nature human behaviour

Article

https://doi.org/10.1038/s41562-026-02504-6

Stochastic models indicate rapid smallpox spread and mass mortality of Indigenous Australians after colonial exposure

Received: 20 December 2024

Accepted: 15 May 2026

Published online: 9 July 2026

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The impact of smallpox (variola) on Aboriginal communities in Australia beginning in 1789 was catastrophic and continues to cause intergenerational trauma. Historically biased perspectives and contemporary misinformation of the disease's introduction and spread impede modern-day truth-telling and efforts towards reconciliation and national healing. Understanding whether the disease entered and spread from pre-colonial Makassan (Indonesian) trade along the northern coast, or from the British First Fleet's arrival in southeastern Australia in 1788, is necessary to estimate the demographic impact. Here we developed stochastic, multipatch epidemiological models supported by systematic evaluation of historical observations to test hypotheses regarding possible disease entry points, spread rate and demographic impacts. Our models support the hypothesis that entry of the disease was in southeastern Australia. Even under ideal conditions and with higher-than-probable infection rates, simulations show that smallpox was unlikely to reach Sydney from a northern entry. We found no evidence that the 1789 epidemic was Australia-wide. Assuming 60% lethality based on global data, the loss of approximately 220,000 people would have occurred in these regions. While catastrophic to traditional Indigenous lifeways in the southeast, the disease also provided the catalyst for population decline and marginalization of Indigenous people in the face of expanding European populations. Our models indicate that it is unlikely that other parts of Australia were affected by the initial epidemic. We warn readers that the content of this study might be distressing.

The impact of introduced diseases to Aboriginal peoples in Australia as a result of colonial invasion in the eighteenth century was catastrophic, resulting in population collapse and disruption of knowledge transmission and reproduction[1]. Today, the uncertainty surrounding the introduction site(s) and effects of disease remain an obstacle to reconciliation and truth-telling initiatives, with competing views on the origin, spread and deliberate use to aid European invasion dominating contemporary debate[2–6]. Some views have been influenced by frontier violence in the early nineteenth century in which European colonists deliberately offered Aboriginal people poisoned food and supplies (for example, Kilcoy Station)[7], and from North America when European settlers proposed giving disease-laden blankets to First Nations people[8]. In the current period of truth telling, it is imperative that we understand the spatial origin of various diseases, their spread rates and likely death toll to quantify the full extent to which Indigenous populations were affected.

Although several diseases ultimately affected Indigenous peoples following colonial invasion, the appearance of smallpox (variola) in

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https://doi.org/10.1038/s41562-026-02504-6

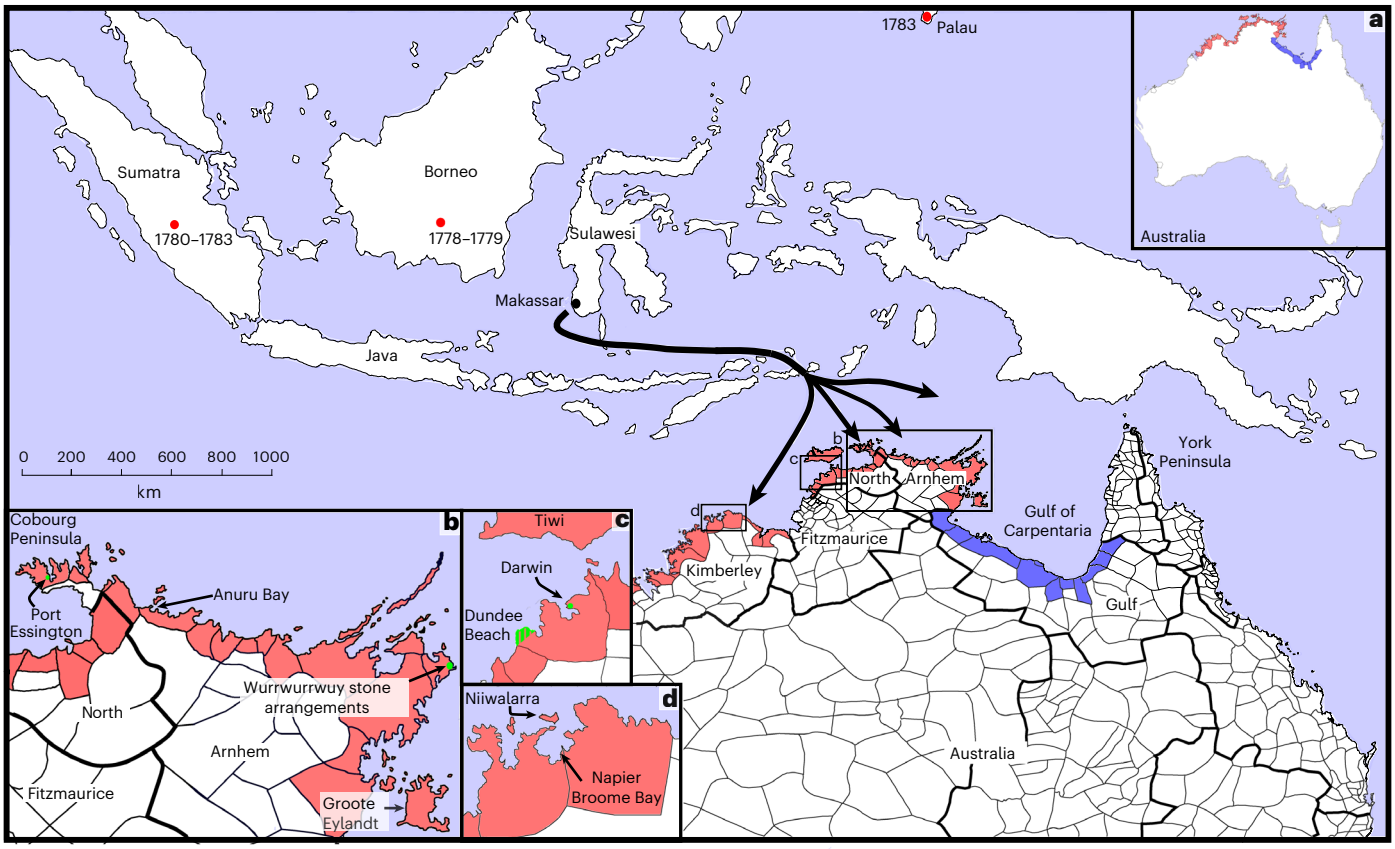


Fig. 1 | Possible contacts made by Makassans during their first frequent voyages to Australia. a, The most probable areas of contact, supported by archaeological evidence, are the Kimberley and northern-most coasts (in red). However, other areas in the Gulf of Carpentaria (in blue) are also potential sites. **b**–**d**, Detailed areas show Arnhem Land that includes the Cobourg Peninsula and Port Essington, Anuru Bay, Groote Eylandt and the location of the Wurrwurrwuy stone arrangements (**b**), the north region including Dundee Beach (**c**) and the Kimberley coast that includes Niiwalarra Island and Napier Broome Bay (**d**). Several well-documented smallpox outbreaks occurred in the late eighteenth century (around the time of the First Fleet's arrival) at various islands throughout the Malay Archipelago (red dots). Thicker black lines indicate the boundary of geographically distinct regions of Australia (see also Fig. 6).

