GENOMICS

Genomic signatures of disease resistance in endangered staghorn corals

Steven V. Vollmer†*, Jason D. Selwyn†, Brecia A. Despard, Charles L. Roesel

White band disease (WBD) has caused unprecedented declines in the Caribbean *Acropora* corals, which are now listed as critically endangered species. Highly disease-resistant *Acropora cervicornis* genotypes exist, but the genetic underpinnings of disease resistance are not understood. Using transmission experiments, a newly assembled genome, and whole-genome resequencing of 76 *A. cervicornis* genotypes from Florida and Panama, we identified 10 genomic regions and 73 single-nucleotide polymorphisms that are associated with disease resistance and that include functional protein-coding changes in four genes involved in coral immunity and pathogen detection. Polygenic scores calculated from 10 genomic loci indicate that genetic screens can detect disease resistance in wild and nursery stocks of *A. cervicornis* across the Caribbean.

Increased global CO_2 emissions and the resulting ocean warming have devastated tropical reef corals by increasing the frequency and severity of thermal bleaching events (*1*–*3*) and disease outbreaks (*4*, *5*). Emergent infectious diseases in Caribbean corals cause high levels of coral mortality (*6*–*8*), with white band disease (WBD) killing up to 95% of the critically endangered Caribbean *Acropora* corals (*8*), and stony coral tissue loss disease (SCTLD) is, at present, decimating more than 20 key reef-building coral species (*6*, *7*). The rising toll of thermal bleaching and diseases on coral reefs globally has focused scientific efforts on identifying thermally resilient and disease-resistant coral species, individuals, genes, and symbionts that allow reef corals to adapt to future climate scenarios (*9*–*11*), including through human intervention (*9*). Adaptive polygenic variation for thermal tolerance with relatively high heritability exists in corals (*11*), although the identification of genetic markers that strongly influence thermal tolerance is more elusive (*12*). Less is known about the genetics of coral disease resistance. Phenotypic variation in disease resistance to WBD has been documented in the staghorn coral, *Acropora cervicornis* (*13*, *14*), and may be heritable and adaptive.

WBD is a highly transmissible, host-specific disease that infects the two sister species of Caribbean *Acropora* (*8*, *15*–*17*): the staghorn coral, *A. cervicornis*, and the elkhorn coral, *Acropora palmata*, as well as their hybrid species, *Acropora prolifera* (*18*, *19*). Since it was first observed in 1979 (*15*), WBD has killed up to 95% of Caribbean *Acropora* and is found throughout the greater Caribbean (*8*). WBD is caused by a bacterial pathogen or pathogens that can be arrested with antibiotics (*16*, *20*) and quorum-sensing inhibitors (*21*, *22*). Multiple putative bacterial pathogens have been associated with WBD (*20*, *23*), including *Vibrio* spp. (*24*, *25*), *Aquarickettsia* (*26*, *27*), and other bacteria (*23*). *A. cervicornis* genotypes display strong phenotypic variation in disease resistance (*13*, *14*) and mount a vigorous immune response to WBD infection (*28*, *29*), with highly resistant genotypes up-regulating genes involved in microRNA-induced posttranscriptional gene regulation (*29*).

We conducted a genome-wide association study (GWAS) to identify genetic variants associated with disease resistance to WBD in *A. cervicornis* from two geographically distant populations: Florida and Panama. Disease resistance was assayed in tank-based transmission experiments that were conducted separately in Florida and Panama with 50 coral genotypes from each location (Fig. 1A). Florida *A. cervicornis* were sourced from Coral Restoration Foundation nursery stocks, and the Panama *A. cervicornis* were sampled from wild populations in Bocas del Toro. A Cox proportional hazards model was used to calculate normalized disease-resistance scores across all 100 genotypes (Fig. 1B) while accounting for experimental and/or population effects of the two independent transmission experiments. Coral genotype explained 6.1% of the variation in disease resistance [χ^2(effective df = 65, reference df = 99) = 2392, $p < 0.0001$; df, degrees of freedom], experimental tank explained 8.4% of the variance [χ^2(7.8, 9) = 4779, $p = 0.00008$], and experiment location and/or population explained 28.8% of the variance [χ^2(0.99, 1) = 23,127, $p < 0.00001$]. The experiment location and population variation in the transmission experiments could result from a variety of factors (*30*), including differences in microbial exposure doses and host microbiomes as well as spatiotemporal environmental variation between the two study sites.

