Examining Density, Size, and Depth Associations of Balding Sea Urchins

University of California, Santa Cruz
Department of Ecology and Evolutionary Biology

by

Anisa Rizo, Cristal Romero Vasquez, Jack Parks, & Zen Kelly

INTRODUCTION

Primary grazers are quintessential species in every biological community. No matter what type of forces control said community, be it top down, bottom up, or a combination of the two, the primary grazer is always in direct play with the primary resources in the community. If the density of the primary grazer is left unchecked, the species can quite readily deplete resources. This unchecked grazing can cause catastrophic repercussions within the community for not only the species but the environment as a whole. For example, a study conducted on the effects of unfettered grazing of primary resources along the U.S, Mexican border in the Sonoran Desert showed detrimental side effects in both plant diversity and ecological health. The Mexican side of the border, with its uninhibited grazing, showed not only a drastic decrease in vegetative diversity, but a resultant, drier soil, less conducive to growth. This decrease in soil moisture also caused the surrounding area to be more arid and increased the air temperature of the area around it. (Nevin et al. 1990). In this example we discover that if the primary resource is completely depleted the community may fail. Without the primary resource there is no movement of energy up the trophic levels of the community which will can lead to a loss of biodiversity. Therefore, to maintain the structure of the community, the density of primary grazers needs to be suppressed. Nature suppresses the density of primary grazers through various methods: predation, limited resources, stochastic events, and disease; for the purpose of our study we will focus on the last of these. Diseases is a very effective method for thinning a species' density or even a community's density depending on the potency of the disease itself. An example of this would be White-nose syndrome a disease that began devastating bat populations in the Northeastern United States from 2006-2007, causing an unprecedented 75% decrease in population size and general health (Blehert et al. 2009).

The primary grazer in California kelp forest ecosystems has gone unchecked for far too long and we are beginning to see the repercussions: "researchers at California Dept. of Fish and Wildlife have documented a 94 percent reduction in kelp abundance and found that urchins are grazing all available algae, reducing the ocean floor to bare rock" (Clark 2018). The primary grazer in the kelp forest ecosystem is *Strogylocentrotus purpuratus*, commonly known as the purple urchin. The primary focus of this paper is to isolate both environmental and sociological factors which help propagate the communicability of a specific disease that affects these urchins. Urchins when kept in relatively low abundance tend to be sedentary and consume mostly free-floating drift kelp, that has detached from its holdfast. When their natural suppressant, predation from sea stars specifically, *Pisaster giganteus* and *Pycnopodia helianthoides,* was

removed due to a sea-star wasting disease that caused a 75% decline for *Asterina miniata* and 56% decline in species density for *Pisaster giganteus*, it allowed the urchin population to explode (Eckert et al.1999). When the number of urchins in an area outgrows the amount of available resources, the normally sessile urchins begin to move, leaving their holes and crevices to become grazers, feasting on all types of kelp and algae in the area. This leads to vast amounts of urchins moving in a herd to clear out vast areas of kelp, leaving the area barren (Harrold and Reed 1985).

Kelp forests are naturally occurring sanctuaries for several species of fish, as resources are high and competition is low. The forests provide massive amounts of nutrients and protection, thus, removing said kelp completely shifts the balance of the community. In search of a mechanism that could reverse local kelp forest damage, we looked to a disease that the urchins have begun to contract, called *balding*.

The effects of this disease leave the urchin with lesions and missing spines. We don't know the long-term effects of the disease or the specific reasons for its transmission. We do know that the disease is bacterially specific: *Listonella anguillarum* and *Aeromonas salmonicida*. (Jangoux 1987) which means that it is transmitted not only to the urchins but possibly the environment around the urchins as well (Becker et al. 2008). As a result, we sought to glean more insight on the disease as a whole. More specifically, we investigated characteristics that may be associated with the disease spreading. To do so we tested three hypotheses: 1) that there will be more symptomatic individuals in areas of higher urchin density 2) that there is a difference in the size distribution of normal and symptomatic individuals and 3) that there are more symptomatic individuals at deeper depths. In short, we looked to see if the disease was more prevalent in areas of high density, among individuals of differing age or health, or at certain depths.

