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Genetic variation in resistance and high fecundity impede viral biocontrol of invasive fish

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Abstract

1. Common carp *Cyprinus carpio* is one of the top global invasive vertebrates and can cause significant ecological damage. The Australian Government's National Carp Control Program (NCCP) proposes to release Koi herpesvirus (KHV) to eradicate feral carp in one of the largest ecological interventions ever attempted. Ecological and human health risks have been highlighted regarding the release of a highly pathogenic viral biocontrol for an aquatic species. The efficacy of KHV has also been questioned, and it has not been demonstrated to produce lasting population reductions.
2. We developed an individual-based model (IBM) to examine the ecological and evolutionary response of a carp population after KHV release. This simulated the interaction between fish life history, viral epidemiology, host genetic resistance and population demography to critically evaluate the impact of KHV release under optimal conditions and a 'best-case scenario' for disease transmission.
3. KHV will rarely result in prolonged reductions or population extinctions. Crucially, realistic scenarios result in a rapidly rebounding population of resistant individuals. Additional measures aimed to reduce carp population recovery rate (e.g. with genetic engineering) require rapid efficacy to significantly reduce carp numbers alongside KHV.
4. Fish fecundity has an overwhelming influence on viral efficacy as a biocontrol agent when combined with genetic resistance within a population. A high probability of population extinction is only met when carp fecundity is reduced to 1% of biological observations.
5. *Synthesis and applications*. We use an individual-based model to evaluate the efficacy of Koi herpesvirus biocontrol in Common Carp, and find that high host fecundity combined with genetic resistance results in rapid population rebound after initial large fish kills. Biocontrol approaches relying on natural selection lose efficacy over successive generations as resistance genes increase in frequency. Given the intense logistical effort and risks to ecosystems and human health associated with large fish kills after viral release, we suggest that sustained manual removal, alongside ecological restoration to favour recovery of native species, provides a risk-free approach to reducing populations.

KEYWORDS
biocontrol, carp, disease ecology, invasive, Koi herpesvirus, pest, population control, virus

1 | INTRODUCTION

Introduced species can disrupt evolutionary and ecosystem processes, and drive declines in native fauna (e.g. David et al., 2017). However, population control programmes can be complex, posing further risks to ecosystem health, and minimizing these risks requires thorough planning. Biocontrol involves the intentional release of a natural enemy (e.g. an exotic predator or parasite), which targets a pest species while avoiding negative impacts on native fauna. Some attempts have been successful (e.g. removing feral cats from Marion Island; Bester et al., 2002), while others have been catastrophic failures (e.g. the introduction of cane toads to Australia; Shine & Phillips, 2014). Only 10% of 6,158 programmes that introduced insects as biocontrol were considered success (Cock et al., 2016), illustrating the difficulties associated with such approaches. Understanding the efficacy and impact of biocontrol requires broad biological analysis across targeted populations, from molecular to ecosystem processes. This is especially important when considering the release of an infectious agent to induce rapid uncontrolled mortality in a target species.

Ecological modelling is an important component in risk and efficacy assessments of proposed control measures (Forbes et al., 2011; McLane, Semeniuk, McDermid, & Marceau, 2011). Modelling is crucial in developing strategies to release a highly pathogenic virus into natural populations, which may have many unexpected or unintentional ramifications for ecosystems that cannot be safely tested in natural systems before deployment. Such is the case for a proposal by the Australian Government's National Carp Control Program (NCCP), which aims to release *Cyprinid herpesvirus-3*, or Koi herpesvirus (KHV), into the continent's largest water system, the Murray–Darling Basin, in attempt to eradicate feral Common Carp *Cyprinus carpio* (Figure 1).

Common Carp is a globally widespread introduced species, driven by its popularity in aquaculture (Rahman, 2015). While the majority of carp populations reach an ecosystem equilibrium and become naturalized, they can disrupt ecosystem processes and have negative impacts of native flora and fauna (e.g. Kloskowski, 2011). Indeed, carp can cause significant ecological damage in the Murray–Darling (Angeler, Álvarez-Cobelas, Sánchez-Carrillo, & Rodrigo, 2002; Vilizzi, Thwaites, Smith, Nicol, & Madden, 2014), but evidence suggests that their role in Australian ecosystems is varied and complex (Weber & Brown, 2009), and can even be beneficial (Marshall et al., 2019). A century of mismanagement has degraded this complex mosaic of ecosystems, arguably reducing favourable habitat for comparatively more sensitive native fish species (e.g. Murray Cod, *Maccullochella peelii*, Silver perch, *Bidyanus bidyanus*). These man-made changes have favoured the more ecologically tolerant and adaptable carp populations (Cadwallader, 1978; Gehrke, Brown, Schiller, Moffatt, & Bruce, 1995; Koehn, 2004; Marshall et al., 2019; Murray–Darling Basin Ministerial Council, 2003; Nicol, Lieschke, Lyon, & Koehn, 2004; Weber & Brown, 2009). To date, the NCCP has produced multiple reports to inform policymakers on whether to release KHV (Beckett, Caley, Hill, & Henderson, 2019; Brookes & Hipsey, 2019; Brown, Wisniewski, & Gilligan, 2019; Durr et al., 2019; Nichols, Gawne, Richards, Lintermans, & Thompson, 2019; Roper & Ford, 2018; Silva, Bell, & Baumgartner, 2017; Stuart et al., 2019; Tilley et al., 2019; Todd et al., 2019; Wedekind, 2019). However, many important questions still remain unanswered in the development of a safe and effective species control programme

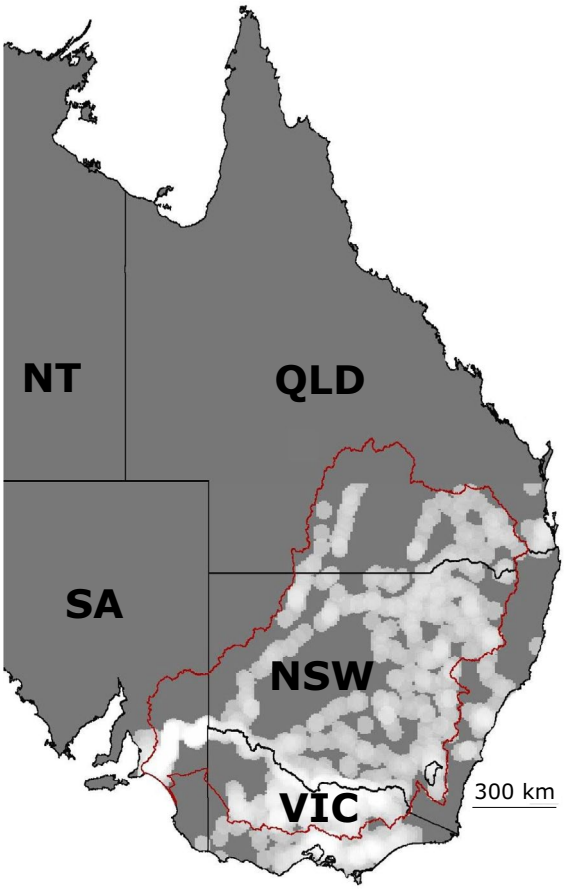

FIGURE 1 Carp densities (white shading) vary throughout the Murray–Darling Basin (red outline), within New South Wales (NSW), Queensland (QLD), Victoria (VIC) and South Australia (SA). The highest densities of carp are seen in the lower reaches of the drainage, bordering NSW and VIC, and especially towards the river mouth, which drains into the Great Australian Bight through SA. Carp are absent from the Northern Territory (NT), with relatively few isolated populations in Western Australia. Map data www.nationalmap.gov.au/about.html. Carp density data (2000–2018) www.ala.org.au

