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TOPICAL REVIEW

Land-use and land-cover changes and emerging non-tuberculous and tuberculous infectious diseases in humans and animals: a mixed-methods review of research findings from global and spatio-temporal perspectives

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Supplementary material for this article is available online

Abstract

Despite recent interest in land-use and land-cover (LULC) change effects on emerging infectious diseases (EIDs), the debate on global potential health threats remains polarizing. These depend on diverse LULC changes, different types of infectious disease systems, and spatio-temporal scales of studies. Here, using both a bibliometric and scoping review method, we summarize the reliability and availability of published relevant studies on LULC effects on mycobacteria, an important group of infectious bacteria that affect humans and both wild and domestic animals. We make connections of LULC with environmental changes (e.g. soils) that likely lead to an increased risk of mycobacteria spillover to human and other animal populations. An important feature of our review is a focus on research from the richest countries of the world, though some studies have been done in Africa, Asia and South America. Geographically, regions experiencing important LULC transformations, such as many tropical regions of Meso- and South America and Southeast Asia, have been given little or no attention in this important topic. Research on *Mycobacterium bovis,* and to a larger extent on *M. ulcerans,* constitutes convincing illustrations of the importance of acknowledging shifts in spatio-temporal scales, from local to global and inter-annual to decadal ones, when evaluating responses of mycobacteria to LULC changes. However, studies on other pathogenic mycobacteria remain very much confined to local and dispersed scales. To date, the role of LULC change effects has not been adequately studied for many human and animal pathogens, and more research and attention to this issue is clearly needed. This review provides a comprehensive set of data on the updates of LULC change and their impact on animal and human mycobacterial infections. It also proposes several research recommendations, in particular to better understand the emergence of mycobacteria in context, by multiplying study sites in different regions of the world and in adopting an ecosystem-based perspective, in order to encourage interdisciplinary research better linking environmental microbiology, veterinary science and medical research.

1. Introduction

Landscape patterns and processes directly and indirectly influence infectious disease emergence and spillover through effects on associated pathogens, hosts, vectors and environmental reservoirs [1]. However, the extent to which these affect infectious disease varies with geography, scale, definitions of disturbance, and ecological-anthropogenic conditions [2]. While land-use and land-cover (LULC) changes are often associated with increased spillover risk and transmission of zoonotic diseases, the effects of LULC change on disease dynamics are little understood [3]. There is a need for additional research into the conditions and mechanisms that influence how LULC change mediates disease risks, especially in areas of the world, e.g. the tropics, with some of the most neglected infectious diseases and vulnerable human populations.

LULC change is collectively recognized as a contributor to global climate change and has affected, based on high-resolution satellite imagery and long-term inventories of land use, 17% of natural habitats from 1960–2019 through tropical rainforest destruction [4]. When multiple changes are accounted, tropical rainforest destruction reaches 32%, accounting for 720 000 sq-km of land surface changed annually since 1960 (i.e. an area nearly twice the size of Germany) [4]. Global trade affecting agriculture (e.g. coffee, palm oil, cacao, lumber and herd production) represents a main driver of expansive tropical rainforest destruction [4].

Emerging infectious diseases (EIDs) are associated with human impacts on natural habitats, especially in rainforest ecosystems [3]. These associations have collectively led to the development of the landscape epidemiology discipline [5], and its extension to infectious disease ecology called pathogeography [6]. For instance, forest fragmentation due to agriculture development may increase the interface between natural and human-modified habitats, which in turn increases human contact with wildlife and their pathogens [7, 8]. In addition, it has been hypothesized that natural habitat disturbance may lead to changes in local diversity and the taxonomic composition of potential host reservoirs or vectors [9]. Host taxa that harbor more zoonotic pathogens may be more likely to occupy human-managed or human-natural interface ecosystems [9]. These relationships are not restricted to zoonotic infections, but are also important for environmental pathogens that cause disease in humans and other animals [10–12]. A major group of such pathogens includes the *Mycobacterium* species that cause disease in animals and humans [12].

Tuberculosis, caused primarily by *M. tuberculosis* and *M. bovis* (both grouped into the *Mycobacterium tuberculosis* complex, or MTBC) in humans and animals, respectively, serves as a paradigmatic case study for the impacts of habitat modification affecting disease dynamics. Urbanization and subsequent overcrowding may enhance human-to-human tuberculosis (i.e. Tb) transmission, while *M. bovis* causing bovine tuberculosis (i.e. bTb), is impacted differently due to changes in the relationships of humans, wildlife, and livestock [13]. Deforestation and habitat fragmentation also result in increased human–wildlife contacts, thereby heightening the risk of zoonotic transmission of Tb [14]. Similarly, factors associated with local environmental conditions, ecological communities and farming management practices can facilitate the spread of *M. bovis* among cattle, with spillover from cattle to wildlife, spillback from wildlife to livestock, and the potential for zoonotic transmission to humans [15].

Mycobacterium constitutes an important genus of Actinobacteria (or Actinomyceta in the recent systematic revision of bacteria; see https://lpsn.dsmz.de/phylum/actinobacteria), which is a group of large acid-fast eubacteria and one of the richest phyla of bacteria [16]. Most *Mycobacterium* species, as other groups of the same phylum, are saprophytic or commensal and ubiquitously distributed across aquatic and terrestrial ecosystems, but are predominantly telluric organisms. Nearly 190 mycobacterial species have been described [17]. Most mycobacterial diseases in animals and humans are sapronoses (or sapronotic diseases), i.e. they are primarily caused by saprophytic microbes living in soil or freshwater, occasionally infecting susceptible vertebrates by chance, with rare cases of zoonoses (or zoonotic disease) [17, 18].

Mycobacteria are divided into two subgroups. The first subgroup includes the clonal genotypes that cause tuberculosis in humans and animals (i.e. *M. tuberculosis sensu stricto, M. bovis, M. mungi* and *M. microti),* and *M. leprae,* which is the main agent of leprosy in humans and does not have a free-living saprophytic life stage [19]. The second subgroup includes all other mycobacteria and is considered the non-tuberculous mycobacteria (NTM hereafter). Bacteriologists have considered *M. avium paratuberculosis* (MAP), responsible for causing Johne's disease in a large diversity of vertebrates, a NTM because of its ability to infect a wide array of vertebrate species and also to survive as a saprophyte [18]. Transmission from an environmental origin is the hallmark of NTMs [18, 19]. MAP is mostly known by veterinarians for its indirect parasitic lifecycle, including alternation of soil saprophytic life-stages and infections of livestock intestinal cells [20]. While vertebrate-driven selection pressures have optimized MTBC pathogenicity, the natural source(s) of the selection pressures for the pathogenicity of most NTMs (including MAP) and *M. leprae* have not yet been identified [19, 20].

Knowledge of *Mycobacterium* species ecological niches, spatial distributions, and ecological drivers in soil, freshwater and estuarine habitats, and dust is very poor [21], and this knowledge should help identify novel approaches to disease prevention for animals and public health [22]. There is growing interest in soil-borne agents associated with increases in immunocompromised populations [17], as well as with significant resurgences of bTb and Johne's disease in cattle [23, 24]. Environmental factors, such as climate change, soil erosion, deforestation, and pollution of surface and groundwater used for agricultural irrigation, are all thought to play important roles in increasing animal and human exposure to these environmental bacilli [8].