To test these hypotheses, we performed surveys in an urchin barren, off the coast of Hopkins Marine Lab in Monterey Bay, California. We hope that by illuminating possible causes and stimuli for the spreading of this disease, we might aid its propagation. If spreading the disease, is a viable method of cutting back urchin density as a whole then this reduction of urchins can allow kelp forests to regrow in currently barren areas.

METHODS

General Approach

We conducted an observational field study to determine whether there was evidence of urchin balding disease amongst purple urchins. Observations were conducted on SCUBA in November 2019 at Hopkins Marine Station. Almost all surveys were done in barrens with a few conducted in more forested areas. Transects ran onshore, offshore, and perpendicular to shore. Of note, for our purposes we will use the terms "diseased" and "symptomatic" synonymously.

System Description

Our study was conducted at the Hopkins Marine Station (36.6203° N, 121.9042° W), a Marine Protected Area, located in Pacific Grove, California. The site maintains a permanent transect cable through a temperate kelp forest ecosystem, running 170m in length from north to south. The Hopkins subtidal ecosystem possesses substrate characterized by high relief granite reef outcroppings intermixed with sand, cobble fragments, and bedrock. We conducted surveys primarily in the urchin barrens which are located around the point from the kelp forest (Figure 1). This is an area dominated by red turf algae and crustose species with no *Macrocystis pyrifera present*. The barrens are dominated by purple sea urchins. The study was conducted in November 2019, roughly between 9:00am and 1:00pm. The visibility ranged approximately between 10-25 feet with water temperatures ranging from 53 and 57 degrees Fahrenheit.

Figure 1: Dive site with kelp forest and barrens surveyed adjacent to Hopkins Marine Station.

Species Description

The kelp forest present at Hopkins is dominated primarily by giant kelp, *Macrocystis pyrifera*. Giant kelp is known to be able to grow up to 30m high and are found in cold, clear waters ranging from Alaska, all the way down to Baja California. These highly productive kelp plants support a diverse ecosystem of fish and invertebrate species, as well as our focal species, *Strongylocentrotus purpuratus*, the purple urchin. Purple sea urchins are found in shallow areas of water, usually along a coast with strong waves. They are mainly found along the coastline of Alaska all the way down the Pacific Coast to Mexico. They tend to live in kelp forests where they ingest their main source of food, kelp. These sea urchins are very important to their habitat because they help maintain the amount of algae in the ocean (Vickery and Fam 2008). As a primary grazer of kelp forests however, they can, if left unchecked, consume entire forests and in turn reduce a once productive, dense kelp ecosystem into an urchin barren. An urchin barren is an area of the shallow part of the ocean where the population growth of sea urchins has gone unrestrained, causing destructive grazing of kelp forests.

Study Design

Hypothesis 1: There will be more symptomatic individuals in areas of high urchin density

We hypothesized that, as the density of urchins increased, so would the probability of finding diseased urchins. In order to examine this, we collected two sets of data, observed and expected, and compared them to each other. For our observed data, we searched opportunistically for balding urchins within a thirty meter by four meter survey area, located in the barrens described above. When a diseased urchin was found, it was centered in a one meter by one meter quadrat and the surrounding urchins were counted as well to determine urchin density. For our expected data, we measured the density every two meters along the center-line of the survey area using the same-sized quadrat.

To test whether the number of diseased urchins found differed by density, we ran an analysis of variance (ANOVA). Once significant results were confirmed, we then used a Tukey Honest Significant Difference test to determine specifically where those differences lay. To run the Tukey test, four groups were defined, based on the number of urchins found: 0,1,2, or 3. These groups were then used in pairwise comparisons. In other words, each possible pair of means were analyzed for statistically significant differences.