We randomly selected 48 genotypes from each population for whole-genome sequencing (WGS) and assembled and annotated a high-quality, de novo genome using adult tissue from the K2 genotype from Coral Restoration Foundation, Florida, with high-quality nanopore sequencing and short-read polishing. The 308-Mb scaffolded *A. cervicornis* K2 genome had an N50 of 2.8 Mb (where N50 is the minimum scaffold length needed to cover 50% of the

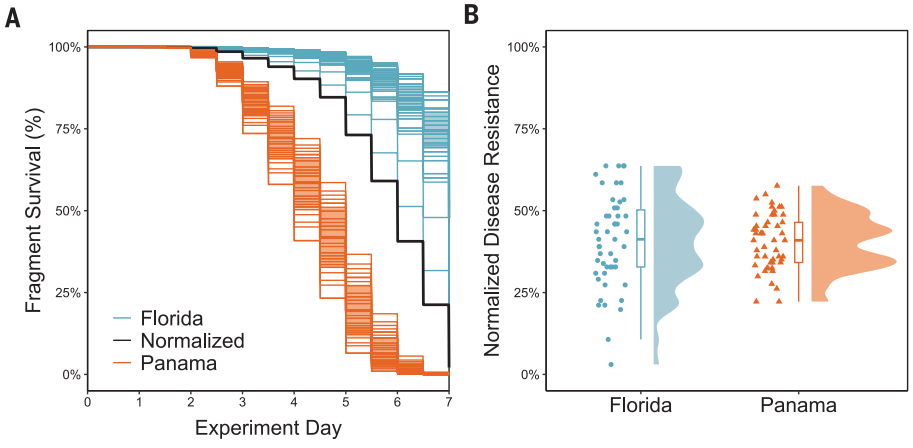

Fig. 1. Tank-based infection, survival, and disease resistance. (**A**) Fragment survival rates (%) from the two independent tank-based transmission experiments conducted in Florida (blue) and Panama (orange). A Cox proportional hazard model detected significant effects of genotype [χ^2(effective df = 65, reference df = 99) = 2392, $p < 0.0001$], experimental tank [χ^2(7.8, 9) = 4779, $p = 0.00008$], and experiment location and/or population [χ^2(0.99, 1) = 23,127, $p < 0.00001$] on fragment survival. Genotype explained 6.1% of the variation in survival, whereas experiment location and/or population (i.e., Florida versus Panama) explained 28.8% of the variation. (**B**) Normalized disease resistance (one minus the probability of infection; see methods for details) at day 6 of exposure for the 50 Florida and 50 Panama *A. cervicornis* genotypes that were surveyed. The center line represents the median, box limits are upper and lower quartiles, and whiskers are minimum and maximum values.

Department of Marine and Environmental Sciences, Northeastern University, 430 Nahant Road, Nahant, MA 01908, USA.
*Corresponding author. Email: s.vollmer@northeastern.edu
†These authors contributed equally to this work.

