Infection Potential of Sudden Oak Death Pathogen *Phytophthora ramorum* in Urban Spaces of San Mateo and Santa Clara counties

Emilie R. Maddison

ABSTRACT

Sudden Oak Death is a tree disease deadly to many oaks (*Quercus spp.*) and tanoaks (*Notholithocarpus densiflorus*) in coastal California and Oregon. Mapping its spread has been a key part of managing the disease as it infects new populations of trees, but the management focus is on forest environments, where the disease thrives. Urban environments, though at lower risk, still see infections of SOD, but little is understood about how the urban environment affects its spread. Using orthologous parameters to those found in the forest-based literature, I modeled SOD presence in urban environments, using GIS mapping techniques. I assessed (1) proximity to wildlands, (2) proximity to known infection, (3) percent tree coverage, and (4) urban street density for their predictive abilities and then used suitability analysis to predict urban spaces susceptible to the disease. All known disease in my study site occurred within 4.1 km of a wildland, and 70% fell within 1 km. Areas within 200m of a known infection are at very high risk of infection, and within 1000m there is elevated risk. (3) Areas with more than 30% tree coverage are five times more likely to support infection. (4) Urban street density below 33 intersections/km^2 are twice as likely to support infection.

KEYWORDS

Tree pathology, epidemiology, microbiology, GIS mapping, suitability analysis

INTRODUCTION

Sudden Oak Death, or SOD, is a tree disease caused by the fungus-like brown algae *Phytophthora ramorum*, and has the potential to kill huge numbers of native trees across the US (Venette and Cohen 2006). Since its arrival to the US in 1995, Sudden Oak Death has killed hundreds of thousands of trees in central California and Oregon, especially various oaks, *Quercus spp.*, and tanoaks, *Notholithocarpus densiflorus*, which represent key parts of the Californian ecosystem (Davidson et al. 2003, Rizzo and Garbelotto 2003). Symptoms of SOD in oak trees include oozing black sap on the tree's trunk, stem cankers, loss of foliage, and death (Davidson et al. 2003). The name, Sudden Oak Death, comes from the rapid death of an infected oak—sometimes in as little as a matter of weeks—with few or no warning signs. However, these trees are usually infected with the disease for months or years before becoming symptomatic and dying (Ockels et al. 2004). As well as killing oaks and tanoaks, Sudden Oak Death harmlessly infects dozens of other "secondary hosts", most notably California bay laurels, *Umbellularia californica* (Rizzo and Garbelotto 2003).

U. californica often grow alongside oak trees, and are major infective vectors for Sudden Oak Death in oak trees (Alexander and Lee 2010)—the disease can pass between *U. californica*, and then from *U. californica* to oak trees, where its path terminates with the death of the oak (Garbelotto et al. 2003). Transmission of the pathogen can occur through direct tree-to-tree contact, soil, or movement of water (Davidson 2002). Oaks do not transmit the disease between each other (Garbelotto et al. 2003). Studies of the movement of the disease must therefore track movements among secondary hosts—mostly *U. californica*, but also any other species which can transmit the disease, including *N. densiflorus*, toyon (*Heteromeles arbutifolia*), and nursery stock such as camellia and azalea. These species serve as the major "reservoir" of *P. ramorum* spores (DiLeo et al. 2009), allowing the disease to persist. Many of these "secondary hosts" exhibit distinctive color disfiguration patterns on their leaves, making it possible to visually identify the disease in the field; further laboratory testing can then confirm the infection is present. (Anacker et al. 2007).

Many groups—university labs and forest management groups primarily—collect data on the presence of *P. ramorum* throughout coastal California. The Garbelotto Lab at UC Berkeley undertakes one of the largest of these projects. Since 2008, it has been working on the "SOD Blitz",

a large, citizen-science based mapping project which aims to track the presence of Sudden Oak Death throughout California, where the largest outbreak of the disease in the US persists. Leaf samples are identified and collected by hundreds of volunteers, then lab-tested for infection with *P. ramorum*. Each year, this collection results in about 1,000-2,000 georeferenced samples, and the lab publishes a map visualizing their distribution. The SOD Blitz provides practical information to forest managers, landowners and the public about Sudden Oak Death.

Using this georeferenced data, maps predicting Sudden Oak Death behavior are produced, analyzing the effects of various climate and spatial distributions on an area's potential for infection (Fowler and Magarey 2005). Most studies focus on wildlands and forests, where the bulk of the infections occur. However, a small but significant minority of infections occur in urban spaces. Using the data from the SOD Blitz projects, my research aims to evaluate the risk of Sudden Oak Death in urban spaces, looking at landscape characteristics within to the urban environment. Habitat fragmentation and distance from forest canopies are well-described factors predicting Sudden Oak Death presence in wildlands areas (Condeso and Meentemeyer 2007). However, no research currently exists on the disease's behavior in urban spaces, research that could be valuable to homeowners and land managers monitoring their properties for the disease (Lee 2011). I describe four parameters which I hypothesize should define the infection potential of urban spaces to SOD: proximity to wildlands (positive correlation), proximity to a known infection (positive correlation), percent tree coverage (positive correlation), and urban street density (negative correlation). After assessing each parameter for its relationship to known infections using ArcGIS and statistical analyses, I use suitability analysis to produce a model capable of assessing the infection potential of urban spaces.

METHODS

This study categorized the infection potential of urban spaces to the Sudden Oak Death pathogen *P. ramorum.* The pathogen infects many *Quercus spp.* and a number of "secondary hosts", primarily *U. californica,* as well as *N. densiflorus*, *H. arbutifolia,* and a number of nursery stock, such as Azalea and Rhododendron. *P. ramorum* needs moist environments to survive, and most infections are a result of localized (<250m) transmission (Meentemeyer 2011). *P. ramorum* lives well in continuous canopies, such as wildland forests, and less well in non-continuous areas

(Ellis 2010), such as open fields or urban spaces. Despite this, some incidences of Sudden Oak Death do occur in urban spaces, in non-ideal conditions. By examining variance in urban environments in comparison to known cases of Sudden Oak Death, I determined the characteristics of urban spaces capable of supporting the disease. I hypothesized that such characteristics would include: proximity to wildlands, proximity to known infections, urban street density, and percent tree coverage. It is also possible that cases of *P. ramorum* within urban spaces may be as a result of the anthropological introduction of infected nursery stock (especially Rhododendron and Azalea) into those areas.

Study System

My study system consisted of the Sudden Oak Death "secondary hosts" sampled in the annual "SOD Blitz" from 2008 to 2013. The majority of trees sampled were *U. californica* or *N. densiflorus*. The sampling range of the SOD Blitz encompassed an area from Fort Bragg to San Luis Obispo, between 40.33506748^{O} to 35.20213^{O} N and -123.9900934^{O} to -120.4316^{O} W.

For the purposes of my study, however, I limited my analysis to an area contained by the counties of San Mateo and Santa Clara (Figure 1). I selected this area because of the extensive number of tree samples (1927), and the presence of Sudden Oak Death in the urban area, which together are unique to the dataset. This region represents the majority of urban samples in the SOD Blitz dataset. Furthermore, the relative climate homology within this narrow region allowed me to omit climate models from my study. I defined "Urban spaces" using the Census bureau's definition of "urbanized areas" from 2000 (UA Census 2000), and refer to areas outside of this range as "Wildlands". I limited my study to the 1078 trees within these urban spaces, from the 1927 tree samples within San Mateo and Santa Clara counties.