Hypothesis 2: There is a difference in the size distribution of normal and symptomatic urchins

Our second hypothesis predicts a size difference between normal and symptomatic urchins. To test this hypothesis, thirty meter by four meter transects were laid out in the barrens and we conducted observed and expected surveys. For the observed, we opportunistically sized all symptomatic urchins found within the study area. For the expected, we randomly generated 15 points along a 30 meter transect line and measured the closest urchin at each point. ANOVA was used to detect whether there was a difference in the size distributions between normal and diseased urchins. Furthermore, we considered the f-statistic, as opposed to a t-test, so as to compare size distributions rather than just the size means.

Hypothesis 3: There are more symptomatic individuals at deeper depths

Our last hypothesis predicts the increased prevalence of symptomatic urchins at deeper depths. Data was collected by creating thirty by four meter survey areas in both the shallow and deep sides of the barrens and tallying the number of diseased urchins found in each area. A whole model test, which is analogous to an ANOVA report for a continuous response model, was then used to determine whether the model was a better fit than a constant response probability. Simply put, it tested whether the depth variable elicited different responses in terms of the number of symptomatic urchins found.

Results

Statistical tests revealed significant results in all three of our hypotheses. Namely, that there are indeed correlations between diseased symptomatic urchins and the variables: density, size, and depth. While the strengths of the correlations vary with each hypothesis, the predicted directionality of our hypotheses were confirmed, and the graphed results clearly illustrate this.

Hypothesis 1: *There will be more symptomatic individuals in areas of high urchin density*

An f-test showed there to be a statistical difference (f-value = .0030) in the distribution of diseased urchins based on density. The Tukey test then identified where the significant differences were found. As we see Table 1 and Figure 2, there were only two significant pairwise differences: between areas with 3 urchins when compared with areas having 0 or 1 diseased urchins. In other words, there was no significant difference in densities between areas with 0 or 1 or 2 diseased urchins nor between areas with 2 or 3. This signifies that the relationship between density and disease does not become significant until it reaches a threshold of 3 diseased urchins. While the relationship between the variables appears to be quite gradual, it still supports our hypothesis: that disease is more prevalent in areas with high density.

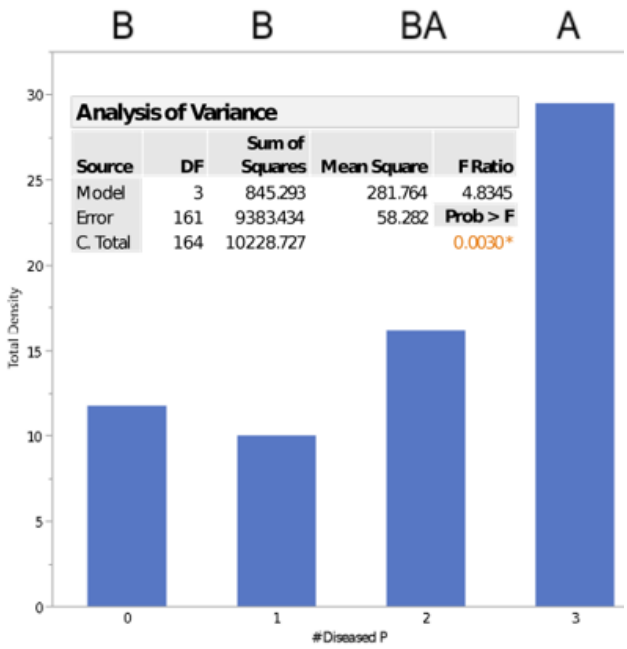


Figure 2: The positive relationship between the number of diseased urchins and density. Columns with different letters are statistically different from each other: Column 3 is statistically different from column 0 and column 1. No statistical differences between column 0 and 1 nor 2 and any other column.

Table 1: The honest significance difference (HSD) is evaluated for every possible pair of means. Only the level pairs 3,0 and 3,1 were statistically different from each other.