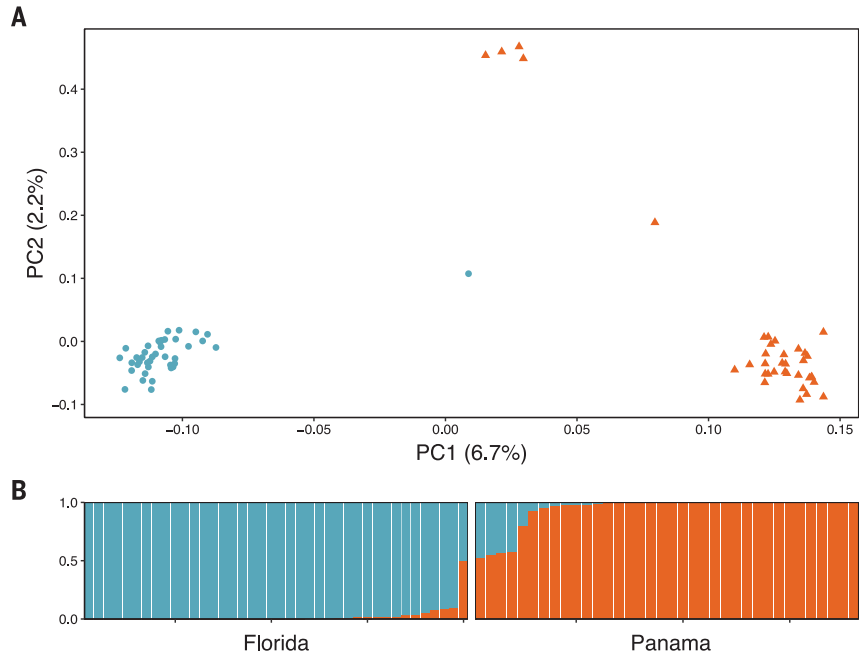

Fig. 2. Population structure and admixture between Florida and Panama. (**A**) PCA showing the strong genetic differences between *A. cervicornis* from Florida (blue) and Panama (orange) (F_{ST} = 0.04, p < 0.001). (**B**) Structure-based assignment analysis (k = 2 populations) showing greater than 10% admixture in six individuals.

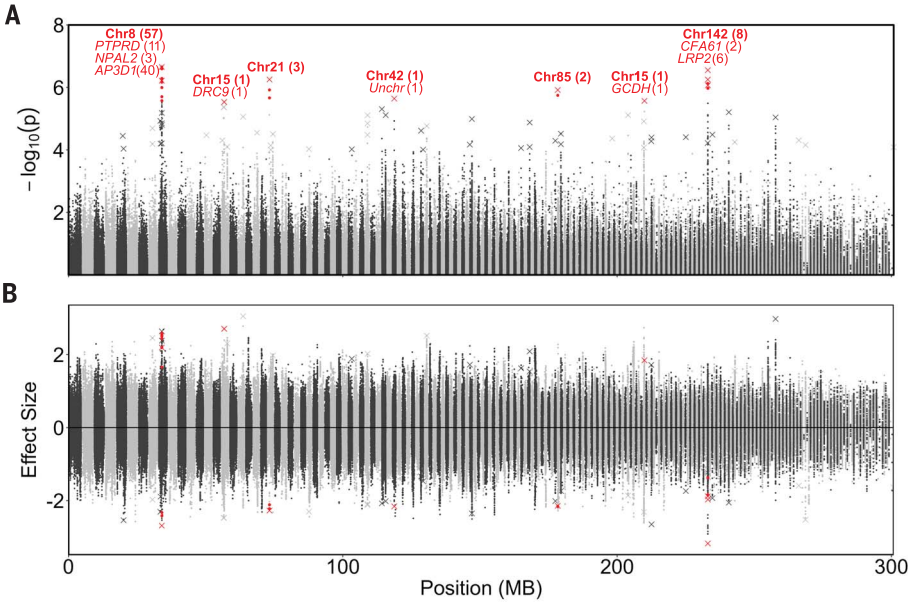

Fig. 3. Genome-wide SNP association analyses of disease resistance. (**A**) Manhattan plot of p values across 1.2 million high-quality SNPs in the *A. cervicornis* genome. (**B**) Effect-size plot across the genome, in which positive effect sizes indicate that the rare allele is associated with increased disease resistance and negative effect sizes indicate that the common allele is associated with increased disease resistance. Seventy-three significant disease-associated SNPs (adjusted p < 0.05) are labeled in red. The numbers of significant SNPs per chromosome (chr) and gene are summarized and labeled in red. The × symbol identifies the index SNP from each linkage group that was used to calculate polygenic scores, with red indicating the top 10 index SNPs from the significant linkage groups and gray indicating the 53 additional SNPs that were used to calculate the expanded polygenic score (p < 0.0001).

genome) (fig. S1) and a BUSCO completeness of 92% and contains 28,059 genes on 398 scaffolded contigs (hereafter, chromosomes), 54% of which were annotated to known SwissProt reference proteins (E-value < 10^{-6}). The 308-Mb *A. cervicornis* genome is somewhat smaller than that of Pacific Acroporids (384 to 475 Mb) and has an intermediate number of gene annotations [23,467 to 41,860 (*31*)].