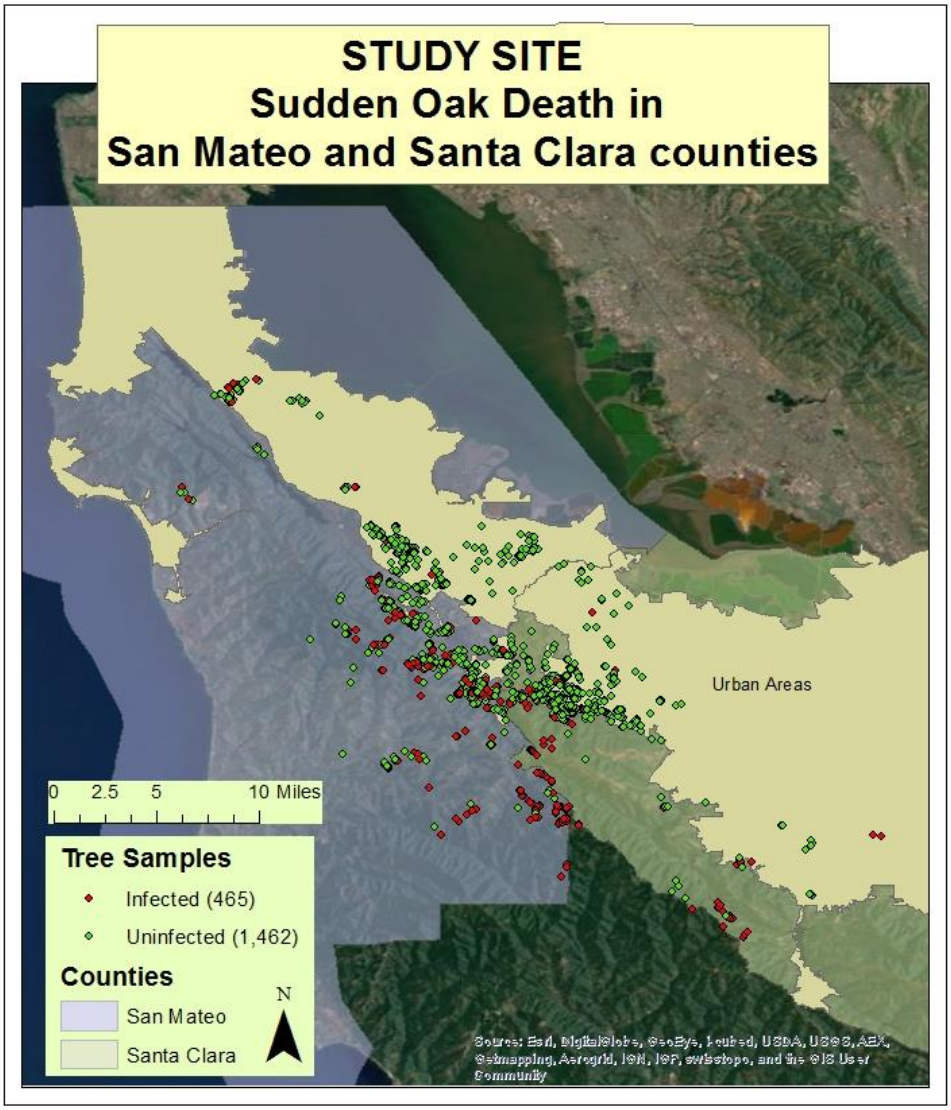


Figure 1. Study Site—Sudden Oak Death in San Mateo and Santa Clara counties, in the South Bay in California, USA. This map includes both urban and wildland samples. Red points represent trees infected with *P. ramorum*, and green points represented uninfected tree samples.

Data Collection

SOD Blitz data were collected from 2008 to 2013, annually, between April and June of each year. "Citizen scientists", or the volunteer public, were provided with training on identifying disease symptoms on the foliage of *U. californica* and *N. densiflorus*, as well as materials for sample collection (Figure 2). Each sample consisted of 6-10 leaves from a single tree which showed disease symptoms. Trees which were sampled were then marked with blue flagging tape.

Along with each leaf sample, citizen scientists were asked to provide information on tree species, location of the tree (preferably GPS coordinates), and whether the sample was taken from public or private land. The majority of samples came from *U. californica* trees, while a minority of samples came *N. densiflorus*, *H. arbutifolia*, and nursery stock.

Sample Processing

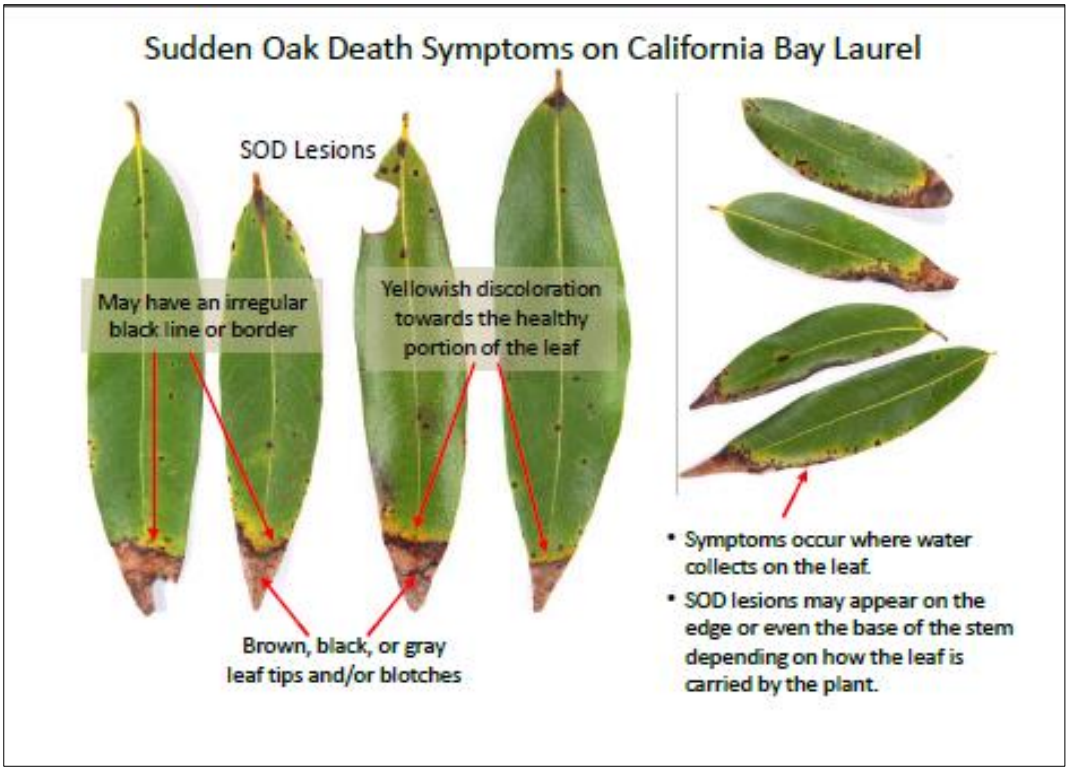
We processed samples from May to September in the laboratory. In 2013, the methods were as follows: We added 6 hole-punch sized samples of the leaf tissue to 1.5ml Eppendorf tubes with a glass bead and ground to a powder. We then extracted the DNA of this material using the ROSE (Rapid One Step Extraction) Extraction method (Osmundson et al. 2013). We then used an Enzyme Linked Immunoabsorbant Assay (ELISA) to test all samples for presence of the genus *Phytophthora* (Vettraino et al. 2010), and further tested samples which returned positive for *Phytophthora* for *P. ramorum* using a single round of PCR. Earlier years followed a similar procedure, and in all sampling years, testing was rigorous.

We then correlated this infected/uninfected data with the GPS points of the original samples. Where citizen scientists could not provide GPS locations, they instead provided addresses or written descriptions of the sample's location, and we estimated GPS location from that information in Google Earth. A minority of samples included incorrect GPS coordinates (e.g. "trees" in the middle of the Pacific Ocean), and these samples were excluded from my analysis.

Map Creation

Using the software ArcGIS 10.2, I mapped the infected/uninfected data from 2008-2013 over a map of California, using the NAD 1983 geographic coordinate system (Figure 1). Since I was interested in incident locations of Sudden Oak Death and not their relationships with each other or their spread over time, I did not differentiate between samples from the 6 collection years (2008-2013) during my analyses. I acquired and overlaid layers mapping urban spaces (UA Census 2000), and county lines (2013 TIGER), and limited my dataset to the 1078 tree samples which were within urban spaces in San Mateo or Santa Clara counties (Figure 3).

(a)



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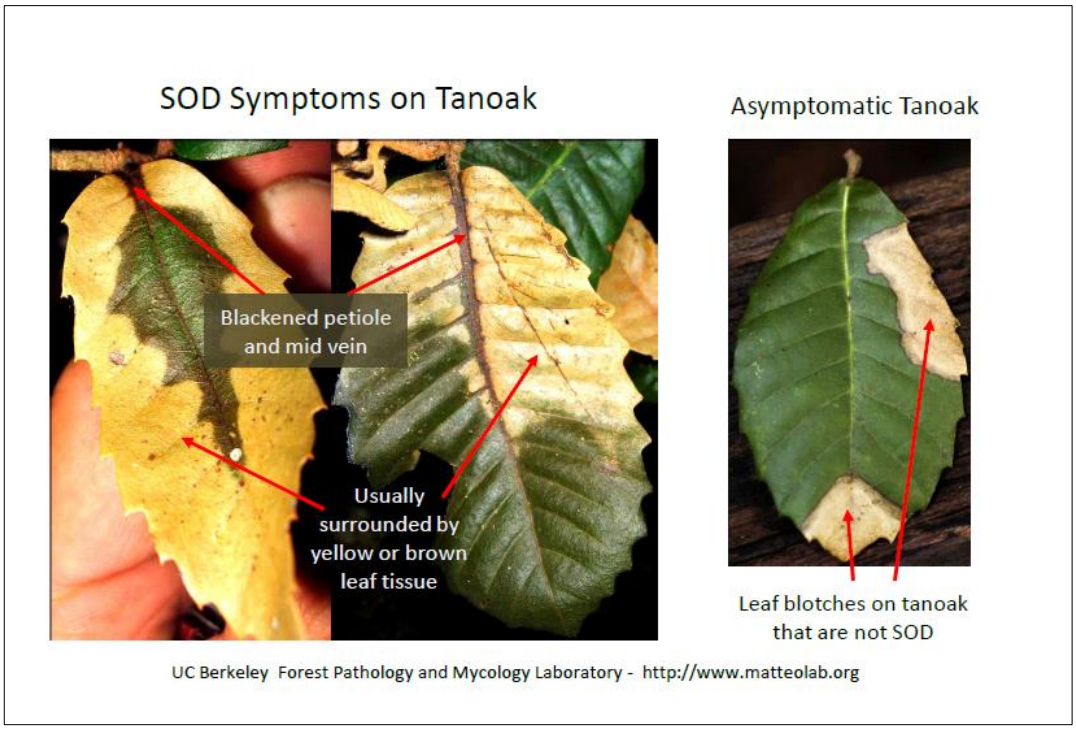


Figure 2: Symptoms of *Phytophthora ramorum* on common "secondary hosts". Identification cards provided to citizen scientists on identifying symptoms on (a) California bay laurels, *Umbellularia californica*, and (b) Tanoak, *Notholithocarpus densiflorus*.

