever spreads in humans through feces (Houhamdi and Raoult, 2005) and, as with epidemic typhus, primarily affects military and civilian populations disrupted by war

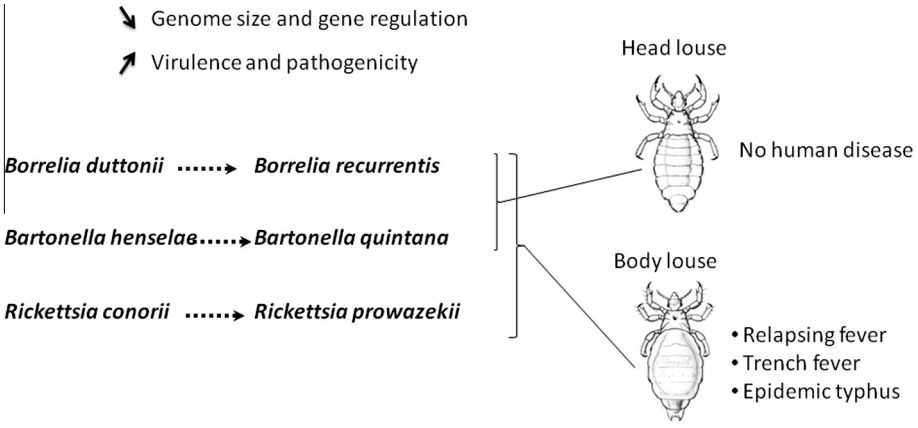

Fig. 3. Reductive evolution of the highly pathogenic bacteria associated with *Pediculus humanus*.

and other disasters. After becoming infected by the ingestion of an infected blood meal, a louse remains infected throughout its lifetime (Raoult and Roux, 1999). As the disease frequently causes jaundice, it has been reported as the yellow plague, which ravaged Europe in 550 AD (Bryceson et al., 1970). During World War I, a half million people contracted relapsing fever in Serbia. During civil war in Russia and eastern Europe between 1919 and 1923, 13 million cases were reported, leading to 5 million deaths (Bryceson et al., 1970).

7. Other louse-associated diseases

The role of body lice in the transmission of pathogenic bacteria to humans is being investigated. *Acinetobacter baumannii* was found in 21% of the 622 body lice collected worldwide (La Scola and Raoult, 2004), although no *A. baumannii* infections are known to be transmitted by body lice. *Yersinia pestis*, an etiological agent of plague, was recovered from a body louse collected from a septicemic patient in Morocco in the 1940s (Drancourt et al., 2006; Houhamdi and Raoult, 2008) and was observed in one head louse and two body lice in the Democratic Republic of the Congo (Piarroux et al., 2013). Louse-mediated plague transmission has been experimentally demonstrated in our laboratory (Houhamdi et al., 2006). One infected blood meal was sufficient to infect lice, in which *Y. pestis* multiplied and produced a generalized infection, and viable *Y. pestis* were excreted in feces (Houhamdi et al., 2006).

8. Pathogenic bacteria in head lice

Body lice are much more potent vectors of pathogens than head lice, likely because of the reduced phagocytic activity of their immune system (Kim et al., 2011). Yet, although it is not clear if head lice can act as vectors of human pathogens, they can carry pathogens. DNA from *B. quintana* was collected in head lice from Nepalese children in 2006 (Sasaki et al., 2006), in head lice from homeless individuals in the USA in 2009 (Bonilla et al., 2009), in head louse nits collected from a Marseille homeless person (Angelakis et al., 2011), in head lice from Ethiopian patients (Angelakis et al., 2011) and in 19 head lice collected from 7 patients in Dakar (Senegal) (Boutellis et al., 2012). In Ethiopia, *B. recurrentis* DNA was detected in head lice; because these patients were also infested with body lice. We hypothesize that the presence of the bacterium in the head lice was the result of the ingestion of contaminated blood (Boutellis et al., 2013). *A. baumannii* DNA was detected in 33% of the head lice collected from 245 children from 74 Parisian schools (Bouvresse et al., 2011).

