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Two decades of tuberculosis surveillance reveal disease spread, high levels of exposure and mortality and marked variation in disease progression in wild meerkats

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Abstract

Infections with tuberculosis (TB)-causing agents of the *Mycobacterium tuberculosis* complex threaten human, livestock and wildlife health globally due to the high capacity to cross trans-species boundaries. Tuberculosis is a cryptic disease characterized by prolonged, sometimes lifelong subclinical infections, complicating disease monitoring. Consequently, our understanding of infection risk, disease progression, and mortality across species affected by TB remains limited. The TB agent *Mycobacterium suricattae* was first recorded in the late 1990s in a wild population of meerkats inhabiting the Kalahari in South Africa and has since spread considerably, becoming a common cause of meerkat mortality. This offers an opportunity to document the epidemiology of naturally spreading TB in a wild population. Here, we synthesize more than 25 years' worth of TB reporting and social interaction data across 3420 individuals to track disease spread, and quantify rates of TB social exposure, progression, and mortality. We found that most meerkats had been exposed to the pathogen within eight years of first detection in the study area, with exposure reaching up to 95% of the population. Approximately one quarter of exposed individuals progressed to clinical TB stages, followed by physical deterioration and death within a few months. Since emergence, 11.6% of deaths were attributed to TB, although the true toll of TB-related mortality is likely higher. Lastly, we observed marked variation in disease progression among individuals, suggesting inter-individual differences in both TB susceptibility and resistance. Our results highlight that TB prevalence and mortality could be higher than previously reported, particularly in species or populations with complex social group dynamics.

Long-term studies, such as the present one, allow us to assess temporal variation in disease prevalence and progression and quantify exposure, which is rarely measured in wildlife. Long-term studies are highly valuable tools to explore disease emergence and ecology and study host–pathogen co-evolutionary dynamics in general, and its impact on social mammals.

KEYWORDS
individual life-history trajectories, long-term surveillance, meerkats (*Suricata suricatta*), South Africa, tuberculosis (*Mycobacterium tuberculosis* complex), wildlife disease

1 | INTRODUCTION

Pathogens, particularly those causing emerging diseases, can be a major driver of mortality in wildlife (Fereidouni et al., 2019; Fisher & Garner, 2020; McCallum, 2008), which can be aggravated by changing environments and climate change (Dwyer et al., 2020; Paniw et al., 2022). Cross-sectional studies are often employed to study patterns of pathogen prevalence and transmission, yet their power to assess long-term patterns of pathogen emergence, dynamics and overall pathogen prevalence are limited (Reis et al., 2021). The advantage of longitudinal surveillance studies rest in their ability to identify factors underpinning pathogen transmission, assess the impact of pathogens on individual hosts and populations, and track host–pathogen dynamics (Ryser-Degiorgis, 2013; Walton et al., 2016; Watsa and Wildlife Disease Surveillance Focus Group, 2020). Since long-term projects are difficult to sustain and pathogen monitoring and detection can be challenging (reviewed in Thomas et al., 2021), such long-term data sets populations are still exceptional in natural (McDonald et al., 2018; Patterson et al., 2017).

Pathogens of the *Mycobacterium tuberculosis* complex (MTC), the causative agents of tuberculosis (TB), are among the most significant emerging pathogens globally (CSFPH, 2019). Generally, *Mycobacterium* infections are chronic, progressive and characterized by prolonged latent (i.e. non-infectious) or subclinical (i.e. without clinical signs but potentially still infectious to others) periods (CSFPH, 2019; Dwyer et al., 2020; Houben & Dodd, 2016; Jolma et al., 2022). If the disease progresses to the development of overt clinical signs, their onset is usually followed by health decline and death (Alexander et al., 2010; Fairbanks et al., 2015). A wide variety of mammals can be infected with MTC bacteria (CSFPH, 2019; Reis et al., 2021), which can easily be transmitted between multiple hosts species, with implications for entire ecosystems (Hardstaff et al., 2014; Michel et al., 2006). Thus, a well-founded understanding of transmission and host–pathogen dynamics is imperative for the development of adequate TB surveillance and management strategies (de Lisle et al., 2002). However, despite intense research efforts in several TB host systems (reviewed in Reis et al., 2021), quantitative data on TB exposure, prevalence and progression are still rare for many wildlife species affected by TB.

Meerkats (*Suricata suricatta*) are highly social, cooperatively breeding small carnivores (Clutton-Brock & Manser, 2016) native to southern Africa, a hotspot region for TB in wildlife (Michel et al., 2006; Tanner et al., 2015). Within the Kalahari Meerkat Project (KMP; Clutton-Brock & Manser, 2016), a natural population has been routinely monitored since 1993. First evidence of infections with *Mycobacterium suricattae*, previously believed to be *M. bovis*, was reported in the late 1990s (Drewe, Foote et al., 2009; Parsons et al., 2013). Meerkats have a complex social system characterized by small to medium sized social groups (2–50 individuals). Reproduction is largely monopolized by dominant breeding pairs (Clutton-Brock & Manser, 2016), which differ from subordinates in physiology (Smyth et al., 2018; Young et al., 2006), social network position (Drewe, 2010) and mortality risk (Cram et al., 2018). Social interactions within and between group are frequent (Clutton-Brock & Manser, 2016), making meerkats an excellent model to investigate pathogen transmission in a social context (Drewe, 2010).