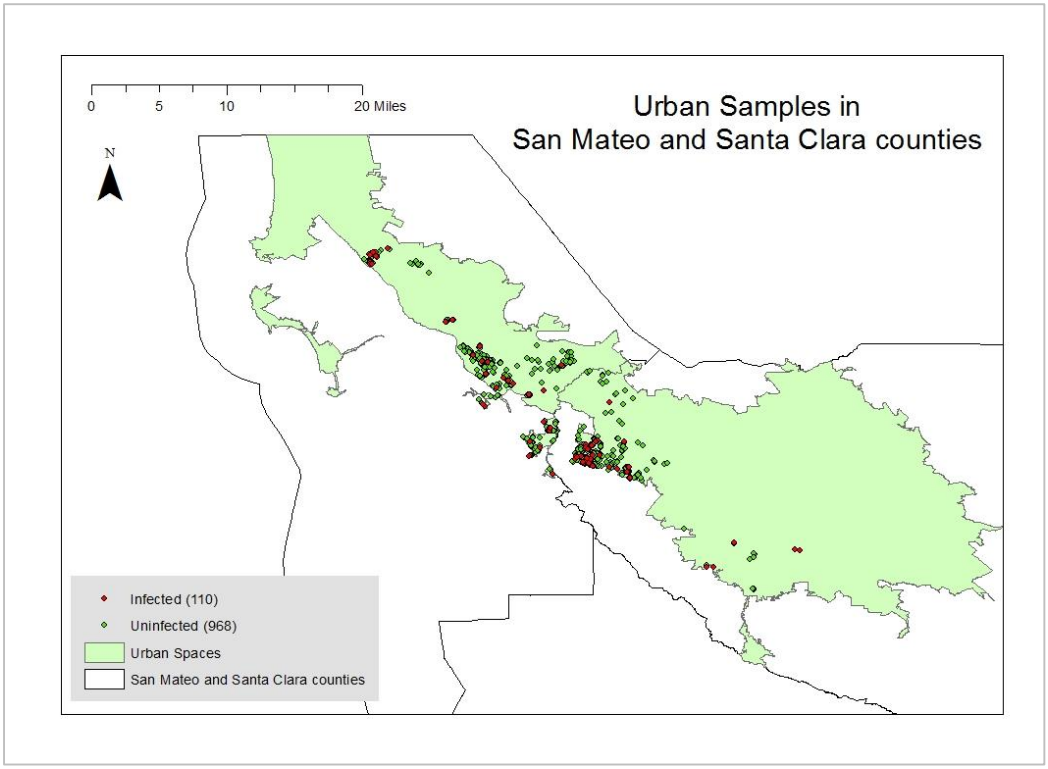


Figure 3. Urban samples in San Mateo and Santa Clara counties. 1078 samples in total. Green areas demark urban spaces, and the red/green points indicate infected/uninfected samples within those urban spaces.

Analysis

I examined a number of variables (Table 1) for their relationship with infection status to determine characteristics of urban spaces capable of supporting Sudden Oak Death: (1) Proximity to Wildlands, (2) Proximity to Known Infection, (3) Percent Tree Coverage, and (4) Street Density.

Table 1. Independent and dependent variables defining Infection Potential. Expected correlation and the metrics I used to measure the relationship.

Independent Variable	Dependent Variable	Correlation	Metrics
Proximity to Wildlands	Infected vs. uninfected	Positive	1km intervals from wildlands
Proximity to Known Infection	Infection presence	Positive	<200m very high risk, <1000m high risk
Percent Tree Coverage	Infected vs. Uninfected vs. Random	Positive	0-100% Tree coverage, determined by study
Street Density	Infection presence	Negative	Areas below and above mean density

Urban vs. Wildland Infections

Urban spaces are at lower risk of infection than wildlands are. I examined this within my dataset by graphing the number of infected and uninfected trees by land type in Excel, and then used chi-squared distributions to determine the significance of the differences.

Proximity to Wildlands

Since wildlands are more likely to be infected than urban spaces, proximity to wildlands should be a good indicator of infection potential. I defined the polyline "Wildland boundary" as the western edge of the "Urban spaces" in San Mateo and Santa Clara counties. I defined tree samples as "Infected" or "Uninfected" in separate map layers and then projected them in ArcMap using the NAD 1983 California (Teale) Albers Projected coordinate system in order to calculate distances in meters. I then used the "Near" tool to calculate the distance between the trees and the nearest point on the "Wildland boundary" polyline. I tested for significance in Excel using a heteroscedastic, one-tailed t-test. I accepted the hypothesis that infected trees were located more closely to the "Wildland boundary" than uninfected trees if the t-test returned $t<0.05$. I then graphed the data with a histogram and boxplot using R to visualize the distribution of the data.

For suitability analysis, I used 1km intervals from the wildland boundary, using the percent of samples which fall within each interval to reclassify values on a 0-10 scale. I generated a

Multiple Ring Buffer to the east of the wildland boundary, which I then converted to a raster with a 30x30m cell size for "Weighted sum" analysis.

Proximity to Known Infection

Proximity to known infection is a well-defined parameter of infection potential—the closer a given tree is to an infected tree, the more likely it is to be infected. Since *P. ramorum* is dispersed by wind and water, and typically needs moist environments to survive, it is extremely unlikely that the disease will spread beyond 1km of a known infection. In the context of this study, "very high risk" was defined as within 200m of a known infection, and "high risk" was defined within 1000m of a known infection. Beyond 1000m (1km), areas were treated as "not at risk".

Using this information, I again created multiple ring buffers in ArcMap around the 110 known infections within the urban spaces of San Mateo and Santa Clara counties. Since presence of a single infection increases the likelihood of multiple infections, I did not value overlapping buffers more highly. This means that if two infections were immediately next to each other, the 1000m radius surrounding those infections were weighted the same as a single infection.

These multiple ring buffers were then clipped to the extent of the urban area, and converted to rasters, using a 30x30m cell size. For the suitability analysis, I reclassified distances on a 0-20 scale, with 10 equaling "high risk" and 20 equaling "very high risk".

Percent Tree Coverage

Since the location of trees capable of hosting *P. ramorum* would necessarily increase the risk of finding the disease in a given location, I analyzed tree coverage for its relationship to known infections. Good data on urban tree species distributions do not exist, so I instead relied on data for "woody vegetation" (GLCF 2013). This data came from the Global Land Cover Facility, and is a product based on Landsat data of the US. This particular dataset encompassed the south bay, and estimates the "percentage of horizontal ground in each 30-m pixel covered by woody vegetation greater than 5 meters in height". The 30-m resolution was fine enough to approximately evaluate individual trees, and the 5-m height of vegetation meant that most *U. californica* and *N. densiflorus* were represented, with mature trees of both species exceeding 5m in height, while

excluding grasses and shrubs which are less likely to carry the disease (Fryer 2008, Howard 1992). Since most *Quercus spp.* also exceed 5m in height, this data can also show areas likely to have *Quercus spp.* susceptible to Sudden Oak Death.

I generated 110 random points within the urban spaces in ArcMap, and then extracted the percent tree coverage value at (1) random, (2) uninfected, and (3) infected points. In Excel, I analyzed the relationship between "point type" and percent tree coverage, using ANOVA and t-tests to determine the significance of variation in averages, accepting significant differences in the mean percent tree coverage with t-values under 0.05. I then graphed the three point-types in a histogram and broke the data into 4 "percent classes"—(1) unlikely to support infection, (2) average likelihood to support infection, (3) medium likelihood to support infection, and (4) high likelihood to support infection. The reclassified values for suitability analysis were based on the elevated risk of infection within these "percent classes", on a 0-10 scale.

Street Density

Street Density is a proxy for habitat fragmentation. Sudden Oak Death is typically found in areas with high moisture and dense foliage, and does not survive well in areas which are exposed or have breaks in vegetation. Higher street densities, a characteristic of urban fragmentation, should therefore be negatively correlated with infection location—Infected trees should be found in lower-density areas than both uninfected trees and random sites within the urban spaces.

