Whole genome resequencing reveals signatures of rapid selection in a virus affected commercial fishery

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Abstract

Infectious diseases are recognised as one of the greatest global threats to biodiversity and ecosystem functioning. Consequently, there is a growing urgency to understand the speed at which adaptive phenotypes can evolve and spread in natural populations to inform future management. Here we provide evidence of rapid genomic changes in wild Australian blacklip abalone (Haliotis rubra) following a major population crash associated with an infectious disease. A genome wide association study on H. rubra was conducted using pooled whole genome re-sequencing data from commercial fishing stocks varying in historical exposure to haliotid herpesvirus-1 (HaHV-1). Approximately 25,000 SNP loci associated with virus exposure were identified, many of which mapped to genes known to contribute to HaHV-1 immunity in the New Zealand pāua (H. iris) and herpesvirus response pathways in haliotids and other animal systems. These findings indicate genetic changes across a single generation in H. rubra fishing stocks decimated by HaHV-1, with stock recovery determined by rapid evolutionary changes leading to virus resistance. This is a novel example of rapid adaptation in natural populations of a non-model marine organism, highlighting the pace at which selection can potentially act to counter disease in wildlife communities.

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Abstract

Infectious diseases are recognised as one of the greatest global threats to biodiversity and ecosystem functioning. Consequently, there is a growing urgency to understand the speed at which adaptive phenotypes can evolve and spread in natural populations to inform future management. Here we provide evidence of rapid genomic changes in wild Australian blacklip abalone (*Haliotis rubra*) following a major population crash associated with an infectious disease. A genome wide association study on *H. rubra* was conducted using pooled whole genome re-sequencing data from commercial fishing stocks varying in historical exposure to haliotid herpesvirus-1 (HaHV-1). Approximately 25,000 SNP loci associated with virus exposure were identified, many of which mapped to genes known to contribute to HaHV-1 immunity in the New Zealand pāua (*H. iris*) and herpesvirus response pathways in haliotids and other animal systems. These findings indicate genetic changes across a single generation in *H. rubra* fishing stocks decimated by HaHV-1, with stock recovery determined by rapid evolutionary changes leading to virus resistance. This is a novel example of rapid adaptation in natural populations of a non-model marine organism, highlighting the pace at which selection can potentially act to counter disease in wildlife communities.

KEYWORDS

Infectious diseases, genetic adaptation, genome wide association study, blacklip abalone, haliotid herpesvirus-1, south-eastern Australia

1 | INTRODUCTION

The spread of infectious diseases is recognised as one of the most pressing global threats to biodiversity and ecosystem function (Daszak*et al.* 2000; Tompkins *et al.* 2015; Cunningham *et al.* 2017). In recent decades, infectious diseases have devastated a range of wildlife groups (Berger *et al.* 1998; Kim & Harvell 2004; Hansen *et al.* 2005; Lorch *et al.* 2016), often exacerbating species declines in ecosystems already stressed by climate change and habitat destruction (Harvell *et al.* 2002; Brearley*et al.* 2013; Bosch *et al.* 2018). The future persistence of many species will likely depend on their ability to adapt to environmental changes associated with increased disease prevalence, although selection for disease resistance or tolerance may not keep pace with rates of pathogen evolution and the emergence and turn-over of novel diseases (Hawley *et al.* 2013; Ujvari *et al.* 2014).

Detecting evolutionary changes in disease affected populations is challenging but has been greatly assisted by modern genomic technologies (Blanchong *et al.* 2016; Storfer *et al.* 2020). These technologies now allow for rapid and cost-effective estimates of genome wide variation among populations spanning disease infection gradients and individuals with distinctive phenotypes related to disease response (Elbers *et al.* 2018; Grogan *et al.* 2018; Margres *et al.* 2018; Auteri & Knowles 2020). Importantly, a number of studies using these technologies have reported rapid evolutionary changes across several generations in natural populations of non-model organisms impacted by disease, including Tasmanian devils (*Sarcophilus harrisii*) (Epstein *et al.* 2016; Hubert *et al.* 2018; Margres *et al.* 2018) and North American house finches (*Carpodacus mexicanus*) (Bonneaud *et al.* 2011). Additionally, recent studies have reported evidence of rapid selection for disease resistant genotypes across a single generation in North American sea stars (*Pisaster ochraceus*) and little brown bats (*Myotis lucifugus*), following rapid and severe population crashes due to infectious diseases (Schiebelhut *et al.* 2018; Auteri & Knowles 2020). Such studies are pivotal in highlighting the pace at which selection can act to counter disease in wildlife communities and opening up opportunities for interventions, such as deliberate translocations of adaptive phenotypes, that can increase the adaptability of threatened populations (Hohenlohe *et al.* 2019; Hoffmann *et al.* 2020). Despite this progress, the number of studies demonstrating rapid evolutionary responses to infectious diseases in natural populations remains limited and biased towards terrestrial systems.