Meerkats are a particularly good model system to investigate TB epidemiology because transmission, progression, and pathology of *M. suricattae* infection mirrors patterns reported in other wildlife hosts: Transmission occurs mostly via aerial routes or potentially bite wounds, and is thus linked to social interactions (Drewe, 2010; Drewe et al., 2011; Gallagher & Clifton-Hadley, 2000). Infections are initially characterized by long latent or subclinical periods (Drewe et al., 2011; McDonald et al., 2018; Tomlinson et al., 2013), followed by rapid progression to terminal stages upon onset of clinical signs (Alexander et al., 2010; Fairbanks et al., 2015). Typical signs of TB infection, including lymphadenopathy, particularly of submandibular, inguinal and cervical lymph nodes, and physical deterioration 1990s (Drewe, Foote et al., 2009; Parsons et al., 2013), usually develop ~12 months post infection (Donadio et al., 2022). After progression to clinical TB, meerkats usually become terminally ill with open lesions at affected lymph nodes and die within several months, with no recovery once clinical stage was reached (Patterson et al., 2021). Thus, TB was reported to impact life history and survival, and can lead to group extinctions (Duncan et al., 2021), a pattern aggravated by climate change (Paniw et al., 2022).

Despite some detailed knowledge gained from previous cross-sectional studies regarding TB in meerkats (Table 1, S1), the dynamics around its spread across the population, and how TB disease manifests over a meerkat's lifetime, remain obscure (see Table S1). To fill these gaps, we leverage an extensive long-term data set with over 25 years of detailed individual data ($n = 3420$ individuals) to examine TB spread across the population. Specifically, we quantify TB exposure,

TABLE 1 Previous studies on *Mycobacterium* infections in wild meerkats in the Kalahari, South Africa with highlighted key findings

Reference	Study type	Data collection	Sample size	Key findings
Alexander et al. (2002)	Pathogen identification	1998/1999	*N* = 20	Immigration of TB afflicted individual followed by development of signs and death/ disappearance of all group members
Drewe, Foote et al. (2009)	TB pathology	2005–2007	*N* = 57	Detailed pathology of 52 individuals euthanized for TB, lesions in multiple organ systems
Drewe, Dean et al. (2009)	TB detection assay validation	2005–2007	*N* = 240	Combining multiple diagnostic tools increases TB detection reliability
Drewe (2010)	TB transmission study	2006/2007	*N* = 110	TB transmission is impacted by social network parameters. Giving grooming and receiving aggression main risk factors
Drewe et al. (2011)	TB transmission study	2006/2007	*N* = 134	TB transmission impacted by social network parameters. Exposure driven mainly by grooming interactions
Parsons et al. (2013)	Molecular description of TB	Not reported	*N* = 4	Identification and description of *M. suricattae*, closely related to *M. mungi* and dassie bacillus
Clarke et al. (2016)	TB detection assay validation	2014/2015	*N* = 108	IP-10 release assay as viable method of TB detection in meerkats
Patterson et al. (2017)	TB transmission study	2001–2015	*N* = 2388	TB infection risk increased by older age, group history of TB, prior immigration into social group
Patterson et al. (2021)	TB detection assay validation	2014–2016	*N* = 268	TB infections double mortality risk; combining multiple diagnostic tools increases TB detection reliability
Patterson et al. (2022)	Intervention study	2014–2016	*N* = 135	Vaccinations of high contact individuals or highly susceptible individuals reduces TB infection rates
Duncan et al. (2021)	TB effect on demography	1993–2019	*N* = 98 groups	TB linked to group failure, prior immigration into social group increases TB risk
Paniw et al. (2022)	TB effect on demography	1997–2018	*N* = 2691	Impact of adverse climate exacerbated by TB, prior immigration into social group increases TB risk
Donadio et al. (2022)	TB detection assay validation	1998–2018	*N* = 66	Detection of *M. suricattae* from faeces viable, marked variation in disease progression
Present study	Quantification of TB	1993–2020	*N* = 3420	High levels of TB exposure, marked variation in TB progression

prevalence of clinical TB, and mortality, provide timelines of typical disease progression in individuals and groups and describe inter-individual and temporal variation in TB progression. These findings provide crucial context by which to understand TB epidemiology and ecology.