early 1789 and its subsequent epidemics had the largest demographic and cultural impacts. Indeed, the earliest colonial observations of smallpox recorded bodies of Aboriginal people floating in Sydney Harbour and an 'emptying' of the landscape[9]. Many have put forward hypotheses regarding the origin of this first outbreak, with the associated conceptual models falling into three main categories: that smallpox (1) originated from the Indonesian archipelago and was brought to Australia by exposure to Makassan fishermen making sustained contact with Aboriginal groups along the northern coastline well before European arrival, subsequently spreading across the continent[4,10,11]; (2) was spread accidentally by the First Fleet in 1788 (11 British ships under naval command also transporting convicts)[12]; or (3) was introduced deliberately by members of the First Fleet to assist invasion and displacement of Indigenous people for easier land acquisition[6,13–15]. Others argue that there is no way to know[1,16–18] or that the outbreak originated from an entirely different source such as chickenpox or some other disease[19].

Most of these ideas are difficult to investigate or test owing to limited primary evidence. We therefore constructed a stochastic, multipatch epidemiological model that incorporates the dynamics of smallpox spreading among small populations distributed across Australia and systematically evaluated historical data as corroborating evidence. We used the model to examine evidence in support of hypothesized entries of the disease in the late eighteenth century and how they would have affected the Indigenous population both spatially and demographically, thereby providing a foundation to explore origin hypotheses (1) and (2) above. It is not possible with our current model to test hypotheses regarding the probability of deliberate introduction of the disease to Indigenous Australians.

Hypothesized entry points of smallpox

Makassan entry. From at least 1720 and probably earlier, trepang-focused industries (harvesting Holothuroidea sea cucumbers) arose mainly out of the port of Makassar on the island of Sulawesi in modern Indonesia[11,20–22] (Fig. 1). The exact date of their initial arrival is unknown; however, recent work suggests that they have been visiting Australia from at least the seventeenth century and probably made sporadic visits hundreds of years before then. By 1754, Makassans were established in the Kimberley region in the northwest, and by 1780 were regular visitors to Arnhem Land (the north of modern Northern Territory)[20,23]. While in Australia, the Makassans established onshore camps where they processed their catches and often lived with Aboriginal people; there is also evidence that some Aboriginal men and women returned to Sulawesi with the fishermen[10,24]. The archaeological remains suggest a large, pre-European trepanging industry in northern Australia, including processing camps along the coastlines, Wurrwurrwuy stone arrangements on the northern tip of Arnhem Land (Fig. 1b), pottery on the islands of Niiwalarra in the west (Fig. 1d) and the South Wellesley group in the east, and swivel cannons near Napier Broome Bay and Dundee Beach near Darwin (Fig. 1c).

Several documented reports identify smallpox epidemics occurring in the Malay Archipelago north of Australia in the late eighteenth century. There was an epidemic in southern Borneo in 1778–1779[25], Palau was hit in 1783[26] and southern Sumatra lost one-third of its

population to an epidemic in 1780–1783[27]. Because most islands of the Archipelago had populations too small to support endemic smallpox[26], the disease appeared in intermittent waves throughout that period. Sulawesi experienced several major smallpox epidemics throughout the century: in 1700–1703, 1724–1726, 1763–1764 and 1792. However, there is no specific mention of an epidemic on the island in the 1780s[27,28].

An idea first proposed by Cleland[29] and Stirling[30], and elaborated by Campbell[4], proposed that Makassan contact in Arnhem Land and the Gulf of Carpentaria brought smallpox to Australia in the late 1780s (see also refs. 12,31). According to this proposal, smallpox moved through the Australian continent via 'chains of connections' (after Mulvaney[32]), such as major intergroup gatherings, down-the-line exchanges and continental-scale trade networks[4]. Mulvaney[32] and Campbell[4] both argued that southward movement through the Channel Country and into the Murray–Darling river system could have provided transmission pathways across much of the eastern half of the continent (Supplementary Fig. 1). Thus, Campbell[4] proposed that the colonial observations of the disease in Sydney in 1789 were part of a larger epidemic that was affecting most of the continent. Indeed, one of Campbell's strongest arguments was the oral history obtained by Péron[33] who wrote that Indigenous people had "religious terror for the Blue Mountains (west of Sydney); they think them the residence of an evil spirit". This would be the probable direction of the disease if arriving initially from the north and west. However, there is no contemporary evidence of that transmission route, with the hypothesis apparently supported by later colonial observations of older people with smallpox scars. However, we cannot rule out the possibility that such scarring could have been the result of subsequent epidemics that occurred in the nineteenth century.

A central tenet of the hypothesis for a northern entry as the source of the outbreak was the widespread assumption[4,26] that it was impossible for smallpox to have survived the journey of the First Fleet due to the high temperatures experienced on the voyage through the tropics (because heat can destroy the virus). However, subsequent research[13,34] has demonstrated that the variola virus could have survived the temperatures experienced by the First Fleet, and that variola matter could have been procured en route when in Cape Town, South Africa, after the voyage through the tropics.

Entry via the First Fleet. In January 1788, the British First Fleet led by Governor Arthur Phillip arrived in Sydney Cove to establish a penal colony. Although many of the new colonists had contact with smallpox before the 36-week journey, there were no reports of major disease on the voyage or for >12 months after invasion[35]. From January to March 1788, two ships of the French expedition led by Jean-François de Galaup, comte de La Pérouse, briefly anchored at Botany Bay. However, there had been no sign of sickness on board the French vessels and no further sign in Australia for 12 months after their departure. Smallpox could not therefore have been incubated in the bodies of these new arrivals. Beyond this visit, there were no other European colonists until the Second Fleet in 1790[35].

There is historical evidence of a widespread epidemic that affected Sydney 15 months after the British landed, in March–April 1789. The new sickness, known to local Indigenous people as 'gal-gal-la', had a high mortality rate and spread quickly along the many coves and bays following established Indigenous pathways[16]. The first sign of the epidemic reported by the colonists were dead bodies found in coves and inlets around the harbour in April 1789[36,37], suggesting an initial exposure some weeks earlier (~24 days). In early May 1789 when Lieutenant William Bradley returned to Sydney after spending time at the Cape of Good Hope, he was struck by the unusual absence of Indigenous people from the harbour[9]. David Collins described the path to Broken Bay "covered with skeletons", with more bodies "to be met with in the hollows of most of the rocks of that harbour"[38]. Governor Phillip observed in June 1789 that the southern path to Botany Bay was also lined with bodies. The fact that the dead were left unburied counter to traditional practices highlights the speed and magnitude of the epidemic. Wangal man Bennelong—a survivor of the disease—reported that "one-half of those who inhabit this part of the country died"[39]. There was one non-Aboriginal death reported at the time: a Native American sailor who had joined the fleet in Rio de Janeiro, Brazil[38].