(Falconer, Coleman, Loh, Rocliffe, & Lighten, 2018), and this requires extensive empirical and simulated data to critically assess potential impacts and efficacy of high-risk approaches to control carp populations (Boutier et al., 2019; Kopf et al., 2017; Lighten & van Oosterhout, 2017; Marshall et al., 2018). Manual removal of carp using cage traps and nets, combined with breeding ground sabotage may be a successful strategy, and carp have now been eradicated from Tasmania using this approach, although this represents a 25-year continual effort in just a few isolate lakes (Kelly, 2020). Thus, targeted and sustained manual removal certainly holds the potential to significantly reduce carp numbers from Australian waters (Stuart & Conallin, 2018). The NCCP has speculated on the use of genetic manipulation of carp populations as a complementary approach to biocontrol (e.g. the introduction of genetically modified daughterless carp; Thresher et al., 2014, or the use of CRISPR Cas-9; McColl, Sunarto, & Neave, 2018, Wedekind, 2019), which can skew sex ratios, resulting in population declines. However, these genetic tools are complicated, requiring years of dedicated research and development, risk assessment and public consultation associated with the release of genetically modified organisms, something which the NCCP is currently not perusing. The entire focus of the NCCP mandate comprises the targeted release of KHV on spawning aggregations, with localized clean-up efforts, followed by unmanaged spread of the virus through the occupied range of carp to produce a significant knock-down in carp numbers.

Koi herpesvirus is a DNA virus, which induces a disease that is notifiable under the World Organization for Animal Health (OIE). The virus emerged in European carp aquaculture facilities during the late 1990s (Haenen, Way, Bergmann, & Ariel, 2004) and rapidly spread around the world causing millions of dollars of losses among the industries surrounding carp for consumption, ornamental pets (Koi) and angling. Substantial risks to ecosystem and human health have been raised since the announcement of the NCCP's proposal (Becker, Ward, & Hick, 2018; Falconer et al., 2018; Lighten & van Oosterhout, 2017; Marshall et al., 2018). The NCCP's plans have been defended (McColl, Sheppard, & Barwick, 2017; McColl et al., 2018), which, in turn, has also been criticized (Boutier et al., 2019). Moreover, infection trials conducted on fish from the Murray–Darling Basin (McColl, Sunarto, et al., 2017) were of inadequate size to draw any meaningful conclusion on the efficacy of the KHV strain proposed for deployment across the genetically diverse carp populations in Australia. Shortcomings of the research plan have also been identified and discussed in the Australian Senate (Falconer et al., 2018). Importantly, the final report produced by the NCCP for the Government to inform a decision on viral release has been kept from public view, yet the constituent reports leave crucial scientific questions unanswered. Critically, the NCCP has released no genetic-based epidemiological modelling to assess the short- and long-term impacts of the release of KHV in carp populations. Given the significant risks associated with the release of foreign pathogens, we believe this should be a prerequisite in any biocontrol programme.

This study focuses on understanding how Australian carp populations may be affected by the release of KHV under favourable conditions producing a 'reasonable best-case scenario'. Carp biomass can reach extraordinarily high densities in Australia, exceeding 3,000 kg/ha (Harris & Gehrke, 1997) and so represent unprecedented epidemiological conditions for KHV in natural environments. The spread of a directly transmitted pathogen like KHV through the host population is determined by its reproductive ratio (*Ro*), which is the product of transmission efficiency, the contact rate, and the duration that an infected host is contagious (Dietz, 1993). The very high host density in Australia is likely to elevate *Ro*, potentially resulting in boom-bust population dynamics that could eradicate the virus before eradicating the host. Given the high reproductive potential of carp, the population number could rapidly rebound after the virus has gone extinct. In addition, significant variation exists in the world's carp population in tolerance and (partial) resistance to KHV, which could result in adaptive evolution of KHV tolerance and/or resistance. To address the impact of these important unknowns on biocontrol efficacy, we developed the first individual-based model (IBM) that incorporates both the ecological and population genetic characteristic of Australian carp populations, and KHV epidemiology. In particular, we employ this IBM to simulate a targeted viral release in a single population, testing the hypothesis that natural genetic resistance to KHV combined with the high fecundity of carp results in KHV deployment having a negligible long-term effect on carp numbers. To this end, we have examined the rate of population recovery after different amounts of mortality, and tracked the evolutionary response of this population under the known genetic model of KHV resistance.

2 | MATERIALS AND METHODS

An IBM was developed in NetLogo 6.0.1 (Wilensky, 1999) to simulate realistic population dynamics of carp in Australia, and to assess the population-level effects of KHV exposure. Specifically, the model explored the compensatory role of population density of disease-resistant individuals in the resilience of populations exposed to KHV. The model is described following the overview, design concepts and details (ODD) protocol (Grimm et al., 2006, 2010). The Overview section is described in the main paper, and the 'Design Concepts and Details' sections in Appendix S2.

2.1 | Entities, state variables and scales

The entities in the model are the spatial units comprising a grid-based waterscape with non-overlapping habitat patches and individual fish, and the time step is one day. The overall environment is characterized by breeding season, implicitly representing seasonal temperature variation, which affects fish movement, reproduction and KHV epidemiology (Gilad et al., 2003; Yuasa, Ito, & Sano, 2008). During the breeding season, water temperature in the model remains constantly within the optimum temperature range to induce carp breeding behaviour and viral replication;

22–24°C (Gilad et al., 2003; Hedrick et al., 2000). Because water temperature is the most important factor affecting ectothermic fish physiological processes and KHV replication, excluding temperature variation allowed us to explicitly examine the effects of genetic resistance and fecundity on population recovery under a best-case scenario for viral spread. In contrast to European carp populations, the Australian breeding season is peculiarly long, spanning 6 months. Rather than just a single spawning event, Australian carp populations can spawn three times each year (Sivakumaran, Brown, Stoessel, & Giles, 2003; Smith & Walker, 2004). Spatial units are characterized by the state variables habitat type: non-breeding ground and breeding ground, as carp will congregate in shallower areas when aggregate broadcast spawning is facilitated by physical agitation from male harassment. Spatial units are additionally characterized by presence/absence of KHV; infected patches maintain the virus so that individuals inhabiting that patch can become infected with a given probability (see below). The scale of the waterbody is user defined, and here represents a section of river/lake measuring 0.1 ha divided into 1,000 patches, each measuring 1 m (length) × 1 m (width). The breeding ground makes up 25% of the waterscape, which can only be accessed by spawning adult fish in the breeding season. The model is representative of an enclosed population, or species management unit observed in the Murray–Darling Basin (Haynes, Gilligan, Grewe, & Nicholas, 2009).

Individual fish have three life stages: egg/sperm, juvenile and adult. All carp are characterized by the state variables age [days post-fertilization (dpf) for eggs, and days post-hatch (dph) for the remaining life stages] and position within the waterscape grid. Juveniles and adults are characterized by length (cm, total length from the snout to the tip of the tail), body weight (wet weight, g) and sex (male or female). Adults possess the state variable breeding status (spawning or non-spawning), which is determined by an inter-spawning interval. Females possess the state variable batch size (eggs per spawning event), which is determined from fish length (cm), and males are characterized by sperm number (a constant; independent of body length in the model).