Marine infectious diseases are responsible for incremental and mass mortalities in a variety of wildlife groups, including keystone and habitat forming taxa (Harvell *et al.* 2007; Clemente *et al.*2014; Martin *et al.* 2016; Montecino-Latorre *et al.* 2016; Harvell & Lamb 2020), and species supporting wild commercial fisheries (Marty *et al.* 2010; Cawthorn 2011; Lafferty *et al.* 2015; Crosson *et al.* 2020). The Australian blacklip abalone (*Haliotis rubra*), a species targeted by the world's largest wild abalone fisheries and a rapidly

expanding aquaculture industry (FAO Fishstat 2021), was heavily impacted by disease between 2006 and 2010 (Mayfield *et al.* 2012). Abalone viral ganglioneuritis (AVG) caused by the haliotid herpesvirus-1 (HaHV-1) spread along the western coastline of Victoria in south-eastern Australia, causing rapid and severe population collapses ($>$ 90% mortality in some areas) and devastating both wild and farmed abalone stocks (Hooper*et al.* 2007). Despite the impact of AVG, abalone stocks in the Western Zone fishery have seen significant recovery, and are considered sustainable (Mundy *et al.* 2020). Given the short generation time of the species (˜4 years; Andrews 1999), large population sizes, and high levels of genetic variability that contribute to existing patterns of adaptation across the fishery (Miller *et al.* 2019), it is possible that rapid evolutionary responses have contributed to this recovery.

Previous research has demonstrated heritable genetic variation relating to herpesvirus immunity in Haliotid species. Challenge tests performed on New Zealand paua (*H. iris*) and Japanese black abalone (*H. discus*), involving controlled exposure to Haliotid herpesvirus-1 (HaHV-1), indicated complete immunity to AVG (Chang*et al.* 2005; Corbeil *et al.* 2017), with complementary transcriptomic analyses helping to characterise the genetic basis of the resistance (Bai *et al.* 2019b; Neave *et al.* 2019). Similar tests on *H. rubra* yielded no evidence of resistance to AVG (Crane*et al.* 2013; Corbeil *et al.* 2016), however, these experiments were performed on a small number of animals from a limited number of locations affected by AVG. While complete immunity may not occur in *H. rubra* , the presence of AVG immunity in sister taxa hints at the potential for some level of resistance developing through standing genetic variation following AVG exposure.

In this study, we explore the possibility of a rapid evolutionary response in recovering *H. rubra* fishing stocks devastated by AVG. Specifically, we performed a genome wide association study (GWAS) using pooled whole genome re-sequencing data on *H. rubra* specimens from fishing stocks varying in disease exposure. Our findings point to rapid changes in population-level allele frequencies over a single generation time-scale in virus affected fishing stocks, with stock recovery determined by rapid evolutionary changes leading to virus resistance. This study highlights the pace at which adaptive phenotypes can potentially evolve and spread in wildlife communities to counter threats from infectious diseases. We discuss these findings in the context of future biosecurity management of Australian abalone fisheries and wildlife conservation more generally.