2 | MATERIALS AND METHODS

Data used for this study was collected within the KMP on wild meerkats living at the Kuruman River reserve (26°580’S, 21°490’E), Northern Cape, South Africa, between the 20 October 1993 and the 29 December 2020. During this time, several meerkat groups well habituated to human observers were visited multiple times a week and detailed data on individually recognizable meerkats collected by trained volunteers and researchers following standardized protocols (for details see Clutton-Brock & Manser, 2016). We included data on all 3420 individuals encompassed in the study for individual level analyses. As it is common for newly founded meerkat groups to quickly fail (Duncan et al., 2021), we limited the analyses on the group level to 91 groups with a group duration longer than 6 months.

For each individual, we recorded first date as birthdate or first entry date into the population and final date as death date or last observed date as well as available information on clinical signs of TB infection (see Table 2 for definitions). Individuals can be infectious even prior to the onset of clinical signs (Donadio et al., 2022; Wilkinson et al., 2000), and exposure can vary with social behaviour (Drewe, 2010; Drewe et al., 2011), so exact time of exposure for each individual cannot be easily determined without elaborate TB diagnostics (which is not available for the majority of the study population). To use a conservative and clearly identifiable estimate for TB exposure, individuals were classified as TB exposed from the first day of cohabitation with conspecifics displaying externally visible signs of clinical TB, as individuals with overt signs likely contribute the most to TB transmission (McDonald et al., 2019). Exposure could thus occur either by clinical TB emerging in the resident group or by individuals being born or

TABLE 2 TB-related terminology and definitions of life-history parameters used in the present study

Term	Definition
TB classification	
Exposure	Meerkats are classified as exposed to TB if they (a) are cohabiting with an individual expressing clinical signs of TB and (b) are born into a group with past or ongoing TB history. The exposed status does not change if individuals, e.g. emigrate and enter a TB-free group; rather it signifies a high likelihood that individuals had contact with *M. suricattae* at some point in their life. Exposed individuals may be infected, but do not (yet) express clinical signs.
Clinical TB signs	Meerkats are classified as clinical TB cases from the first day they express clear signs of *M. suricattae* infection, specifically lymphadenopathy of submandibular, cervical or inguinal lymphnodes, or when they were classified as displaying TB signs by field observers. Individuals with clinical TB are exposed by definition.
Group TB	Past or present cases of clinical TB within the respective group. Based on the long subclinical phases, individuals in groups with past, but not current clinical TB, are considered exposed to TB. Current clinical cases are not required for a group to be defined as a group with TB cases.
Susceptibility	The time taken for an exposed meerkat to develop clinical signs of TB, used as proxy of individual susceptibility to *M. suricattae* infection upon exposure. While disease transmission is determined by both exposure and susceptibility to a pathogen, the susceptibility term here encompasses both mechanisms, so 'exposure' can be used to describe whether individuals are considered in contact with *M. suricattae*.
Resistance	The time-period between development of clinical signs and death, representing the capability of an individual to withstand negative effects of the infection. Please note that here, the term resistance is not referring to immune function or implying the nature of immune responses of the individual against *M. surciattae* but signifies individual capacity to survive with clinical TB.
Survival	Time period between exposure to TB and death, irrespective of development of clinical disease.
TB death	Individual was euthanized with terminal TB infection or infection was confirmed by post-mortem examination. Individuals clinically ill with TB but dying of other causes are not included here.
Life-history parameters	
First date	First recorded date for the respective individual, i.e. birthdate for individuals born into the study population, or date of first inclusion in the study in case of newly habituated or immigrated individuals.
Last date	Last recorded date for the respective individual, i.e. date of death or last record of the individual before disappearance.
Group start	First date of records for the respective group.
Group end	Recorded date of group failure, irrespective of the nature of this failure (group split, group extinction or group abandoned).

immigrating into a group with visibly infected individuals. Especially for the first individuals in groups to develop clinical TB, the exposure might per definition be underestimated, yet most individuals were either born into a TB afflicted group or came into contact with TB upon immigration into a TB afflicted group (see below). Individuals were considered presenting clinical signs from the date they were recorded with TB by observers or displayed clear signs of TB infection (submandibular, inguinal or cervical lumps). If individuals had records of suspected TB, that is, lumps or swelling with no alternative explanation, and progressed to develop clinical TB, the date of first putative TB signs was considered as onset of clinical TB. Clearance of clinical TB has not been recorded in meerkats, so we considered infections to be purely progressive with no return to prior states (Patterson et al., 2021). Within KMP, terminally ill individuals were euthanized to curb the spread of the disease (Duncan et al., 2021; Patterson et al., 2021), with infections being confirmed in post-mortem examinations (Patterson et al., 2021). We classified these individuals as having died of TB and included them in our data set of TB progression (see Table 2), as terminally ill individuals do not recover and are expected to die within a few days or weeks (Patterson et al., 2021). To be conservative in our interpretations, individuals displaying clinical signs at time of death but dying of other or unknown causes (e.g. predation or disappearance) were not considered as having died of TB. At the group level, groups were considered as TB exposed upon the first observation of a clinically ill individual within the group. Groups that did not persist until the end of the study are considered extinct, irrespective of the reason of group termination (group abandoned, disintegrated, etc.).