Every individual (juvenile or adult) that comes into contact with the virus (via direct contact with infected individuals or patches) will get infected. Infected individuals will spread KHV to all other individuals within the same patch, and they will make a patch 'infected'. Each infected individual is classed as either resistant (i.e. individuals survive infection) or susceptible to disease (individuals succumb to infection and die). Individuals are diploid and possess a user-defined number of immune genes at non-linked loci; here between 1 and 32 (recombination rate between loci: $r = 0.5$). Alleles are additive and offer partial resistance to KHV, and each has a resistance value equivalent to $1/2L$, where L is the number of immune genes simulated. KHV resistance of an individual (ω) is equal to the additive effect of alleles across these immune genes, that is, resistance values of all alleles are summed across all 2–64 alleles of an individual. This means that individuals with all 'resistant' alleles were completely resistant to KHV, and individuals with all 'susceptible' alleles were completely susceptible. Importantly, Common Carp hybridize freely with Goldfish in the Murray–Darling Basin (Haynes, 1999), and with goldfish being resistant to KHV (Yuasa, Sano, & Oseko, 2013), experimental studies have shown that this results in around 95% of Goldfish × Common Carp hybrids in being KHV resistant (Hedrick, Waltzek, & McDowell, 2006). Moreover, selective breeding for KHV resistance has shown that within a few generations the heritable component of KHV resistance can result in the majority of family member being resistant to KHV (Tadmor-Levi, Hulata, & David, 2019), and thus some individuals in populations are assumed to be completely resistant to KHV at the genetic level. Experimental trials of carp infected with KHV show that resistance alleles act largely additively (Palaiokostas et al., 2018; Tadmor-Levi et al., 2019), and our model is consistent with these data. If ω is less than a randomly drawn number between 0 and 1, the individual is susceptible and dies. We modelled between 50% and 95% mortality rates of infected fish, and monitored the adaptive evolution of allele frequencies and resistance over time.

2.2 | Process overview and scheduling

Each of the following processes (in bold) will occur over each time step in sequential order. The environment (date and breeding season) is updated. Juveniles and adults are subjected to **viral interactions/effects and disease survival** where the immunocompetence of an individual determines its likelihood of surviving KHV exposure. Juveniles and adults **move** across the landscape randomly, occupying appropriate habitat patches, where multiple individuals can occupy a patch per time step. All individuals occupy non-spawning patches outside of the breeding season, and only spawning adults occupy spawning patches in the breeding season. All life stages undergo **general survival** where an individual's daily mortality rate is determined by four main factors: developmental mortality (eggs only), senescence (adults only), density-dependent recruitment (juveniles only), and a general size-dependent mortality rate, which represents all other sources of mortality except KHV-induced mortality (adults). Juveniles and adults **grow** by increasing body length and mass each day, where length is determined by age, and mass is allometric to length. Adults undertake **reproduction** in the breeding season. Gametes receive a randomly drawn allele of each of the L immune genes of the parent, which randomly associate during broadcast spawning events to determine offspring genotype and resistance. If an adult individual has spent more than the between-spawning interval time not breeding, they will enter the spawning area and release haploid gametes once a day for 7 days. After 7 days, they return to the non-spawning area for the duration of their inter-spawning interval. The number of eggs produced by a female is proportional to body length, and ranges from 0.12 to 0.18 million eggs per year per female. A random sperm from the spawning area is chosen to fertilize an egg to produce a diploid embryo at a rate of 0.79 (Bozkurt & Öğretmen, 2012). Total egg survivorship was estimated at 1% (see Appendix S1). Finally, eggs and juveniles **develop** and age. Eggs develop into juveniles after 4 days post-fertilization. Juveniles develop into adults once they reach the specified length at maturity. Full model equations are given in Table S1 (Appendix S1).

2.3 | KHV transmission and simulation experiments

We modelled direct exposure of 10% of random adult individuals as the route of viral introduction, simulating the introduction of KHV-injected individuals into high-density breeding aggregations. Observations of KHV incubation period varies in accordance with water temperature, occurring over 7 days in the upper range of permissible temperatures, and as long as 21 days at lower temperatures (Haenen et al., 2004). We set the viral incubation period to 5 days for a best-case scenario of viral spread at a constant optimal temperature. Individuals spread the virus during this time and died after 5 days of infection if carrying a susceptible genotype. Viral particles were transmitted between individuals that occupy the same grid space, and particles that were shed into the water column, remained in the grid space for 3 days (Shimizu, Yoshida, Kasai, & Yoshimizu, 2006), which could then enter individuals that co-occupied the same position. If an individual died from the virus, it remained in the same position for 3 days and continued to spread the virus to individuals that came into contact with the infected grid space. On the last day of the breeding season, infected individuals carrying the virus but had not succumbed before the end of the breeding season then exited the breeding grounds and carried the virus as a latent infection until it could then be transmitted to a naïve individual on the first day of the next breeding season. We found that due to its rapid spread, the model was insensitive to the number of individuals that carried the latent virus between breeding seasons (Appendix S1). Individuals that survived infection were then resistant to future infections irrespective of genotype, in accordance with antibody production and acquired immunity observed after KHV infections (Adkison, Gilad, & Hedrick, 2005; Perelberg, Ilouze, Kotler, & Steinitz, 2008; Ronen et al., 2003). We did not include any costs in physiology, reproductive success or survivability in carp due to resistance, as previous studies have indicated that selection for KHV resistance in carp has no negative impact on survival, growth and size (which is allometrically correlated with fecundity; Ødegård et al., 2010; Zhao et al., 2020), similar to disease studies in other fish (e.g. Bassini et al., 2019; Yáñez et al., 2014). Full details are shown in Appendices S1 and S2.

3 | RESULTS

Even when 95% of all KHV-susceptible carp (adults and juveniles) were killed annually by the virus, our model shows that the populations recover to circa 80% of the original population densities within 10 years when averaged across simulations (Figure 2a). Under this extreme high kill rate, the probability of population extinction is just 29%. All surviving populations recover to 100% of their original biomass in under 10 years. With annual kill rates of 50%–80% of adults and juveniles, there are significant knock-downs of carp, yet populations would fully recover within 5 years, and there is no eradication of carp.

