



Profiling a possible rapid extinction event in a long-lived species

Ricky-John Spencer^{a,b,*}, James Van Dyke^a, Kristen Petrov^a, Bruno Ferronato^c, Fiona McDougall^a, Murray Austin^a, Claudia Keitel^d, Arthur Georges^c

^a School of Science and Health, Western Sydney University, Locked Bag, 1797, Penrith South DC 2751, NSW, Australia

^b Hawkesbury Institute for the Environment, Western Sydney University, Locked Bag, 1797, Penrith South DC 2751, NSW, Australia

^c Institute for Applied Ecology, University of Canberra, ACT 2617, Australia

^d School of Life and Environmental Sciences, University of Sydney, 380 Werombi Road, Brownlow Hill, NSW 2570, Australia



ARTICLE INFO

Keywords:

Turtle
Myuchelys georgesi
Pathogen
Virus
Climate change
Epidemic

ABSTRACT

Infectious disease is a contributing factor to species extinction or endangerment. Extinction is most likely to occur when a pathogen is evolutionarily novel, or when it utilizes an alternate reservoir, or when hosts have small pre-epidemic populations. Here we focus on such a case where a mystery disease almost drove the range restricted, Bellinger River Snapping Turtle (*Myuchelys georgesi*), in north-eastern NSW, Australia, to extinction in less than a month. The disease did not affect other turtle species, and the juvenile population of *M. georgesi* appears unaffected. The cause of the disease remains unknown, but may be a novel virus and whether the species can recover with or without human intervention is currently unknown. Here, we review the biology of *M. georgesi* and explore the epidemiology of the disease. We highlight circumstantial evidence of the potential role of recent environmental change in the susceptibility of *M. georgesi*. We show that long-term regional warming and localised drying reduced both water levels in the river and the number of flooding events that occurred prior to the disease outbreak. We also provide evidence that the food web may have been disrupted, possibly causing malnutrition and reduced immunocompetence of sub-adult and adult turtles. We hypothesize that these factors may have exacerbated the virulence and contagiousness of a novel, but as-yet unidentified pathogen, and must be also mitigated in any future recovery actions. The identity of the pathogen is necessary for managing the recovery of the species, however, understanding the processes that rendered the species susceptible to infection is of equal importance for planning the recovery of the species from the brink of extinction.

1. Introduction

Infectious disease is infrequently listed as a contributing factor to species extinction or endangerment. The IUCN Red List (Baillie et al., 2004) reports that in the past 500 years, 100 plant and 733 animal species are known to have gone extinct. Of these 833 known extinctions, only 31 cases (3.7%) have been attributed, at least in part, to infectious disease. Whereas some forces, such as habitat loss or over-exploitation, are listed as the single causal driver of a species' extinction, in no case is infectious disease listed alone (Baillie et al., 2004). Amphibian pandemics caused by Chytrid fungus (*Batrachochytrium dendrobatidis*) may be an exception, but Chytrid extinction risk may also be worsened with climate change (Pounds et al., 2006). This raises the possibility that infectious disease is less likely than other drivers of species extinction to act in isolation. It is critical that we combine evidence with theory to identify the circumstances under which infectious disease is most likely to serve as an agent of extinction.

Extinction is most likely to occur when a pathogen is evolutionarily novel to a susceptible host species, or when it utilizes an alternate reservoir (biotic or abiotic), or when hosts have small pre-epidemic populations (De Castro and Bolker, 2005; Gerber et al., 2005).

Identifying the cause of wildlife diseases is difficult because single factors can rarely be identified as solely responsible. In addition to immune suppression related to elevated stress responses and pollutant exposure, environmental change can impinge directly on wildlife health, and may affect population viability in intricate ways. For example, climate-related shifts in pathogen and host ranges, and pathogen spillover among species brought into contact in novel ways, can increase exposure to new diseases (reviewed in Smith et al., 2009). Similarly, changes in habitat size or quality can lead to reductions in prey populations and increased competition for resources (Ryall and Fahrig, 2006), which in turn can cause malnourishment or starvation and increased susceptibility to disease. Effects may be further complicated if the genetic diversity of the species is low, as low genetic diversity has

* Corresponding author at: School of Science and Health, Western Sydney University, Locked Bag, 1797, Penrith South DC 2751, NSW, Australia.
E-mail addresses: r.spencer@westernsydney.edu.au, <https://twitter.com/emydura5> (R.-J. Spencer).

been correlated with reduced fitness and lowered evolutionary potential (Spielman et al., 2004).

In the summer of 2014/2015 in Australia, two closely related species of turtle suffered mass mortality event. An unusual mortality event in Johnstone River Snapping turtles (*Elseya irwini*) in Far North Queensland, Australia, occurred during the summer months of December 2014 and January 2015 (Ariel et al., 2017). Moribund animals appeared lethargic with variable degrees of necrotising dermatitis and while the primary etiology of the disease remains unknown, environmental conditions at the time may have played some part, as the conditions were hot and dry and water levels were extremely low (Ariel et al., 2017). Similarly, on the North Coast of NSW, over 2000 km away from the North Queensland mortality event, over 400 Bellinger River Snapping Turtle (*Myuchelys georgesi*) were also found lethargic with variable degrees of necrotising dermatitis and similarly, the primary etiology of the disease remains unknown (Moloney et al., 2015). Importantly, environmental conditions were hot and dry and water levels were extremely low. *Myuchelys georgesi* only exists in the Bellinger River catchment and the mortality event was also a possible extinction event.