Based on this data, we calculated time from exposure to developing clinical signs as proxy of TB-susceptibility, that is, the risk to become clinically ill upon exposure, and time between clinical signs and death as proxy of resistance, that is, the capacity of a clinically ill individual to survive despite disease. We also calculated survival (i.e. time from exposure to death) for all exposed individuals (see Donadio et al. 2022, see Table 2). To assess TB exposure and prevalence as well as progression and mortality, we calculated overall and yearly proportion of exposure, clinical infection and TB-related death at the group and individual level. Plus, the temporal patterns of TB progression, that is, TB susceptibility, resistance and survival (definitions see Table 2), were analysed. Data was retrieved from the KMP database into R using the package RMariaDB (Müller et al., 2020) and processed using the package tidyverse (Wickham et al., 2019); all figures were generated using the package ggplot2 (Wickham et al., 2016). All analyses were performed in R, version 3.6.3 (R Core Team, 2019). Research for this study was conducted with permission of the ethical committee of Pretoria

University and the Northern Cape Conservation Service, South Africa (Permit number: EC031-13, FAUNA 1020–2016).

3 | RESULTS

TB was first recorded in the late 1990s in this population and spread within ~8 years among the majority of social groups, exposing most individuals to the disease. Throughout the 27-year study period (1993–2020), 71% of all individuals were exposed to TB at some point during their life ($n = 2427/3420$), with no TB exposure recorded prior to 1997. The proportion of individuals exposed varied markedly between study years (mean ± SD: 54.8 ± 38.4%). Exposure levels proceeded to rise rapidly until 70%–95% of individuals were exposed in any given year after 2006 and remained at comparably high levels thereon after. In contrast, clinical signs of the disease increased in frequency less steeply. Between 0% and 29.4% of the study population showed clinical signs (mean ± SD: 8.6% ± 9.0%, with 0% to around 6% of individuals dying with confirmed TB (mean ± SD: 4.2% ± 4.6%) in any given year (Figures 1 and S1).

Individuals were most often exposed at birth (75.3%, $n = 1827$), or upon emergence of TB signs in their current resident group (19.8%, $n = 472$). The remaining exposed individuals immigrated into a group already experiencing TB (5.4%, $n = 128$). Only 22.9% of exposed individuals ($n = 555$), corresponding to 16.1% of the study population, developed clinical signs of TB, within 1.5 years (mean ± SD: 520 ± 421 days). The majority of individuals reported with clinical signs were confirmed to have died with or from TB ($n = 398$), leading to a TB-related mortality rate of 11.6% in the population. This does not account for individuals infected, but not showing overt signs of TB, or clinically ill individuals dying of other causes, so it is likely this figure underestimates the real extent of TB-related mortality.

We observed marked inter-individual variation in TB progression and outcomes, with time between exposure and first clinical signs ranging from 0 (for index cases in a group) to 2971 days (Figures 2 and 3). The mean duration between exposure and signs was ~1.4 years (mean ± SD: 520 ± 421 days, median: 440 days). Individuals survived the onset of clinical signs for up to 2756 days, although some individuals were recorded with signs only at the time of their death. On average, individuals survived the onset of clinical TB for ~6.6 months (mean ± SD: 200 ± 328, median: 70 days), indicative of the rapid progression of TB after development of clinical signs (Figures 2, 3 and S2). Comparing exposed individuals progressing to clinical TB with exposed individuals never developing TB signs, we found high variation in survival in both groups: Individuals developing TB signs survive between 5 and 3541 days (mean ± SD: 720 ± 529, median = 603 days) past exposure and asymptomatic individuals survived up to 4123 days (mean ± SD 432 ± 485, median = 269 days) past exposure. Asymptomatic individuals thus seemed to die significantly earlier (MWU-test: $U = 723{,}844$, $p < .001$).

On the group level, we focused on 91 meerkat groups lasting between 196 and 9235 days (mean ± SD: 1513 ± 1935 days, median: 660 days). Individuals with clinical TB were present in 62 (68.1%) groups, only eight of which persisted until the end of the study. Additional four groups present at the end of the study had not been in contact with TB. TB prevalence among groups varied between 0% before TB detection in the study and 96% in 2013 and 2016, with an average of 59.5% ± 33.9% of groups exposed to TB (Figure 1). TB was present in most groups that did not persist ($n = 54$, 68.4% of extinct groups).

After the first detection of clinical TB, groups persistence varied between 46 and 6356 days (mean ± SD: 1200 ± 1352 days, median: 569 days) and notably the two longest surviving groups in our data set persisted despite being exposed to TB since ~17 and 21 years, respectively. Marked variation in group survival past TB exposure is retained when only considering groups that disintegrated after detection of TB, after which these groups survived up to 8243 days (mean ± SD: 1591 ± 1858 days, median: 573 days).