Several soil microbiome analyses have revealed a significant number of NTM taxa [25–27]. The prevalence of the *Mycobacterium* genus in soil samples of Ethiopia and the UK was 2.3×10^7 gene targets per gram for fast growing NTMs and ranged from 2.9×10^5 to 1.2×10^7 cells per gram for slow growing NTMs [25]. There were also high abundances of *Mycobacterium* species reported in soil between 55 and 60 cm in depth [26]. Applying a pyrosequencing approach, targeting 16 S ribosomal RNA genes, to a set of 2173 soil samples distributed throughout France [28], reported the presence of 32 bacterial phyla or subphyla and three archaeal phyla, with 20 of the 35 phyla being cosmopolitan and abundant. In this dataset, *Frigoribacterium, Acidothermus, Conexibacter*, and *Mycobacterium* were the most abundant genera in forest soils, with their abundance correlating with a high C/N ratio [28]. Along with climatic conditions, soil pH, management, texture and nutrients affect bacterial community spatial and temporal change. As high-throughput molecular approaches and microbiological growth media designs improve, surveys of uncultured and non-identified soil microbial diversity will likely reveal substantial increases in mycobacteria species diversity in many ecological habitats and changing environmental conditions, including those associated with LULC. However, some controversies exist regarding the validity and accuracy of results that link LULC change to EID risk [3, 29].

One concern is the relative lack of scientific research to support broad generalities of direct and indirect LULC effects on pathogen spillover and spread [see 8 and 30 for an illustration on deforestation and EIDs]. The topic of LULC-mycobacteria relationships has received very little attention. Local-scale response to land-use change for soil and water microbial communities should differ depending on diverse LULC change and intensity, and different types of diseases. Several hypotheses can be formulated for LULC-mycobacteria relationships: (1) LULC influences changes in soil or water characteristics, microbial community diversity and composition, function and activity, and their potential hosts and vectors, thus having downstream effects on mycobacteria; (2) shifts in soil-environmental conditions associated with LULC change shape microbial community responses in ways that influence the diversity and abundance of mycobacteria, some of which are of human and wildlife health concern; (3) pristine ecosystem destruction, e.g. tropical forest, and spatial homogenization modify microbial community composition, organization and function having indirect effects on environmental mycobacteria, and (4) LULC leads to new selective pressures and genomic feature re-assortments that increase mycobacteria virulence and spread. The present paper aims to assess whether the current state of scientific knowledge allows testing of these functional hypotheses on the relationships of LULC change and mycobacterial infections. Based on an extensive mixed-methods literature review, we highlight gaps in current knowledge, discuss mechanisms of LULC effects on mycobacteria and their potential to emerge and spillover to human and other animal populations. Also, we highlight areas of future research efforts to better understand the relationships of changing environmental conditions and mycobacterial diseases.

2. Materials and methods

2.1. Study design

In this study, we employed a mixed-methods approach, integrating both a bibliometric analysis and a scoping review, to investigate the geographical and temporal trends of scientific literature that have addressed impacts of LULC change on human and other animal mycobacterial diseases and their etiologic agents. To do this, we (1) reviewed the existing literature on mycobacteria-LULC relationships, and (2) tested the reliability and availability of published data related to mycobacteria and LULC change. Inclusion criteria were: (1) peer-reviewed papers in indexed scientific journals; (2) papers with concise information on LULC change

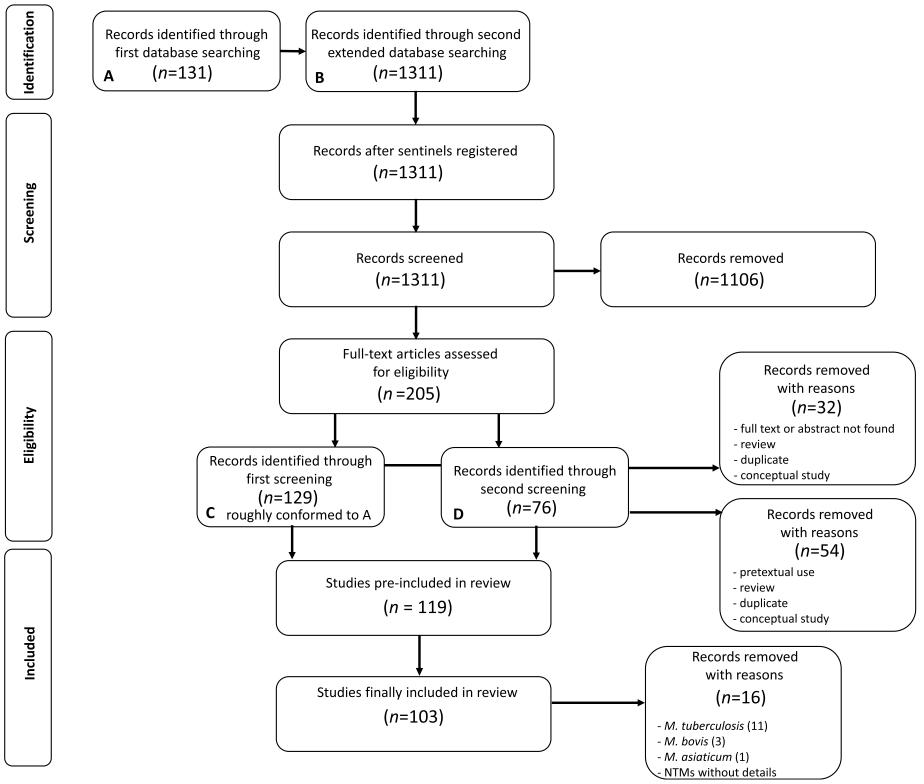

Figure 1. Flow chart diagram for article selection and data processing in the present mixed-methods review.

and mycobacteria; (3) empirical research, and (4) studies published in English, Spanish and French. Exclusion criteria were: (1) papers lacking original empirical data on mycobacterial-LULC relationships, and (2) reviews without original research. Article research was performed by two authors (J-FG and CEVC). Articles were then screened for eligibility via in-depth reading by three authors (J-FG, CEVC and CC) who also retrieved data from retained studies. Discussion and consensus were undertaken among these three authors in order to proceed to final selection.