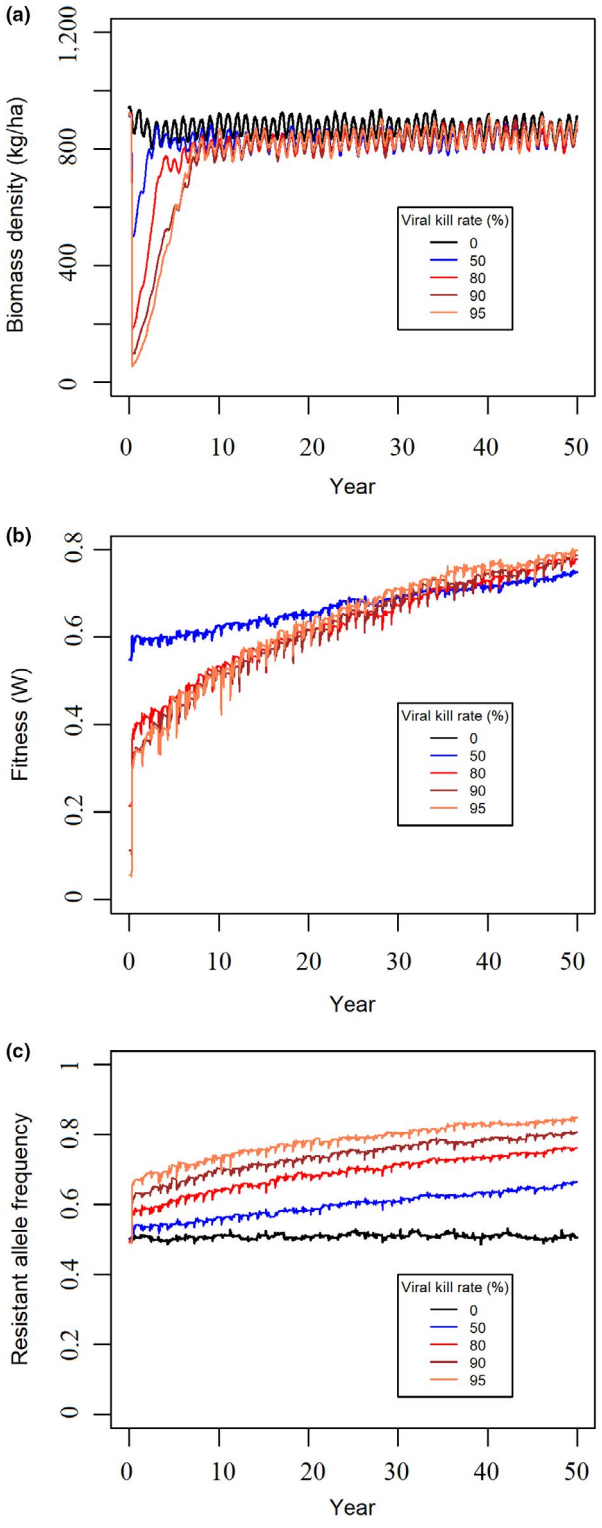

FIGURE 2 (a) Reductions and recovery of the mean total biomass of Common Carp populations after regular annual outbreaks of Koi herpesvirus (KHV), killing between 50% and 95% of susceptible adults and juveniles. (b) Changes in mean individual resistance (ω) in Common Carp populations after regular annual outbreaks of KHV, killing between 50% and 95% of susceptible adults and juveniles. (c) Changes in mean resistant allele frequencies in Common Carp populations after regular annual outbreaks of KHV, killing between 50% and 95% of genetically susceptible adults and juveniles

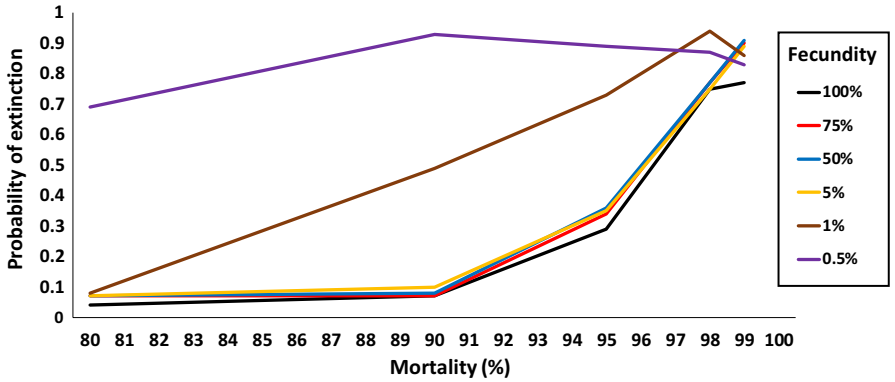

FIGURE 3 The probability of population extinction in Common Carp after regular annual outbreaks of Koi herpesvirus, killing between 80% and 99% of susceptible individuals. There is a negligible effect of reducing female fecundity from the default (100%) to 5% on extinction risk

Increased kill rates augment natural selection acting on resistance alleles, leading to rapid changes in frequencies (Figure 2b). The response to selection is similar in populations experiencing a kill rate of 80% and above, and resistance alleles reach fixation in ~50 years (Figure 2b,c). Moreover, our model suggests that female fecundity needs to be reduced by 99% with a kill rate of 98% before an enclosed Australian carp population is likely to go extinct from KHV infection ($p > 0.9$; Figure 3). Alternatively, a 90% kill rate requires female fecundity to be reduced by 99.5%. Without changes in fecundity, and a 90% kill rate, the mean probability of population extinction is small ($p < 0.1$). The minimum 'tipping point' for increased probability of population extinction requires a combination of 90% total biomass kill rate, and a 99% reduction in fecundity (Figure S1, Appendix S2). At 1% fecundity, the probability of extinction rapidly increases with mortality rate. This is because the population density is high enough for efficient viral transmission, but it is not sufficiently high to buffer the effects of mortality. When the mortality rate increases to 98%, viral transmission becomes less efficient due to the decreased population density, and so not all susceptible individuals will become infected. The efficacy of viral transmission is more severely impaired at 0.5% fecundity due to the rapidly decreasing population density. This means that with 90% or more mortality, there is too little viral transmission, resulting in a lower probability of extinction. This explains the counterintuitive observation that a higher fecundity leads to a higher probability of extinction.

We next explored the effect of the number of loci contributing to additive variation in KHV resistance at this tipping point. Mean disease resistance steadily decreased as the number of independently segregating loci increased (Figure S2a, Appendix S2). With more loci, each contributes a smaller proportion of the variance of the resistant trait, effectively 'watering down' the heritability of genetic resistance. However, population extinction risk is not directly related to changes in the genetic architecture of resistance, that is, changes in the number of loci (Figure S2, Appendix S2).

Finally, we tested if manual targeted manual removal could eradicate carp. A single removal event of up to 95% of all biomass would lead to rapid population recovery in 3–8 years (Figure 4). A sustained effort of removing 80%–95% of all biomass could result in population eradication within 2–5 years (Figure 5). However, if adults are disproportionality removed in a single event, then

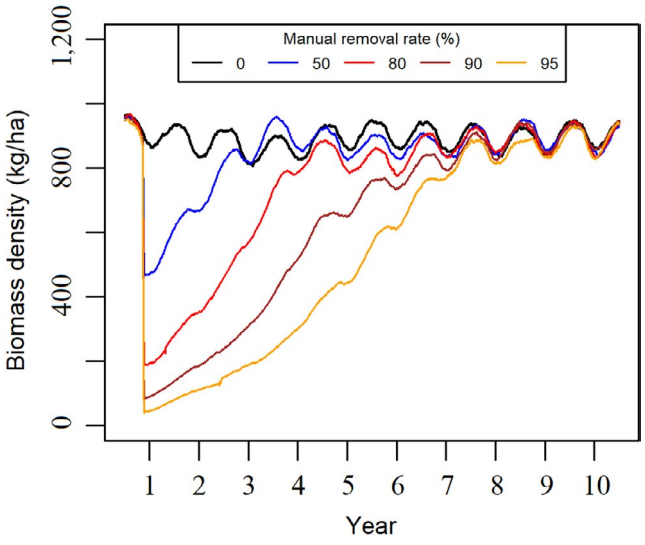

FIGURE 4 Reductions and recovery of the mean total biomass of Common Carp population after a single manual removal event of between 50% and 95% of adults and juveniles