Using Street Centerline data from the San Mateo and Santa Clara county GIS offices, I created a Geometric Network in ArcMap of each street intersection and endpoint to quantify "street density" in terms of "intersections per sq. km.". I produced a Thiessen polygon map of this network, allowing me to calculate the intersections per sq. km. over the region. I then transformed the vector data into a raster layer with 30-30 cell size.

I joined the 110 random points, the 968 uninfected points, and the 110 infected points to this dataset to evaluate street density at each point. In Excel, I then used ANOVA and t-tests to examine the relationships between point-type and street density. I accepted differences in the mean as significant with a t-value<0.05.

For the suitability analysis, I examined patterns in the data and assigned Low and High risk of Sudden Oak Death infection. I reclassified data values on a 0-10 scale based on the increased likelihood of infection, using the mean street density as a benchmark.

Suitability Analysis

For the final Suitability Analysis, I used the "Weighted Sum" tool within ArcMap. My four parameters, (1) proximity to wildlands, (2) proximity to infection, (3) percent tree coverage, and (4) street density were each included, using reclassified values for each layer. This produced a final map of the urban spaces within San Mateo and Santa Clara counties based on their risk for Sudden Oak Death infection. The final map had a 30x30m cell size, and was projected into both the NAD 1983 geographic coordinate system and NAD 1983 California (Teale) Albers projected coordinate system so that future evaluations could be made using both decimal degrees and meters.

RESULTS

Using georeferenced data on the presence/absence of Sudden Oak Death in the Bay area between 2008 and 2013, I assessed urban spaces for their infection potential by *P. ramorum*. This involved taking into account a number of variables which are correlated with *P. ramorum* infection: Proximity to a wildland boundary, proximity to a known infection, percent tree coverage, and street density.

Urban vs. Wildland Infections

Urban spaces are clearly at a lower risk for infection than wildlands (Figure 4), with urban samples infected 10.2% of the time and wildland samples infected 41.8% of the time. The chi-squared test I ran on actual vs. expected values of infection rates returned $X^2=2.55*10^{-58}$, strongly confirming the results that urban spaces have significantly lower numbers of infected trees. This is in line with current literature. The rest of this study examines the areas where urban infections do occur to define urban spaces at risk of infection.

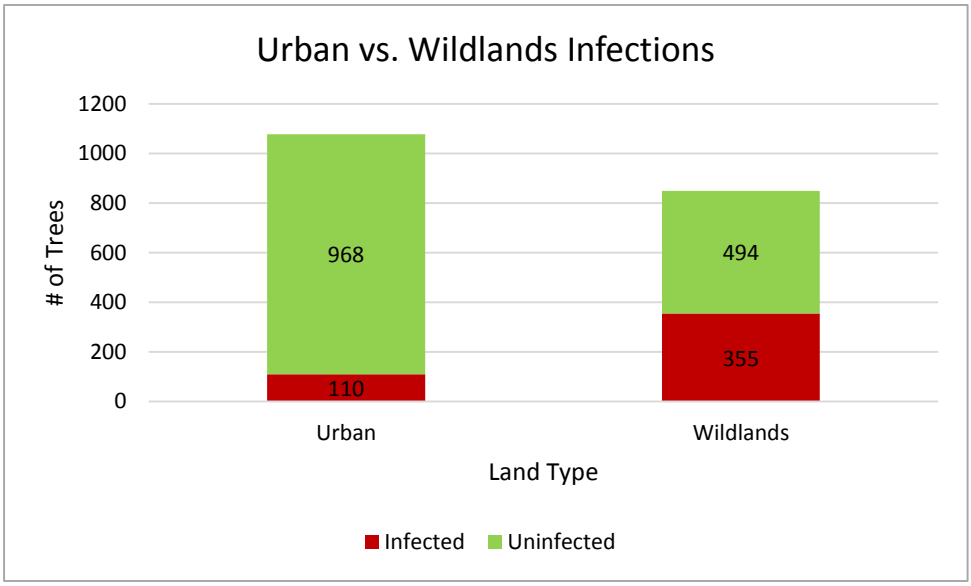


Figure 4. Urban vs. Wildlands infections, counts of tree samples. Infection rates are much lower in urban spaces, but urban infections are still present.

Proximity to Wildlands

I found infected trees were closer to the "Wildland boundary" than uninfected trees, proving my hypothesis. The mean distance of 110 infected trees from the "Wildland boundary" was 808 meters, while the mean distance of 968 uninfected trees from the "Wildland boundary" was 1213 meters, for a difference of 405 meters. I confirmed using a heteroscedastic, one-tailed t-test that these results were significant, with $t=1.74*10^{-5}$. No Infected trees were found outside of approximately 4.1 kilometers (4103 meters) from wildlands, while uninfected trees extended to approximately 5.7 kilometers (5730 meters) from wildlands. The boxplot (Figure 5a) demonstrates that both infected and uninfected trees were heavily weighted towards the "Wildlands boundary", which was expected because Sudden Oak Death is an emerging infectious disease, and has not infected all of the areas it is capable of infecting.

(a) (b)

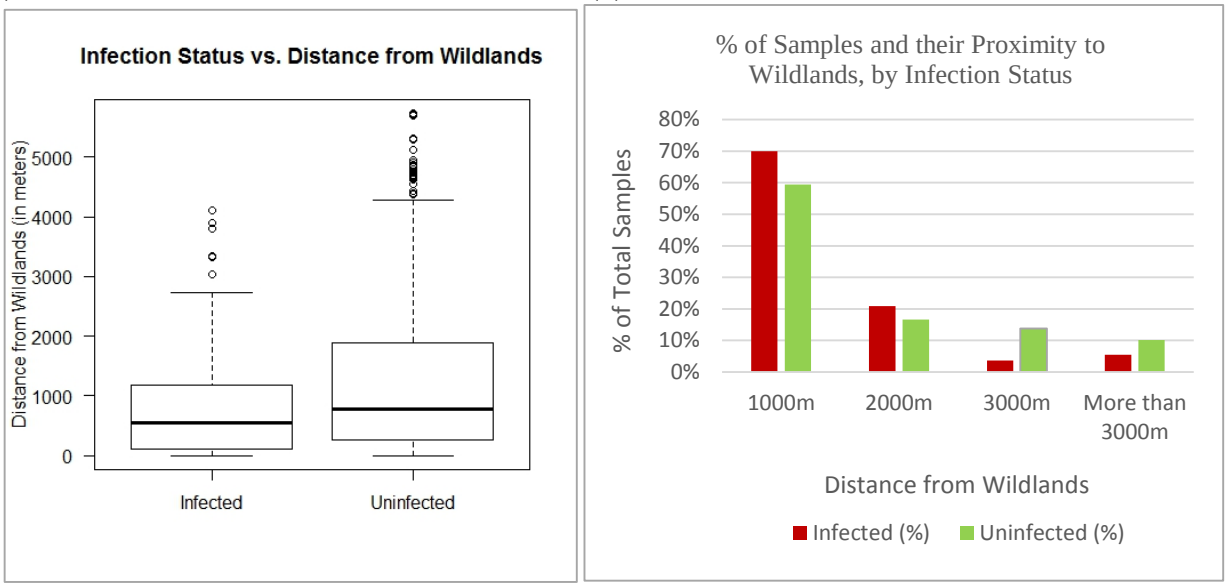


Figure 5. Infection status vs. Distance from Wildlands. (a) Infected trees are found closer to wildlands boundaries than uninfected trees, but there is an overlap between the two groups. (b) You are more likely to find infected trees, as compared to uninfected trees, between 0-1000 and 1000-2000m from a wildland.