2.2. Literature search

In April 2023, we searched the Web of Science Clarivate Analytics, PubMed-Medline and Scopus databases using a combination of search terms related to land use, land-use change, forestry and EIDs of the phylotype *Mycobacterium* spp., both non-tuberculous and tuberculous types, in humans and wild and domestic animals. The information was cross-checked to identify all original research articles published from 1950 to April 2023 (search ended on 25 April 2023). Search terms were: ('Mycobacter*' OR 'Mycobacterium tuberculosis' OR 'Mycobacterium ulcerans' OR 'non-tuberculous mycobacteria' OR 'NTM' OR 'Buruli ulcer' OR 'Tuberculosis' OR 'Leprae' OR "Mycobacterial disease) AND ('land use change' OR 'land cover change' OR 'deforestation' OR 'habitat alteration' OR 'agricultural expansion' OR 'urbanization' OR 'ecological disturbance' OR 'soil degradation' OR 'water resource management' OR 'land cover fragmentation' OR 'landscape ecology'). We first identified $n = 131$ article items (mainly scientific publications) in which one or several of these search terms were initially only present in the article title, summary, or introduction sections (figure 1, see box A). However, this first list failed to identify three specific publications that we had selected as sentinels of our bibliometric request, including: [31]; [32] and [33]. Brou *et al* [31] was used because it clearly matches with the scope of the bibliometric study; [32] is a modeling study that projects scenarios of soil microbial community composition in a context of global change without directly focusing on mycobacteria, and [33] discusses multi-host pathogens of zoonotic interest in wild rodents, but indirectly considers LULC change. Therefore, we extended the list of search terms including 'landscape' OR 'landscape diversity' OR 'landscape modification' OR 'landscape alteration' to capture these three specific publications, which led to a considerable increase in the number of selected publications ($n = 1{,}311$) (figure 1, see box B). Of this total, 1106 articles were excluded from the final database because they did not meet the selection criteria (figure 1). It was particularly the case for molecular and genetic or epidemiological papers using 'landscape' in their title, abstract or introduction (e.g. current landscape of microRNAs, landscape of vaccines). After this step, 205 publications were retained for a second, detailed evaluation of keywords and abstracts, and separated into two groups (figure 1). A first group ($n = 129$) corresponded to the original search (minus two non-eligible articles) (figure 1, see boxes A and C). A second group required a more careful analysis ($n = 76$) (figure 1, see box D). In the former group, additional in-depth reading led to selecting 97 publications ($n = 97$), falling perfectly within the scope of the review. In the latter group, a more stringent evaluation of each article resulted in retaining 22 scientific ($n = 22$) (figure 1). From this database ($n = 119$), seven publications on *M. tuberculosis* were withdrawn from consideration because they focused on environmental (mainly climatic and other more demographic or social) factors in urban settings, and not directly complying with LULC-mycobacteria interactions. For *M. bovis*, nine publications were withdrawn from further analysis since the terms '*M. bovis*' or 'infectious disease' were only given cursory mention in the abstract, introduction or discussion sections but were otherwise not treated in the paper. A final database of 103 records was used as a reference to initiate the bibliometric review (figure 1). The review was focused on the framework of LULC change and animal and human mycobacteria relationships, considering their spatial (i.e. local, regional, continental, global) and time (i.e. day, month, year, decadal) study dimension. The risk of bias was low in this final list of 103 retained articles. The list of selected papers is given with a brief description in supplementary table 1, and is available upon request. The analyses and figures were performed using Systat 13$^{\text{TM}}$ and Stata 15$^{\text{TM}}$.

3. Results and main bibliographic trends

3.1. Literature search tendency and temporal trend

Overall, from our paper selection process, 81 publications (78.6%) were empirical studies and 22 (21.4%) conceptual or theoretical. 39.8% ($n = 41$) were on *M. bovis*, 30.1% ($n = 31$) on *M. ulcerans*, 15.5% ($n = 16$) on *M. tuberculosis*, 6.8% ($n = 7$) on mycobacteria in the environment; the remaining involved several other mycobacteria (e.g. *M. microti, M. mungi,* MAP). Author numbers in publications ranged from one to 27 with a mean of 5.8 and a median of 5.0 (s.d. = ±3.81). Publications came from authors with affiliations from 15 Western countries (Australia, Belgium, Canada, Finland, France, Germany, Ireland, Italy, New Zealand, Portugal, Slovakia, Spain, Switzerland, United Kingdom, United States of America); 12 were from Africa (Benin, Botswana, Cameroon, Côte d'Ivoire, Egypt, Ethiopia, Ghana, Kenya, Republic of South Africa, Tanzania, Togo, Zimbabwe); eight were from Eurasia, East and Southeast Asia (Bangladesh, China, Indonesia, Iran, Mongolia, Pakistan, Saudi Arabia, Taiwan) and one from South America (Brazil). There was also a single publication from French Guiana, a French overseas department located northeast of the southern American continent. There were only six single author publications with all the other publications produced by authors representing nine nationalities from four continents.

Ten particular *Mycobacterium* species were analyzed in relation to LULC change (see figure 2 for an illustration of three of them, and table 1 for a presentation of the seven others). Four additional categories of mycobacteria were also considered: (1) NTMs in a diversity of soils with or without species characterization, (2) NTMs causing pulmonary syndromes in humans but with no species definition, (3) NTMs without any additional details, and (4) phylotype *Mycobacterium* corresponding with [32] (see table 1).

From a subtotal of 84 papers retained for presenting an interest in temporal trends (i.e. sufficient records of the same mycobacterial species for proceeding to temporal analysis), we analyzed the temporal trends in the article production for *M. tuberculosis, M. bovis* and *M. ulcerans* (figure 2). We only found enough papers addressing LULC change impacts on mycobacterial emergence for *M. bovis* and *M. ulcerans*, indicating an important increase in scientific publication records over time (figure 2). Although we found such an increase in research production, they made up a relatively small number compared to studies focusing on medical treatment, disease presentation and genomics on *M. tuberculosis. In aparte*, 36 840 publications have been published on *M. tuberculosis* from 1904–2023, with only 2.7% (i.e. 992) that considered ecological or evolutionary aspects, e.g. LULC-disease relationships, population dynamics or phenology. Current research on LULC change and mycobacteria is clearly dominated by papers on *M. bovis* and *M. ulcerans*.

3.2. Spatio-temporal scales of Mycobacterium records

The spatial distribution of publications on LULC change for the *Mycobacterium* species and categories is illustrated in figure 3. Studies on the effects of LULC change on *M. bovis* were from Europe, North America, Africa and Oceania, but there were geographical gaps, i.e. from Meso- and South America, Eurasia, South and Southest Asia. For *M. tuberculosis*, existing studies were represented from Europe, Africa and Southeast Asia, but were notably absent from other continents and regions. For MAP, rare studies were conducted in Europe and Oceania. For *M. ulcerans,* research was localized in West and Central Africa, Australia and South America. For *M. leprae, M. mungi* and *M. microti*, only one country was represented for each, i.e. Brazil,

Table 1. List of Mycobacterium species and NTMs retained in our systematic bibliometric review on land-use change and mycobacteria. Numbers indicate the number of papers published on this topic for a corresponding year. Letters (a): *Mycobacterium bovis* or *M. bovis*, and (b): *M. tuberculosis* or MTBC. This table is complementary to figure 1 which illustrates the number of publications produced over time for three other mycobacteria, i.e. *M. tuberculosis*, *M. bovis* and *M. ulcerans* on the same topic, and for which the numbers of publications is more substantial in authorizing an illustration.