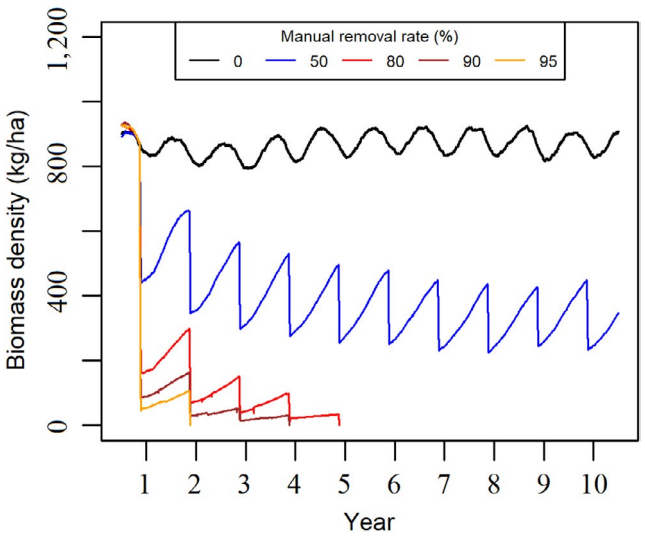

FIGURE 5 Reductions and recovery of the mean total biomass of Common Carp populations after regular annual removal events of between 50% and 95% of adults and juveniles

population recovery times are much more rapid as a consequence of the large remaining biomass of juveniles that reach maturity the following years (Figure S3a, Appendix S2). Importantly, our model suggests that it would take between 2 and 5 years of successive removal of 80%–95% of all biomass to drive a population to extinction (Figure 5), whereas the targeting of adults would require a 95% annual removal rate over more than 5 years (Figure S4a, Appendix S2).

4 | DISCUSSION

Previously, an age-based stochastic model named CARPSIM was developed to investigate the population effects of manual carp removal, poisoning and genetic interference of sex ratios (Brown & Walker, 2004), and the impact of KHV release (Brown & Gilligan, 2014). However, the model does not include disease resistance and viral epidemiology, limiting its usefulness to evaluate the efficacy of KHV biocontrol. Similarly, Thresher et al. (2014) modelled the reduction of carp populations via the introduction of genetically constructed individuals that progressively lead to alteration of sex ratios through the inheritance of disruption to female life-time reproductive output. As with CARPSIM, this model did not include KHV epidemiology or genetic resistance, instead applying arbitrary removal rates across age classes and years as a proxy for annual KHV outbreaks; 80% year 1, 50% year 2, 30% year 3 and 20% every subsequent year. Because no component of natural selection was incorporated, both models overestimate the number of individuals which could be removed by annual mass mortalities as a direct consequence of KHV.

We developed an IBM parametrized to simulate host–pathogen interactions over successive days within Australian carp populations, thereby taking into account the adaptive evolutionary response of carp upon KHV release. We tested the effects of seasonal KHV outbreaks on relative population size and the evolutionary dynamics of disease resistance within a replicated infected carp population. This IBM was developed to provide a 'reasonable best-case scenario' for KHV deployment in Australian populations while maintaining conservative measures of fecundity and recruitment. Under constant optimal temperature conditions for viral replication and disease transmission, all individuals are quickly infected with the virus, and resistance is dependent on heritable additive variation at immunogenetic loci. Our model suggests that carp populations would generally recover to carrying capacity in under 10 years, following rapid increase in resistant individuals, even with successive annual KHV outbreaks that eradicate 95% of all disease susceptible biomass. Our model incorporated reactivation of KHV from latent infections of individuals that survive their first exposure and carry the virus into the next breeding season, which is believed to be a significant avenue in re-emerging infections of KHV (Eide et al., 2011; St-Hilaire et al., 2005). Individuals that are able to survive their first KHV infection gain acquired immunity through antibody production, which provides a significant buffer against recurring mass outbreaks of KHV in carp that are just moderately genetically resistant (Perelberg et al., 2008).

Importantly, we demonstrate that the high reproductive potential of carp (fecundity) can buffer the effects of carp removal. The speed of carp population recovery is exemplified in the 25-year effort to eradicate carp from Tasmania, where after reducing the carp population of a single enclosed lake to just *c*. 40 individuals, a hyper-recruitment event increased numbers to thousands in a matter of weeks (Kelly, 2020). Efforts on Tasmania have only just been deemed successful after two decades of complex management using netting, electrofishing, spawning ground sabotage and ecological restoration. Indeed, our simulations corroborate the requirement for sustained and efficient manual removal to significantly reduce carp numbers, and given the difficulties of removing fish with sustained effort from a fully enclosed habitat, the prospect of significantly reducing carp within interconnected aquatic habitats without ecosystem alterations appears bleak.

Notably, our model does not include the daily variation in water temperature which can lead to punctuated permissive conditionals for viral replication in the Murray–Darling Basin (Becker et al., 2018). Thus, the spread of KHV between carp in Australia is likely to be much less efficient than the scenario modelled here. Fluctuating temperature may lead to disrupted viral replication in many individuals that are not genetically resistant, which then develop antibodies and acquired immunity to KHV, thus reducing the efficacy of biocontrol. Moreover, the extreme fecundity of carp offers a powerful buffering mechanism against population perturbations, especially when using a biocontrol that interacts with a genetic component of disease resistance, thus reducing the efficacy of approaches that are based on natural selection. The reproductive potential of carp would need to be reduced to 1% of that commonly observed in the species to make KHV a plausible option for efficiently reducing populations. Thus, even if complementary genetic control approaches were implemented alongside KHV release, their efficacy would need to be extreme and rapid.

The NCCP recognize that KHV is not a silver bullet and must be used in combination with other removal approaches (McColl et al., 2018). Indeed, KHV alone is unlikely to produce excessive and prolonged carp knock-downs given that for example, among 25 infection trails from 7 separate studies of varying carp strains and experimental conditions, the average mortality after KHV infection was 57.5% $\pm$ 27.8 (Gilad et al., 2004; Hedrick et al., 2006; McColl & Crane, 2013; McColl, Sunarto, et al., 2017; Palaiokostas et al., 2018; Perelberg et al., 2003; Shapira et al., 2005). Genetic manipulation of ratios is commonly referred to as a potential complementary approach to KHV (e.g. McColl et al., 2018; Wedekind, 2019). In principle, this strategy is sound, as it reduces the effective fecundity and population growth rate potential. However, such genetic techniques are extremely complex to implement and require years of dedicated research and development, and risk assessment regarding the release of genetically modified organisms. The power of fecundity in population recovery suggests that such slow-acting approaches would need to be deployed well in advance of KHV to have a complementary effect. To our knowledge, there is currently no research programme in operation or planned, which investigates the complex technicalities

and implementation of genetic control approaches in Australian carp populations.

5 | CONCLUSIONS

The scientific community agree that carp numbers need reducing in Australia but also question the efficacy and safety of biocontrol as a preference over less risky approaches. We have developed an IBM to assess the efficacy of KHV for carp population control, incorporating population genetic and epidemiological principles. We thus assessed the influence of genetic resistance on population recovery following biocontrol, which affirms that KHV alone would not result in lasting population reductions. In our simulations, we have been progressive in our assumptions (i.e. using settings that should improve KHV biocontrol efficacy), but still KHV is unlikely to eradicate carp or produce lasting knock-downs in Australian waters. Our findings support that habitat restoration for native fauna and reduction of carp spawning grounds (e.g. by increasing managed river flow) would provide the first steps to reducing carp numbers in the Murray–Darling Basin. Although this cannot eradicate carp, it poses zero risk to human and ecosystem health, and is a sustainable solution to reduce carp numbers. Furthermore, reducing the fecundity or population growth rate potential, for example, though genetically modification, could increase the efficacy of KHV biocontrol, but requires further research and feasibility studies.