9. Genetic studies and different types of lice

The major phenotypic differences between the head and body louse relate to the ecology and color (Veracx and Raoult, 2012). No congruence among those characteristics could be assessed (Veracx et al., 2012a,b), and phenotypic studies were subsequently challenged by genetic studies (Veracx and Raoult, 2012). Three clades (A, B and C) of head lice were described by analyzing the mitochondrial DNA (cytochrome b and cytochrome oxidase subunit 1), and of them, only one (clade A) was present in body lice (Raoult et al., 2008; Reed et al., 2004) (Fig. 4). Clade A can be subdivided in two subclades: the Eurasian subclade A1 and the Sub-Saharan subclade A2 (Li et al., 2010; Veracx et al., 2012a,b; Yong et al., 2003). A third clade, named A3, was recently characterized using high polymorphic intergenic spacers (Multi spacer typing) in the American lice and represents a specific Amazonian genotype that is putatively pre Columbian (Boutellis et al., 2013a,b,c,d). This multi spacer typing technique was previously used to show a correlation between genotypes and ecotypes (the genotypic distribution in relation with the source of the analyzed lice) (Li et al., 2010). Clade B head lice are found in North and Central America (USA and Honduras, respectively), in Europe, and in Australia (Light et al., 2008). The third type (clade C) is only found in head lice from Nepal (Reed et al., 2004), Ethiopia (Angelakis et al., 2011) and Senegal (Boutellis et al., 2012) and these clade C head lice always have a dark color (Veracx et al., 2012a,b; Veracx et al., 2013). Further complicating the study of the different groups of lice, a recent study showed possible interbreeding events between lice of different clades, and identical genotypes of highly variable spacers were

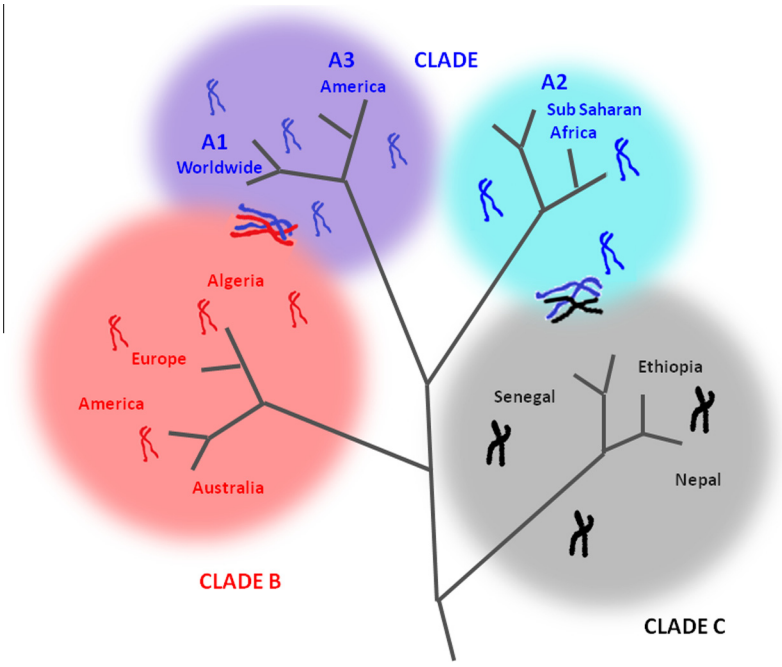

Fig. 4. Schematic representation of the *cytb* gene phylogenetic tree with the different recombinations occurring between plasmids (minichromosomes).

found between lice on different continents (Veracx et al., 2012a,b). Another study based on multi-spacer typing of doubly infested homeless individuals showed that, at least among Clade A lice, head and body lice are ecotypes of the identical species (Veracx et al., 2012a,b). Reed et al. (2004) recently assessed the genetic structure of the human louse through an analysis of variation at 15 new microsatellite loci in 93 human lice from four regions of the world and confirmed a high level of inbreeding in human lice (Ascunce et al., 2013). Finally, using genetic and transcriptomic profiles, recent studies showed that body and head lice can be differentiated based on a difference at a single 752 bp gene (PHUM540560) that encodes a hypothetical protein of 69 amino acids that was found in body lice but not in head lice (Olds et al., 2012).