2 | METHODS

2.1 | Sample collection and DNA sequencing

Tissue biopsies were collected from a total of 343 individual *H. rubra* from 14 locations spanning the Victorian Western and Central Zone fisheries. Locations were selected based on their known virus exposure history according to confidential records held by the Victorian wild fishing sector and the Victorian Fisheries Authority (10 AVG affected and 4 AVG unaffected locations; Table 1, Figure 1). Sampling for 5 of the locations within the Western Zone fishery was coordinated in 2009 by the Department of Economic Development, Jobs, Transport and Resources (DEDJTR). It is expected that animals from these locations during this sampling period survived the virus event and provide a reliable snap shot of the post-virus allele frequency distributions in AVG affected and unaffected populations. Sampling of the remaining 9 locations was performed between 2015 and 2020. To avoid the potential swamping effects of inter-generational gene flow since the disease outbreak, sampling was biased towards fishing stocks expected to be largely self-recruiting based on biophysical connectivity models (Young et al. 2020), and toward large adult animals (expected to be either direct survivors or first generation post-virus survivors). This sampling was performed by contract divers, commercial fisherman, and our research team. At each location, individual abalone were collected within a 100 m^2 area, with tissue biopsies consisting of 20 mg of muscle tissue from the abalone lip obtained using sterile dissection tools to avoid cross-contamination. Biopsied material was transferred to 2 ml microcentrifuge tubes containing 80-100% ethanol and stored at 4 °C until required for genomic analysis.

Total genomic DNA was extracted from 10 mg of tissue using a DNeasy Blood and Tissue Kit (Qiagen, Valencia, CA) following the manufacturer's instructions. Resulting DNA extracts were quantified using a Qubit version 2 fluorometer (Life Technologies, Carlsbad, CA, USA). To obtain population genomic data, we applied the Pool-Seq approach (Futschik & Schlotterer 2010), which involves pooling the DNA of a large

number of individuals from the same population and then sequencing the 'population variability genome'. This was achieved by pooling individual DNA extracts from each sample location equimolar, splitting the 25 individuals per location into 2 x pools per locations consisting of DNA from 12 and 13 individuals, respectively, to account for potential sequencing bias. The resulting 28 pooled libraries were prepared for sequencing using the Nextera^{TM} DNA Sample Preparation kit, and sequenced using the Illumina NovaSeq platform (Illumina, San Diego, CA, USA), with the 150 base pair (bp) paired-end protocol. Sequencing was performed allowing for 3 x genome coverage per individual per pool, equating to 80 - 100 x genome coverage per population.

2.2 | Data preparation

The Illumina NovaSeq sequencing yielded a total of 25 x 10^9 assigned 150 bp reads, and a total of 45 – 100 Gb of sequence data for each of the 28 pooled DNA libraries. Raw DNA sequence reads from the two separate pooled libraries per sample location were pooled for processing purposes. Raw sequences were processed using the Trimmomatic V0.36 program (Bolger *et al.*2014) by removing Nextera^{TM} adaptors and discarding all reads that had a Phred score below 20. All retained reads were subsequently aligned to the *H.rubra* reference genome (NCBI RefSeq QXJH00000000.1; Gan *et al.* 2019) using the PPalign package in the PoolParty pipeline (Micheletti & Narum 2018) with default parameters. Single nucleotide polymorphisms (SNPs) were called using PoolFstat (Hivert *et al.* 2018) where SNPs were required to have a read depth of 20 – 200 reads to be called. SNPs with a minor allele frequency of [?] 0.05 were used for downstream genomic analysis.

2.3 | Estimating overall genetic structure

SNP frequencies over all loci were initially contrasted between all 14 sample locations to determine patterns of overall genetic structure and population connectivity. The software PoolFstat implemented in R (Hivert *et al.* 2018) was used to calculate global and pairwise measures of population differentiation (F_{ST}; Weir & Cockerham 1984).

2.4 | Genome-wide association analysis

To identify SNPs associated with virus exposure status we performed a genome-wide association study (GWAS) using BayPass 2.1 (Gautier 2015). Analyses were performed under the auxiliary (AUX) covariate mode (-covmcmc and -auxmode flags), after scaling the variables with the -scalecov flag. The underlying models explicitly account for the covariance structure among the population allele frequencies that originates from the shared history of the populations through the estimation of the population covariance matrix Ω, which removes the variation associated with demography (Bonhomme et al., 2010; Gunther & Coop, 2013). The auxiliary covariate model specifically involves the introduction of a binary auxiliary variable to classify each locus as associated or not associated. This allows computation of posterior inclusion probabilities (and Bayes Factors) for each locus while explicitly accounting for multiple testing issues. The auxiliary covariate model was applied with default parameters, a 5,000 burn-in of iterations in the Markov chain Monte Carlo (MCMC) simulation, followed by 25,000 iterations. To reduce artefacts due to potential variability between runs, we performed 3 independent BayPass simulations. We then calculated the average Bayes Factor (BF), expressed in deciban units (dB), for each SNP as a quantitative estimate of the strength of association with virus exposure and the standardized allele frequency. For each SNP, the level of effect was assessed based on the Bayes Factor (BF) models according to Jeffrey's rule (Jeffreys 1961). SNPs with BF scores [?] 50 were regarded as decisive associations with virus exposure and were retained as potential candidate loci.