	Mycobacterium taxa										
	M. microti	*M. mungi*	*M. leprae*	*M. orygis*	*M. avium paratuberculosis*	*M. caprae*	*M. asiaticum* in freshwaters	*Mycobacterium spp.*	*Mycobacterium* species in soils	*NTMs* without details	*NTMs* causing pulmonary syndroms in human
Year											
2003					1						
2004											
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2006											
2007											
2008											
2009											
2010											
2011					1				1 (a)		
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2013									1		
2014			1		1		1				
2015											
2016											
2017									1 (a, b)		
2018											
2019		1							2		
2020	1	1	1			1				1	
2021		1		1				1	1		1
2022	1								1		
2023											

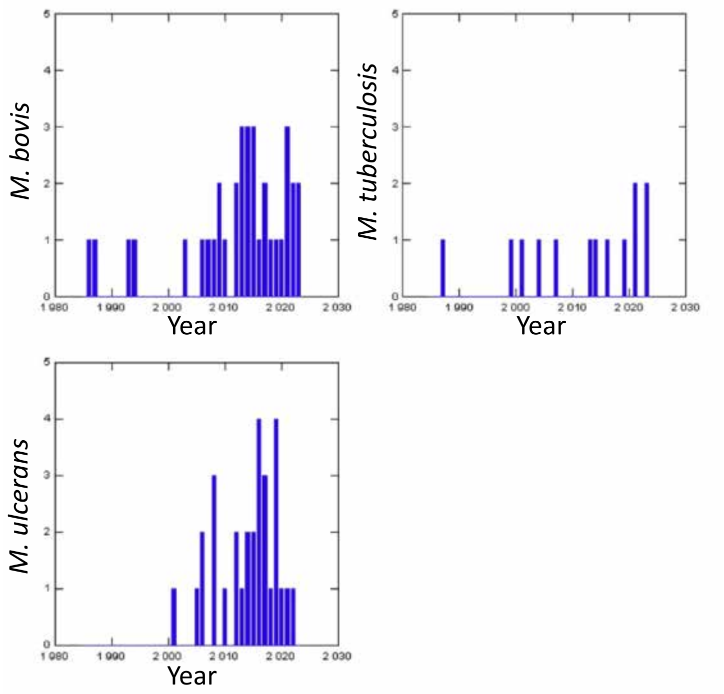

Figure 2. Bar chart illustrations of the number of scientific publications across time for three major *Mycobacterium* species, i.e. *M. bovis, M. tuberculosis* and *M. ulcerans*. For *M. bovis*, nine scientific publications were pretextual and for *M. tuberculosis* seven only analyzed bacilli transmission in urban landscape settings. See text for further explanations and abbreviations. Axes have been set to the same scales for comparison.

Botswana and the United Kingdom, respectively. Studies on mycobacteria discovery in soils were from Europe, North and South America, and China (see figure 3).

Based on our review, we are able to separate studies by spatial scale into three categories (figure 4(a)). A first group includes studies on *M. mungi*, MAP and *M. microti* with low spatial and time scales. A second group is formed by studies on *M. tuberculosis* and *M. bovis*, with local to national scales, and to which we can add a subnational scale of analysis (i.e. Bahia state and Brazil) on *M. leprae*. A third group is composed of studies on *M. ulcerans* and epidemiological studies on Buruli ulcer that consider larger spatial scales among investigations.

In terms of the temporal scale, approximately 55% of the reviewed studies represented inter-annual records (2–5 years), 26% intra-annual datasets (days, weeks, months) but only 19% included decadal-long datasets. These latter studies focused on *M. ulcerans* and cases of Buruli ulcer (figure 4(b)). For instance, as the only exception, a study by [34] on *M. ulcerans* in Australia used Buruli ulcer case data—initially acquired case data from 1939–2008. However, because many studies had incomplete data, only studies from 1981 to 2008 were used in [34] to model climate and LULC change for predicting disease dynamics.

Finally, statistical analysis and multiple regressions are two of the most used tools in LULC-mycobacteria studies, and mechanism-based models are extremely rare. There is a unique study that evaluated non-linear and complex processes among deforestation, change in local species communities, and the emergence of *M. ulcerans* [11].

4. Discussion

This mixed-methods review confirms that LULC change-mycobacteria relationships are generally poorly studied, despite the fact that the ancestral or more recent origin of these disease agents is environmental. It also highlights numerous research trends and gaps, with gaps both in the geographical distribution of studies and in the near impossibility of generalizing the LULC-mycobacteria relationships from studies most often conducted at very local scales. Further, very few studies evaluate both environmental mycobacteria and infected animal or human hosts in the same studies. The research developed on *M. ulcerans* and the skin disease it causes, Buruli ulcer, was found to be representative of interdisciplinary work within this group of pathogens, and on which future research on mycobacteria should be based.

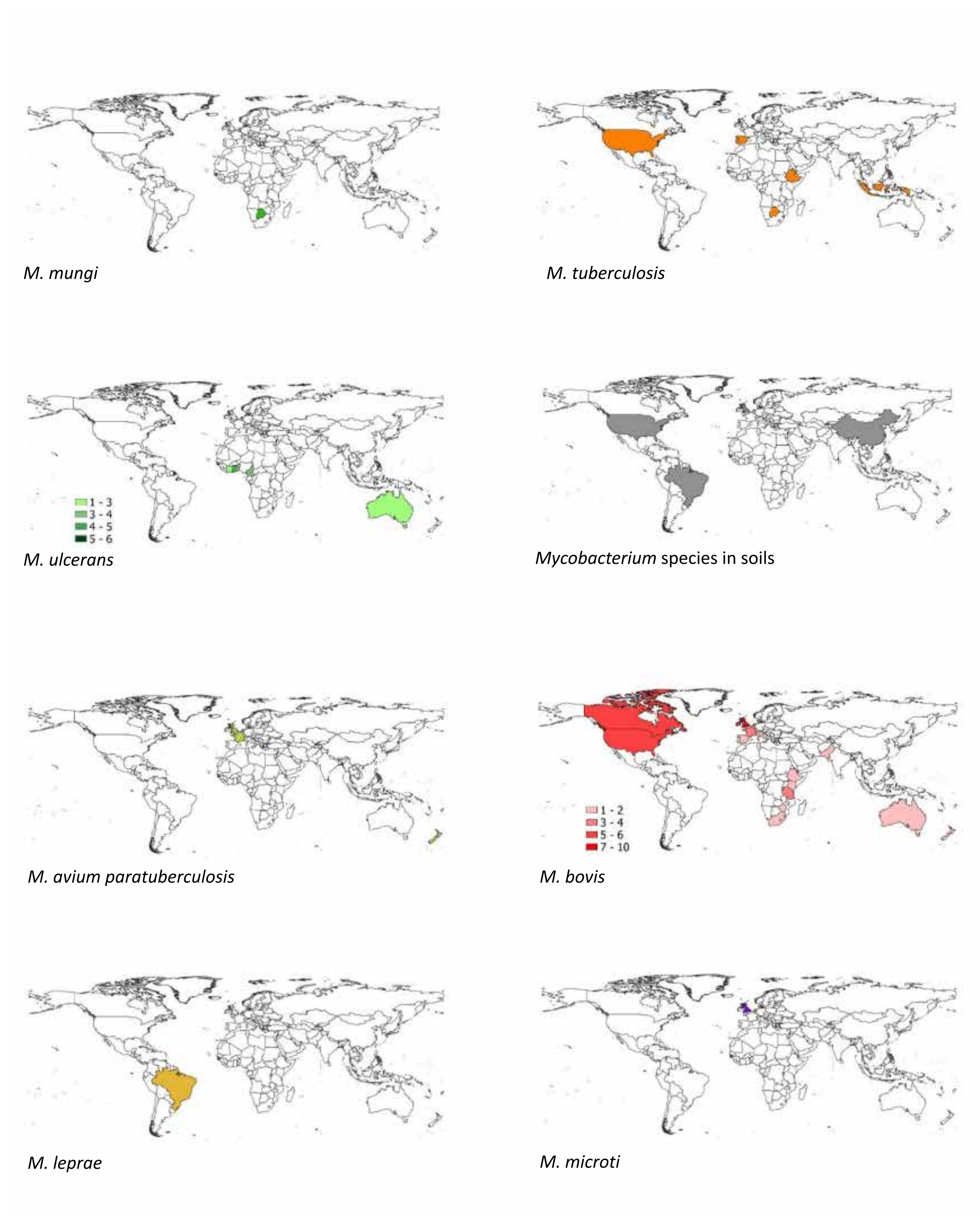

Figure 3. Geographical distribution of research studies that have actually taken place for the different mycobacteria species and categories analyzed in the present review on land-use and land-cover change-mycobacteria relationships. Scales, when available, give the number of published papers per countries. When no scale is illustrated for a *Mycobacterium* species or category, it means that only one paper per country was obtained on LULC-specific disease relationships. This is the case for MAP (only France), *M. leprae* (only Brazil), *M. microti* (only the United Kingdom) and *M. mungi* (only Zimbabwe). Concerning *M. tuberculosis* and *Mycobacterium* species in soils, corresponding subfigures show several countries for which research work has been identified but with one occurrence per country.