Speculation and disagreement on the origin of the outbreak began early. Phillip postulated that the French had brought it, but Watkin Tench[37] questioned whether it originated from variola matter in bottles brought with the First Fleet. The first vaccine was not available until 1796, before which inoculation through variolation (rubbing powdered smallpox scabs or fluid from pustules into superficial scratches made in the skin) was common. Although ships of the British Royal Navy did not routinely carry variola matter[13,40], it is likely that at least one of the surgeons would have brought it along or acquired it en route in Rio de Janeiro or Cape Town[41].

Smallpox transmission via fomites is rare, but there are documented cases of virus transmission from contaminated clothing and bedding[26,42]. There is also contemporary evidence from North America of British commanders calling for smallpox to be deployed against First Nations communities "… by means of Blanketts (sic)" (Amherst[8]). Fenner[26] stated that "… smallpox had an established, if irregular, place in late-eighteenth-century warfare"; as a weapon, it also "… had the advantage of deniability". Many of the officers on the First Fleet, including the officer in charge of the Marines, Major Robert Ross, had served in America and had witnessed the role of smallpox during the Seven Years' War and the American Revolutionary War[43]. The months preceding the Sydney outbreak were marked by "… a new, still more serious phase in deteriorating relations", with a range of confrontations erupting in the colony[16,17,37].

Characteristics of smallpox (variola)

Of the global diseases that have devastated human populations around the world, it was smallpox that eventually replaced bubonic plague as the most deadly. Throughout its long presence, first in Asia and then in Europe, smallpox killed an estimated 0.5 million people per year[26]. However, unlike the bubonic plague, smallpox did not suddenly invade naive populations; rather, in places where it became endemic, many adults survived mild infections earlier in life, and it was therefore mostly considered a disease of childhood, only flaring up with waves of immigration to urban areas[44].

Most of what we know about smallpox comes from laboratory studies done during the 1967–1979 global campaign to eradicate the disease[26]. The first symptoms follow an incubation period of roughly 12 days, which often include high fever and body aches and conclude with a rash on the third day. During the following 14-day phase, characteristic pustules form that eventually hardened. This was the most infectious stage of the disease during which victims were most likely to die. Those who survived experienced a final stage that lasted approximately 6 days where scabs formed and eventually fell off to leave distinctive scars. The disease often left victims with complications including blindness, and as many as 80% of survivors had distinctive pockmarked faces. Epidemiological research suggested that transmission rarely occurred before the first day of the rash appearing and was less likely when scabs began to form[26]. A measure of the speed of transmission is the basic reproduction number, $\mathcal{R}_0$, the average number of secondary cases infected by each primary case. Although some data suggest a wide range ($\mathcal{R}_0$ = 1.5 to >20), a recent review proposed $3.5 \leq \mathcal{R}_0 \leq 6$ (ref. 45). The emergence of the closely related monkeypox virus[46] might potentially yield more precise epidemiological parameter estimates for smallpox, even though monkeypox is zoonotic[47].

Results

If introduced in any of the northern regions including Arnhem, Gulf of Carpentaria, Kimberley or North, it was unlikely that a smallpox epidemic would have reached the southeastern coast. Figure 2 illustrates

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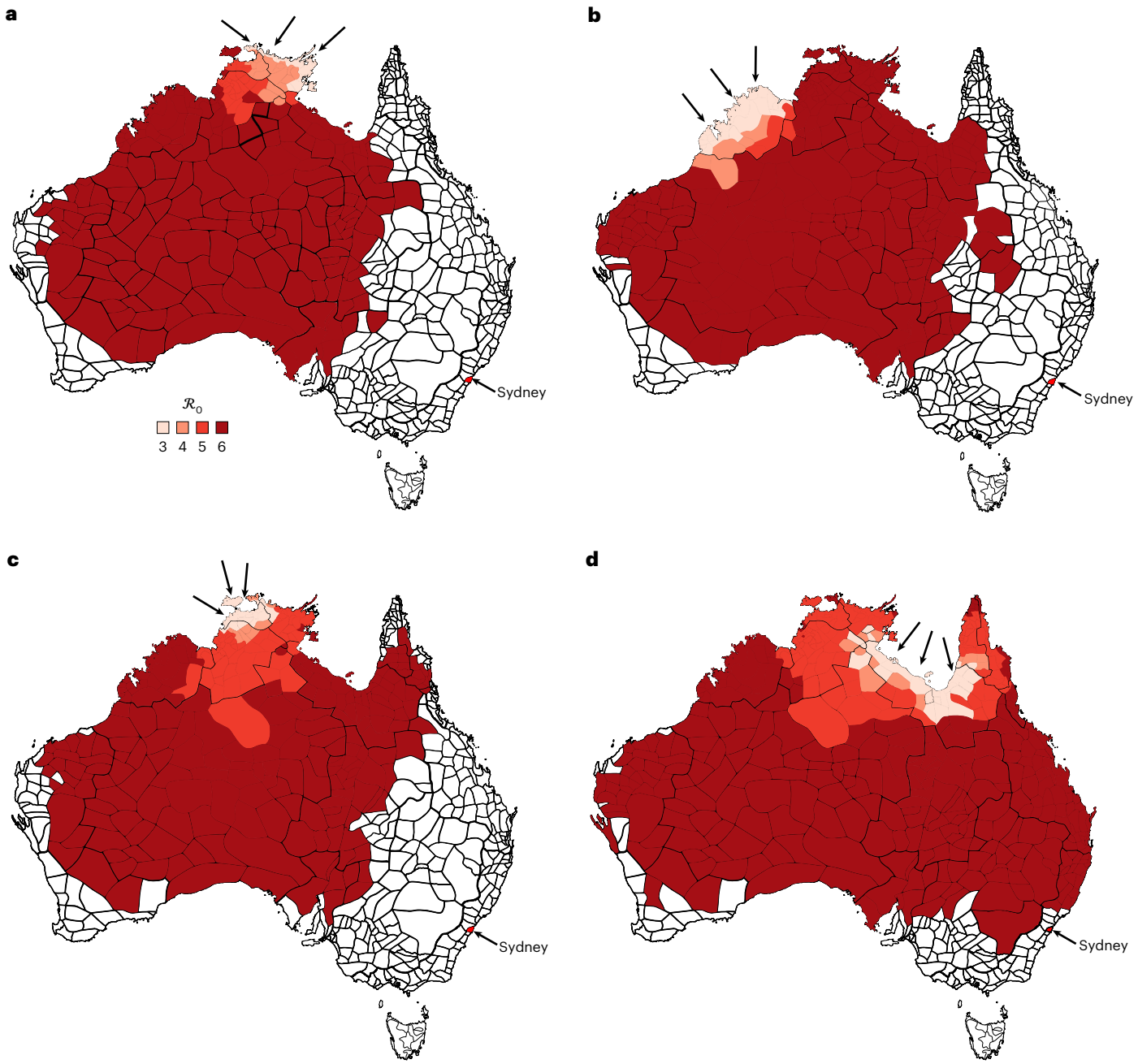