Using the K2 reference genome, we produced WGS data for 96 putative genotypes (48 from Florida and 48 from Panama) using Illumina paired-end 150–base pair sequencing. Four samples were removed because of low coverage, leaving 92 putative genotypes with high-quality WGS data (mean 74× coverage ± 3.6 SE) that included 1.2 million single-nucleotide polymorphisms (SNPs) [minor allele frequency >0.05] and 54,871 unlinked SNPs [linkage disequilibrium (r^2_{LD}) < 0.5, 250 kb]. A total of 76 unique staghorn coral genotypes (40 from Florida and 36 from Panama) were retained after filtering for clones based on 97% genetic similarity (fig. S2). *Symbiodinium* was the dominant algal symbiont in *A. cervicornis* (fig. S3), and the composition of algal symbionts did not differ by host disease resistance ($F_{1,72}$ = 0.04, p = 0.65) or the interaction with location ($F_{1,72}$ = 0.02, p = 0.78). There were significant compositional differences between Florida and Panama ($F_{1,72}$ = 5.65, p = 0.012, r^2 = 0.07), with slight relative increases in *Breviolum* and *Fugacium* in Panama.

Principal components analysis (PCA) and admixture analysis (*32*) clustered *A. cervicornis* from Florida and Panama into two distinct populations (Fig. 2 and fig. S4) separated by an average population genetic difference (F_{ST}) of 0.038 (p < 0.001), which is similar to the population genetic structure that was detected using mitochondrial and nuclear sequencing data (*33*, *34*) and suggests restricted Caribbean-wide gene flow. Six genotypes—one from Florida and five from Panama—had greater than 10% population-level admixture (Fig. 2), but this admixture was not correlated to disease resistance (Kendall's rank correlation coefficient = 0.05, p = 0.53) (fig. S5) or recent introgressive hybridization (fig. S6).

Genetic variants associated with disease resistance

We used a latent factor mixed model to account for underlying population structure between Florida and Panama [inferred populations (k) = 2; fig. S4] and identify SNPs associated with disease resistance (table S1). Clumping and thresholding was used to organize all 1.2 million SNPs into linkage groups (r^2_{LD} > 0.5, 250 kb). Seventy-three SNPs in 10 linkage groups on seven chromosomes were significantly associated with disease resistance after false discovery rate correction (adjusted p < 0.05; Fig. 3 and table S1). Three linkage groups