4 | DISCUSSION

Using a large long-term data set including over 3400 individuals and spanning 27 years, we report the spread of TB in wild meerkats and quantify the extent of TB exposure, clinical TB prevalence and TB-related mortality. We show that TB exposure rose to over 50% within approximately 8 years after first detection in the population and temporarily reached over 90%. Rates of clinical disease and mortality over the same period peaked at around 25% and 6%, respectively, suggesting a degree of resistance to the pathogen. TB prevalence varied strongly between the years, with over one quarter of the population displaying clinical signs in some years. TB prevalence reported in this study is comparable with those reported from other well-studied species [~12% for European badgers (*Meles meles*) to ~32% for red deer (*Cervus elaphus*) and wild boar (*Sus scrofa*); see Reis et al., 2021], both with regard to overall (16.2%) and annual prevalence. Previous studies using diagnostic tools on subset of the study population to detect TB reported prevalence between 24% (Drewe, 2010) and up to 82.4% of individuals of exposed groups (Clarke et al., 2016), implying that by using clinical signs, the true extent of *Mycobacterium* infection prevalence is underestimated.

In line with previous studies, we find TB contributing strongly to meerkat mortality, both on the individual and group level (Duncan et al., 2021; Patterson et al., 2017). At 11.6%, individual TB-related mortality is almost twice as high as previously reported in the same population (Patterson et al., 2017), and comparable with estimated annual mortality of mongooses infected with *M. mungi* (Fairbanks et al., 2014). Most individuals (71.7%) with clinical signs were confirmed to have died of or with TB; yet given the irreversible nature of TB progression, the disease was likely a contributing factor in the death of diseased individuals dying of other causes. As many infected individuals likely die before progressing to clinical stages, subclinical infections are known to increase mortality risk (Patterson et al., 2021) and the impact of *Mycobacterium* infections on the population is likely even higher than reported here.