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AUTHORS' CONTRIBUTIONS

All authors designed the model and analyses; K.S.M. wrote and implemented the code, and undertook model and data analysis; J.L. conceived the study and drafted the paper. All authors had significant input in writing the final version of the manuscript, and gave final approval for publication.

DATA AVAILABILITY STATEMENT

Annotated computer code for the IBM can be found via the Dryad Digital Repository https://doi.org/10.5061/dryad.t4b8gtj0b (Mintram, van Oosterhout, & Lighten, 2020).

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REFERENCES

Adkison, M. A., Gilad, O., & Hedrick, R. P. (2005). An enzyme linked immunosorbent assay (ELISA) for detection of antibodies to the koi herpesvirus (KHV) in the serum of koi *Cyprinus carpio*. *Fish Pathology*, *40*(2), 53–62. https://doi.org/10.3147/jsfp.40.53

Angeler, D. G., Álvarez-Cobelas, M., Sánchez-Carrillo, S., & Rodrigo, M. A. (2002). Assessment of exotic fish impacts on water quality and zooplankton in a degraded semi-arid floodplain wetland. *Aquatic Sciences*, *64*(1), 76–86.

Bassini, L. N., Lhorente, J. P., Oyarzún, M., Bangera, R., Yáñez, J. M., & Neira, R. (2019). Genetic parameters for *Piscirickettsia salmonis* resistance, sea lice (*Caligus rogercresseyi*) susceptibility and harvest weight in rainbow trout (*Oncorhynchus mykiss*). *Aquaculture*, *510*, 276–282. https://doi.org/10.1016/j.aquaculture.2019.05.008

Becker, J. A., Ward, M. P., & Hick, P. M. (2018). An epidemiologic model of koi herpesvirus (KHV) biocontrol for carp in Australia. *Australian Zoologist*, *40*(1), 25–35.

Beckett, S., Caley, P., Hill, M., & Henderson, B. (2019). *Biocontrol of European carp: Ecological risk assessment for the release of Cyprinid herpesvirus 3 (CyHV-3) for biocontrol in Australia*. A report for the National Carp Control Plan, Fisheries Research and Development Corporation.

Bester, M. N., Bloomer, J. P., van Aarde, R. J., Erasmus, B. H., van Rensburg, P. J. J., Skinner, J. D., … Naude, T. W. (2002). A review of the successful eradication of feral cats from sub-Antarctic Marion Island, Southern Indian Ocean. *African Journal of Wildlife Research*, *32*(1), 65–73.

Boutier, M., Donohoe, O., Kopf, R. K., Humphries, P., Becker, J. A., Marshall, J., & Vanderplasschen, A. (2019). Biocontrol of carp: The Australian plan does not stand up to a rational analysis of safety and efficacy. *Frontiers in Microbiology*, *10*, 882. https://doi.org/10.3389/fmicb.2019.00882

Bozkurt, Y., & Öğretmen, F. (2012). Sperm quality, egg size, fecundity and their relationships with fertilization rate of grass carp (*Ctenopharyngodon idella*). *Iranian Journal of Fisheries Sciences*, *11*(4), 755–764.

Brookes, J., & Hipsey, M. (2019). *Water quality risk assessment of carp biocontrol for Australian waterways*. Unpublished Client Report for the Fisheries Research and Development Corporation, Environment Institute, University of Adelaide, Adelaide, South Australia.

Brown, P., & Gilligan, D. (2014). Optimising an integrated pest-management strategy for a spatially structured population of common carp (*Cyprinus carpio*) using meta-population modelling. *Marine and Freshwater Research*, *65*(6), 538–550. https://doi.org/10.1071/MF13117

Brown, P., & Walker, T. I. (2004). CARPSIM: Stochastic simulation modelling of wild carp (*Cyprinus carpio* L.) population dynamics, with applications to pest control. *Ecological Modelling*, *176*(1–2), 83–97.

Brown, P., Wisniewski, C., & Gilligan, D. (2019). *The role of commercial fishing in control of invasive freshwater fish species: Common carp, can we fish them out through overfishing?* Mildura, Australia: Fisheries Research and Development Corporation.

Cadwallader, P. L. (1978). Some causes of the decline in range and abundance of native fish in the Murray–Darling river system. *Royal Society of Victoria Proceedings*, *90*(2), 211–224.

Cock, M. J. W., Murphy, S. T., Kairo, M. T. K., Thompson, E., Murphy, R. J., & Francis, A. W. (2016). Trends in the classical biological control of insect pests by insects: An update of the BIOCAT database. *BioControl*, *61*, 349–363. https://doi.org/10.1007/s10526-016-9726-3

David, P., Thébault, E., Anneville, O., Duyck, P. F., Chapuis, E., & Loeuille, N. (2017). Impacts of invasive species on food webs: A review of empirical data. *Advances in Ecological Research*, *56*, 1–60.

Dietz, K. (1993). The estimation of the basic reproduction number for infectious diseases. *Statistical Methods in Medical Research*, *2*(1), 23–41. https://doi.org/10.1177/096228029300200103

Durr, P. A., Davis, S., Joehnk, K., Graham, K., Hopf, J., Arakala, A., … Gilligan, D. (2019). *Development of hydrological, ecological and epidemiological modelling to inform a CyHV3 release strategy for the biocontrol*

of carp in the Murray–Darling Basin. Part A. Integrated ecological and epidemiological modelling. Canberra, Australia: FRDC.

Eide, K. E., Miller-Morgan, T., Heidel, J. R., Kent, M. L., Bildfell, R. J., LaPatra, S., … Jin, L. (2011). Investigation of koi herpesvirus latency in Koi. *Journal of Virology*, *85*(10), 4954–4962. https://doi.org/10.1128/JVI.01384-10

Falconer, A., Coleman, F., Loh, R., Rocliffe, M., & Lighten, J. (2018). *Issues that need addressingon the conduct and approach of the NCCP*. Retrieved from https://www.aph.gov.au/Parliamentary_Business/Committees/Senate/Rural_and_Regional_Affairs_and_Transport/New_Content_Folder/Fisheries_Research_and_Development_Corporation

Forbes, V. E., Calow, P., Grimm, V., Hayashi, T. I., Jager, T., Katholm, A., … Stillman, R. (2011). Adding value to ecological risk assessment with population modeling. *Human and Ecological Risk Assessment*, *17*(2), 287–299. https://doi.org/10.1080/10807039.2011.552391

Gehrke, P. C., Brown, P., Schiller, C. B., Moffatt, D. B., & Bruce, A. M. (1995). River regulation and fish communities in the Murray–Darling river system, Australia. *Regulated Rivers: Research & Management*, *11*(3–4), 363–375. https://doi.org/10.1002/rrr.3450110310

Gilad, O., Yun, S., Adkison, M. A., Way, K., Willits, N. H., Bercovier, H., & Hedrick, R. P. (2003). Molecular comparison of isolates of an emerging fish pathogen, koi herpesvirus, and the effect of water temperature on mortality of experimentally infected koi. *Journal of General Virology*, *84*(10), 2661–2668. https://doi.org/10.1099/vir.0.19323-0