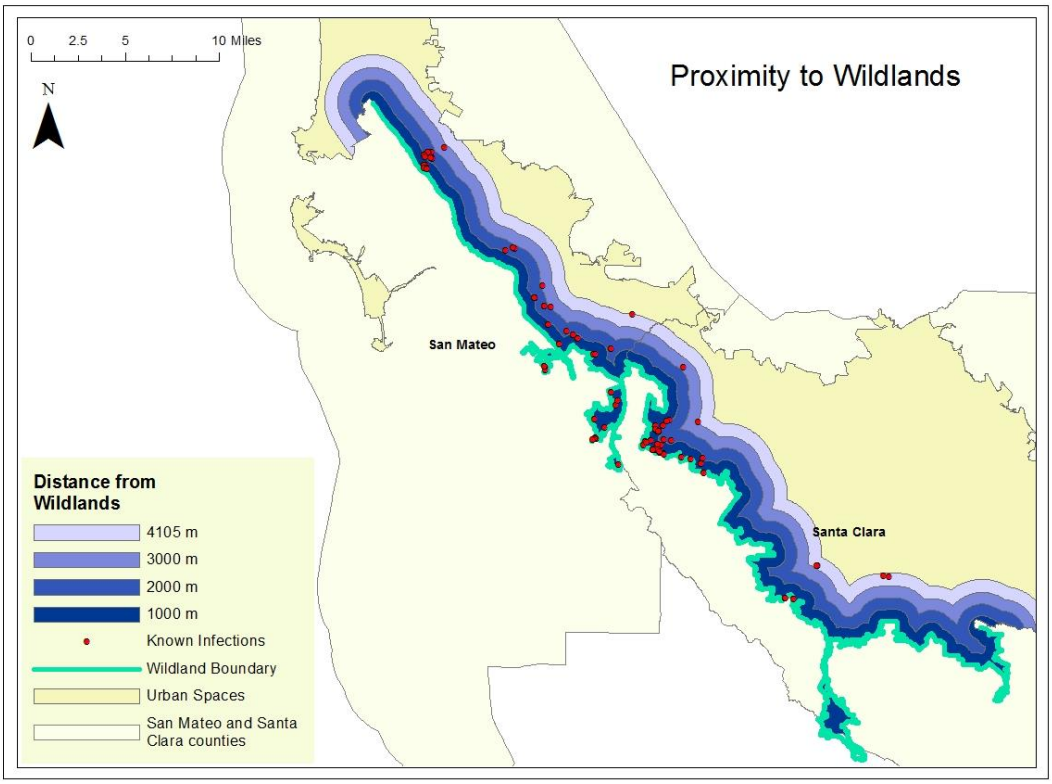
70% of urban infections fall within 1000 m of the wildland boundary. An additional 20% are between 1000 and 2000 m of the wildland boundary, and the last 10% are found between 2 and 4.1 km of the wildland boundary (Figure 5b, Table 2). Trees sampled closer to wildlands are more likely to be infected that trees sampled further away from wildlands.

Table 2. Metrics of Proximity to Wildlands

Distance from Wildland boundary	**0-1km**	**1-2km**	**2-4.1km**
Percent of total infection in each range	70%	20%	10%
Suitability Values	10	3	1

I then created a Multiple Ring Buffer in ArcMap based on this data (Figure 6a), which I then converted to a raster for the final suitability analysis. The suitability values used to reclassify intervals (Table 2) were on a 0-10 scale, with the 0-1km range assigned a 10, and other values generated as a fraction of that value.

(a)



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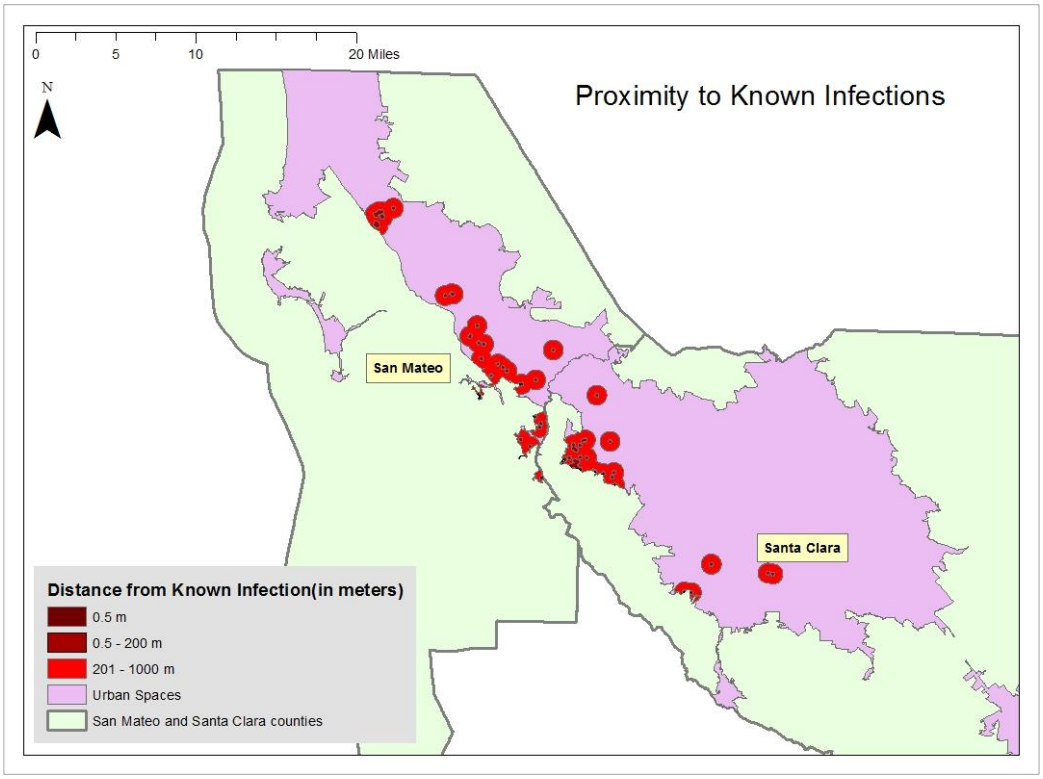


Figure 6. Proximity variables and Sudden Oak Death infection. Proximity to wildlands (a) strongly predicts Sudden Oak Death presence, up to 4.1km from a wildland boundary. Proximity to known infections (b) can predict infection up to 1 km from a wildland boundary.

Proximity to Known Infection

I created a multiple ring buffer around known urban infections, producing a map of susceptible areas (Figure 6b), with <200m representing "very high risk" and <1000m representing "high risk". This was clipped to the boundary of the urban spaces.
Since proximity to a known infection is the strongest indicator of disease presence, I weighted the 200m-radius zone as "20" and the 200-1000m zone as "10" for reclassification purposes.

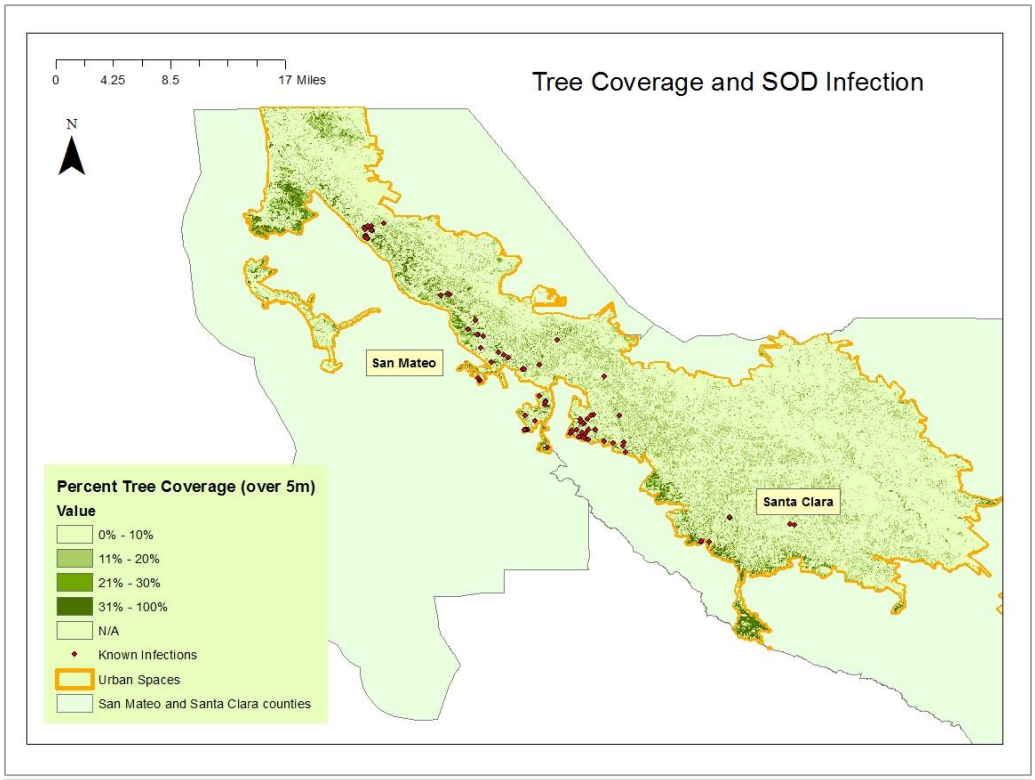
Table 3. Proximity to known infection, by distance. Suitability assessed based on proximity.

Distance from Infection	**<200m**	**<1000m**
Infection risk	"Very high"	"high"
Suitability Value	20	10

Percent Tree Coverage

From data from the map I created of percent tree coverage (Figure 7a), I found infected trees are more likely to be in areas of high tree coverage than uninfected trees or random points. The mean percent tree coverage at infected points was 25.2%, while it was 19.9% at uninfected areas. Compared to uninfected points, infected points had 27% more tree coverage. Random points had a mean tree coverage of just 6.5%, meaning infected trees were almost were in areas almost 4 times more tree-dense than random points in Santa Clara and San Mateo counties (Figure 8a). The p-value of the ANOVA comparing these groups was 0. A single-tailed, heteroscedastic t-test between infected and uninfected points yielded a p-value of 0.001, and so I accepted the hypothesis that infected trees were in more tree-dense areas than uninfected trees. The same test between infected and random points yielded a p-value=$1.4*10^{-18}$, which allowed me to accept that all three point-types were distinct from each other.