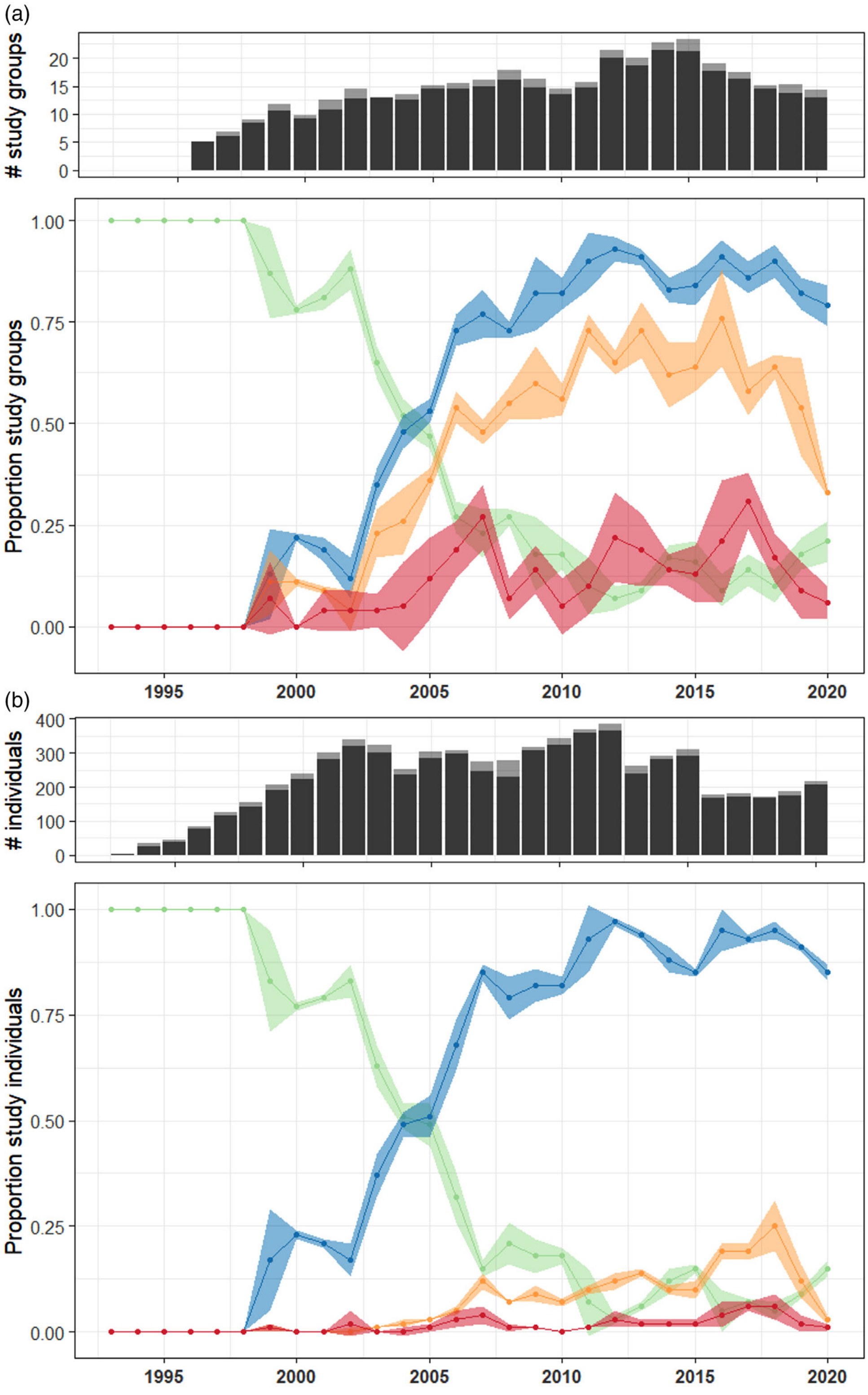

FIGURE 1 Patterns of TB exposure, prevalence of clinical signs and TB-related death over 27 years of research at the Kalahari Meerkat Project (South Africa) illustrating the inter-annual variation and development of TB prevalence within the study population. Absolute numbers (barplots) and proportion of (a) meerkat groups and (b) individuals of the study population being not exposed yet (green), exposed (blue), displaying clinical signs of TB (orange) or dying with confirmed TB (red). Means ± SD of each measure were calculated in 3-month increments

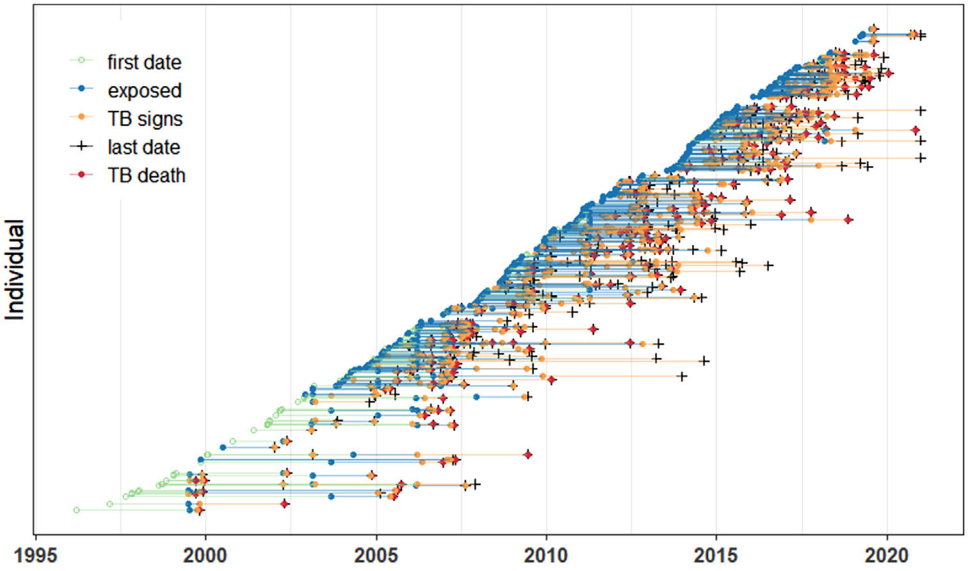

FIGURE 2 Life trajectories of meerkats that developed clinical TB ($n = 555$). For each individual, respective first (birth or entering into the study population) and last (death or disappearance) dates, as well as transitions between TB states are shown. Individuals were on average exposed to TB for 1.4 years before the onset of clinical signs and survived on average until 6.6 months after developing clinical TB. Individuals are sorted by birth date