Unexpectedly, when we amplified the 187 bp partial fragment of the PHUM540560 gene by PCR in our laboratory, a positive amplification was observed for both head and body lice, indicating that at least one portion of the gene is present in both types of lice (Drali et al., 2013). Sequencing revealed that the partial gene fragment of head lice is very different from that of body lice, which may explain why it was not amplified in the Olds et al. study. We established a rapid multiplex real-time PCR assay that differentiates between head and body lice with a 100% specificity and sensitivity (Drali et al., 2013). Based on these studies, the most prudent hypothesis is that clade A head louse has a deleted genome and originated from body louse. The opposite hypothesis was considered evident for years (EWING, 1926; Ferris, 1953; Kittler et al., 2003; Maunder, 1983).

10. The sympatric life of lice and recombination between mitochondrial genes

P. humanus females have lost their spermatheca, the sperm storage organ, and must mate before laying eggs; frequent mating is essential, and this process encourages outbreeding (Maunder, 1983; Mukerji and SEN, 1951). The high mobility of lice is rarely recognized, and lice do not exist as isolated groups but as actively intermingling specimens (Maunder, 1983). This intermingling allows them to mate more frequently, which increases gene exchange and recombination (Veracx and Raoult, 2012).

Two recent studies from our laboratory characterized recombination events that occur in lice: the first used the cytochrome b gene to show that Clade A and Clade C lice are highly divergent, while the second used the intergenic spacer PM2 to show that Clade C lice are not clustered separately from Clade A. These studies suggest that recombinations occur between Clade A and C (Veracx et al., 2013).

An identical analysis was performed on clade B and A head lice, which live in sympatry in Algeria. The phylogenetic analysis of the concatenated sequences of the 4 polymorphic intergenic spacers and the cytb gene sequences of these populations of head lice showed that clade A and clade B head lice recombine, suggesting that interbreeding occurs in sympatric environments (Boutellis et al., 2013a,b,c,d). Potential recombination events between Clade A and B or C further illustrate that their evolution is not dichotomic and that their behavior is closer to that of a rhizome (Georgiades and Raoult, 2011; Raoult, 2010). Given the wide genomic plasticity of louse mitochondria, which is split on 20 minicircular chromosomes, the exchange of a single plasmid was to be expected (Georgiades and Raoult, 2011; Raoult, 2010). An extraordinary feature of the fragmented mt genome of the human body louse is the stretches of identical sequences (26–127 bp long) shared between genes on different minichromosomes; this finding provides unequivocal evidence for inter-minichromosome recombination (Shao et al., 2009). As in body lice, inter-minichromosome recombination occurs in head and pubic lice (Shao et al., 2012).

11. Evolutionary history of human lice

The intimate association of lice with their hosts can explain why lice show more cospeciation with hosts than other groups of insects (Grimaldi and Engel, 2006). The oldest human head louse nit was found on a hair from an archeological site in northeastern Brazil and was dated to 8000 B.C. (Araujo et al., 2000). The oldest such finding in the Old World was 9000 years old, obtained from a hair sample from an individual who lived in the Nahal Hemar cave in Israel (Mumcuoglu, 2008). Head lice have been found at archeological sites in the southwestern USA, the Aleutian Islands, Peru, Greenland and Mexico and on mummies that were Incan sacrifices (Araujo et al., 2000). Recently, another discovery of lice was reported from a Maitas Chiribaya mummy from Arica, in northern Chile, dating to 670–990 calibrated years (Arriaza et al., 2013; Arriaza et al., 2012) (Fig. 5).

The origin and early evolution of lice is obscure because the fossilization of lice requires exceptional circumstances, and their fossils are exceedingly scarce (Wappler et al., 2004). Within *Phthiraptera*, phylogenetic studies showed that sucking lice are monophyletic and derived from chewing lice (Cruickshank et al., 2001; Johnson et al., 2004; Yoshizawa and Johnson, 2010), and thus, sucking lice evolved from a blood feeding chewing lice ancestor with highly modified anopluran mouth parts (Cumming, 1915).