Gilad, O., Yun, S., Zagmutt-Vergara, F. J., Leutenegger, C. M., Bercovier, H., & Hedrick, R. P. (2004). Concentrations of a Koi herpesvirus (KHV) in tissues of experimentally infected *Cyprinus carpio* koi as assessed by real-time TaqMan PCR. *Diseases of Aquatic Organisms*, *60*(3), 179–187. https://doi.org/10.3354/dao060179

Grimm, V., Berger, U., Bastiansen, F., Eliassen, S., Ginot, V., Giske, J., … DeAngelis, D. (2006). A standard protocol for describing individual-based and agent-based models. *Ecological Modelling*, *198*(1–2), 115–126. https://doi.org/10.1016/j.ecolmodel.2006.04.023

Grimm, V., Berger, U., DeAngelis, D. L., Polhill, J. G., Giske, J., & Railsback, S. F. (2010). The ODD protocol: A review and first update. *Ecological Modelling*, *221*(23), 2760–2768. https://doi.org/10.1016/j.ecolmodel.2010.08.019

Haenen, O. L. M., Way, K., Bergmann, S. M., & Ariel, E. (2004). The emergence of koi herpesvirus and its significance to European aquaculture. *Bulletin of the European Association of Fish Pathologists*, *24*(6), 293–307.

Harris, J., & Gehrke, P. (1997). *Fish and rivers in stress: The NSW Rivers Survey*. Sydney, Australia: NSW Fisheries Office of Conservation and the CRC for Freshwater Ecology.

Haynes, G. D. (1999). *Population genetics of common carp (*Cyprinus carpio*) in the Murray–Darling Basin* (PhD thesis). The University of Sydney.

Haynes, G. D., Gilligan, D. M., Grewe, P., & Nicholas, F. W. (2009). Population genetics and management units of invasive common carp *Cyprinus carpio* in the Murray–Darling Basin, Australia. *Journal of Fish Biology*, *75*(2), 295–320.

Hedrick, R. P., Gilad, O., Yun, S., Spangenberg, J. V., Marty, G. D., Nordhausen, R. W., … Eldar, A. (2000). A herpesvirus associated with mass mortality of juvenile and adult koi, a strain of common carp. *Journal of Aquatic Animal Health*, *12*(1), 44–57. https://doi.org/10.1577/1548-8667(2000)012<0044:AHAWMM>2.0.CO;2

Hedrick, R. P., Waltzek, T. B., & McDowell, T. S. (2006). Susceptibility of koi carp, common carp, goldfish, and goldfish × common carp hybrids to cyprinid herpesvirus-2 and herpesvirus-3. *Journal of Aquatic Animal Health*, *18*(1), 26–34.

Kelly, M. (2020). Carp success, after long battle against invasive fish, has trout faithful rejoicing. *ABC News*.

Kloskowski, J. (2011). Impact of common carp (*Cyprinus carpio*) on aquatic communities: Direct trophic effects versus habitat deterioration. *Fundamental and Applied Limnology*, *178*(3), 245–255.

Koehn, J. D. (2004). Carp (*Cyprinus carpio*) as a powerful invader in Australian waterways. *Freshwater Biology*, *49*(7), 882–894. https://doi.org/10.1111/j.1365-2427.2004.01232.x

Kopf, R. K., Nimmo, D. G., Humphries, P., Baumgartner, L. J., Bode, M., Bond, N. R., … Olden, J. D. (2017). Confronting the risks of large-scale invasive species control. *Nature Ecology and Evolution*, *1*, 0172. https://doi.org/10.1038/s41559-017-0172

Lighten, J., & van Oosterhout, C. (2017). Biocontrol of common carp in Australia poses risks to biosecurity. *Nature Ecology and Evolution*, *1*, 0087. https://doi.org/10.1038/s41559-017-0087

Marshall, J. C., Blessing, J. J., Clifford, S. E., Hodges, K. M., Negus, P. M., & Steward, A. L. (2019). Ecological impacts of invasive carp in Australian dryland rivers. *Aquatic Conservation: Marine and Freshwater Ecosystems*, *29*(11), 1870–1889. https://doi.org/10.1002/aqc.3206

Marshall, J., Davison, A. J., Kopf, R. K., Boutier, M., Stevenson, P., & Vanderplasschen, A. (2018). Biocontrol of invasive carp: Risks abound. *Science*, *359*(6378), 877.

Mccoll, K. A., & Crane, M. S. J. (2013). *Cyprinid herpesvirus 3 (CyHV-3): Its potential as a biological control agent for carp in Australia*. Canberra, Australia: PestSmart Toolkit Publication, Invasive Animals Cooperative Research Centre.

McColl, K. A., Sheppard, A. W., & Barwick, M. (2017). Safe and effective biocontrol of common carp. *Nature Ecology and Evolution*, *1*, 0134. https://doi.org/10.1038/s41559-017-0134

McColl, K. A., Sunarto, A., & Neave, M. J. (2018). Biocontrol of carp: More than just a herpesvirus. *Frontiers in Microbiology*, *9*, 02288. https://doi.org/10.3389/fmicb.2018.02288

McColl, K. A., Sunarto, A., Slater, J., Bell, K., Asmus, M., Fulton, W., … Crane, M. S. J. (2017). Cyprinid herpesvirus 3 as a potential biological control agent for carp (*Cyprinus carpio*) in Australia: Susceptibility of non-target species. *Journal of Fish Diseases*, *40*(9), 1141–1153.

McLane, A. J., Semeniuk, C., McDermid, G. J., & Marceau, D. J. (2011). The role of agent-based models in wildlife ecology and management. *Ecological Modelling*, *222*(8), 1544–1556. https://doi.org/10.1016/j.ecolmodel.2011.01.020

Mintram, K., van Oosterhout, C., & Lighten, J. (2020). Data from: Code for Individual Based Model to assess impact of KHV release on carp population, v2. *Dryad Dataset*, https://doi.org/10.5061/dryad.t4b8gtj0b

Murray–Darling Basin Ministerial Council. (2003). *Native fish strategy for the Murray–Darling Basin 2003–2013*. MDBC Publication No. 25/04.

Nichols, S. J., Gawne, B., Richards, R., Lintermans, M., & Thompson, R. (2019). *The likely medium- to long-term ecological outcomes of major carp population reductions*. Australian Capital Territory, Australia: Institute for Applied Ecology, University of Canberra for Fisheries Research and Development Corporation.

Nicol, S. J., Lieschke, J. A., Lyon, J. P., & Koehn, J. D. (2004). Observations on the distribution and abundance of carp and native fish, and their responses to a habitat restoration trial in the Murray River, Australia. *New Zealand Journal of Marine and Freshwater Research*, *38*(3), 541–551. https://doi.org/10.1080/00288330.2004.9517259

Ødegård, J., Olesen, I., Dixon, P., Jeney, Z., Nielsen, H. M., Way, K., … Gjerde, B. (2010). Genetic analysis of common carp (*Cyprinus carpio*) strains. II: Resistance to koi herpesvirus and *Aeromonas hydrophila* and their relationship with pond survival. *Aquaculture*, *304*(1–4), 7–13.

Palaiokostas, C., Robledo, D., Vesely, T., Prchal, M., Pokorova, D., Piackova, V., … Houston, R. D. (2018). Mapping and sequencing of a significant quantitative trait locus affecting resistance to koi herpesvirus in common carp. *G3: Genes, Genomes, Genetics*, *8*(11), 3507–3513.