Fig. 2 | Furthest spatial extent of a smallpox epidemic starting in the north from Makassan contacts. a–**d**, Simulations with four different $\mathcal{R}_0$ values starting with a single exposure on the coast of Arnhem Land (**a**), Kimberley (**b**), North (including Darwin and the Tiwi Islands) (**c**) and the Gulf of Carpentaria (**d**). The arrows indicate introduction points. For each scenario, we simulated multiple sample paths corresponding to approximately 50 realizations per coastal patch, which we combined for each region. Each simulation was terminated only when no infectious individuals remained. Thus, the results reflect the full extent of the epidemics. For completeness, we ran simulations for distinct $\mathcal{R}_0$ values: 3, 4, 5 and 6. The colours represent the likely spatial extent of an epidemic originating in each region. These patches contained at least one infective individual for at least 50% of all simulations ($P \geq 0.5$). In all cases the disease is unlikely to reach the Sydney Basin. The parameters used in the simulations shown are $a = 8$ and $b = 1$.

the most likely spatial extent of an epidemic originating in each of these regions for various potential values of $\mathcal{R}_0$. We simulated multiple epidemics starting with a single exposure from within distinct patches in these regions. Patches that included at least one infective individual for at least 50% of the simulations (probability of $P \geq 0.5$) are highlighted with a colour corresponding to a fixed value of $\mathcal{R}_0$, illustrating the most likely spatial spread of disease (detailed results are shown in Fig. 3). Even under the most ideal conditions with generous movement parameters and an $\mathcal{R}_0$ fixed at the maximum expected value, the disease was constrained largely to regions in the north and central part of the country. Much of the southern and eastern regions of the continent would not have experienced infections of epidemic proportions in most scenarios. Also, alignment with the historical observations of the disease[9,36–39,48] cannot be reproduced with any of the simulations.

If smallpox escaped or was released near Sydney in March–April 1789, the area of greatest impact was in the Sydney Basin and the coastal populations for several hundred kilometres both north and south (Fig. 4 and details shown in Fig. 5). At the highest $\mathcal{R}_0$ (6), the disease extended across a large portion of southeastern Australia, with a distribution indicating movement along the heavily populated Murray

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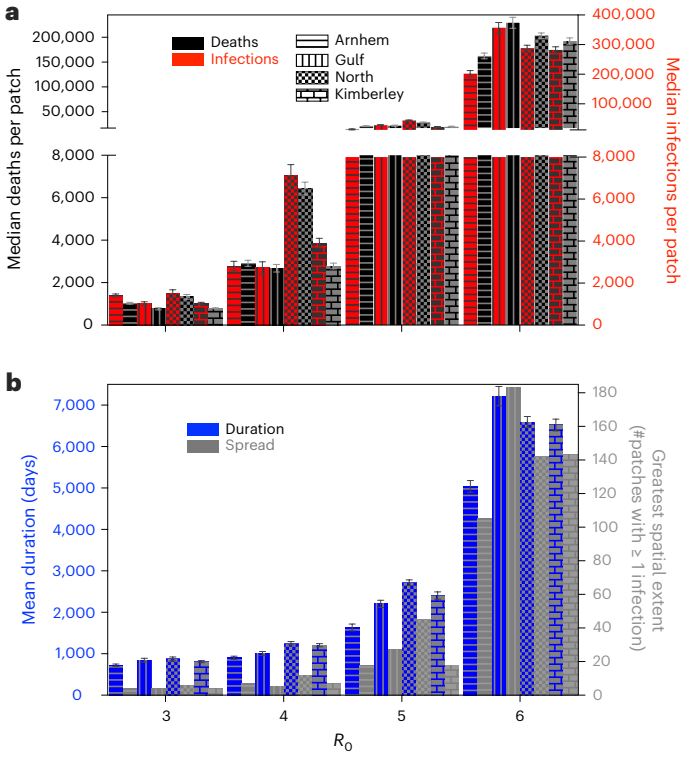


Fig. 3 | Median (± s.e.m. across simulations) results for each of the coastal regions in the north of Australia. a,**b**, Using various values of $\mathcal{R}_0$, we illustrate the potential impact of an exposure in the north by comparing the median total deaths to infections (**a**) and the median duration and spatial spread of the disease corresponding to an exposure in each of these regions (**b**). The total number of patches indicates the full tally of patches that included at least one infectious individual with $P \geq 0.5$, representing the maximum likely extent of epidemic spread. All simulations started with a single exposure and continued until no more infectious individuals remained in the population. We estimated total mortality (deaths) based on the assumption that 60% of infections led to death. Total number of simulations for each region: Arnhem, 925; Gulf, 535; Kimberley, 625; North, 475.

and Lachlan river systems[49] and throughout the Australian Alps. The latter perhaps reflects movement associated with large, quasi-annual regional gatherings in this region, where many Indigenous groups came together to exploit Bogong moths (*Agrotis infusa*) as food[50]. Distribution is more patchy outside the immediate environs of the Sydney Basin, especially in the semi-arid parts of western New South Wales and along the Victorian coastal fringe. Regardless of the $\mathcal{R}_0$ we applied, the disease remains constrained to the southeastern corner of the continent and suggests other parts of Australia would have been unaffected by the outbreak.

On average, with $\mathcal{R}_0 = 6$, the disease failed to propagate in 38.8% of scenarios that ended in <50 infections, further suggesting the northern route was improbable. If released at Sydney, the epidemic would have lasted between 844 and 7,784 days (2–21 years) depending on the $\mathcal{R}_0$ we applied. The larger mortality associated with $\mathcal{R}_0 = 6$ aligns more closely with the findings of Williams et al.[51] and might indicate that a higher number of infections were prevalent in the 1789 outbreak than later observations of the disease imply. This suggests that the epidemic might have still been present, albeit unrecorded by colonists, well into the 1790s.

From a hypothesized northern origin and assuming a 60% mortality, the model predicts that between 77,000 and 324,000 total deaths (40,000–77,000 per year) would have occurred over this period. An epidemic originating in Sydney could have resulted in approximately 220,000 total deaths throughout the epidemic (12,000 per year).

The full results of the global sensitivity analysis are provided in Supplementary Section D. Overall, parameters that primarily determine the basic reproduction number $\mathcal{R}_0$ exhibited the greatest influence on both the final epidemic size and the number of patches affected.