There has been much debate over the age of lice and the origins of parasitism in this group (Lyal, 1985). The Psocoptera order is thought to have originated in the Mesozoic Era (Grimaldi and Engel, 2006). The recent discovery of two important fossils provides information on the age of lice. *Saurodectes vrsanskyi*, a putative louse, was recovered from the *Zaza* formation shales of Bassia, Siberia (ca. 140 MYA): at 17 mm in length. This fossil is approximately 10 times larger than any currently living louse and presumably resided on a massive host. some of its features suggest a link with *Phthiraptera* (Grimaldi and Engel, 2006). The *Megamenopon rasnitsyni*, a well preserved louse fossil showing close phylogenic affinities with the modern feather louse (*Menoponidae*), was found in the oil shale of Eckfeld Maar (ca. 44.3 ± 0.4 MYA): at 6.74 mm in length, it is twice the length of similar present day lice (Wappler et al., 2004).

Humans (*Homo sapiens*) are parasitized by two genera of sucking lice: one shared with chimpanzees (*Pan* spp.) and the other shared with gorillas (*Gorilla gorilla*) (Fig. 6). Human lice

Fig. 5. A pre-Columbian nit isolated from Mummy 23 from the Camarones 15-D, Northern Chile (photograph by Mario A Rivera reproduced with permission).

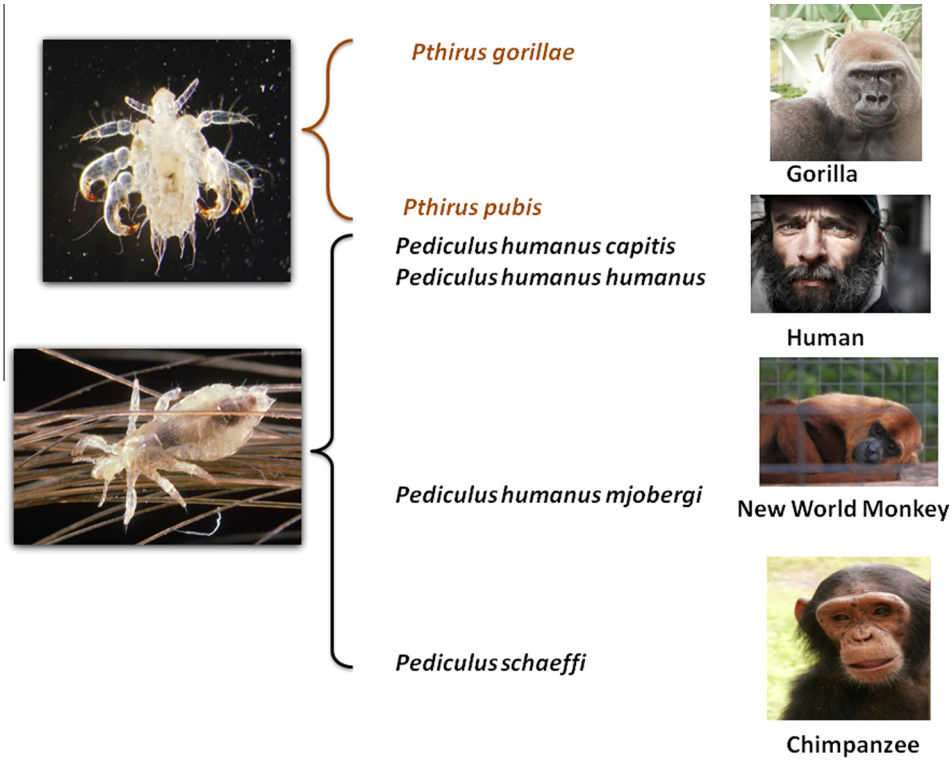

Fig. 6. Classification of *Pthirus* and *Pediculus* and host association.

(*P. humanus capitis* and *P. humanus humanus*) and chimpanzee lice (*Pediculus schaeffi*) are members of the *Pediculidae* family, and human pubic lice (*Pthirus pubis*) and gorilla lice are members of the *Pthiriadae* family (Reed et al., 2007, 2004; Weiss, 2009).