4.1. Regional matches and mismatches between LULC and emerging non-tuberculous and tuberculous infectious diseases

Based on the literature search, research on mycobacteria and the diseases they cause in animals or humans largely focuses on local and subnational scales. Regions from the economically richest countries (United States of America, Canada, Europe and Australia), produced more research on LULC changes and mycobacteria relationships. Several countries of sub-Saharan Africa have been well studied for different groups of bacilli. However, Meso- and South America (except Brazil and French Guiana) and South, Southeast Asia and Insulindia have been less studied (figure 3). Currently, the most vulnerable regions, such

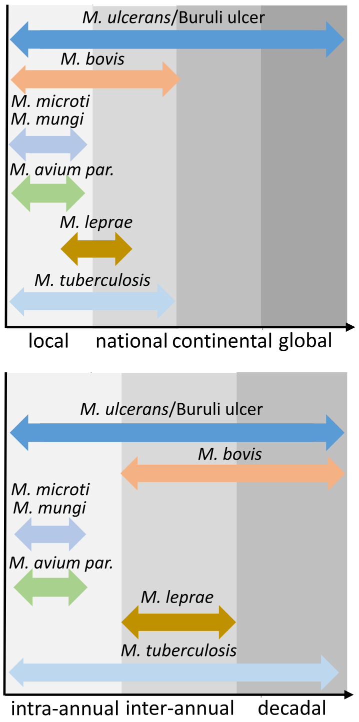

Figure 4. Schematic representation of the trend of different *Mycobacterium* species/land-use and land-cover changes relationships. Upper left: spatial-scale distribution of current studies from local to global scale. Lower left: time-scale distribution of the same studies from intra-annual to decadal timeframe.

as Western and Central Africa, to land-use transformation and notably deforestation of the rainforest [4, 35], continue to be neglected, except for *M. ulcerans*.

Gradual but persistent environmental changes, such as LULC and climate change, may cause shifts in the phenology of soil or water microorganisms [36]; thus, understanding the microbial ecology of soils is important to understanding mechanisms of LULC effects on potential mycobacterial pathogen spillover. LULC and climate change can have complex effects on biota, resulting in modified resilience to additional disturbances [32], but with unknown consequences for aboveground-belowground species' interactions and ecosystem functioning [19, 36]. It is well known that soil microbial communities respond to environmental changes, such as seasonal and medium-term climate dynamics, fire regimens, LULC change for agriculture development, soil pollution and freeze-thaw cycles [37].

In [32] the authors used a global bacterial and fungal database across six continents to estimate past and future trends in soil microbiomes. They used temperature and precipitation with land-use projections between 1950 and 2090 to analyze both direct and indirect effects with structural equation modeling. This work showed that local bacterial species' richness increased in all scenarios of climate and LULC changes. However, a general homogenization of bacterial communities was predicted for >85% of terrestrial ecosystems. Results indicated that the *Mycobacterium* species, belonging to a cluster of species denoted as A in this study, will become more abundant in communities responding to environmental changes [32]. Using these projections, and notably figure 4(c) and cluster A in [32], we reproduce here, in figure 5, a supporting scenario for 2015–2090. Ecological clusters composed of both fungi and bacteria are formed from a correlation network analysis [32] which determines four clusters (A–D), with mycobacteria belonging to cluster A), which are associated with areas with different temperature, precipitation, vegetation structure, and pH and carbon concentration soil conditions.

Results show a massive increase of cluster A soil bacteria across a range of regions, except in boreal areas, deserts, mountains, most of India and Southeast Asia, and Oceania. Several regions also have predicted losses of cluster A soil bacteria in Central Africa, the Great Lakes region of the United States of America, the Lao Republic, eastern Vietnam and several patchy areas of the Insulindia, and New-Guinea. In addition, net gains of *Mycobacterium* species associated with LULC and climate changes by 2015 [32]) are remarkable across continental Europe and central North America and more sporadically on the eastern coast. Net losses for cluster A can be identified in different and more localized regions of the world like the British Isles, Scandinavia, southeastern regions of the United States of America, and regions bordering the African Great Lakes.

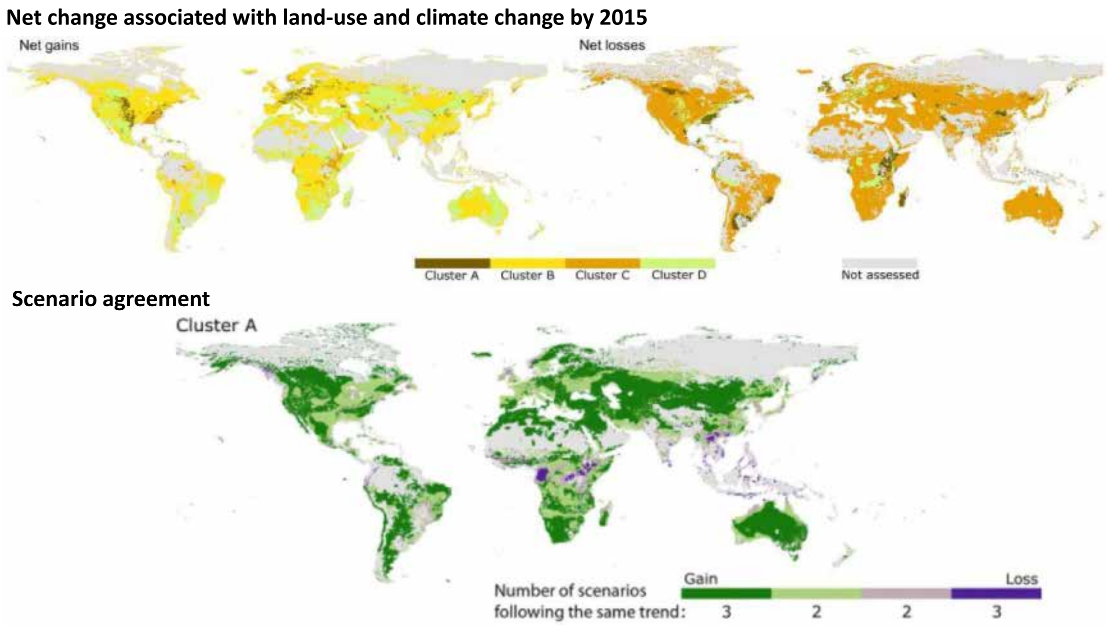

Figure 5. Upper panel. Global projections of soil bacterial and fungal species communities in the context of climate change and land-use transformation. Lower panel. Bacterial cluster A includes Actinobacteria with the phylum *Mycobacterium* spp. Adapted with permission from [32]. © 2021 The Authors. Global Ecology and Biogeography published by Wiley CC BY-NC-ND 4.0.