2.5 | Posthoc analyses including functional annotations

An analysis of principal components (PCA) was implemented in the adegenet package for R (Jombart 2008; Jombart & Ahmed 2011) to obtain a graphical depiction of patterns of genetic structure among virus affected and unaffected stocks based on all candidate SNPs identified by BayPass (BFs [?] 50).

Total linkage disequilibrium (LD) among all candidate loci was calculated using LDx, a package which uses an approximate maximum likelihood approach from pooled resequencing data (Feder *et al.*2012). Linkage

disequilibrium was calculated asr^2, the square of the correlation between alleles of SNP pairs within the paired sequence reads of each population. We subsequently calculated the average LD for each pairwise SNP comparison across sample sites. Next, we assessed the distribution of candidate loci and signatures of selection across the reference*H. rubra* genome consisting of 2,854 annotated scaffolds varying between 1,830 and 1.1 x 10^7 bp in length (Gan *et al.* 2019). This was achieved by regressing the total number of candidate loci against scaffold length using ggpubr package for R (Kassambara & Kassambara 2020). Scaffolds with exceptionally large numbers of candidate loci relative to scaffold length were interrogated further using the package LDBlockShow (Dong *et al.* 2021) to measure pairwise linkage disequilibrium and haplotype blocks using the default -SeleVar option to calculate D ' (the ratio of the difference between the observed and expected frequency of a haplotype, and its maximum value when considering total allele frequencies).

SnpEff v2.0.3 (Cingolani *et al.* 2012) was used to map candidate SNP loci to the *H. rubra* genome and predict variant impacts; high (highly disruptive impact on protein function), moderate (non-synonymous mutations, possible change in protein effectiveness), low (unlikely to change protein behaviour) or a modifier (synonymous mutations, non-coding or intergenic variant). Functional classification of candidate genes was achieved by aligning the peptide sequences for mapped candidate *H. rubra* genes with the annotated genomes for human (NCBI RefSeq IDs NC_000001 - NC_000024), pacific oyster (RefSeq IDs NC_047559 - NC_047568), scallop (RefSeq ID NC_007234.1) and the blue mussel (RefSeq ID NC_006161.1) using DIAMOND (Buchfink *et al.* 2015). A maximum e-value of $1e^{-40}$ was set to conservatively estimate the likelihood of similar gene functions between taxonomies. Protein GI accessions from the top hit of DIAMOND alignments were imported into the web-based version of the DAVID bioinformatics tool (Huang *et al.* 2009a; Huang *et al.* 2009b), where corresponding annotations were generated. Functional annotations were performed by focussing specifically on gene homologs known to be associated with virus-host interactions, in particular herpesvirus response pathways, including those in response to HaHV-1 in the AVG resistant *H. iris* .

3 | RESULTS

3.1 | Genotyping and overall population structure

Pooled whole genome re-sequencing of 384 *H. rubra* specimens from 14 locations distributed across the Victorian Western and Central Zone abalone yielded a total of 7,745,655 SNPs that were used for population genomic analyses. Estimates of overall genetic structure indicated a lack of structure and panmixia across the Western and Central Zone fisheries. Specifically, global F_{ST} did not differ significantly from zero ($F_{ST} = 0$, $P > 0.05$), nor did any pairwise measure ofF_{ST} between sampling locations.

3.2 | Genome-wide association analysis

Our genome-wide association study found 25,854 candidate SNPs with strong associations with virus exposure (BF > 50), with the PCA based on candidate loci revealing clear patterns of genetic structuring between locations varying in historical AVG exposure (Figure 2). Estimates of LD were high at all locations (mean$r^2 = 0.61 \pm 0.01$ *SD*) indicating non-random association of alleles, while comparisons ofr^2 between virus affected and unaffected stocks did not significantly differ ($P > 0.05$). Analyses suggest a genome wide patterns of selection, with regression analyses indicating strong linear relationship between number of candidate loci and scaffold length ($R^2 = 0.83$; Figure 3a). However, scaffolds QXJH01000030.1 and QXJH01000212.1 exhibited a higher number of candidate loci relative to scaffold length (deviating from the linear distribution), with candidate SNPs comprising 0.0062% and 0.0108% of the total scaffold nucleotides, respectively. Pairwise linkage among SNPs across the entirety of these scaffolds was high (D ' [?] 1), with the detection of large haplotype blocks indicating large sections of the genome linked to virus exposure (Figure 3b).