(a)



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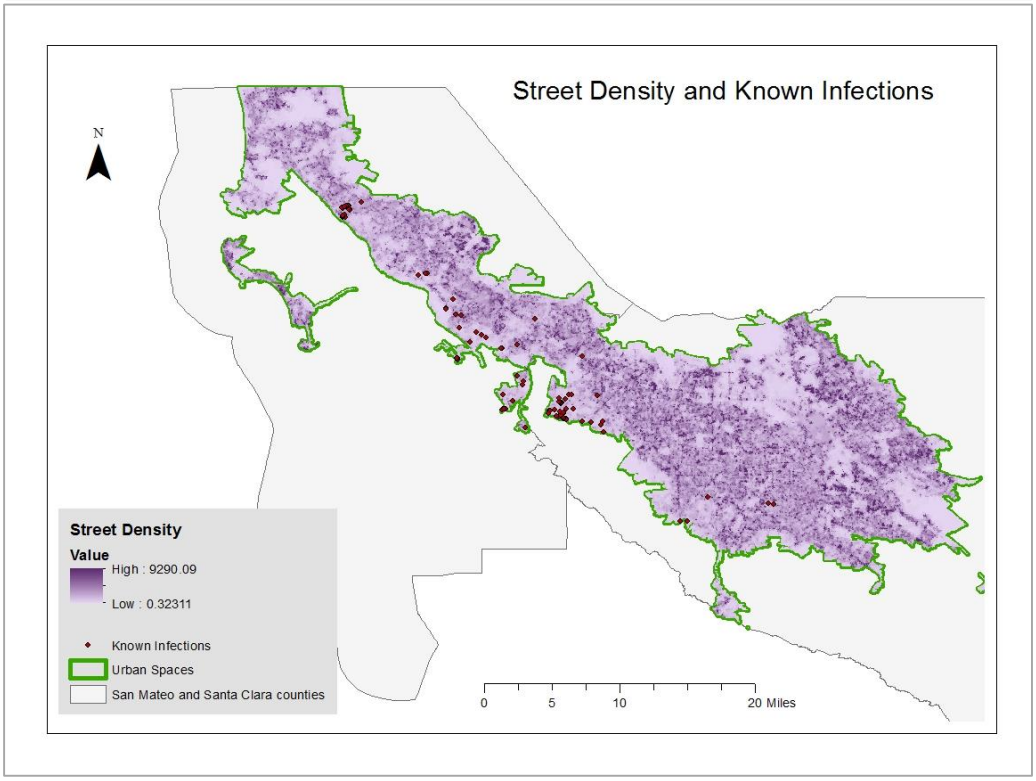

Figure 7. Density variables and Sudden Oak Death infection. (a) represents percent tree coverage, and (b) represents street density.

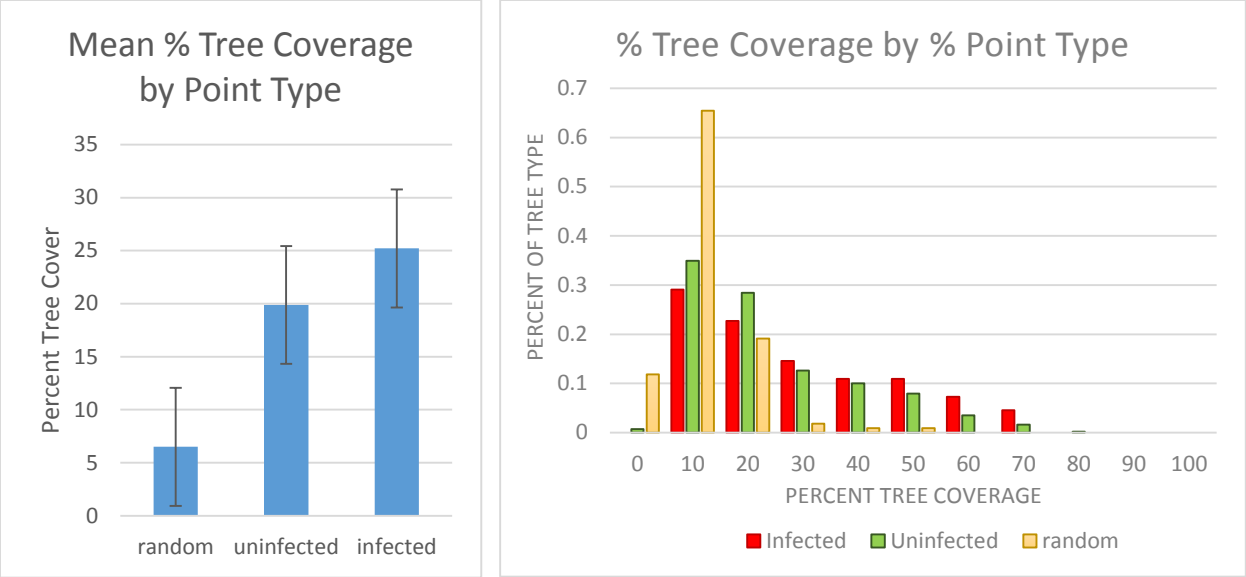

Figure 8. Percent Tree Coverage by Point Type. (a) represents the mean percent tree coverage by point-type, while (b) represents the percent of each point-type at each coverage level, in 10% bins.

In general, all sampled trees were most likely to be in areas exceeding 30% tree coverage, as compared to random points. I evaluated likelihood in 10% bins. ~50% of infected trees occurred in areas exceeding 30% tree coverage, and so this was deemed "high risk" tree coverage. I defined the risk in the rest of the bins linearly from Percent Infected/Percent random in each bin (Table 4). I then used these values to reclassify the percent tree coverage raster for my final suitability analysis.

Table 4. Percent Tree Coverage values for Suitability Analysis, with risk defined by the likelihood of finding an infected tree versus a random point at that tree coverage.

Percent Tree Coverage	**0-10%**	**10-20%**	**20-30%**	**30%+**
% Infected/ % random	~0	~1	~2	~10
Infection Risk	Very low	low	average	high
Suitability Value	0	1	2	10

Street Density

Based on the map I created in ArcMap (Figure 7b), street density also explained variation in infected tree locations. The mean street density (in intersections per km^2) was 33.0 at infected points, 40.5 at uninfected points, and 46.1 at random points (Figure 9). The ANOVA of the three datasets yielded a p-value of 0.04, and so I accepted the differences as significant. A heteroscedastic, one-sided t-test between infected and uninfected points yielded a p-value of 0.0036, and the same test between infected and random points yielded a p-value of 0.0046, and so I could accept the three point-types as statistically distinct from one another in terms of difference in means. Infected trees, on average, were located in areas with 30% less street density than random points in the study area.

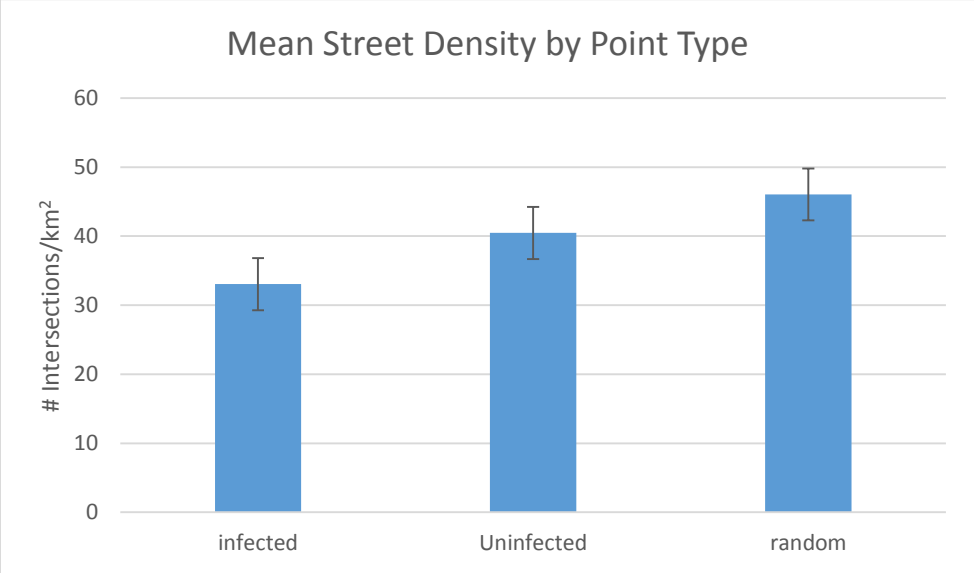

Figure 9. Mean Street Density by Point Type, in intersections/km^2

64% of the infected points fell below the mean, and 36% fell above. Areas below 33 intersections/km as twice as likely to support infection as areas above 33 intersections/km^2. Confounding reasons for low street density meant I evaluated this layer as lower risk overall. For the purposes of the suitability analysis, I therefore defined "high risk", at a value of 5, below the mean, and "low risk", at a value of 2.5, as above the mean (Table 5).