Comparing the projections of [32] and our mapping of LULC-mycobacteria relationships (figure 3), an important contrast in research foci and interests exists globally. Even for the two species with the most spatially distributed research, *M. bovis* and *M. ulcerans*, the spatial extent of studies retained in our review does not cover areas of mycobacterial emergence highlighted by [32]. Studies on the interactions between LULC and *M. bovis* are still rare, and research teams in North America and continental Europe should mobilize to better integrate research on soil bacteria and their potential pathogenicity to domestic animals. Interestingly, projections made in [32] for North America show central regions of the Mississippi and Missouri catchment areas to be at high risk for mycobacteria increases in abundance in soil bacteria communities. These findings support our recent detections of *M. ulcerans* and related mycolactone-producer mycobacteria in several watersheds of the southern United States of America (Dogbe *pers. comm.*). However, according to predictions in [32], *M. ulcerans* should become less common in areas of the world where it is currently highly prevalent in freshwater ecosystems, notably those of Western and Central Africa.

One important message that comes from our review is the need for stronger collaborations among disciplines that tend to operate in isolation, i.e. environmental microbiologists, health bacteriologists, ecologists and clinicians [30, 38]. New and stronger interdisciplinary collaborations, based on an ecosystem-based approach, considering in the same sites soil and water microbiology and potential infectious risk for animals and humans would be ideal and more advantageous for understanding environment-disease interfaces [38]. This would help identify possible known mycobacteria that have emerged in new areas of the world under LULC change, e.g. *M. canettii*, as well as currently undescribed bacterium ecological variant forms and taxa that could be responsible for causing new infections in humans and animals, e.g. lower infectious pneumonitis and cutaneous infections.

4.2. Do LULC and non-tuberculous and tuberculous mycobacteria relationships change over spatio-temporal scales?

From local to global scales, agreements on the response of mycobacterial diseases to LULC change are very difficult to determine, except for *M. ulcerans* and to a lesser extent, *M. bovis*. Despite a growing level of research and political interest in characterizing LULC effects on these tuberculous and non-tuberculous pathogens [8], very little empirical data are still available from the international literature. Both LULC change and mycobacterial disease outbreaks occur at multiple spatial and temporal scales. Studies of land transformation at large scales over decades (e.g. deforestation in the Brazilian Amazon), and LULC change at more local and short-term scales, produce different results of ecosystem and mycobacterial pathogen relationships, and thus, models of epidemic foci [30]. Whether associations between LULC change and EIDs in general are constant over space and time still remains unknown [8].

Except for *M. tuberculosis* and *M. leprae,* most mycobacterial diseases are not communicable and do not spread from individual to individual; but even for *M. leprae* there are questions that direct transmission may not always be the only means of infection [39–41]. For most mycobacteria with pathogenic outcomes in humans and other animals, small spatial scale land transformations and their local specificities may be important to their emergence. However, the scarcity of scientific studies does not allow us to understand, or predict, if and how the spatial extent of LULC change, i.e. from local to more global, influences mechanisms of new epidemic foci emergence.

Most research on mycobacteria and LULC change has been conducted over short time scales (e.g. 1–4 years), which is the most common duration of research in these fields, and most often conducted to answer animal or human health questions, with much less interest on environment-health relationships. In general, long-term studies spanning multiple decades are very rare, and near unique only for *M. ulcerans* [11, 34, 42], *M. tuberculosis* [43] and *M. bovis* [44]. Research on *M. ulcerans* and Buruli ulcer accomplishes a continuum of spatiotemporal scales. These several studies have allowed important insight into the impacts of LULC change, demonstrating the driving role of deforestation, for example in Cameroon, Côte d'Ivoire and French Guiana, on the ecology of this disease agent and its transmission to exposed individuals [8, 11, 45].

4.3. Do LULC and non-tuberculous and tuberculous mycobacteria relationships change over specific types of land-use transformation?

We may be able to make inferences from studies of forest ecosystem loss in temperate and boreal regions whose soils constitute the origin of mycobacteria [46], and where these studies show increases in NTM infections, and notably actinobacteria, for animals and humans (see figure 5). This assertion is commensurate with projections made in [32]. It was reported that deforestation, compared to that of agricultural land, was more largely associated with *M. ulcerans* increases in French Guiana [11]; deforestation and forest fragmentation severely disrupting the dynamical equilibria of these ecosystems, and in particular those of microbe-soil or -water interactions. Studies in Ghana have also shown that proximity to mining sites is associated with higher Buruli ulcer prevalence [47]. It has also been suggested that an interaction between natural land cover and human mediated landscape changes are important to the ecology of Buruli ulcer [48]. Impoundment by a dam in Cameroon was implicated in the emergence of Buruli ulcer [49]. One study showed that the central region of the Côte d'Ivoire had the highest occurrence of Buruli ulcer cases; it is a region with abundant hydro-agricultural dams, which are mainly used for agricultural activities like semi-intensive rice farming and banana production [31]. In Australia, from 1990 an important change was observed in the epidemiology of Buruli ulcer in the Victoria province with most of the cases observed within a very small region surrounding the development of a golf course and a shallow lake in an area previously dominated by wetlands [50]. These studies indicate that land-use types (e.g. the conversion of rainforests into land for abattis, rice fields, palm trees or crops such as maize) are important to the temporal aspects of habitat change, and need increased consideration for mycobacterial diseases throughout the world.

Interestingly, a past study examined the impact of the introduction of a non-native woody species, *Prosopis juliflora*, on the occurrence of bovine tuberculosis in cattle and revealed that the proportion of *P. juliflora* is a significant contributing factor to the increased prevalence of bTb in cattle. The higher prevalence in these areas can be attributed to the decline in the diversity of the host species and the increased movement of cattle resulting from the scarcity of palatable grasses in *Prosopis*-infested regions [51]. Another recent investigation [52] demonstrated that plant community diversity and composition was significantly different between areas of high and low Buruli ulcer endemicity, suggesting that local environmental changes affecting the plant species communities may be important for predicting the ecological conditions most suitable for the disease agent *M. ulcerans*. Such work supported previous research showing that *M. ulcerans* was found more often on certain types of aquatic plant species than others [53]. There have been additional country-wide studies that corroborate local and regional landscape processes which are statistically associated with Buruli ulcer disease [48, 54]. Taken together, these local and country-scale level studies indicate that LULC changes affect Buruli ulcer risk at multiple scales; however, additional quantitative studies are needed to test these relationships with plausible sources of transmission [13, 45], knowing that different socio-environmental contexts might also favor different transmission pathways.

The cool, wet and acidic soils associated with peatland and peat-rich boreal forests are considered to be hot spots of mycobacterial diversity [46, 55]. Changes of forest to shrub land, forest to grassland, shrub land to grassland, and grassland to degraded grassland were described broadly for mycobacteria [56]. This indicates that deforestation of primary tropical and sub-tropical forests in Western and Central Africa, southern America and southern Australia, could clearly play a major role in the emergence of mycobacteria. It would be interesting to know how specific tuberculous and NTM respond to these different scales and directions of LULC changes [57]; and in particular to study the role of different types of crops on the preferential development of mycobacteria in soils and peripheral water. Another study suggests that mycobacteria are sensitive to land disturbance and are a core genus in forest ecosystems in the cycling of soil nutrients [56]. Small-scale changes in agricultural sites have been broadly investigated for bacilli in Africa, but not specifically for mycobacteria [58]. These studies indicate that spatial and temporal LULC specificies, e.g. types of land transformation, are important to consider for a more comprehensive understanding and future ability to manage mycobacterial emergences.