3.3 | Functional annotations

Candidate SNP loci which showed significant associations with virus exposure were successfully mapped to the annotated *H. rubra*genome. SNPeff analyses predicted 333 candidate loci to have moderate effect on protein function (involving non-synonomous mutations), while 489 candidates were predicted to have low effect, and 24,722 candidates were recognised as non-coding or intergenic variants. Candidate loci that

successfully mapped to *H. rubra* genome peptides sequences, were found to correspond with gene homologs in other animal systems including haliotids, non-haliotid marine molluscs, crustaceans, and humans. These include 13 gene homologs linked to HaHV-1 immunity in New Zealand pāua (*H. iris*), and 13 genes associated with herpes virus response pathways in Japanese disk abalone (*H. discus hannai*), decapod crustaceans (*Penaues monodon* and*Procambarus clarkia*), and humans (Table 2). An additional 10 peptides mapped to gene homologs associated with host-virus interactions in various haliotids (*H. discus hannai* , *H. laevigata* , and*H. ruscefens*), decapod crustaceans (*Penaues monodon* and*Procambarus clarkia*), and humans (Table 2). All gene homologs and known functions are provided in Table 2. Notable findings include several genes linked to chitin-binding peritrophin-A domain, and the cytochrome P450 (CYP) 3A family, which have recognised associations with immune responses in aquatic molluscs (Badariotti *et al.* 2007; Zhao *et al.* 2017) and humans (Fattahi *et al.* 2018), respectively. Also, the CREB-binding protein (CBP), which is associated with herpesvirus responses in humans (Gwack *et al.* 2001; Chen*et al.* 2020), as well as immune pathways for C-type lectins that are important contributors to innate immune responses in invertebrates (Nam *et al.* 2016; Zhang *et al.* 2018; Qin *et al.*2019).

4 | DISCUSSION

We provide evidence of rapid genetic changes over a single generation in wild *H. rubra* populations decimated by HaHV-1. Specifically, our GWAS identified SNP loci associated with virus exposure, many of which mapped to genes known to contribute to HaHV-1 immunity, herpes virus response pathways, and host-virus interactions in haliotids and other animal systems. These findings require experimental validation but point to rapid evolutionary changes in *H. rubra* fishing stocks impacted by disease, with stock recovery influenced by evolved resistance. This study highlights the pace at which selection can act to counter disease in wildlife communities by leading to an increased frequency of adaptive genotypes. The implications of these findings are discussed in the context of future infectious diseases management in abalone fisheries and wildlife conservation more generally.

4.1 | Evidence of rapid evolution in *H. rubra*

Our analyses identified approximately 25,000 SNP loci associated with AVG exposure in *H. rubra* , suggesting that selection has driven advantageous genetic variants to higher frequencies in virus affected populations. The number of positively associated candidates is a substantial proportion of the total number of SNPs included in our analyses (0.4%). The strong linear relationship between genome scaffold length and density of candidate SNPs suggests a genome wide response to selection pressure. However, only some SNPs identified here may contribute to pathogen resistant phenotypes, given linkage disequilibrium among loci and the fact that alleles contributing to resistance will be embedded in large haploblocks. Nevertheless, we did find a higher incidence of significant SNPs on two scaffolds, which could be indicative of important regions of adaptation. The genes annotated within these regions are not known for providing virus resistance, but further work is needed to test for possible functional significance of these regions.

Despite the potential for linkage between loci, our analyses indicate that some candidate loci may be directly involved in disease adaptation in *H. rubra* . In particular, functional annotations of candidate loci point to associations with genes and protein domains that contribute to HaHV-1 immunity in the New Zealand pāua (*H. iris*). Neave *et al.* (2019) first characterised genes associated with HaHV-1 immunity in *H. iris* through transcriptome analyses of animals subject to HaHV-1 immersion challenge tests. This study was the first to characterise the molecular basis of HaHV-1 immunity in a haliotid species, and our study complements these findings by identifying a common set of genes involved in haliotid host response to HaHV-1 exposure. Functional annotations of candidate loci also point to associations with genes and protein domains contributing to herpes simplex virus responses and immune responses in various haliotids and other animal systems. These findings strongly support the notion that emergent genomic architectures have resulted in divergent adaptation and possible selection for disease resistance phenotypes in *H. rubra*fishing stocks impacted by AVG.