Table 5. Street density at infected points, defined by the mean (33 intersections/km^2)

Street Density (in intersections/km^2)	**>33 i/km^2**	**<33 i/km^2**
Infected points	36%	64%
Suitability Value	2.5	5

Final Suitability Analysis

Areas closer to wildlands are at highest risk for Sudden Oak Death infection, but some sites within urban spaces, especially those with high percent tree coverage and low street density, are at elevated risk. Summing together suitability values from each individual risk factor (Table 6, Figure 10), I produced my final Infection Potential map for Sudde Oak Death (Figure 11). The risk ranged from 0-45, with 45 being the absolute highest risk of infection. This was met if a site was within 1000m of a wildland boundary, 200m of a known infection, and was in at least 30% tree coverage, with less than 33 intersections/km^2. The lowest risk is characterized by a site more than 4100m from a wildland boundary, more than 1000m from a known infection, in an area with less than 10% tree coverage and with more than 33 intersections/km^2. The final map represents a range between these two extremes, using Standard Deviation from the mean to define the spread of data within ArcMap.

Table 6. Final Suitability Analysis, with Proximity to Wildlands, Proximity to Known Infection, Percent Tree Coverage, and Street Density valued based on the predescribed study.

	Very low risk	**Low risk**	**Medium risk**	**Medium-high risk**	**High risk**	**Very high risk**
Proximity to Wildlands	0	1	1	3	10	-
Proximity to Known Infection	0	-	-	-	10	20
Percent Tree Coverage	0	1	2	-	10	-
Street Density		-	2.5	5	-	-

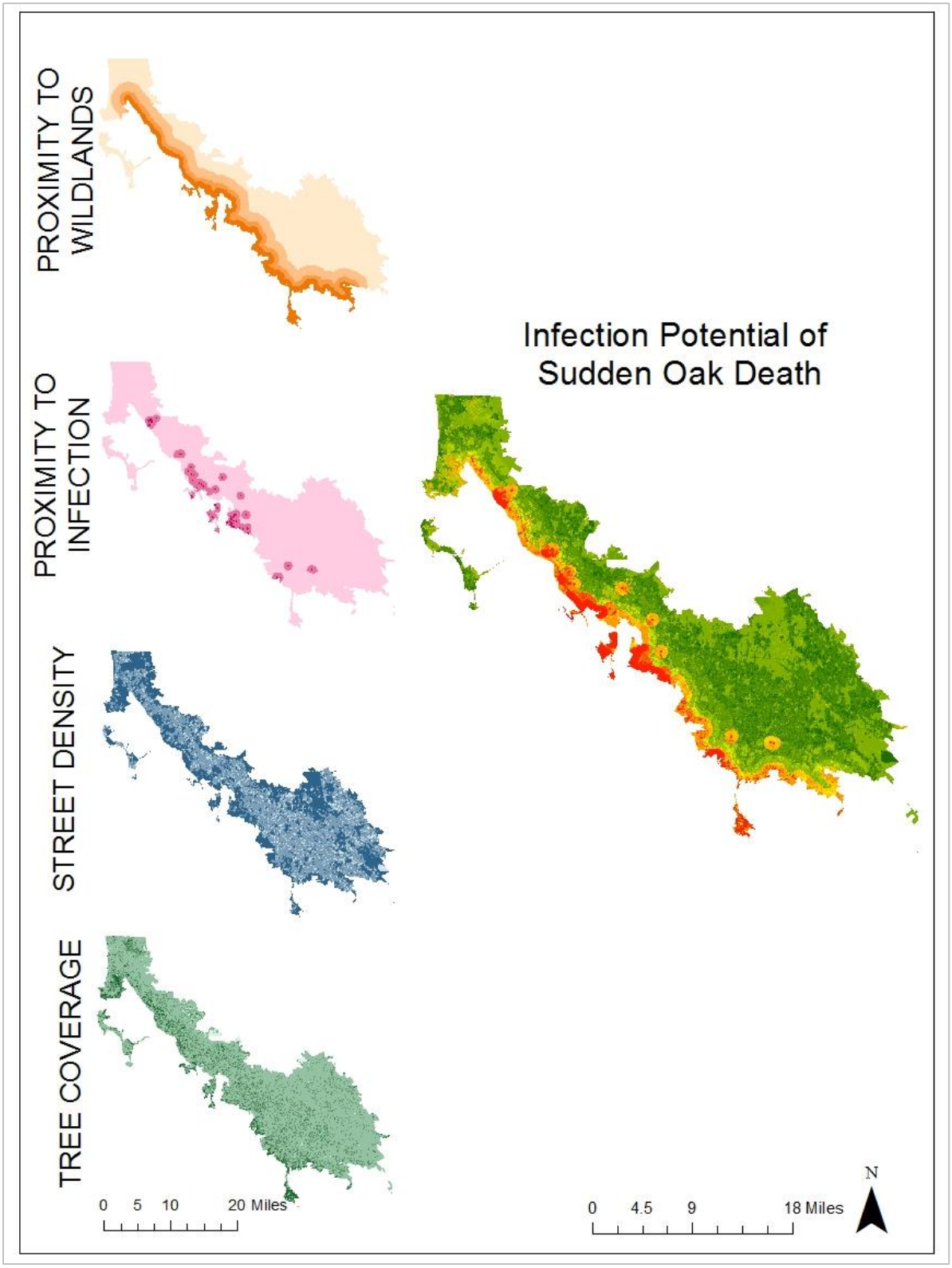

Figure 10. Each of the variables building the final Infection Potential model.

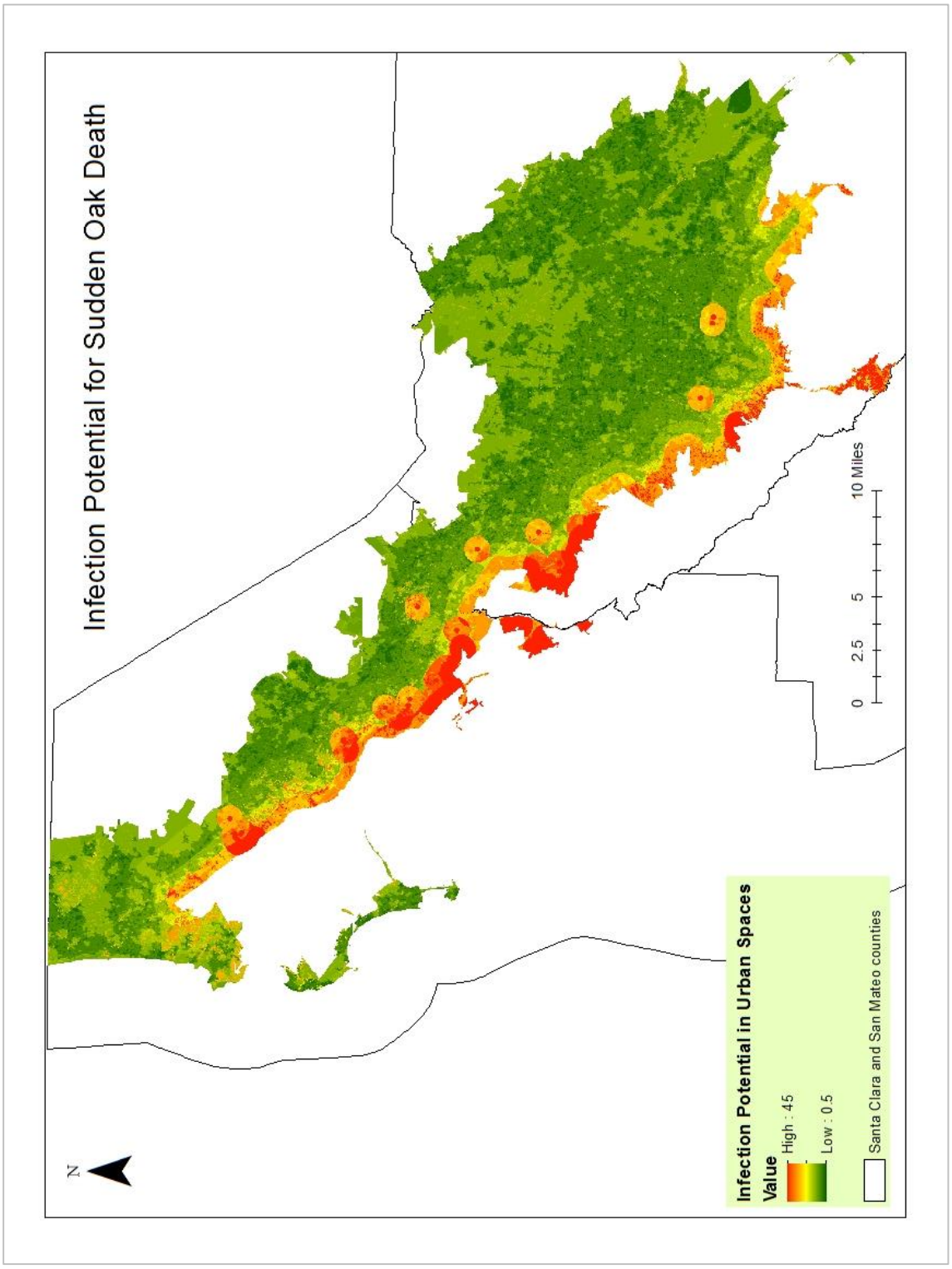

Figure 11. The Final Infection Potential for Sudden Oak Death

DISCUSSION

I created a predictive model of urban Sudden Oak Death infection based on the parameters I identified, accepting the hypothesis that each parameter represented a significant factor in Sudden Oak Death distribution in urban areas. This model will provide useful information about SOD to people in urban environments, especially landowners, as well as urban park managers and public officials. Existing research has focused on spatial suitability of wildlands, where there is much higher risk for Sudden Oak Death, and urban spaces have been broadly described as "low-risk" areas for the disease (Meentemeyer et al. 2011). However, as my research demonstrates, some areas within urban landscapes are at higher risk than others, and this information could prove useful to landowners who otherwise have little information for interpreting their risk within urban environments. Urban park managers with limited resources can use this information to focus on high-risk areas rather than attempting to address the disease across a broader region. I accepted my hypothesis that some urban spaces were at higher risk for Sudden Oak Death.