Based on the divergence estimations made by Reed et al. (2007), the most probable evolutionary history for the genera is that *Pthirus* and *Pediculus* diverged on an evolutionary ancestor of the chimpanzee, human, and gorilla approximately 13 MYA. Gorillas retained *Pthirus* with an extinction of *Pediculus* on the branch leading to humans and chimpanzees. Conversely, *Pediculus* was maintained on the lineage leading to humans and chimpanzees and was lost from the gorilla lineage, and the resulting species (*P. schaeffi* and *P. humanus*) diverged in tandem with their primate hosts approximately 6 MYA. Approximately 3–4 MYA, a *Pthirus* species switched from the gorilla lineage to the lineage leading to modern humans (Reed et al., 2007).

Based on nuclear and mitochondrial DNA markers, human and chimpanzee lice evolved approximately 5.6 MYA. At this time, evolving humans diverged from ancient chimpanzees (Reed et al., 2004). This finding was confirmed by a direct comparison of chimpanzee and human DNA, which showed that the two species diverged over a period of time and that the final divergence of chimpanzees and humans occurred no earlier than 6.3 MYA (Patterson et al., 2006). The divergence date estimates for the gorilla and human pubic lice averaged 3.32 MYA and are noticeably more recent than the divergence between the two *Pediculus* species (Reed et al., 2007).

The separation of *P. humanus* clade A into distinct "head" and "body" lice was estimated to have occurred approximately 72,000 ± 42,000 years ago, according to a mtDNA molecular clock analysis from a global sample of 40 head and body lice (Kittler et al., 2003). Because head lice DNA shows more diversity than body lice DNA, this finding suggests that head lice represent the source population (Kittler et al., 2003). The gene content and arrangement of the 20 mitochondrial minichromosomes are identical in head and body lice (Shao et al., 2009), indicating that the characteristics of these minichromosomes remained unchanged since the two types of louse separated (Shao et al., 2012). The theory that body lice evolved from head lice is not consistent with recent molecular data, and a likely alternative model is that clade A was constantly evolving. This evolution was facilitated by high infestations, and the deletion of head louse genome showed that the body louse represents the source population (Veracx and Raoult, 2012; Veracx et al., 2012a,b).

A third member of the Pediculidae family was reported and it was first described as *Pediculus affinis*, but because the name *affinis* had already been used by Burmeister for a louse that was later shown to be in the genus *Polypax*. In 1916, Ferris changed the name *P. affinis Mjöberg* to *P. humanus mjobergi* (Ferris, 1951). This species is not a human parasite but is found instead on certain South American monkeys of the Cebidae family (Locker Pope, 1966) (Fig. 6). Ewing (1926) hypothesized that Mjoberg's description indicated that his specimens were *P. humanus* (Ewing, 1938). Maunder speculated that when the first humans migrated to the New World across the Bering Strait, the New World monkeys had no indigenous lice and acquired the human head louse, which developed into the third lice species (Maunder, 1983).

12. Distribution of lice before globalization and association with the different human migrations

An exciting aspect of studying the diversity of lice is its application to the history of human evolution. Lice have been associated with humans for millions of years and dispersed throughout the world by early human migrants (Ascunce et al., 2013). *P. humanus* shows genetic evidence of population expansion from Africa approximately 100,000 years ago, which is consistent with the host evolutionary history because humans originated in Africa (Reed et al., 2004, 2007) and dispersed to four continents within the pre-