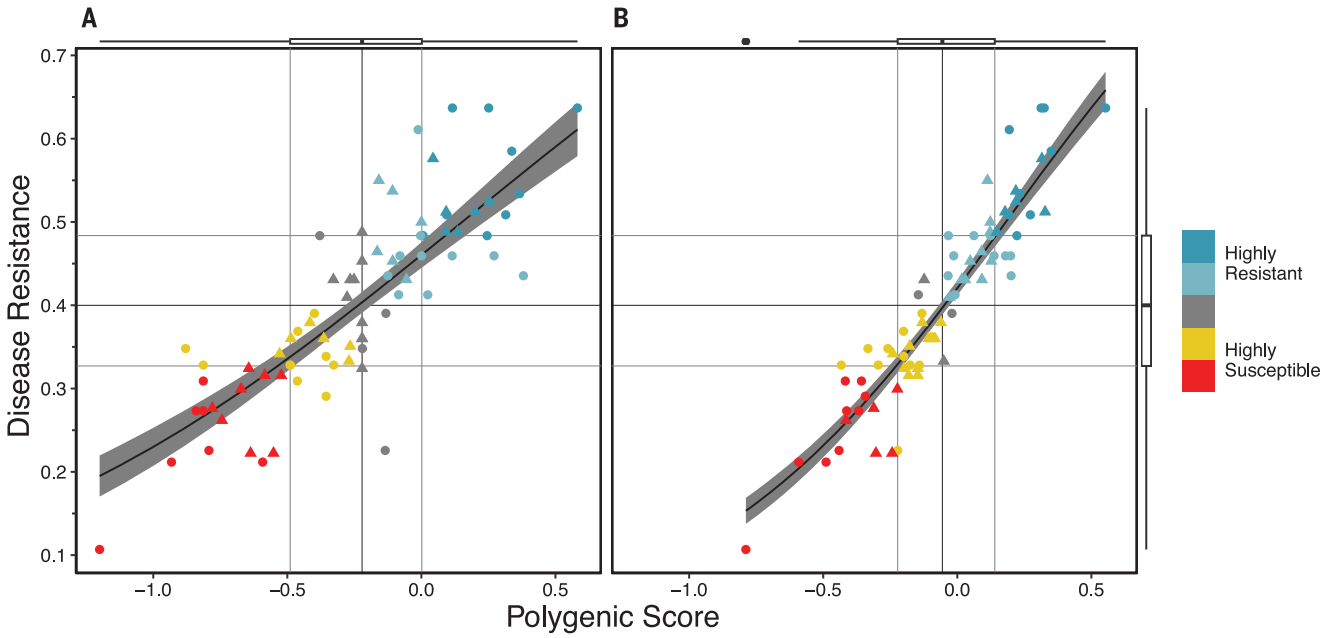

Fig. 4. Polygenic scores that predict disease resistance. (**A**) Polygenic scores for the top ten SNPs and genomic regions explain 66.9 ± 5.4% of disease resistance ($p < 0.0001$). (**B**) Polygenic scores based on the most important 63 SNPs and genomic regions ($p < 0.0001$) explain 85.3 ± 2.3% of the variation in disease resistance, showing that the inclusion of 53 additional loci improved disease-resistance predictions based on polygenic scores. Individuals represented by blue symbols have above-average polygenic scores and disease resistance; the dark blue points indicate individuals that are highly disease resistant, with polygenic scores and disease-resistance values in the top quartiles. Individuals represented by yellow and red symbols have lower-than-average polygenic scores and disease resistance, with red symbols indicating highly susceptible individuals. Coral genotypes from Florida are labeled with a circle and those from Panama are labeled with a triangle. The gray shaded area indicates ±1 standard error.

had SNPs with positive effect sizes, indicating that the rare allele is associated with increased disease resistance, whereas the remaining seven linkage groups had negative effect sizes in which the common allele was beneficial. Sixty-five disease resistance–associated SNPs occurred in annotated genes, including 54 SNPs in three genes—*AP3D1* (40), *PTPRD* (11), *NPAL2* (3)—on chromosome 8, and eight SNPs in two genes—*LRP2* (6) and *CFA61* (2)—on chromosome 142. Eight out of the 65 significant SNPs in annotated genes occurred in protein-coding regions. Only one significant SNP in *PTPRD* resulted in a functional, nonsynonymous (i.e., amino acid) change. *PTPRD* and *AP3D1* also contained one and three significant SNPs that resulted in synonymous changes, respectively. However, multiple nonsynonymous changes occurred in linkage groups that contained significant disease resistance–associated SNPs (fig. S7), including nonsynonymous changes in *PTPRD* (2), *AP3D1* (1), and *SECG* (1) on chromosome 8 and *LRP2* (7) and *CFA61* (1) on chromosome 142. The eight remaining significant SNPs that fell outside of annotated genes included three SNPs linked to the significant SNPs on genes in chromosome 8, three SNPs linked to an uncharacterized gene on chromosome 21, and two SNPs on chromosome 85 that were not linked to significant SNPs on genes.

Four of the five candidate disease-resistance genes with functional, protein-coding changes—*PTPRD*, *AP3D1*, *SECG*, and *LRP2*—have roles in coral immunity. Protein tyrosine phosphatase receptor type D (*PTPRD*) is a tumor suppressor gene (*35*) in the receptor protein tyrosine phosphatases (PTPs) family that regulates cell signaling through dephosphorylation and acts as an immune regulator (*36*). AP-3 complex subunit delta-1 (*AP3D1*) is part of the AP-3 complex, which regulates lysosomal trafficking and endocytosis and is specifically involved in the sorting of cargo molecules into clathrin-coated vesicles during endocytosis (*37*). Ankyrin repeat, PH and SEC7 domain–containing protein secG (*SECG*) is a cytohesin protein involved in signal transduction, immune regulation, and phagocytosis (*38*). Low-density lipoprotein receptor–related protein 2 (*LRP2*) is an innate immune gene that activates the complement system and nuclear factor κB (NF-κB) to initiate endocytosis (*39*). The fifth gene, cilia- and flagella-associated protein 61 (*CFA61*), is part of the calmodulin and spoke-associated complex that is involved in cilia motility (*40*).