Proximity to Wildlands

Proximity to wildlands proved to be a predictor of Sudden Oak Death infection. Though this is visually apparent based on known infections, the parameter is not addressed in the Sudden Oak Death literature. I believe this is because prior research has not tried to make a distinction between urban/wildlands spaces, and has instead focused on wildlands. Some research indicates that forest/wildland edges are more susceptible to the disease, however, which could explain the increased risk to urban spaces on the other side (Kelly and Meentemeyer 2002). I believe that this distinction that I made between Urban and Wildlands infections is important, and warrants further research. It emphasizes the relatively low-risk any homeowner has for Sudden Oak Death, but it also defines communities that are likely to be at a somewhat-elevated risk, such as those bordering wildlands. Sudden Oak Death control efforts could be targeted towards these communities, which could then serve as an active barrier between the heavily-infected wildlands and urban spaces, which are currently mostly unaffected by the disease.

My model found 100% of infections within 4100m of a wildland boundary. This does not exclude the possibility that infections could occur more deeply in an urban space, but because of

the trajectory of the disease (localized infections), it is most likely to slowly progess over years. Furthermore, 70% of infected trees were within 1000m of a wildland boundary. With the overwhelming majority of infections clustered close to wildlands, it is reasonable to assume that urban infections, even if they spread, are unlikely to be supported enough by the urban environment to establish themselves as densely as they do in wildlands. Given that most of the South Bay is now built out, the wildland-urban interface is basically a fixed parameter. This is useful from a model perspective because it is not dynamic—being within 1000 meters of a wildland space will signify increased risk, even as the landscape of Sudden Oak Death changes dynamically into the future.

Proximity to Known Infection

Proximity to a known infection is one of the strongest predictors of infection potential, as found in the literature (Meentemeyer et al. 2008). The SODmap Mobile application, produced by the Garbelotto lab and based on the same SOD Blitz data I used in this study, currently provides on-the-ground risk predictions to the public. It uses proximity as its only parameter (unpublished data). As Sudden Oak Death is caused by a water- and wind- dispersed pathogen, requiring proximity between hosts to jump between them, it is reasonable to treat local areas as high-risk (Kelly and Meentemeyer 2002). The distances I used to demonstrate that risk are in line with current literature on the subject, at 200m for very high risk, and 1000m for high risk (Kelly and Meentemeyer 2002).

The vast majority of the risk areas I defined in this way had direct contact with either a wildland boundary or another infection risk area (Figure 6b). Only 6 of the 110 infected samples appeared outside of these ranges. This reconfirms the importance of both proximity to wildlands and proximity to known infections in defining an area's infection potential for Sudden Oak Death. The "independent" infection sites either represent undersampled areas or infections due to anthropological introduction of infection through nursery stock.

Since known infections are constantly increasing, this part of the model has the potential to change rapidly with new information. One new primary infection could significantly change the risk landscape in my study site (Meentemeyer 2008). For this reason, if it were to be part of a dynamic predictor for the public, mechanisms would need to be in place to regenerate risk with

each new known infection. While within the capabilities of software, this is outside the scope of my study, though something I would like to see developed if this project continued.

Percent Tree Coverage

Areas with higher percent tree coverage are more susceptible to Sudden Oak Death. This makes sense, since dense foliage has been shown to be more hospitable to the disease (Davidson 2002). Areas with more than 30% tree coverage in a 30m by 30m region are at high risk of infection, which could represent about one mature *U. californica* or *N. densiflorus* (Fryer 2008, Howard 1992). Most urban areas have tree coverage of just 10-20%, and so infected areas represent a significant deviation from average. However, without comprehensive tree species knowledge, the percent tree coverage parameter could potentially include large areas of species which are not susceptible to Sudden Oak Death, which could under or over-predict risk in an area if left unaddressed (Meentemeyer et al. 2011).

Urban Street Density

Previous research has demonstrated that "patch size" of wildlands is a key predictor of Sudden Oak Death viability, with small patches incapable of supporting the disease (Condeso et al. 2007). This has been hypothesized to be due to lack of local humidity within small areas, as well as the "isolation" of smaller patches—the disease has a harder job "jumping" between breaks in the landscape (Ellis et al. 2010). It is for this reason that Sudden Oak Death generally does so poorly in urban spaces—the highly fragmented environment inhibits spread of the pathogen, which requires moisture and short-range wind dispersal mechanisms to spread (Ellis et al. 2010, Meentemeyer et al. 2011, Meentemeyer et al. 2008).

The disease thrives in moist, dense foliage; dense areas of urban streets, such as downtown areas, are unlikely to meet those needs. Regardless, I found a correlation between urban street density and (1) presence of sampled trees, as well as (2) urban infections, with infected samples in areas, on average, 30% less dense than random sites within the study area. Most infections were found in areas with less than 33 intersections per sq. km. However, this parameter is confounded by low street density around coastal bay areas and especially industrial parks. For this reason, it is

important to take it into consideration alongside percent tree coverage to eliminate areas of "unfragmented" urban space which is inhospitable to tree species.

Limitations and Future Directions

In summary, this model enhances current risk-analysis for infection by Sudden Oak Death. It is based on known risk factors, and does a good job of defining urban spaces at elevated risk of infection by the pathogen. This risk is lower than the risk of infection within wildlands, but this model provides information on more nuanced differences in risk within urban spaces, where individuals may be able to target the disease.

One alternative explanation for some of the points of known infection is transmission by infected nursery stock—as I mentioned earlier in my study, nursery plants such as Azalea and Rhododendron are susceptible to Sudden Oak Death, and considerable monitoring effort is put into testing nursery stock for the pathogen (Werres et al .2001). There is some risk that novel infections in an area can be introduced by these stock (Goss et al. 2009). This could explain some of the most isolated cases of Sudden Oak Death in my dataset.

This model, used in parallel with climate models, could potentially further define risk areas in my study site. Generally, temperature and precipitation are the leading predictors of an area's suitability to Sudden Oak Death, and play large roles in many existing models (Ellis et al. 2010, Meentemeyer 2011). Since my study area was within a relative homogenous region in terms of climate, and because I wanted to focus on non-climate-based predictive modeling, these pieces were not included in my model. Furthermore, ground conditions and microclimate data for urban landscapes would necessarily need to be highly detailed, as urban spaces alter local environments so much (Arnfield 2003).

My definition of wildlands is defined as any areas not encompassed by the Census Bureau's definition of urban spaces. Other approaches could include defining wildlands by watersheds, or by whether wildlands are infected with Sudden Oak Death. Watershed definitions could offer some information about water- or wind- dispersal in areas, although high quantities of water (streams etc.) are not correlated with infection rates (Davidson et al. 2002). The wildland-urban interface (WUI) is also used, especially for wildfire management, to define this boundary. It is usually characterized as a region of wildlands and low residential density, and it could be applied to

Sudden Oak Death distributions using similar techniques to those in this study (Radeloff et al. 2005).

Broader Implications

Since urban spaces are generally at a reduced risk of infection, they are generally of low-priority in disease management schemes. However, with public interest in protecting oak species, providing information on urban infection rates is valuable to a range of people. With this model, private property owners could have a better tool to evaluate the risk their property has to the disease. High-phosphite treatments exist for preventing oak tree infection by Sudden Oak Death, but the cost and labor of applying these treatments make them economically unviable on the wildland scale (Garbelotto et al. 2007). Individual trees on private property, however, are perfect candidates for this kind of protective treatment. Also, urban park managers can better prepare for potential infections, knowing which areas are the most vulnerable to Sudden Oak Death (Meentemeyer 2011). New plantings can take into consideration the location of known infections, and clearances of susceptible plants can be evaluated likewise. In conclusion, characteristics of the urban environment which correspond to known requirements for Sudden Oak Death infection can be used to predict infection potential within urban green spaces.

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