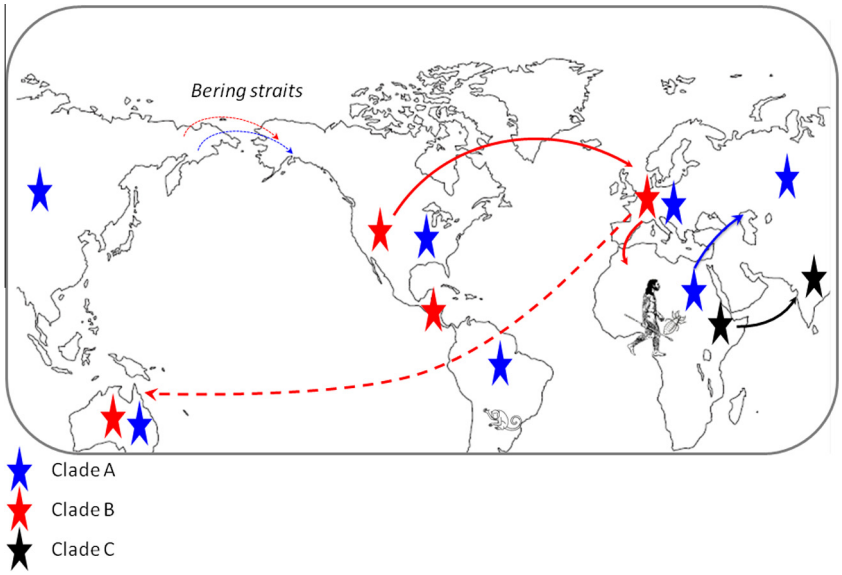

Fig. 7. Proposed migration route of human lice based on human migrations. Solid lines represent the results based on our molecular studies, and dashed lines represent the distribution cited by Light et al. (2008).

vious 80,000 years (Forster, 2004; Light et al., 2008). The comparison of the nucleotide diversities of African and non-African lice based on the PM2 spacer and the *cytb* gene shows that the DNA diversity is higher in African lice than in non-African lice (Veracx et al., 2013). Reed et al. (2004) found that the A and B clades of human lice separated approximately 1.18 MYA. While the former is a worldwide clade, the latter is a New World clade. Zinsser noted that the hair of ancient Peruvian mummies and the scalps of pre-Columbian Native Americans contained nits or lice (Zinsser, 1935), and the DNA analysis of lice from similar remains indicated that they belong to the worldwide clade A, so this clade was most likely present in pre-Columbian American populations before the arrival of European colonists (Raoult et al., 2008) (Fig. 7). Louse mitochondrial haplogroup B is found in the New World, Europe and Australia but not in Africa. Reed et al. (2004) suggested that its evolutionary origins might be found in the archaic hominids from Eurasia (such as the Neanderthals or Denisovans) and that it became associated with modern humans via a host switch during the periods of overlap (Ascunce et al., 2013). In our laboratory, we confirmed that the source of clade B head lice was American and putatively pre-Columbian by the analysis of two head lice nits from a 4,000-year-old Chilean mummy of the Camarones and the identification of clade B on one of the two operculated nits (Boutellis et al., 2013a,b,c,d) (Fig. 5). A third clade of head lice has been delineated in Ethiopia and Nepal, and this clade C, diverged from clades A and B approximately 2 MYA (Weiss, 2009). Given the age of this clade, it is possible that it evolved in archaic humans in Africa or Asia and was passed to modern humans, which is consistent with the close proximity of the interactions of modern and archaic humans in Africa and Asia (Ascunce et al., 2013). The presence of deeply divergent and regional mitochondrial lice clades is thus possibly the result of multiple colonization events of lice on their modern human hosts from now extinct archaic hominids (Reed et al., 2004).

Clade A lice most likely migrated from Africa to Eurasia and subsequently to Europe, Asia and the New World. Theoretically the first peoples of the Americas could have brought lice during migration to the New World, where lice remained in situ for thousands of years (Light et al., 2008) (Fig. 7). European colonists returning to the Old World from the Americas would have taken Clade B lice back to Europe beginning in the 16th Century (Raoult et al., 2008). European colonization of Australia approximately 150 years later may account for the presence of Clade B lice in Australia and potentially in other geographic regions affected by globalization (Reed et al., 2004).

13. Conclusion

The combination of phenotypic characters and genetic data is crucial to understand lice epidemiology, and efforts to obtain more data on that parasite are essential to prevent disease reemergence because body lice can carry and spread severe diseases in human populations and head lice can serve as a reservoir. From a genetic standpoint, the most important paradigm is that there are three major clades of head lice with one that also comprises a body louse, which represents a different ecotype of the same species (Drali et al., 2013). Understanding the processes shaping the genetic structure of human parasite populations is significant to basic and applied evolutionary biology, and defining the processes and mechanisms that maintain and generate human louse genetic diversity within and among host populations is critical for the design of effective control methods and for predicting how louse-borne diseases can spread through human populations.

Conflict of interest

None.

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Author contributions

D. Raoult conceived and designed the review.

A. Boutellis conducted the literature search and wrote the drafts of the review.

L. Abi Rached critically revised all of the draft.

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