4.4. Mycobacterium taxa of soil

Considering the origin of pathogenic mycobacteria is obviously crucial with regard to LULC change, and also requires a more interdisciplinary mindset than just looking at human or animal diseases. A worldwide survey on the genetic diversity and abundance of soil-dwelling NTMs reported their presence in almost 143 soil types representing worldwide climatic, chemical, and geographical gradients [27]. This research reported higher mycobacterial abundance and genetic diversity in cooler, wetter, and more acidic soils, and demonstrated many more soil-dwelling species than the 190 currently described (representing only 3% of the phylotypes detected in this soil survey). When comparing the worldwide distribution of soil-dwelling NTMs relative to other dominant soil bacterial groups, *Mycobacterium* stands as the 12th most represented genus among all soil samples (top 3% of bacterial genera) [27]. A larger worldwide survey of soil bacterial communities evaluated the effects of vegetation cover (grasslands, forests), climatic areas, and 15 chemical characteristics (e.g. *pH*, humidity, abundance of K, N, P elements or C/N ratio) on the species assemblage present in soils [59]. Such an association of *Mycobacterium* and soil characteristics is commensurate with regional scale distributions of NTMs and soils. For instance, *M. abscessus* was present in 27% of Hawaiian soils with a positive correlation reported between NTM abundance in soils and different abiotic factors (pH, water-balance, and Fe-oxides/hydroxides concentration) [60]. In Ethiopia, there were significant correlations between the relative abundance of different soil NTM species and altitude [61]. Altogether, these results suggest that the distribution of most soil-dwelling NTMs is predictable at different geographical scales and using multiple molecular markers.

Several studies found that specific NTMs shared physiological traits for human intracellular infection, and such information may inform assessments of their environmental distribution and responses to ecological perturbations [21, 62]. First, their common preference for acidic and micro-aerobic habitats aids switching from environmental to host intracellular habitats. Second, NTMs share tolerance across a wide range of temperatures (0 °C–55 °C), desiccation, and starvation conditions. Other shared physiological traits demonstrate trade-offs of structure and composition of the mycobacterial cell wall. This cell wall includes long chain fatty acids (C_{50}–C_{80}) making NTM cells highly hydrophobic, facilitating their attachment to many surfaces as well as their aerosolization (hence inhalation by mammals, including humans) at air-water or soil-air interfaces. The production of such long chain fatty acids likely explains the common mycobacteria ability to efficiently metabolize complex organic molecules (e.g. humic and fulvic acids, molecules with aromatic motives). However, production of long chain fatty acids has a physiological cost resulting in slow growth rates. This may explain the ability to efficiently metabolize complex organic molecules that are common in mycobacteria, but rare in other bacterium phyla. Understanding these mycobacterial characteristics from environmental distributions and adaptations will help inform how some non-pathogenic and pathogenic ancient or new mycobacterial taxa may respond to LULC change.

Diverse observations are congruent with the expectations driven by assuming NTMs as K-strategists and other bacteria as *r*-strategists [63]. For instance, manipulating the intensity of grazing in the Pampa grassland of Brazil showed that moderate and high grazing intensity significantly increased, relatively to control, the abundance of mycobacteria in soils compared to many bacterial groups on a multi-decadal time scale [64]. Similarly, NTM abundance in forest soils significantly increased after diverse ecological perturbations (e.g. fire, windthrow and extraction of wood mass) relative to nearby preserved forest areas [65]. Conversely, comparing changes in rhizosphere communities between undisturbed control and one-hour heated soils demonstrated that although NTMs were less sensitive to heat than many other bacteria, the heat treatment was too high so the efficiency of recolonization by fast growing bacteria (*r*-strategists) overwhelmed the maintenance of NTMs in the heated rhizospheres [66]. According to [67], peat-rich soils and natural waters are well documented sources of NTMs driving the emergence of NTM lung disease.

M. tuberculosis clones also display soil survival with expected variability among clones supporting the epidemiological importance of temporary soil reservoirs. However, such effects are likely minimal in *M. mungi* epidemiology since its transmission among mongoose animals primarily relies on direct contact between the nose and anal glands involved in the chemical communication of these social animals [68]. By contrast, survival in temporary soils is a key determinant in interspecific transmission of *M. bovis* among susceptible animal species [69]. For instance, *M. bovis* circulation in the United Kingdom involves two primary hosts: European badgers, *Meles meles*, and cattle. These hosts rarely have direct contact with each other, making interspecific bacterial transmission mediated by soil reservoirs and badger latrines. A similar scenario is suspected in Michigan (United States of America) between cattle and local deer species [70]. However, *M. bovis* survival in Michigan soils may be low since an extensive survey aimed at characterizing the local temporary reservoir soils failed to detect any environmental *M. tuberculosis* [70]. *M. tuberculosis* cell survival within temporary soil reservoirs likely depends on soil properties, but also increases up to one year in the presence of urine and feces, so that sewage stations represent serious and long-lasting infectious sources for waste workers and local human communities [71]. This raises the possibility that variation in soil composition and organization of latrines may have contributed to the absence of a correlation between tuberculous prevalence and human population sizes in medieval Denmark [72]. Furthermore, a recent *M. leprae* phylogenomic analysis found that human and animal infections rely on common infectious sources with unknown soil organisms likely acting as intracellular reservoirs [39]. Such a scenario may explain the reduced likelihood of person-to-person *M. leprae* transmission relative to alternatives such as arthropod-borne transmission [39].

4.5. A comparative and retrospective vision for understanding new mycobacterial emergence

From a historical perspective, extensive changes in human ecology from the Pleistocene to the present through controlled use of fire has been considered to have created ideal conditions for the emergence and increased exposure of humans to the natural reservoir of ancestral tuberculosis agents, likely from soil [73]. In Cameroon, ecological habitat transformations and new cultivation practices used bush fires during the late 1980s; people said that Buruli ulcer disease (due to *M. ulcerans*) appeared after the beginning of these bush fires for agriculture development [74]. Such information suggests that plant cover or species diversity changes may directly or indirectly be associated with increased human exposure to *M. ulcerans* (and other soil mycobacterial agents) through burning associated with agriculture expansion [74]. Epidemiological, evolutionary genetics, paleoanthropological and anthropological findings support this plausible scenario [73, 75]. In general, these studies suggest a need for additional, multidisciplinary avenues of inquiry to better understand the potential of new or more extreme emergences of mycobacterial infections with global LULC and climate change. We argue that there is a need to go beyond traditional clinical studies of mycobacterial disease (that dominate the literature) to include a broader understanding of more complex ecological relationships and evolutionary histories of this important group of environmental bacteria that often opportunistically affect humans and wildlife [19].