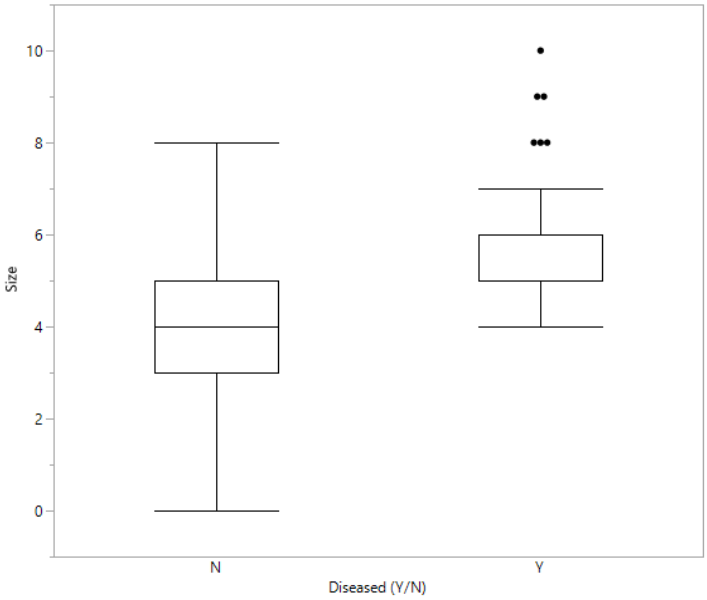
Hypothesis 2: There is a difference in the size distribution of normal and symptomatic urchins

An f-test showed there to be a statistical difference (f-value < .0001) in the size distributions of normal and diseased urchin groups. Each group's size range was very different in scale

Analysis of Variance

Source	DF	Sum of Squares	Mean Square	F Ratio	Prob > F
Diseased (Y/N)	1	88.24596	88.2460	37.3562	<.0001*
Error	89	210.24306	2.3623		
C. Total	90	298.48901			

(see Figure 2). While normal urchins were found in a wide range of sizes,



only larger urchins were found to be symptomatic. In fact, there were no diseased urchins found under 4cm, though there were several large outliers. Furthermore, the interquartile ranges of each distribution had no overlap with each other. Based on the above, we can confidently say the symptoms of the disease are more likely to be found on larger individuals.

***Figure 3*:** Distribution of sizes for normal and symptomatic urchins. Normal urchins have an interquartile range of 3 to 5cm while symptomatic urchins have an interquartile range from 5cm to 6cm. Overall, the size range was much smaller for diseased urchins with no symptomatic urchins found under 4cm.

Hypothesis 3: There are more symptomatic individuals at deeper depths

A whole model test shows that there is a significant positive relationship between diseased urchins and depth (p-value = 0.0006). We see in Figure 3 that the likelihood of finding a disease urchin rises from 9% at a depth of 20ft to 33% at 35ft. Therefore, our hypothesis that there are more symptomatic urchins at deeper depths is well supported.

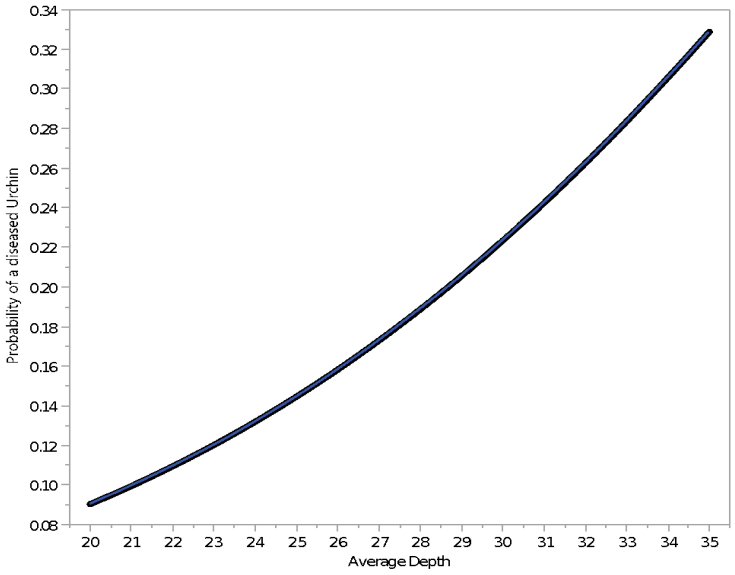


Figure 4: The probability of finding at least one diseased urchin by depth. We see a positive monotonic relationship between the two variables, with probability increasing by 24% when comparing depths of 20 to 35ft.