PTPRD and *LRP* have previously been shown to be differentially expressed in WBD-infected *A. cervicornis* (*28*) and *A. palmata* (*41*). *LRP* genes have also been reported to be up-regulated in multiple Caribbean corals infected with white plague (*42*). However, none of the five candidate disease-resistance genes with functional variants were constitutively or differentially expressed in six genotypes from the Florida transmission experiment (fig. S10 and table S6) as a result of the observed disease resistance; this effect was also not reported in prior research that examined the links between gene expression and disease resistance in *A. cervicornis* (*29*). The lack of gene expression polymorphisms at these five candidate disease-resistance genes suggests that the functional protein-coding changes in *PTPRD*, *AP3D1*, *SECG*, *LRP2*, and *CFA61* influence disease resistance rather than changes in their gene expression.

Shared polygenic variation explains disease resistance

Polygenic scores calculated for the 76 *A. cervicornis* genotypes, by using the top index SNP from each of the 10 linkage groups with significant SNPs, explained 67.9 ± 5.4% of the variation in observed *A. cervicornis* disease resistance (Fig. 4A and table S2). Thirty-three genotypes (44.6%; 19 from Florida and 14 from Panama) had above-average polygenic scores and displayed disease resistance, with 15 genotypes (20.2%; nine from Florida and six from Panama) scoring as highly disease-resistant and falling in the top 25% for both polygenic score and disease resistance. By contrast, 31 genotypes (41.9%; 17 from Florida and 14 from Panama) had below-average polygenic scores and disease resistance, with 15 genotypes (20.2% total; seven from Florida and eight from Panama) scoring as highly disease susceptible.

Extending the polygenic score to 63 loci ($p < 0.0001$) captured 85.5 ± 2.3% of the observed variation in disease resistance (Fig. 4B and table S2), indicating that these 53 additional genetic loci have small, but measurable, effects on observed disease resistance.

Our data show that, similar to coral thermal tolerance (*43*), disease resistance within *A. cervicornis* is polygenic across multiple genomic regions and chromosomes. Although gene variants strongly associated with thermal tolerance have been difficult to identify with GWASs (*12*), we identified 10 genomic regions and 73 loci that were associated with *A. cervicornis* disease resistance, including linked functional protein-coding variation within five coral disease-resistance genes. Thus, natural selection for increased coral immunity and disease resistance may be higher than it is for thermal tolerance.

The shared adaptive polygenic variation in *A. cervicornis* disease resistance that is observed in Florida and Panama indicates that genetic variation for disease resistance is widely dispersed across the Caribbean, despite the restricted gene flow that we and others have found (*33*, *34*), and thus could be used to improve disease resistance through natural or assisted selection. *Acropora* populations throughout the greater Caribbean are under active conservation management and restoration because of their critically endangered status (*44*, *45*). Florida is a hotspot for *Acropora* restoration, with hundreds of *A. cervicornis* genotypes being actively propagated in land-based and in situ nurseries (*46*, *47*), including the Coral Restoration Foundation nurseries from which our Florida genotypes were sourced. More than 25,000 *A. cervicornis* fragments have been outplanted across Florida (*47*) and elsewhere in the Caribbean.

Our research shows that polygenic scores from the top 10 SNPs and genomic regions identified disease-resistant *A. cervicornis* genotypes. Future research is required to establish whether there are fitness trade-offs between disease resistance and other key metrics such as growth, fecundity, thermal tolerance, and survival (*48*). Although fitness trade-offs are, as of now, largely unknown, WBD prevalence and susceptibility often increase with temperature stress (*13*). Recent evidence indicates that *A. cervicornis* varies in its thermal tolerance (*46*), and thus future efforts need to determine how disease resistance, thermal tolerance, and their genetic bases influence the fitness and future resiliency of *A. cervicornis* across the Caribbean.

