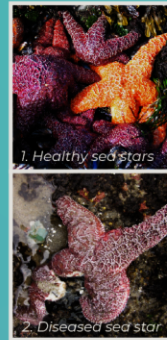


Heterozygosity of *Pisaster ochraceus* in Association with Sea Star Wasting Disease

ABSTRACT

Sea star populations alongside the Pacific intertidal zones have been decimated by a mass mortality event known as the sea star wasting disease (SSWD) in recent years. Symptoms start with starvation, followed by listlessness, and finally growths of white lesions cause rapid tissue decay leading to death. SSWD constitutes a variety of pathogenic factors including viral, bacterial, and fungal infections, and environmental factors such as high sea temperatures, oxygen depletion, and low salinity levels.

My poster focuses on the ochre sea star (*Pisaster Ochraseus*) and how heterozygotes for the elongation factor 1- α locus (EF1A) are less susceptible to SSWD.



METHODS

Site	SSWD symptomatic +/+	SSWD symptomatic +/-	SSWD asymptomatic +/+	SSWD asymptomatic +/-	Effect size
Nanaimo, BC	8	4	7	5	0.089
Olympic Peninsula, WA	5	0	11	4	0.31
San Juan Island, WA	25	17	15	18	0.14
Cape Meares, OR	1	0	4	5	0.2
Seal Rock, Or	1	2	1	6	0.25
Coquille Point, OR	7	0	1	2	0.88
Damnation Creek, CA	1	1	4	4	0
Sonoma County, CA	8	1	22	18	0.21
San Francisco Bay, CA	0	0	7	13	0
OVERALL	56	25	72	75	0.19

Table 1: Regional sample sizes of specimens listed by health status (+/+ denotes wild type, +/- denotes heterozygote genotype)

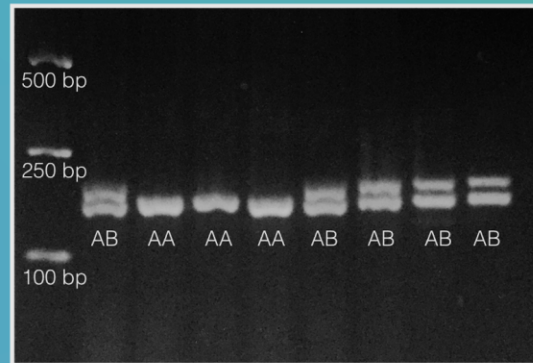


Figure 3. Primers PisEF1-F and PisEF1-R were used to determine individual EF1A genotypes by scoring length-polymorphic PCR products on agarose gels

RESULTS + SIGNIFICANCE

Table 1 Results:

- ★ Analyzed with Fisher's exact test
- ★ Each regional sample showed that SSWD was more prevalent amongst homozygotes than heterozygotes
- ★ Combining all results lead to a p-value of .0035

Despite the hypothesis that increased fitness for EF1A heterozygotes and apparent overdominance are supported, there is no current evidence that this trait is evolutionarily selected for in the regional sample sizes. However, a larger sample size may be needed to decide whether this is the case. Overall, this finding focuses more on the future demographics of *P. ochraceus* in an increasingly warmer and disease-affected environment, rather than a single cure.

Although heterozygote advantage remains a popular explanation for the maintenance of genetic variation associated with disease resistance, this may not necessarily equate that all ochre sea stars afflicted with SSWD would be homozygotes nor would all healthy individuals be heterozygous. Other possibilities could show that regulation and expression of EF1A can alter an individuals' tolerance or capacity for heat stress. This is critical as warming climates and ocean temperatures cause more disease and higher mortality rates due to physiological stress modifying an organism's response to pathogens.

references

<https://aquarium.org/ochre-sea-stars-come-different-colors/>

By Oregon State University - Dying sea star, CC BY-SA 2.0, <https://commons.wikimedia.org/w/index.php?curid=50435037>

Wares, J., & Schiebelhut, L. (2016, March 29). *What doesn't kill them makes them stronger: An association between elongation factor 1- α overdominance in the sea star *Pisaster ochraceus* and "sea star wasting disease"*. PeerJ. Retrieved June 3, 2022, from <https://peerj.com/articles/1876/?td=wk>