The ubiquity of pathogenic mycobacteria in soils and waters throughout the world should raise new insight and interest associated with mycobacterial infection risks in humans and animals. Asthma, an atopic epidemic in western societies, has raised the hypothesis that a decrease in human contact with soil bacteria that convey immune-modulatory properties may have caused a dysregulation of the immune system involved in such diseases [75]. Given the proven immunomodulatory potential of many mycobacteria, frequent contact with NTMs—which has been constant throughout human history given the ubiquity of soil-dwelling NTMs—could be important for improving human immunity to such pathogens [33]. As research in this area moves forward, the disciplinary silos that persist in current infectious disease research will likely erode, providing important interdisciplinary solutions to global infectious disease problems. Finally, there is an urgent need for the development of empirical studies to better clarify the trends in the risk of LULC-mycobacterial disease outbreaks and EID events.

5. Final remarks and recommendations

This mixed-methods review has revealed several important considerations for research progress related to mycobacterial diseases and projected changes in local and regional land transformation and global changes. Our review covered 10 different *Mycobacterium* species plus four categories of NTMs analyzed with what was published on LULC changes. Results showed clear evidence that there are very few studies at multiple spatial and temporal scales to understand LULC-mycobacteria relationships, especially in some of the most vulnerable areas of the world. Most landscape ecology and LULC research has been on *M. bovis* and *M. ulcerans*, demonstrating disparate disciplinary foci regarding mycobacterial pathogen diversity. Of the four hypotheses cited in the introduction to explain the links between LULC change and mycobacteria, it appears from the present review that none of them are sufficiently supported by empirical evidence. In this respect, the work on *M. ulcerans* and Buruli ulcer is exemplary and tends to support the first and third hypotheses, i.e. shifts in community diversity and composition, and natural habitat conversion. Several studies comparing different soils and their diversity and composition of mycobacteria tend to support the second hypothesis, i.e. shifts in soil- or water-environmental conditions. However, these studies are comparative in nature and thus do not account for longer term LULC and related changes in the composition and organization of mycobacterial communities. Finally, empirical work is clearly lacking for testing the fourth hypothesis, i.e. new selective pressures and genomic evolution potentially driven by LULC change. Thus, there is a need for additional research on tuberculous and NTM taxa within the context of landscape ecology and pathogeography, where new information will likely provide additional perspectives into disease prevention and risk management.

In addition, concerning *M. tuberculosis*, it appears that very few studies interpret how either LULC change has been historically a promoter of the emergence of this pathogen, or that LULC modifications allow pathogen persistence in these habitats and their retransmission to animals or humans. In the Melbourne area, Australia, a combination of environmental factors driven by European colonizers during the XIXth century and the local ecology led to tuberculosis in the human population [76]. Both human-driven changes to the local Melbourne landscape and the effect, in terms of epidemics due to human population size and density, combined to foster high levels of infections in a region that was once free of these organisms [76]. Beyond these features, resident practices, e.g. golf venues and drainage systems, contributed to the tubercular environment, and livestock production in the area established new conditions for bovine tuberculosis [76]. This explanation is reminiscent of the one described today in the same Melbourne region, but for another *Mycobacterium*, *M. ulcerans*, causing Buruli ulcer in humans [50].

Our review and supporting analyses suggest responses in *Mycobacterium* species diversity and abundance to landscape change will depend on the geographic region and temporal scale of research interest. Similarly, elucidating the mechanisms' associated relationships between mycobacteria responses to LULC change (i.e. the relative weight of the four hypotheses proposed in the introduction) will depend on the *Mycobacterium* species of interest. Indeed, *M. mungi* and MAP have mostly been studied at small spatial and time scales, while *M. ulcerans* literature is better represented by larger spatio-temporal scales. It is also clear that multi- and interdisciplinary approaches to studying mycobacteria pathogen emergence and transmission to humans and other animals is needed [38]. For example, studies of genomes and phylogenomic relationships among mycobacteria, combined with ecological studies that evaluate environmental existence and responses to change will provide novel insights into predicting disease emergences across spatial and temporal scales [77]. Such an approach can help place collective infectious disease research outside of historically-separated conventional disciplines to establish new collaborative networks that can help address some of the grand challenges associated with predicting disease responses to global, regional, and local changes [30].

One limitation of our work concerns the bibliometric search insofar as it appears to be sensitive to the keywords used. In fact, keywords such as landscape, for example, are used in different search fields and have absolutely nothing to do with the theme dealt with in this work. We may not have selected all the articles dealing with LULC change–mycobacterial disease relationships, but we are confident in the bibliometric results obtained.

The main strength of our study lies in demonstrating that this topic receives little or no attention in current research, despite the fact that a very large number of mycobacterial species are present in the environment. It also shows that the One Health approach is not being applied in practice, as some of us have already demonstrated [8]. Weaknesses include an obvious lack of interdisciplinary research, which should be carried out on research objects in places and subject to environmental pressures from human activities, rather than on research subjects using the monodisciplinary approach which is now showing its limits.

This review demonstrates both trends and gaps in current knowledge of LULC relationships with the occurrence and spatial distribution of non-tuberculous and tuberculous mycobacteria in soils and waters. Based on this review of the literature and additional analyses, we suggest several avenues for future research:

1. New information is needed on the survival of NTMs and MTBC in soil and water, and their spatiotemporal persistence and dynamics with LULC change. No study has explicitly addressed the survival of these bacilli and the driving factors influencing their ecology, evolution or potential to emerge from natural environments to human and wildlife populations. Therefore, there is an urgent need to delineate their ecological niche under laboratory, field and modeling conditions [78, 79] in order to gain a better understanding of the mycobacteria-LULC relationships, and their potential pathogenicity in humans and animals.
2. We know little about the potential for environmental modifications to impact mycobacterial pathogen prevalence at different biogeographic scales, as these disturbances are known to contribute to underlying population genetic variation and may also impact host–parasite interactions.
3. A deeper understanding is needed of the role of LULC change in the emergence and spread of ancient and future mycobacterial diseases, and the characterization of LULC types and intensity of change that are associated with higher risk for disease emergence in animals and humans [80]. Additional needs include the implementation of preventive measures to potentially reduce or eliminate new risks of infection, especially in areas of the world with rapid changes in LULC.

4. There is a need to ascertain the full pathogen (both MTN and MTBC) occurrence and concentration in soil and water, and potential pathways and modes of transmission through direct contact or indirect contact via LULC change in specific habitats shared by animals and humans.
5. Through interdisciplinary research, additional medical literacy about mycobacteria should also include information on their ecology and evolutionary history to better understand where they came from and what they are capable of in terms of current and future risk management in the global context of massive LULC changes.
6. There is an important need to improve surveillance methods of environmental-, veterinary- and clinical microbiology research to track the occurrence, diversity, and abundance of these bacteria from terrestrial and aquatic environments within the context of global LULC changes. Additional longer-term and interdisciplinary studies are needed for many other mycobacteria known to cause infectious disease.

Data availability statement

All data that support the findings of this study are included within the article (and any supplementary information files).

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Conflict of interest

The authors declare that they have no conflict of interest.

Authors' contributions

JFG and MEB are responsible for the conceptualization of this scholarly review paper, for formulation and evolution, and for writing the initial draft. They are also responsible for leadership and coordination of contributions by other coauthors. JFG, CEVC and CC did the bibliometric analysis and synthesis review of the scientific literature. JFG, CEVC, CC, AS, MD, KMF, JP, AR, MWS, MES, HJ and MEB contributed to writing previous and final drafts through critical review, substantial additions in sections and commentary.

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