REFERENCES AND NOTES

1. T. P. Hughes *et al*., *Science* **359**, 80–83 (2018).
2. J. F. Bruno *et al*., *PLOS Biol.* **5**, e124 (2007).
3. O. Hoegh-Guldberg *et al*., *Science* **318**, 1737–1742 (2007).
4. C. D. Harvell *et al*., *Science* **285**, 1505–1510 (1999).
5. C. A. Burge *et al*., *Annu. Rev. Mar. Sci.* **6**, 249–277 (2014).
6. L. Alvarez-Filip, F. J. González-Barrios, E. Pérez-Cervantes, A. Molina-Hernández, N. Estrada-Saldívar, *Commun. Biol.* **5**, 440 (2022).
7. W. F. Precht, B. E. Gintert, M. L. Robbart, R. Fura, R. van Woesik, *Sci. Rep.* **6**, 31374 (2016).
8. R. B. Aronson, W. F. Precht, in *The Ecology and Etiology of Newly Emerging Marine Diseases*, J. W. Porter, Ed., vol. 159 of *Developments in Hydrobiology* (Springer, 2001), pp. 25–38.
9. M. J. van Oppen, J. K. Oliver, H. M. Putnam, R. D. Gates, *Proc. Natl. Acad. Sci. U.S.A.* **112**, 2307–2313 (2015).
10. J. Kleypas *et al*., *Biol. Conserv.* **257**, 109107 (2021).
11. E. Mcleod *et al*., *J. Environ. Manage.* **233**, 291–301 (2019).
12. Z. L. Fuller *et al*., *Science* **369**, eaba4674 (2020).
13. E. M. Muller, E. Bartels, I. B. Baums, *eLife* **7**, e35066 (2018).
14. S. V. Vollmer, D. I. Kline, *PLOS ONE* **3**, e3718 (2008).
15. W. B. Gladfelter, *Bull. Mar. Sci.* **32**, 639–643 (1982).
16. D. I. Kline, S. V. Vollmer, *Sci. Rep.* **1**, 7 (2011).
17. S. A. Gignoux-Wolfsohn, C. J. Marks, S. V. Vollmer, *Sci. Rep.* **2**, 804 (2012).
18. M. J. Van Oppen, B. L. Willis, H. W. Van Vugt, D. J. Miller, *Mol. Ecol.* **9**, 1363–1373 (2000).
19. S. V. Vollmer, S. R. Palumbi, *Science* **296**, 2023–2025 (2002).
20. M. J. Sweet, A. Croquer, J. C. Bythell, *Proc. Biol. Sci.* **281**, 20140094 (2014).
21. R. H. Certner, S. V. Vollmer, *Sci. Rep.* **5**, 11134 (2015).
22. R. H. Certner, S. V. Vollmer, *Environ. Microbiol.* **20**, 645–657 (2018).
23. S. A. Gignoux-Wolfsohn, F. M. Aronson, S. V. Vollmer, *FEMS Microbiol. Ecol.* **93**, (2017).
24. D. L. Gil-Agudelo, G. W. Smith, E. Weil, *Rev. Biol. Trop.* **54**, 59–67 (2006).
25. K. B. Ritchie, G. W. Smith, *Rev. Biol. Trop.* **46**, 199–203 (1998).
26. L. J. Baker *et al*., *ISME J.* **16**, 400–411 (2022).
27. V. Casas *et al*., *Environ. Microbiol.* **6**, 1137–1148 (2004).
28. S. Libro, S. T. Kaluziak, S. V. Vollmer, *PLOS ONE* **8**, e81821 (2013).
29. S. Libro, S. V. Vollmer, *PLOS ONE* **11**, e0146636 (2016).
30. R. Vega Thurber *et al*., *Front. Ecol. Evol.* **8**, 575927 (2020).
31. C. Shinzato *et al*., *Mol. Biol. Evol.* **38**, 16–30 (2021).
32. E. Frichot, F. Mathieu, T. Trouillon, G. Bouchard, O. François, *Genetics* **196**, 973–983 (2014).
33. E. M. Hemond, S. V. Vollmer, *PLOS ONE* **5**, e8652 (2010).
34. S. V. Vollmer, S. R. Palumbi, *J. Hered.* **98**, 40–50 (2007).
35. S. Veeriah *et al*., *Proc. Natl. Acad. Sci. U.S.A.* **106**, 9435–9440 (2009).
36. T. Mustelin, T. Vang, N. Bottini, *Nat. Rev. Immunol.* **5**, 43–57 (2005).
37. S. J. Neuffer *et al*., *Pigment Cell Melanoma Res.* **35**, 495–505 (2022).
38. R. Müller *et al*., *Cell Commun. Signal.* **11**, 54 (2013).
39. A. Beenken *et al*., *Cell* **186**, 821–836.e13 (2023).
40. P. Urbanska *et al*., *Mol. Biol. Cell* **26**, 1463–1475 (2015).
41. B. D. Young *et al*., *PLOS ONE* **15**, e0228514 (2020).
42. N. J. MacKnight *et al*., *Sci. Adv.* **8**, eabo6153 (2022).
43. E. J. Howells, L. K. Bay, R. A. Bay, in *Coral Reef Conservation and Restoration in the Omics Age*, M. J. H. van Oppen, M. Aranda Lastra, Eds., in vol. 15 of *Coral Reefs of the World* (Springer, 2022), pp. 55–70.
44. National Marine Fisheries Service, *Federal Register* 71, no. FR26852 (8 June 2006), pp. 26852–26872.
45. J. Crabbe *et al*., *Acropora cervicornis*, The IUCN Red List of Threatened Species 2022: e.T133381A165860142 (2022); https://www.iucnredlist.org/species/133381/165860142 [accessed 5 April 2023].
46. R. Cunning *et al*., *Proc. Biol. Sci.* **288**, 20211613 (2021).
47. R. van Woesik *et al*., *Restor. Ecol.* **29**, e13302 (2021).
48. W. C. Million *et al*., *Proc. Natl. Acad. Sci. U.S.A.* **119**, e2203925119 (2022).
49. S. V. Vollmer, J. D. Selwyn, B. A. Despard, C. L. Roesel, Genomic signatures of disease resistance in endangered staghorn corals, Zenodo (2023); https://doi.org/10.5281/zenodo.8095056.