Discussion

Our stochastic, multipatch model that tests hypotheses about the origin of the first recorded smallpox epidemic in Australia indicates that the epidemic began in Sydney in March 1789. On the basis of our simulations, this initial dissemination would have encompassed much of the southeastern corner of Australia (>500,000 km^2), primarily along the coastal fringe and via major river systems such as the Murray River, Lachlan River and their tributaries. This distribution aligns with historical observations of the disease that were concentrated in the Sydney Basin[9,38,48], but that it extended to Jervis Bay[52] and Port Phillip Bay[53,54]. Many smallpox-scarred people were observed later in the nineteenth century in semi-arid parts of New South Wales and within the simulated extent of the epidemic[1,55]. Indeed, our model is further corroborated by the observations of Angas, Taplin and Curr[56] who documented that "… before the advent of the (colonists) a strange disease came down the Murray and carried off many of the natives" when recording oral history of the Ngarrindjeri at the outflow of the Murray River; the Murray appears to have been one of the main conduits for the disease beyond the Sydney Basin.

By contrast, even under optimal conditions of infection and transmission with extensive movement, ideal humidity—unlikely for most of the year in northern Australia—and an $\mathcal{R}_0$ value tending towards the extreme of its plausible range, our results are not consistent with an entry from the northern regions of Australia. In all scenarios, the epidemic abates a considerable distance from any historical observations of the disease in 1789.

Our results indicate the disease was constrained to the southeast, with the impact and lethality of the disease (regardless of the assumed $\mathcal{R}_0$) limiting the opportunity for people to move long distances and/or interact with neighbours before becoming infected. The time between becoming infective and being incapacitated was generally in

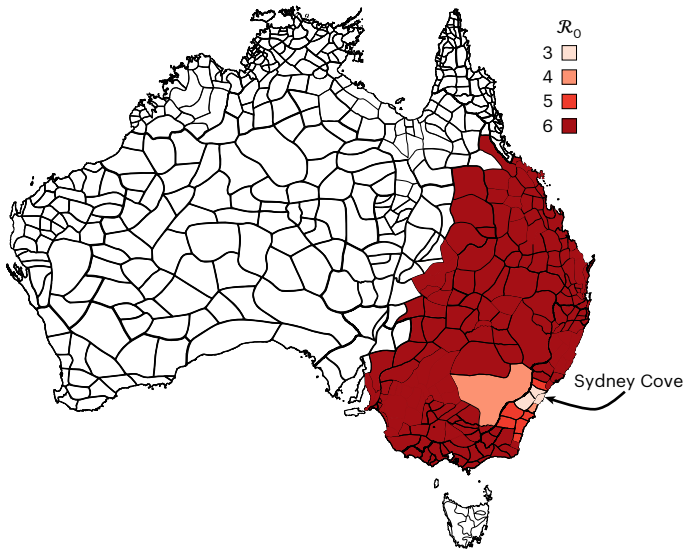


Fig. 4 | Sydney-originated epidemic. Model results when beginning with a single exposure in the patch containing Port Jackson and the Sydney Cove. For each of the four $\mathcal{R}_0$ values shown in the figure, we simulated 50 sample paths starting with one exposure. We simulated each sample until no individuals remained in the infective compartment of our model. Colours illustrate the patches that contained at least one infective individual for at least 50% of the simulated epidemics (corresponding to a probability of $P \geq 0.5$), which represents the maximum likely spread of an epidemic. The parameters used in the simulations shown are $a = 8$ and $b = 1$.

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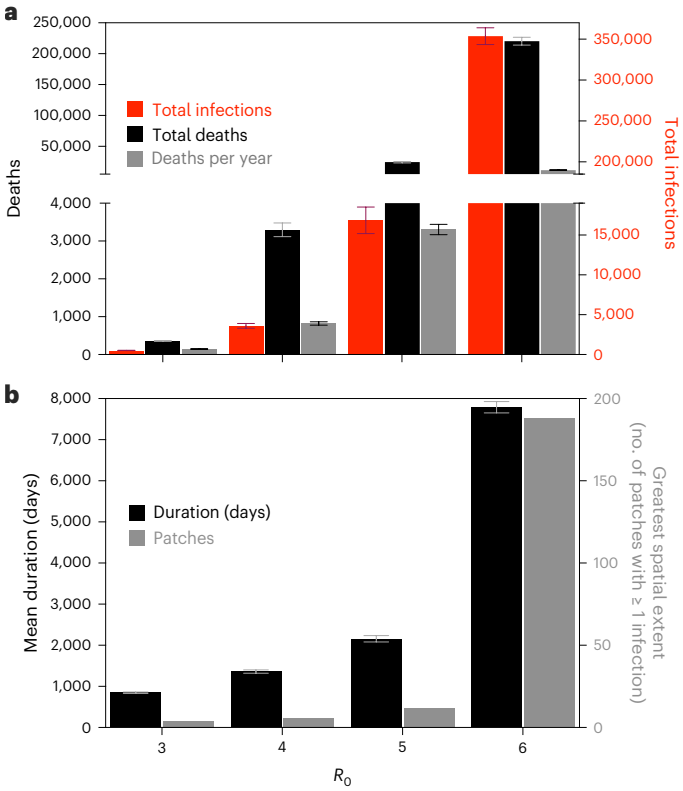


Fig. 5 | Median (± s.e.m. across simulations) results for an exposure in Sydney Cove. a,**b**, Using various values of $\mathcal{R}_0$, we illustrate the potential impact of an exposure of smallpox in the Sydney area by comparing the median total infections and deaths (**a**) and the average duration of the disease and the likely extent of the spatial spread (**b**). The total number of patches indicates the full tally of patches that included at least one infectious individual with $P \geq 0.5$, representing the maximum likely extent of epidemic spread. All simulations started with a single exposure and continued until no more infectious individuals remained in the population. We estimated total mortality (deaths) based on the assumption that 60% of infections led to death. Total number of simulations: 50.

the order of 2–4 days. Movement of the disease through long-distance trade networks was therefore improbable, with infected people physically unable to travel long distances usually required for such activities[13]. It was therefore more likely spread within the family and/or small groups in regular contact, in tandem with larger (but local) gatherings. Historical observations suggesting a breakdown of social cohesion and capacity, where burial of the dead was unachievable, are consistent with the rapid onset of symptoms within the Indigenous population. These limitations on transmission are also consistent with the observation that although there were huge demographic effects in the southeast, other parts of Australia—some only a few hundred kilometres from Sydney—were completely unaffected by the epidemic. At a lower $\mathcal{R}_0$, the epidemic might not have extended outside of the Sydney Basin. This has important ramifications for early ethnographic observations and their interpretation, which are often considered to reflect societies in a state of rupture[57,58]. While evident in early accounts of Sydney and Port Phillip, our models indicate that similarly impacted Indigenous societies were less likely in other parts of Australia around the time of the first smallpox epidemic.

The restriction to the southeast also raises questions about when and how most other Indigenous populations were affected by colonists. Given that the next major smallpox epidemic affecting the Indigenous population did not occur until around 1830, the implication is that other diseases, including tuberculosis[59,60], leprosy[61], influenza[62], chlamydia[1], syphilis[1], gonorrhoea[1] or measles[63], along with colonial expansion and frontier violence[64,65], played a role in the decline of Indigenous populations throughout the nineteenth century[51].