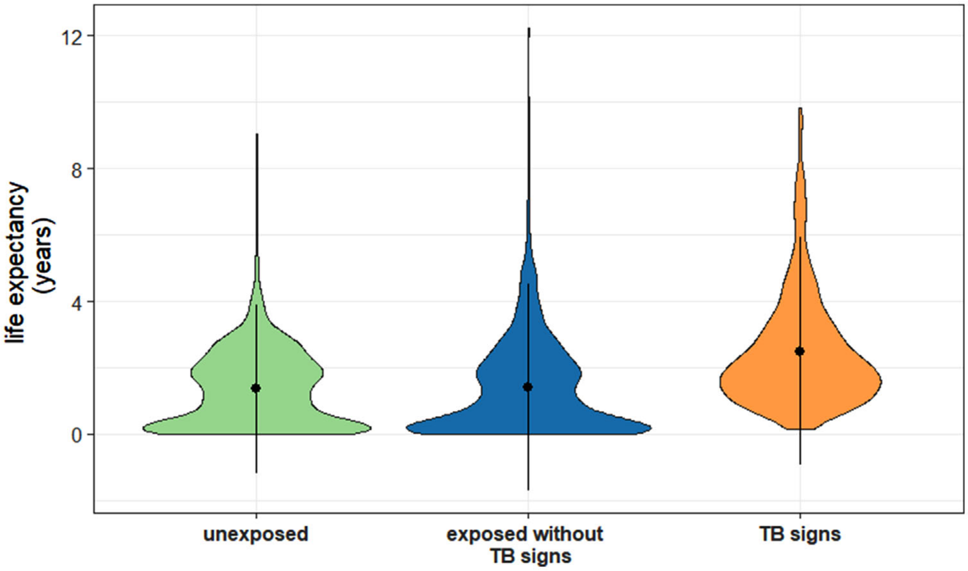

FIGURE 3 Meerkat life expectancy. Variation in life expectancy of meerkats with known final age (i.e. age at death or disappearance, $n = 3113$), based on their TB status. Points indicate the mean, vertical lines the standard deviation

We observe a high extend of variation of TB susceptibility, resistance and progression patterns across the study population. Only ~22% of exposed individuals progress to clinical TB, on average within 1.4 years of exposure. Our findings confirm the generally long latent or subclinical period of *M. suricattae* infections (Donadio et al., 2022; Drewe, Dean et al., 2009), with individuals developing TB signs as long as 8 years past first exposure. As infection occurs on average 1 year before the onset of clinical signs (Donadio et al. 2022), most exposed individuals are likely infected for most of the time between exposure and clinical TB manifestation. Lacking precise *Mycobacteria* shedding information to infer the actual infection state of most individuals prior to onset of clinical signs (Donadio et al., 2022), exposure might have been underestimated for some individuals. The proportion of individuals not susceptible to TB has been estimated at around 25.8% in a recent study (Donadio et al. 2022), implying that ~ 50% of exposed individuals are infected without displaying overt signs. This finding is comparable to results in badgers, where up to 80% of infected individuals do not present visible TB lesions (Gallagher & Clifton-Hadley, 2000). Given the parallels, we consider our classification as useful, since clinically ill individuals likely contribute most to transmission, and we are using a conservative, objective measure unlikely to introduce systematic errors.

After onset of clinical TB, meerkats died on average within ~6.6 months, confirming the rapid progression to terminal stages reported by previous studies of the same population (Donadio et al., 2022; Patterson et al., 2021). This pattern of prolonged subclinical or latent infection with rapid progression upon onset of clinical signs is a common feature of wildlife TB (Alexander et al., 2010; CFSPH, 2019; Tomlinson et al., 2013). Individuals survived for up to 7.5 years post first TB signs, suggesting factors facilitating natural TB resistance. The capacity of some individuals to survive TB for a long period is another common feature of wildlife MTC pathogen infections (Ezenwa et al., 2010; Tomlinson et al., 2013). Paradoxically, individuals eventually developing TB signs die at older ages than individuals that never proceed to clinical TB stages, a phenomenon most likely attributable to the long latent period compared to meerkat life expectancy. Baseline mortality rates in meerkats are high, with subordinates suffering increased mortality risk upon evictions (Cram et al., 2018), so while KMP meerkats can live up to ~12 years, median life expectancy is only 2.3 years (Drewe, 2009), making death of infected individuals prior to clinical manifestation of TB a likely occurrence. Another factor that might contribute to the high variability in TB progression in meerkats but to date is entirely unresolved is the role of co-infections. Co-infections with nematodes, for example, affect TB mortality in African buffalos (Ezenwa et al., 2010), and both nematode and virus infections impact TB severity in wild boar (Risco et al., 2014). While we lack detailed information on pathogen communities in wild meerkats, nematode infections are present in our study population (Smyth & Drea, 2016) and thus might contribute to variation in TB progression in this species. Moreover, climate change and environmental or social disturbances can drive co-infections and might even facilitate the emergence of novel pathogens or the re-emergence of endemic, but historically unproblematic pathogens via shifts in host-associated gut microbiota (Risely et al., 2022; Schmid et al., 2022).

The origin of *M. suricattae* in the study population and when it first emerged is currently unclear. Furthermore, whether meerkats are the original host, or the pathogen was transmitted from another, yet unknown reservoir species, is not known to date (Alexander et al., 2010; Parsons et al., 2013). While the disease was probably present in the study population from the beginning of data recording, and exposure as well as prevalence are likely underestimated in the first years of the long-term project, clinical TB is a highly conspicuous and easily recognizable disease, allowing the conclusion that the rapid increase in detected cases after the late 1990s is reflective of increased disease prevalence and transmission rather than an artefact of observation bias. This conclusion is also supported by the observation that both bovine and non-bovine TB seems to become increasingly prevalent in southern Africa (Alexander et al., 2010; Michel et al., 2006; Parsons et al., 2019; Tanner et al., 2015). Originally, inter-species transmission of *M. bovis* was suspected to cause the TB outbreaks in meerkats (Drewe, Dean et al., 2009; Drewe, Foote et al., 2009) before identification of *M. suricattae* (Parsons et al., 2013, 2019), which is now considered to be endemic in meerkats (Patterson et al., 2022).