The results of this study point to heritable genetic changes in *H. rubra* fishing stocks impacted by disease.

However, experimental validation will be needed to link any genetic changes to disease resistance. Challenge tests involving the exposure of animals with putatively resistant genotypes to HaHV-1 will help to determine if, and how much, HaHV-1 resistance is determined by the candidate genotypes (Crane *et al.* 2013; Corbeil *et al.* 2016). Given that a large number of loci appear to be responding to the virus, we suspect that changes in resistance will represent a polygenic response that can be followed by controlled breeding studies (Guarna *et al.* 2017; Gutierrez *et al.* 2018). The response to selection in these studies will depend on levels of heritable variation as well as the intensity of selection which will determine the impact of the phenotypic change. Breeding studies and experimental trials will also be essential to assess trait heritability and genotype-environment interactions, particularly if industry intend to control for resistance traits in a culture environment for breeding purposes (discussed below).

4.2 | Implications for fisheries management

Re-emergence of AVG remains a significant threat to the economic viability of *H. rubra* fisheries in south-eastern Australia (Lafferty *et al.* 2015; Corbeil 2020). Therefore characterising the spatial distribution and prevalence of disease resistant genotypes will help managers identify stocks expected to be either resilient or vulnerable to AVG re-emergence. Previous population genomic research has indicated a lack of biological stock structure in these fisheries (Miller *et al.* 2016), suggesting that gene flow could contribute to the spread of adaptive genotypes and resilience of naïve fishing stocks. However, gene flow from unaffected parts of the fishery could eventually reduce the frequency of adaptive genotypes over time in the absence of ongoing selection. Whether selection for resistance will be a recurring process is still unclear. However, for the first time in a decade AVG was recorded in 2021, leading to abalone mortalities at a few proximal fishing locations heavily impacted by AVG in the early 2000s (Agriculture Victoria 2021). Unlike the first outbreak, animal mortality and disease spread has been minimal. While environmental and epidemiological factors may be contributing to the suppression of the disease (Bai *et al.* 2019a; Corbeil 2020), it is possible that the presence of adaptive phenotypes has already reduced the number of susceptible animals and overall viral load within affected fishing stocks.

Evidence of panmixia in *H. rubra* (Miller *et al.* 2016) suggests that standing genetic variation is likely to persist within disease naïve populations allowing for *in situ* adaptation to HaHV-1. However, strategic stock augmentation activities, involving the translocations of animals with AVG resistant genotypes, could potentially assist the spread of genotypes to reduce risks of vulnerability across wild fisheries. Also, there may be future opportunities to biosecure farm fisheries through the establishment of AVG resistant breeding programs, similar to disease related breeding programs in other farmed mollusc, crustacean and finfish fisheries around the world (Ragone Calvo *et al.* 2003; Kjøglum *et al.*2008; Moss *et al.* 2012; Potts *et al.* 2021). Overall, these results add to those of Miller *et al.* (2019) demonstrating patterns of genetic adaptation across environmental gradients and the adaptability of *H. rubra* populations to new environmental conditions. This is pertinent in south-eastern Australia where rapid changes in the physical marine climate are threatening commercial fisheries through shifts in species distributions (Ling 2008; Johnson*et al.* 2011), changes in habitat and trophic interactions (Holland *et al.* 2021), and risks of infectious diseases (Oliver*et al.* 2017).

5 | Conclusion

The spread and impact of existing and emerging infectious diseases is expected to intensify as a result of ongoing land use change, climate effects and anthropogenic vectors (Daszak *et al.* 2001; Tompkins*et al.* 2015; Price *et al.* 2019). Disease responses in individual species have the potential to influence wider patterns of biodiversity, ecosystem function, human health and agriculture, particularly those of high functional or economic importance (Daszak*et al.* 2001; Wiethoelter *et al.* 2015; Cunningham *et al.* 2017). Consequently, understanding the ability of such species to adapt to infectious disease is becoming increasingly important. While it has been proposed that selection in some species may fail to keep pace with rates of pathogen evolution and the emergence and turn-over of novel diseases (Hawley *et al.* 2013; Ujvari *et al.* 2014), our study demonstrates that rapid evolutionary responses within a single generation are possible. This study highlights the value of genome scans for identifying signatures of adaptation among natural populations spanning infection gradients and characterising putative genes that contribute to disease resistant phenotypes. Future

studies of this nature will be critical for understanding the adaptability of other species threatened by infectious diseases, informing the management new outbreaks, and future proofing populations with little or no resistance.