Our results suggest that an average of 220,403 total deaths (mean ± 1 s.e.m.: 213,530–227,276) would have resulted from the epidemic, equivalent to approximately 11,600 annual excess deaths between 1789 and 1808 (11,238–11,962). Continentally, such losses of life might reflect as much as 10% of the Australia-wide population estimated before European invasion, and all limited to one of the most populous parts of Australia[49,51,66].

The societal changes imposed by such a high mortality rate would have been catastrophic to the traditional lifeways of Indigenous populations and regional sociopolitical and socioeconomic (for example, trade and ceremonial gatherings) networks. The elderly, pregnant and young were the most susceptible to the disease, meaning this disruption disproportionately affected Knowledge holders as well as subsequent generations. This selective mortality would have altered the age, sex and household composition of affected communities, with widespread social and economic implications. It is likely that the disease, coupled with the subsequent removal of people from their lands later in the nineteenth century, would have reduced their capacity to care for Country[67]. The pace of the disease would have limited knowledge transfer to beyond the immediately affected communities. Not only did the disease directly disrupt Indigenous populations, survivors would have been more susceptible to colonial expansion, including frontier violence, pastoralism and assimilation in the early nineteenth century[1,17,68]. Our model cannot provide any direct evidence of the intent of the colonists during that time (Supplementary Information).

Despite the insights our results provide, they should be interpreted in light of several limitations. Our multipatch compartmental model is necessarily a first approximation and relies on simplifications of population structure and mobility, including the standard assumption of a well-mixed population within patches. The model also incorporates uncertainty in some important epidemiological parameters and does not account for behavioural responses to disease. Given these constraints, together with the incompleteness of the historical record, our findings are best understood as plausible scenarios rather than precise reconstructions.

Conclusion

Since 2017, Australian governments have been leading a process of truth-telling with Indigenous communities to acknowledge the historical harm caused by colonialism, explore and recognize the past, and promote reconciliation. The smallpox epidemic of 1789 is arguably one of the most devastating events resulting from colonialism, yet there remains widespread disagreement on its origin, scale, impact and spread. Our spatial epidemiological models combined with historical observations provide a quantitative overview, at the continental scale, of the first smallpox epidemic in Australian history. The ensuing mortality event estimated by our models would have been catastrophic to Indigenous society and had far-reaching ramifications for how societies resisted colonization in the face of frontier violence and altered the capacity of communities to manage Country. It is important for Australians to come to terms with this traumatic legacy to inform the national process of healing.

More detailed research is needed to appreciate how the expansion of colonialism affected broader Indigenous society who endured major population reduction[51]. Our model raises the question: was the magnitude of the depopulation a result of introduced diseases and/or was frontier violence more extensive than documented? Our results suggest that although southeastern Australia was heavily impacted by the initial outbreak, large parts of the continent were not. This has implications for how early ethnographic observations and data are interpreted, particularly where they were previously thought to reflect partially assimilated or impacted communities. From an Indigenous perspective, the absence of smallpox is an important

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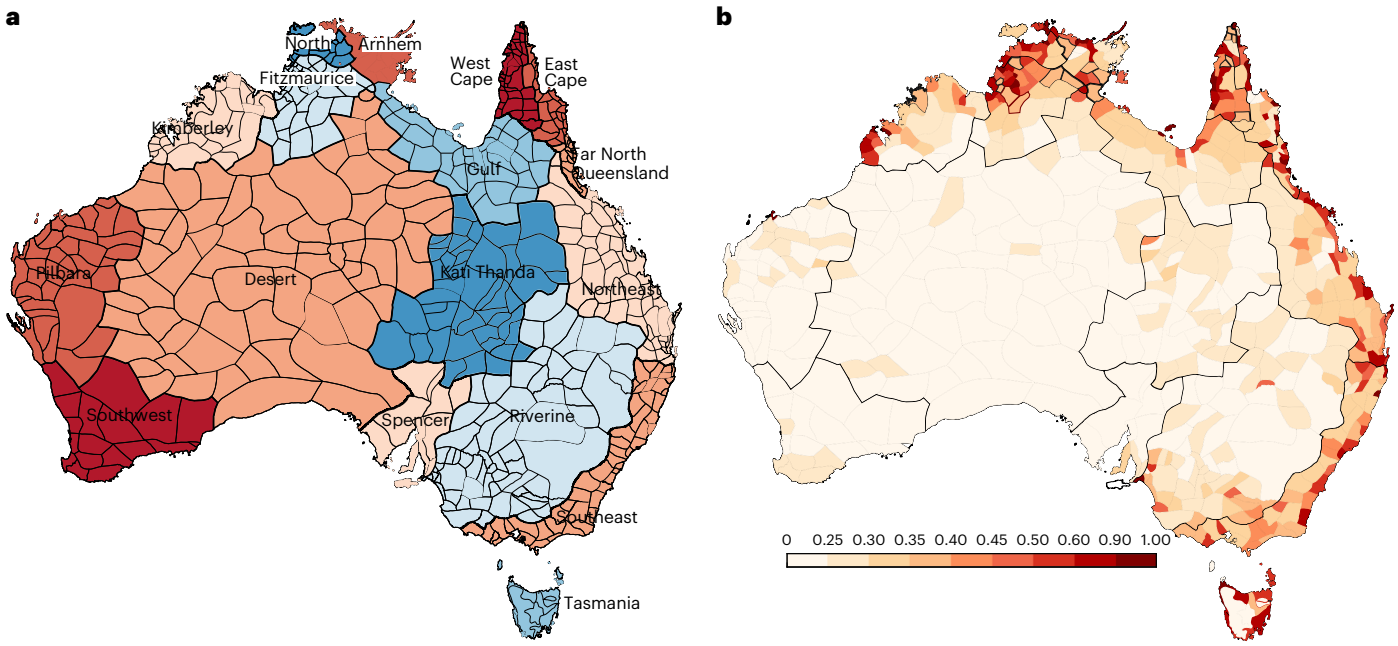


Fig. 6 | Map of Aboriginal Australia. a, The Tindale (1974)[94] map includes 592 distinct language and cultural groups, separated into patches collected into 17 geographic regions represented by the colours. **b**, The population density for each of the 592 language groups with an overall median population estimate of ~2 million[51], measured in persons km^{-2}. The darkest shading indicates densities ≥ 0.9 persons km^{-2}.