The mechanisms underlying this increased TB prevalence and transmission are likely complex and multi-faceted, and their detailed discussion is beyond the scope of this study. However, based on our findings and previous research, we can identify factors that likely contributed to the rapid spread of TB, both inherent to the host–pathogen system and external factors and discuss the implication of the establishment of TB within the population. Even though clinical TB is highly contagious, there are no obvious behavioural defences against TB transmission in meerkats. Banded mongooses (*Mungos mungo*), which are closely related to meerkats, apparently do not avoid TB affected conspecifics (Fairbanks et al., 2015), and there is no evidence for avoidance behaviour in meerkats. In fact, social contact behaviour is linked to increased risk of TB (Drewe, 2010; Weber et al., 2013), which could imply behaviourally driven differences in exposure level based on contact patterns. Aggression, for instance, increased TB risk in meerkats (Drewe, 2010), which could indicate that TB transmitted via bite wounds could lead to more severe cases, mirroring results from badgers (Gallagher & Clifton-Hadley, 2000). Additionally, subclinically infected individuals have been shown to shed *M. suricattae* via their faeces (Donadio et al., 2022) and are potentially already capable of transmitting TB as observed in badgers (Gallagher & Clifton-Hadley, 2000; Graham et al., 2013; Tomlinson et al., 2013). In meerkats, migrating males are of particular importance for TB transmission between groups (Duncan et al., 2021; Paniw et al., 2022). Consequently, long subclinical infection periods, limitations in behaviourally avoiding TB exposure and frequent intra- and inter-group social contacts can facilitate rapid TB transmission within a population based on high levels of exposure.

Transmission can further be facilitated by increased susceptibility to a pathogen. Adverse climate conditions have been suggested to exacerbate the negative effects of TB on individuals and populations (Dwyer et al., 2020; Paniw et al., 2022), potentially indicative of such increased susceptibility to TB of individuals affected by environmental stressors. In the study population, above average temperatures increasingly occurred after the early 2000s and correlate with clinical TB occurrence (Paniw et al., 2022). Both adverse climate conditions and clinical TB reduce meerkat survival and can lead to group failure, particularly of small groups (Duncan et al., 2021; Paniw et al., 2022), which can lead to higher social mobility, as remnant individuals form new groups or immigrate into other groups. With immigration increasing the risk of TB transmission into previously TB-free groups, these may become more vulnerable to adverse environmental conditions and group extinction once individuals progress to clinical TB and the group suffers from TB-related mortality (Duncan et al., 2021; Paniw et al., 2022). Thus, the negative effects of climate change and TB emergence can enforce each other, facilitating TB transmission throughout the study population and affecting population dynamics. Potentially, the effect of *M. suricattae* infections transcends meerkats and has implications for entire ecosystems, based on the high inter-species transmissibility of MTC pathogens and a wide TB presence in South African mammals (Clarke et al., 2016; Hlokwe et al., 2014; Parsons et al., 2019). Predation is one of the main causes of meerkat mortality (~25%, NMK, unpublished data), so meerkat predators are likely exposed to *M. suricattae* via infected prey, potentially facilitating transmission via ingestion. This possibility highlights the need for detailed and systematic surveillance studies to understand the impact of TB on not only single species, but entire ecosystems.

Our study is based on purely observational data and thus not suitable to investigate determinants of TB susceptibility, resistance and progression. Individuals differ strongly in how they are affected by TB, and the high level of exposed but never clinically ill individuals is suggestive of non-susceptible individuals within the population. Future studies should investigate the impact of hosts' immune genetics and responses (Ezenwa & Jolles, 2015; Ezenwa et al., 2021; Jolma et al., 2022; Marjamäki et al., 2021) in explaining the variation in TB susceptibility, resistance and progression. Furthermore, assessing for effects of pathogen-mediated selection on meerkat population genomics and risk of co-infections will be highly informative furthering our understanding of the effect of TB on meerkat ecology and evolution.

AUTHOR CONTRIBUTIONS

Conceptualization: NMK, SS. Formal analysis: NMK. Investigation: NMK. Resources: SS, MBM, THCB. Data curation: MBM, THCB. Writing – original draft: NMK, AR, DWS. Writing – review & editing: NMK, AR, DWS, SS. Project administration: SS. Funding acquisition: SS, AR, MBM, THCB.

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CONFLICT OF INTEREST

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

Data used for this publication is available at https://github.com/Nadine-MK/TB-characterization-paper.

ETHICS STATEMENT

Research for this study was conducted with permission of the ethical committee of Pretoria University and the Northern Cape Conservation Service, South Africa (Permit number: EC031-13, FAUNA 10202016). All procedures adhere to the ASAB/ABS guidelines for the Treatment of Animals in Behavioural Research and Teaching (ASAB Ethical Committee/ ABS Animal Care Committee, 2012) (Buchanan et al., 2012).

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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