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Author Contributions

This project was conceived by A.D.M, while O.J.H, M.T, A.D.M, and L.C were responsible for generating and analysing the data, with assistance from C.A and A.A.H. Writing of the manuscript was led by O.J.H and A.D.M with assistance from all authors.

Data accessibility

All genomic data sets are available upon request and will be made available in the DRYAD archives.

Table 1. Site location details and corresponding codes for 14 collection locations of *Haliotis rubra* from Victorian Western and Central Zone fisheries. Sample sizes and AVG exposure history are also provided.

Zone and Location	Code	Year Sampled	Sample Size	GPS Location	GPS Location	AV
				Latitude	**Longitude**	
Western Zone fishery	Western Zone fishery					
Port Macdonell	**PMC**	2020	25	–38.054	140.881	una
Inside Murrels	**MUR**	2009	25	–38.407	141.524	affe
Inside Nelson	**ISN**	2009	25	–38.409	141.558	affe
The Crags	**CRG**	2009	25	–38.390	142.135	affe
Lady Julia Percy	**LJP**	2009	23	–38.422	141.993	una
Killarney	**KIL**	2015	20	–38.363	142.321	affe
Levies	**LEV**	2009	25	–38.385	142.235	affe
Central Zone fishery	Central Zone fishery					
Childers Cove	**CHC**	2019	25	–38.490	142.672	affe
Bay of Islands	**BIP**	2019	25	–38.582	142.827	affe
Cat Reef	**CAT**	2015	25	–38.741	143.188	affe
White Cliffs	**WCF**	2015	25	–38.758	143.330	affe
Castle Cove	**CCV**	2020	25	–38.783	143.422	una
Parker River	**PKR**	2020	25	–38.855	143.538	affe
Blanket Bay	**BLK**	2015	25	–38.827	143.586	una

Table 2. List of predicted genetic variant impacts, and genes that candidate loci mapped to. Table also includes gene functions, as well as the species from which these functions have been reported and their respective references.

Number of candidate SNPs	Predicted variant Impact
Genes involved *H. iris* HaHV-1 immune response	**Genes involved *H. iris* HaHV-1 immune re**
17	MODERATE, LOW, MODIFIER

Number of candidate SNPs	**Predicted variant Impact**
35	MODERATE, LOW, MODIFIER
6	MODERATE, MODIFIER
2	MODIFIER
3	MODIFIER
2	MODIFIER
1	MODIFIER
1	MODIFIER
17	MODIFIER
15	MODIFIER
1	MODIFIER
14	MODIFIER
1	MODIFIER
Gene homologs associated with herpesvirus response pathways	**Gene homologs associated with herpesviru**
2	MODIFIER
1	MODIFIER
1	MODIFIER
7	MODIFIER
2	MODIFIER
6	MODIFIER
2	MODIFIER
2	MODIFIER
2	MODIFIER
1	MODIFIER
1	MODIFIER
4	MODIFIER
1	MODIFIER
Gene homologs associated with host-virus interactions	**Gene homologs associated with host-virus**
4	MODIFIER
1	LOW
1	LOW, MODIFIER
1	MODIFIER
4	MODIFIER
8	LOW, MODIFIER
1	MODIFIER
3	MODIFIER
1	MODIFIER
2	MODIFIER