Table 1 | Events, transitions, rates and propensity functions that define the within-patch model

Event	Transition	Rate
Exposure in patch i	$S_i \to S_i - 1, E_i \to E_i + 1$	$\beta S_{ij} I_{ij} A_i^{-1}$
Susceptible movement from patch i to j	$S_i \to S_i - 1, S_j \to S_j + 1$	$\mu_i \sigma_{ij} S_{ii}$
Susceptible return from patch j to i	$S_i \to S_i + 1, S_j \to S_j - 1$	ρS_{ij}
Exposed movement from patch i to j	$E_i \to E_i - 1, E_j \to E_j + 1$	$\mu_i \sigma_{ij} E_{ii}$
Exposed return from patch j to i	$E_i \to E_i + 1, E_j \to E_j - 1$	ρE_{ij}
Infection in patch i	$I_i \to I_i + 1, E_i \to E_i - 1$	αE_{ij}
Recovery in patch i	$I_i \to I_i - 1, R_i \to R_i + 1$	γI_{ij}
Recovered movement from patch i to j	$R_i \to R_i - 1, R_j \to R_j + 1$	$\mu_i \sigma_{ij} R_{ii}$
Recovered return from patch j to i	$R_i \to R_i + 1, R_j \to R_j - 1$	ρR_{ij}

Only components of the state that change in a given transition are shown; all other components are fixed.

factor in demonstrating ongoing connection to place and a determinant in the process of acquiring Native Title. Our findings indicate that outside the southeast, many Indigenous societies would not have experienced infections of epidemic proportions into the early nineteenth century.

The findings we present here may also raise questions about how Indigenous societies reacted to the epidemic, especially in terms of how affected versus unaffected communities resisted domination and attempted assimilation. Recognizing that different Indigenous groups probably experienced colonialism in distinct ways and at different times is an important foundation in the process of truth-telling, and the development of more effective social and environmental Indigenous policies and reforms in the future.

Methods

We constructed a stochastic, multipatch epidemiological model that incorporates the dynamics of smallpox spreading among small populations distributed across Australia. The approach includes a generalized human mobility model with parameters inferred from conservative estimates of movement, intergroup gatherings and trade between groups, to which we applied a standard, compartmental disease-transmission model based on the susceptible–exposed–infected–recovered (S–E–I–R) framework[69,70].

Pre-invasion populations largely consisted of traditional, non-agropastoralists comprising local residence groups of 20–50 people[71–73]. To incorporate the effects of small populations and a network of frequent interactions, we modelled both disease transmission and movement between groups with a probabilistic agent-based model. Thus, our model has two parts: (1) a model of human mobility between discrete regions in space, which requires a population structure and estimated movement parameters; and (2) an epidemiological model for the spread of smallpox between individuals in a local region using the S–E–I–R compartmental approach[74]. Unlike more traditional models with frequency-dependent transmission, we incorporated the spatial heterogeneity of Indigenous Australia by including a density-dependent force of infection.

Developing a pre-invasion population

We incorporated the spatial heterogeneity of pre-colonial Australia using a map of Indigenous groups and languages first constructed in 1940 by Norman Tindale based on his decades of anthropological research[75]. The map consists of 592 distinct language and cultural groups divided into 17 regions based on drainage basins and climate types[76,77] (Fig. 6). Although there are alternative maps, and the boundaries and accuracy of this map are often disputed, the heterogeneity provided by the Tindale map allows for an approximation of disease spread throughout the country within which regional populations were established. At the continental scale, the choice of map does not affect our general conclusions.

Our map includes an estimate of population density, based on an overall median human population of ~2 million in 1788 as suggested by Williams et al.[51]. We made the simplifying assumption that visitors only move to adjacent patches and their preferred directions align with major migration pathways reflecting proximity to water and other resources, evaluated previously with archaeological data[78–80]. In addition, vast trading networks between cultural groups existed in pre-invasion Australia, where individuals and groups regularly travelled long distances for trade and ceremonial gatherings[32,77,81–83]. Large congregations were observed, with estimates ranging from 100 to >2,500 individuals containing between 20 and 120 members from each nation or group[32,84]. Trading networks covered distances as great as 6,000 km and traversed some of the most inhospitable regions of the continent[32,85,86]. Thus, we incorporated this aspect into the model to predict the mobility between patches.

Mobility process

We consider M total patches. Individuals from patch i leave on visits to other patches at a per capita rate μ_i per unit time. The visitors are distributed among the $M-1$ destinations with probabilities σ_{ij} to each destination patch j. In our model, only neighbouring patches have non-zero entries. The σ_{ij} are conditional probabilities of visiting another patch, thus $\sigma_{ij} \in [0, 1]$ for $i \neq j$ and $\sigma_{ii} = 0$. In addition, $\sum_j \sigma_{ij} = 1$. Travellers from patch i to j have a per capita return rate to region i of ρ_{ij} and, by definition, $\rho_{ii} = 0$. When an individual is in the infectious stage, we assume no movement occurs; therefore, we set both μ and ρ to zero in these cases (Supplementary Section A).

The mobility model captures the effects of regular movement between cultural groups for trade and ceremony, but not for long-term migration. Therefore, each person is assigned a residency patch. Each patch is further partitioned into family groups or households (hearths) that represent the clusters of individuals who sit and sleep together[87]. At a fixed rate, individuals then move to neighbouring patches to visit and mix with both residents and other visitors, eventually returning to their home patch. To account for both regular trade and less-frequent, larger intergroup gatherings, we define the probability of movement as a sum of two components[87–89]. The first component represents regular movement between patches for trade that occurred each year. We approximate this for individuals in the ith patch as the quotient $(a\ T_t)/(365\ N_i)$, where a is the average number of trips per year per participating household and T_t is the total number of individuals from each patch who engage in trade (Supplementary Section C). The second part represents ceremonial gatherings that occurred less frequently but included more individuals. This is approximated for the ith patch as the quotient $(b\ T_c)/(365\ N_i)$, where b is the average number of ceremonial trips per biennium per participating household and T_c is the total number of individuals from each patch that participate in ceremonies. Thus, the expected number of movements per person per day in patch i is

$$\mu_i = \frac{a}{365}\frac{T_t}{N_i} + \frac{b}{730}\frac{T_c}{N_i}. \quad (1)$$

In both components, the effective number of participants who engage in either activity from patch i is defined as $T_l = (f_l N_i / s)^v$, $l \in \{t, c\}$, where f_l is the fraction of representatives from each household that participate in the activity, s is the mean household size and v is a scaling exponent. Throughout our analysis, we assume that $f_t = 0.25, f_c = 0.80, s = 6, a = 8$, $b = 1$ and $v = 0.75$ [89,90] (a full discussion of the details is provided in Supplementary Section C)

The movement of exposed and infective individuals is a main component of the spread of smallpox and the conclusions of our model. It is likely that individuals moved differently during times of fear; however, this aspect would be difficult to capture in a model of this size. Our aim is to show a worst-case scenario for the spread of smallpox from potential entry points to Australia in 1789. We therefore decided to incorporate this 'shock and fear' into the movement probability by imposing exaggerated values.

Modelling the spread of smallpox

We consider an S-E-I-R multipatch epidemic model with movement of healthy individuals between patches. In this framework, $S_{ij}(t)$, $E_{ij}(t)$, $I_{ij}(t)$ and $R_{ij}(t)$ denote the number of susceptible, exposed, infective and removed ('recovered') individuals, respectively from patch j who are present in patch i at time t; thus, if we set $N_{ij}(t) = S_{ij}(t) + E_{ij}(t) + I_{ij}(t) + R_{ij}(t)$, $i, j = 1, 2, \ldots, M$, the total population in patch i at time t is $N_i(t) = \sum_j N_{ij}(t)$ and the area of patch i is denoted A_i. We simplified the analysis by considering the compartment R as 'removed' (cf. 'recovered'), which includes all those who are no longer part of the infection dynamics. Disease transmission is density dependent, so the rate of contact between individuals depends on the population density and size of each patch.[91,92]. We summarize a complete list of all events and their rates in Table 1 (full details in Supplementary Section C).