Whole Model Test

Model	-LogLikelihood	DF	ChiSquare	Prob>ChiSq
Difference	5.85759	1	11.71518	0.0006*
Full	108.48093			
Reduced	114.33852			

RSquare (U)	0.0512
AICc	221.012
BIC	227.923
Observations (or Sum Wgts)	240

Discussion

Each of our three hypotheses were supported, as evidenced by the statistical tests conducted on the collected data. As the density of urchins increased, we found there to be more diseased individuals. We believe that this may be a result of the mode of transmission for the bacteria that is linked to urchin balding disease. In an experiment testing the transmission of the disease, it was found to be spread by both direct contact and by being in the vicinity of a diseased individual (Maes and Jangoux 1984). Thus, if a diseased urchin is in a region of high urchin density, it is more likely to have direct contact with another individual and to spread the disease which is consistent with our results.

Data supports our hypothesis that diseased individuals are often larger than non-diseased individuals. We found that diseased urchins have a narrow size range consisting of larger individuals than those that are not diseased. Diseased urchins were observed in the range of five to seven centimeters, with outliers reaching up to ten centimeters. Normal urchins had a much wider size range from less than one to a maximum of eight centimeters. We believe that diseased urchins may be larger in size because they are older. One study that examined the types of bacteria linked to balding disease concludes that the bacteria are opportunistic (Becker et al. 2008). Bacteria that are opportunistic are innocuous but will become pathogenic once the individual (urchins, in this case) develops a weakened immune system (Porta 2014). Since immunity is known to decline with age, we conclude that large, old sea urchins are more likely to become diseased.

We found that there were more diseased urchins at our deep transects compared to our shallow ones. We believe this pattern exists as a result of storm and surge intensity; this type of activity is more prevalent in shallow areas and can damage individuals, decreasing the urchin population. At depth, the water is generally calmer and can allow urchins to survive for a longer period of time. This conclusion is further supported by our results regarding the size and assumed age of diseased individuals as discussed above. Additionally, one study suggests that the bacteria related to this disease could be driven by temperature change (Greech et al. 2019). Because temperature changes with depth, our findings may also suggest the bacteria is triggered by temperature.

All the data collected for this project was done in a marine protected area and may not be consistent with results found elsewhere. Therefore, we think it's very important for this study to be replicated in a non-marine protected area in order to understand if having a protected status influences the patterns observed. This study focused only on *S. purpuratus* because it was the most abundant at our location and was the only species observed to be symptomatic. The disease, however, is seen across other species of urchins and we do not know if our results would be consistent with other urchin species. Another aspect that was disregarded in this project was gonad index of urchins. An urchin that has regular access to food and is generally healthy will have more gonads (a higher gonad index) than an urchin who is starved (Sonnenholzer et al. 2011). Since we surveyed urchins in a barren where there is little to no kelp the urchins, we surveyed probably had a low gonad index. We wonder if a low gonad index is indirectly associated with balding disease.

Considering the recent loss of California kelp forests from overgrazing of sea urchins, we believe that our results can provide some insight on the recovery of kelp forests. Because we know that disease is found at greater depths, it may be these areas could be the first to transform out of an urchin barren and have an increased chance of becoming a kelp forest again. Since there is more disease in areas of high urchin density, denser areas of urchin populations may experience a large crash due to disease outbreaks. Additional field surveys and controlled

experiments are necessary to get a deeper understanding of how this disease affects kelp forest ecosystems.

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