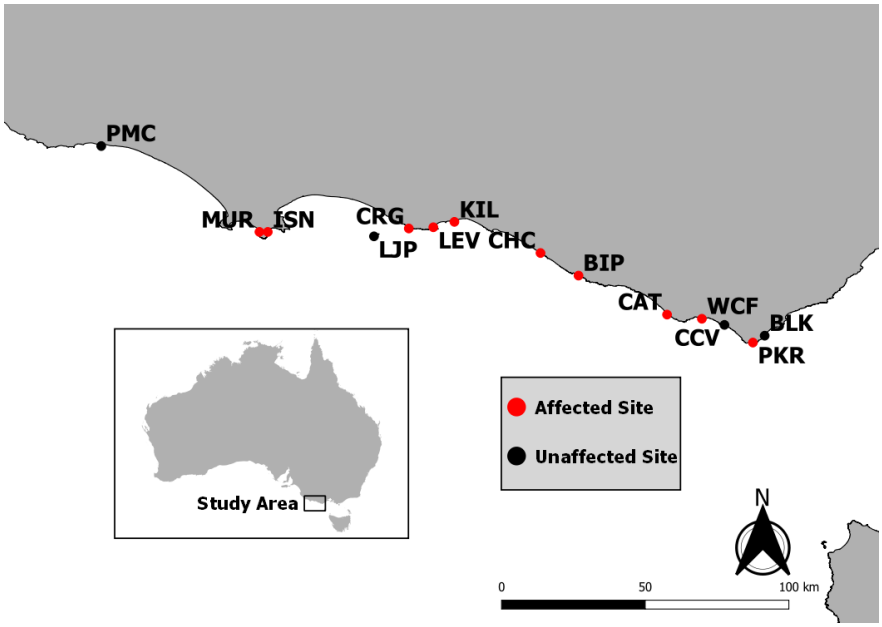

Figure 1. Sampling sites selected for population genomic analysis from the south-eastern Australia. Figure legend and colour coding of mapped sites indicate history of virus exposure. Refer to Table 1 for sample codes.

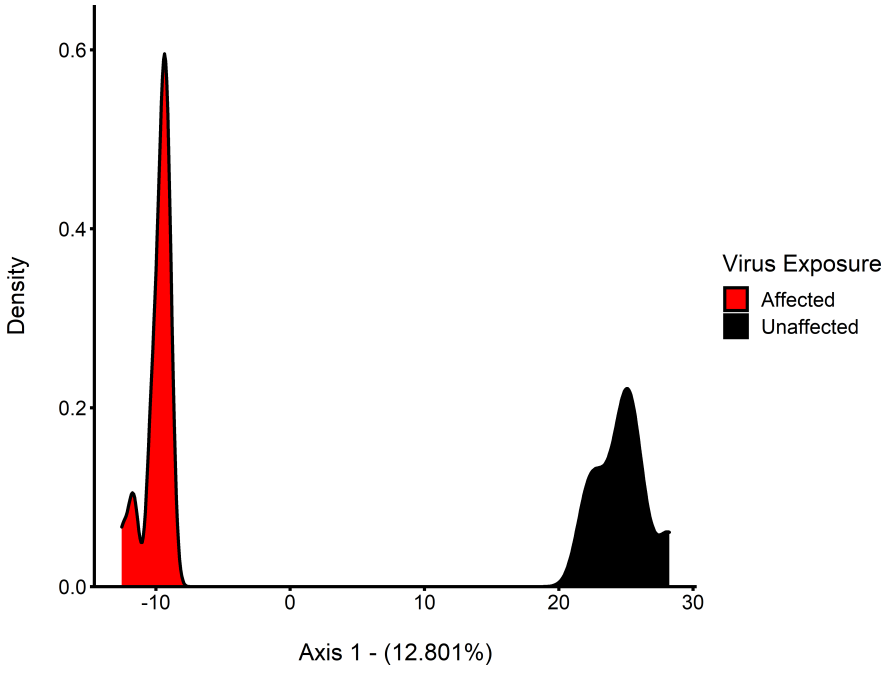

Figure 2. Density plot of axis 1 eigenvalues from the Principal Components Analysis. Plots are based on candidate SNP genotypes from each of the 28 pooled whole genome resequencing libraries representing virus affected (red) and unaffected (black) fishing stocks.

Figure 3. **(a)** Regression analysis indicating a positive linear relationship between number of candidate

SNPs (BF > 50) and scaffold length. Outlier scaffolds with a greater frequency of candidate SNPs relative to scaffold length are plotted in red. **(b)**Heatmaps of outlier scaffolds generated with the package LDBlockShow (Dong, et al., 2021) depicting the pairwise linkage disequilibrium measure of *D* ' (refer to colour key) between each SNP with a BF [?] 50. In addition, black triangle sections represent detected haplotype blocks; genomic regions of low recombination.

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