Victims of smallpox experience several distinct stages of symptoms during the infectious period with varying infectivity. To incorporate this fluctuating infectivity, we approximate the period to be 18 days, which includes 14 days of maximum infectivity, 1 day of the early stage with low infectivity and 3 days of the last stage with medium infectivity. Thus, the transition rate from the infectious to recovered compartments is $\gamma = 1/18$ (parameter estimates listed in Supplementary Table 3).

We applied the model to all the patches within each of four regions along the northern coast of Australia (Kimberly, North, Arnhem and Gulf) and from Sydney in the southeast to test the two primary hypotheses for smallpox introduction. In each patch, we introduced a single, exposed individual with several fixed parameters to determine the most likely path of an epidemic and the conditions necessary for it to be observed in Sydney in 1789.

Sensitivity analysis

We completed a global sensitivity analysis using Latin hypercube sampling to generate 200 joint samples of the 12 parameters governing disease transmission and spatial dispersal between patches and evaluated the model for each sample (full details in Supplementary Section D). The epidemiological parameters α, γ and β control the duration of infectious stages and the successful contact rate between susceptible and infectious individuals. Dispersal-related parameters ρ, a, b, v, f_t, f_c and s represent movement out of a patch, the rate of return home and the average household size. To examine the role of long-distance movement, we also relaxed the assumption of strictly adjacent dispersal by introducing two additional parameters: λ, which governs the spatial decay of movement probability with distance, and ε, which determines the proportion of movement that is designated as long-distance.

Overall, parameters strongly associated with the basic reproduction number $\mathcal{R}_0$, β and γ, contributed the most to variability in epidemic outcomes, namely the total number of infected individuals and the number of infected patches. Mobility scaling v and transmission rate β also had substantial influence through nonlinear interactions, highlighting the interdependence between contact processes and movement. In contrast, distance-related dispersal parameters λ and ε had minimal influence in both linear and nonlinear analyses, suggesting that long-distance movement within reasonable bounds has limited impact on outbreak size and spatial extent in this model.

Ethics and permissions

We obtained ethical approval for this study from the Flinders University Human Research Ethics Committee before data extraction and analysis (ethical clearance no. 8389). Australian university Human Research Ethics Committees are appointed legal authorities under Commonwealth of Australia law to assess and approve research in accordance with the National Statement on Ethical Conduct in Human Research and are

registered with the National Health and Medical Research Council of Australia. This authority includes responsibility for determining the ethical acceptability of research involving, or having implications for, Aboriginal and Torres Strait Islander peoples.

Consistent with the AIATSIS Code of Ethics for Aboriginal and Torres Strait Islander Research principles of Indigenous self-determination, Indigenous leadership, impact and value, and sustainability and accountability, Aboriginal community representatives and Indigenous scholars identified the need for the project and contributed to its design, including refinement of research aims, ethical considerations and dissemination pathways. Indigenous co-authors Shane Ingrey, Kirsty Beller, Lynette Russel and Christopher Wilson contributed cultural knowledge, historical context and guidance on ethical engagement throughout the study. Lynette Russell, drawing on her Aboriginal heritage and extensive expertise in Indigenous histories, also contributed to the interpretation and contextualization of the research. Their involvement ensured that the research questions, the analysis of findings and the consideration of community impacts were grounded in Indigenous perspectives.

The author team includes both Indigenous and non-Indigenous researchers working across disciplines including ecology, mathematics, archaeology, history, epidemiology and Indigenous studies. In addition to their scholarly contributions, Indigenous collaborators provided cultural and historical expertise and guidance on ethical engagement, while non-Indigenous authors recognize their positionality as researchers working within Australian academic institutions and the responsibilities associated with doing research on Indigenous histories.

Data availability

All relevant data for the analyses and results presented are available via GitHub at github.com/mcnitschke/Smallpox-in-Aboriginal-Australia.

Code availability

All relevant MATLAB code for the analyses and results presented are available via GitHub at github.com/mcnitschke/Smallpox-in-Aboriginal-Australia.

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Acknowledgements

We thank the sovereign Traditional Owners and custodians (First Nations) of the unceded lands, seas and skies where we live and work, including Kaurna in Tarndanya/Adelaide (M.C.N., C.J.A.B., F.S. and S.H.F.), Dharawal/Sydney (A.N.W., N.P. and A.B.), Dharawal in Kamay/Botany Bay (K.B. and S.D.I.), Kulin Nation in Naarm/Melbourne (B.G., I.J.M. and L.R.), Yirrganydji and Gimuy Walubara Yidinji in Gimuy/Cairns (S.U. and M.I.B.), Dharawal in Woolungah/Wollongong (F.S.), Ngarrindjeri of Murrundi/lower Murray River, Kurangk/Coorong and eastern Fleurieu Peninsula (C.W.), Palawa in Nipaluna/Hobart, lutruwitra/Tasmania (C.W.), and Peramangk in Bukatila/Mount Lofty Ranges (C.J.A.B.). We also recognize the deep historical and cultural harm our truth-telling exposes, and we commiserate with all First Nations peoples of Australia. This study was approved by the Flinders University Human Research Ethics Committee (project no. 8389). Computational support was provided by Deep Thought, the high-performance computing facility at Flinders University[93]. We also thank J. Llewelyn for valuable early discussions.

Author contributions

M.C.N. performed the analysis and wrote the first draft of the paper, with contributions from C.J.A.B. and A.N.W.; S.U., S.D.I., N.P., A.B., L.R., I.J.M. and B.G. provided data. S.U., L.R., M.I.B., C.J.A.B., F.S., A.B., I.J.M. and C.W. obtained funding. All authors reviewed, edited and provided contributions to the final manuscript.

Funding

Funded jointly by the Australian Research Council Centre of Excellence for Indigenous and Environmental Histories and Futures (grant no. CE230100009) and the Australian Research Council Centre of Excellence for Australian Biodiversity and Heritage (grant no. CE17010001). L.R. is supported by an Australian Research Council Laureate Fellowship (grant no. FL190100161). B.G. is supported by an Australian Research Council Discovery Early Career Researcher Award (grant no. DE220100203). A.B. and N.P. are supported by an Australian Research Council Laureate Fellowship (grant no. FL200100144).

Competing interests

The authors declare no competing interests.

Additional information

Supplementary information The online version contains supplementary material available at https://doi.org/10.1038/s41562-026-02504-6.

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Peer review information *Nature Human Behaviour* thanks George Taiaroa and the other, anonymous, reviewer(s) for their contribution to the peer review of this work. Peer reviewer reports are available.

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