Our study reviews the published literature on *M. georgesi* and explores the epidemiology of the unknown disease to determine whether Bellinger River Snapping turtles were particularly susceptible to viral infection. In lieu of peer-reviewed information regarding the novel pathogen itself, we aimed to identify potential associated risk factors that may have reduced immunocompetence of the species. In particular, we sought to identify trends in environmental factors, including temperature, rainfall, and river level, which may have created poor biophysical conditions for *M. georgesi* survival. We also use stable isotope and body condition analyses to determine whether turtles have experienced nutritional deficits prior to the disease outbreak. Finally, we use population viability modelling to determine the chances of extinction of the species under several management scenarios. Our study is admittedly preliminary, and our ultimate aim is to identify any potential environmental deficits that must be mitigated simultaneously with pathogen management to prevent extinction of *M. georgesi*. We hope that our approach and results may also be useful in preventing similar rapid extinction events in other species.

1.1. The event: epidemiology of the disease (summarized from Moloney et al., 2015)

A severe mortality event in Bellinger River Snapping Turtles (*Myuchelys georgesi*) was investigated on February 18, 2015 by Bellingen Shire Council (BSC), Environment Protection Authority (EPA), and National Parks and Wildlife Service (NPWS) following a report of dead or dying turtles on the side of the Bellinger River from local kayakers. An estimated 432 *M. georgesi* were captured with symptoms of a disease or were found dead. Most were slow moving and apparently blinded by inflammation of the eye and surrounding tissues. No other species appeared affected (e.g., native *Chelodina longicollis*, exotic *Emydura macquarii*).

Turtles were initially treated by veterinarians but because of high rates of mortality and potential biosecurity risk, ill turtles were humanely euthanased (Moloney et al., 2015). The Australian Registry of Wildlife Health at Taronga Conservation Society Australia conducted gross and histological examinations of affected animals collected from the riverbank, and coordinated the diagnostic investigation. Turtles displaying initial signs of disease were emaciated and had swollen eyes (Fig. 1), had slight clear nasal discharge, and some animals had hind limb paresis. Necropsy and histopathology revealed turtles were thin and had numerous additional symptoms associated with bacterial infection, but these infections were assumed to not be the primary cause of mortality (Moloney et al., 2015). A range of infectious pathogens were excluded as the primary pathogen for the disease including Ranavirus, adenovirus, paramyxovirus (ferlavirus), herpesvirus, mycoplasma, chlamydia, and trichomonas (Moloney et al., 2015). No toxins



Fig. 1. External symptoms of the disease in *M. georgesi*. (Source: Photo Credit: Rowan Simon).

or environmental contaminants were detected, but only water quality was investigated, and the potential for *M. georgesi* to bioaccumulate contaminants via diet (e.g., Hopkins et al., 2002) is unconfirmed. Scientists at the Elizabeth MacArthur Agricultural Institute (EMAI) Virology Laboratory have recently (July 2015) detected high levels of a novel virus in tissues of affected turtles (Moloney et al., 2015). However, the identity of the virus, and the data supporting its discovery, have not been published at this point.

After initial discovery, the disease appeared to be propagating upstream at a rate of ca 2 km per day (Moloney et al., 2015). Surveys began in the upper sections of the River to remove healthy individuals from 8th–12th April 2015. Seventeen healthy individuals of a range of sizes and sexes were removed from the top of their range in the River and relocated to a quarantine facility for holding and breeding. Subsequent surveys not long after the removal of these virus-free or asymptomatic adult turtles revealed dead turtles from the same waterholes.

1.2. Post-mortality developments

From a scientific point of view, very little has emerged about the remaining population and the disease since the release of Moloney et al. (2015). A press release suggested the disease was a “Mystery Virus” in September 2015, based on the Moloney et al. (2015) report (ABC News, 2015). However, data supporting this assertion remain unpublished. Twenty juveniles were collected in limited surveys of the river in November 2015 (Bellingen Courier, 2015) and a survey in March 2016 confirmed that the remaining turtles in the River are predominately juveniles (Bellingen Courier, 2016).

Myuchelys georgesi is restricted to the Bellinger and adjacent Kalang rivers, and its total population size in the larger Bellinger River was previously estimated at only 4500 (Blamires et al., 2005). Subsequent density estimates from surveys in 2007 and 2014 (Spencer et al., 2007; Spencer et al., 2014) suggested a population size half of that value. Regardless, decreasing population sizes and a severely restricted distribution may have combined to make the Bellinger River Snapping Turtle vulnerable to a novel pathogen, particularly if populations were stressed or malnourished prior to infection.

2. Material and methods

We combine a classical ecological theory approach with forensic analyses by 1) summarizing all current literature on *M. georgesi*, including both scientific and public media reports about the disease 2) drawing from historical data on the species to determine potential long-term changes in population dynamics and individual health of turtles, and 3) reviewing long-term environmental data on temperature, rainfall, and river levels.

Historical data on the species stems from a range of studies that have occurred inconsistently since 2000. The last systematic survey was

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