Perelberg, A., Ilouze, M., Kotler, M., & Steinitz, M. (2008). Antibody response and resistance of *Cyprinus carpio* immunized with *cyprinid*

herpesvirus 3 (*CyHV-3*). *Vaccine*, *26*(29–30), 3750–3756. https://doi.org/10.1016/j.vaccine.2008.04.057

Perelberg, A., Smirnov, M., Hutoran, M., Diamant, A., Bererano, Y., & Kotler, M. (2003). Epidemiological description of a new viral disease afflicting cultured *Cyprinus carpio* in Israel. *Israeli Journal of Aquaculture – Bamidgeh*, *55*(1), 5–12.

Rahman, M. M. (2015). Role of common carp (*Cyprinus carpio*) in aquaculture production systems. *Frontiers in Life Science*, *8*(4), 399–410.

Ronen, A., Perelberg, A., Abramowitz, J., Hutoran, M., Tinman, S., Bejerano, I., … Kotler, M. (2003). Efficient vaccine against the virus causing a lethal disease in cultured *Cyprinus carpio*. *Vaccine*, *21*(32), 4677–4684. https://doi.org/10.1016/S0264-410X(03)00523-1

Roper, K., & Ford, L. (2018). *Cyprinid herpesvirus 3 and its relevance to human health*. Deakin West, Australian Capital Territory, Australia: Fisheries Research and Development Corporation.

Shapira, Y., Magen, Y., Zak, T., Kotler, M., Hulata, G., & Levavi-Sivan, B. (2005). Differential resistance to koi herpes virus (KHV)/carp interstitial nephritis and gill necrosis virus (CNGV) among common carp (*Cyprinus carpio*) strains and crossbreds. *Aquaculture*, *245*(1–4), 1–11.

Shimizu, T., Yoshida, N., Kasai, H., & Yoshimizu, M. (2006). Survival of koi herpesvirus (KHV) in environmental water. *Fish Pathology*, *41*(4), 153–157. https://doi.org/10.3147/jsfp.41.153

Shine, R., & Phillips, B. L. (2014). Unwelcome and unpredictable: The sorry saga of cane toads in Australia. In G. I. Holwell, A. Stow, & N. Maclean (Eds.), *Austral ark: The state of wildlife in Australia and New Zealand* (pp. 83–104). Cambridge, UK: Cambridge University Press.

Silva, L., Bell, K., & Baumgartner, L. (2017). *Clean-up procedures applied for fish kill events: A review for the National Carp Control Plan*. New South Wales, Australia: Charles Sturt University.

Sivakumaran, K. P., Brown, P., Stoessel, D., & Giles, A. (2003). Maturation and reproductive biology of female wild carp, *Cyprinus carpio*, in Victoria, Australia. *Environmental Biology of Fishes*, *68*(3), 321–332.

Smith, B. B., & Walker, K. F. (2004). Spawning dynamics of common carp in the River Murray, South Australia, shown by macroscopic and histological staging of gonads. *Journal of Fish Biology*, *64*(2), 336–354. https://doi.org/10.1111/j.0022-1112.2004.00293.x

St-Hilaire, S., Beevers, N., Way, K., Le Deuff, R. M., Martin, P., & Joiner, C. (2005). Reactivation of koi herpesvirus infections in common carp *Cyprinus carpio*. *Diseases of Aquatic Organisms*, *67*(1–2), 15–23. https://doi.org/10.3354/dao067015

Stuart, I. G., & Conallin, A. (2018). Control of globally invasive common carp: An 11-year commercial trial of the Williams' cage. *North American Journal of Fisheries Management*, *38*(5), 1160–1169. https://doi.org/10.1002/nafm.10221

Stuart, I., Fanson, B., Lyon, J., Stocks, J., Brooks, S., Norris, A., … Bennett, A. (2019). *A national estimate of carp biomass for Australia*. Unpublished Client Report for the Fisheries Research and Development Corporation. Arthur Rylah Institute for Environmental Research, Department of Environment, Land, Water and Planning, Heidelberg, Victoria.

Tadmor-Levi, R., Hulata, G., & David, L. (2019). Multiple interacting QTLs affect disease challenge survival in common carp (*Cyprinus carpio*). *Heredity*, *123*(5), 565–578. https://doi.org/10.1038/s41437-019-0224-0

Thresher, R., Van De Kamp, J., Campbell, G., Grewe, P., Canning, M., Barney, M., … Fulton, W. (2014). Sex-ratio-biasing constructs for the control of invasive lower vertebrates. *Nature Biotechnology*, *32*, 424–427. https://doi.org/10.1038/nbt.2903

Tilley, A., Colquhoun, E., O'Keefe, E., Nash, S., McDonald, D., Evans, T., … Howieson, J. (2019). *Options for utilisation of carp biomass*. Bentley, Perth, Western Australia, Australia: Curtin University.

Todd, C. R., Koehn, J. D., Brown, T. R., Fanson, B., Brooks, S., & Stuart, I. (2019). *Modelling carp biomass: Estimates for the year 2023*. Heidelberg, Victoria, Australia: Institute for Environmental Research, Department of Environment, Land, Water and Planning.

Vilizzi, L., Thwaites, L. A., Smith, B. B., Nicol, J. M., & Madden, C. P. (2014). Ecological effects of common carp (*Cyprinus carpio*) in a semi-arid floodplain wetland. *Marine and Freshwater Research*, *65*(9), 802–817. https://doi.org/10.1071/MF13163

Weber, M. J., & Brown, M. L. (2009). Effects of common carp on aquatic ecosystems 80 years after 'carp as a dominant': Ecological insights for fisheries management. *Reviews in Fisheries Science*, *17*(4), 524–537. https://doi.org/10.1080/10641260903189243

Wedekind, C. (2019). *Synergistic genetic biocontrol options for common carp (*Cyprinus carpio*)*. Deakin West, Australian Capital Territory, Australia: Fisheries Research and Development Corporation.

Wilensky, U. (1999). *NetLogo*. Evanston, IL: Center for Connected Learning and ComputerBased Modeling Northwestern University. Retrieved from http://ccl.northwestern.edu/netlogo/

Yáñez, J. M., Lhorente, J. P., Bassini, L. N., Oyarzún, M., Neira, R., & Newman, S. (2014). Genetic co-variation between resistance against both *Caligus rogercresseyi* and *Piscirickettsia salmonis*, and body weight in Atlantic salmon (*Salmo salar*). *Aquaculture*, *433*, 295–298. https://doi.org/10.1016/j.aquaculture.2014.06.026

Yuasa, K., Ito, T., & Sano, M. (2008). Effect of water temperature on mortality and virus shedding in carp experimentally infected with koi herpesvirus. *Fish Pathology*, *43*(2), 83–85. https://doi.org/10.3147/jsfp.43.83

Yuasa, K., Sano, M., & Oseko, N. (2013). Goldfish is not a susceptible host of koi herpesvirus (KHV) disease. *Fish Pathology*, *48*(2), 52–55. https://doi.org/10.3147/jsfp.48.52

Zhao, J., Prchal, M., Palaiokostas, C., Houston, R. D., Kause, A., Vandeputte, M., … Kocour, M. (2020). Genetic relationship between koi herpesvirus disease resistance and production traits inferred from sibling performance in Amur mirror carp. *Aquaculture*, *520*. https://doi.org/10.1016/j.aquaculture.2020.734986

SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

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