ACKNOWLEDGMENTS

We thank the staff of the Coral Restoration Foundation and Florida Keys Marine Lab in Florida and the staff of the Smithsonian Tropical Research Institute in Bocas del Toro, Panama, for their help with this project. We also thank S. Belliveau, A. Moura, and the Coral Restoration Foundation volunteers who assisted with the transmission experiments and E. Trytten, R. Aronson, and R. Gatins, who helped review the manuscript. Sample collections were permitted with the approval of Autoridad Nacional del Ambiente, Panama (CITES permit nos. SEX/A-03-08 and SEX/A-98-19), and samples were provided by the Coral Restoration Foundation (permit no. CRF-2021-001). **Funding:** Grant funding was provided to S.V.V. by a National Science Foundation Division of Ocean Sciences grant (NSF OCE-1924145). **Author contributions:** Conceptualization: S.V.V.; Methodology: S.V.V., J.D.S., B.A.D., C.L.R.; Investigation: S.V.V., J.D.S., B.A.D., C.L.R.; Visualization: S.V.V., J.D.S.; Funding acquisition: S.V.V.; Project administration: S.V.V.; Supervision: S.V.V.; Writing – original draft: S.V.V., J.D.S.; Writing – review and editing: S.V.V., J.D.S., B.A.D., C.L.R. **Competing interests:** The authors declare that they have no competing interests. **Data and materials availability:** Genome assembly and WGS reads can be accessed from NCBI BioProject with accession numbers PRJNA948411 and PRJNA950067, respectively. Analysis code with disease resistance calculations is available at Zenodo (*49*).

SUPPLEMENTARY MATERIALS

science.org/doi/10.1126/science.adi3601
Materials and Methods
Figs. S1 to S10
Tables S1 to S6
References (*50*–*103*)
MDAR Reproducibility Checklist

Submitted 21 April 2023; resubmitted 30 June 2023
Accepted 9 August 2023
10.1126/science.adi3601

Genomic signatures of disease resistance in endangered staghorn corals

Steven V. Vollmer, Jason D. Selwyn, Brecia A. Despard, and Charles L. Roesel

Science **381** (6665), . DOI: 10.1126/science.adi3601

Editor's summary

Bleaching and disease outbreaks are the biggest threats to coral reefs. Although heritable variation in coral thermal tolerance is documented, the genetic basis of disease resistance is less well understood. Vollmer *et al.* identified 10 genomic regions and 73 single-nucleotide polymorphisms that are strongly associated with white band disease resistance in the endangered Caribbean staghorn coral *Acropora cervicornis* (see the Perspective by Mydlarz and Muller). Ten gene regions were associated with disease resistance, including functional protein coding variation in four genes involved in coral immunity and pathogen detection. Polygenic scores from the top 10 genomic loci could accurately predict observed disease resistance and be applied to improving disease resistance in the wild and nursery stocks of A. cervicornis for reef outplanting. —Caroline Ash