

# Gallery Characteristics of the Invasive Shot Hole Borer and Extent of Accompanying *Fusarium* Dieback Disease Spread in Relation to the CODIT Model and Principles in a London Plane Tree

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*Figure 1. The London plane tree in Lakewood, CA with invasive shot hole borer presence selected for dissection. Image courtesy of Google Street View.*

## Introduction

For urban forests to provide extensive benefits to residents including urban cooling, particulate pollution reduction, mental health improvements and raising property values, the trees within the forests must be healthy (Livesely et al., 2016). Annually, huge investments of time and money are spent trying to develop and maintain urban forests. In 2018, the landscape sector alone of the green industries in the US, of which the management of urban forests comprises a significant amount, accounted for \$221.89

billion in output, \$119.18 billion in GDP added value, and employed 1.5 million people (Hall et al., 2020). Part of the maintenance of urban forests includes understanding, treating, and preventing the diseases and pests that cause tree mortality and ill health, and planning forests for disease- and pest-resistant future forests.

*Fusarium* dieback is a disease that commonly afflicts urban trees. The complex of fungi causing *Fusarium* dieback is symbiotic with and vectored by invasive shot hole borers (ISHB), which is a collective

term for two small ambrosia beetles distinguishable only through DNA analysis: the polyphagous shot hole borer (*Euwallacea fornicates*) and the Kuroshio shot hole borer (*Euwallacea kuroshio*). Polyphagous shot hole borers are known to be present throughout Southern California. First identified in Los Angeles County in 2003, polyphagous shot hole borers have since been observed in San Diego, Riverside, and Orange Counties (Eskalen et al., 2013; Umeda et al., 2016).

These ambrosia beetles bore holes into trees, creating galleries that they inoculate with fungi to feed their young. While providing nutrients for the beetles, the fungi also invade the vascular parts of the tree and inhibit the transport of water and nutrients to the branches and leaves (Eskalen et al., 2012; Freeman et al., 2013). This disease can cause discoloration of bark and leaves, wilting, branch dieback and breakage, and death of both young and mature trees (Mendel et al. 2012; Umeda et al., 2016). The extensive ISHB galleries by themselves weaken tree wood and can lead to breakage.

Nealy half of street tree species are vulnerable to colonization by the complex of fungi that causes *Fusarium* dieback disease (Eskalen et al., 2013), including *Platanus × hispanica* (London plane tree), the second most planted street tree in California (California Urban Forest Inventory, 2022). We had conducted an evaluation of pesticide treatments to control the ISHB on London plane trees in Lakewood, California from 2018 to 2020 (results not yet published). We dissected an infested London plane tree to learn more about the movement of ISHBs, the extent and structure of their galleries and *Fusarium* dieback disease spread, and how the principles of the Compartmentalization of Decay in Trees (CODIT) model (Shortle, 2000) affected ISHB behavior and subsequent disease spread (Figure 1).

### Methods

For this case study, we chose a London plane tree in Lakewood, CA that was part of an evaluation



**Figure 2.** Cross-sectional cookies of the destructively sampled London plane tree. *Image courtesy of Don Hodel.*

of pesticides to control ISHB. On July 7, 2020, we destructively sampled the tree to observe the galleries and the extent of any disease invasion. Using a chainsaw, we cut the trunk into 30-cm-long sections. For three weeks, we stored the sections outdoors in the shade at a milling yard in Ontario, CA until we cut them with more precise angles.

On July 28, 2020, we dissected the sections to track the galleries. Using a mix of three different angled cuts, we tracked five galleries. To select the galleries for the dissection, we identified five entry/exit holes in the sections that were formed between 2018 and 2020. To track holes 1 to 3, we cut across seventeen cross-sectional “cookies” 1.25 cm thick (Figures 2-3). In each cookie, we measured gallery depth from the bark surface and gallery length. In cases of branched galleries, we measured each fork separately. For hole 4, we cut 0.625 cm slices parallel to the trunk’s axis and perpendicular to the entry hole. For hole 5 we cut five 0.625 cm thick slices through the center of the trunk parallel to the entry hole. For holes 4 and 5, we measured the width and length of both the galleries and the surrounding wood discoloration, the latter of which is evidence of *Fusarium* disease.

### Results

The dissected tree grew from 37.8 cm DSH (diameter at standard height) in January 2018 to 39.05 cm DSH July 2020 (an increase of 1.25 cm DSH). Over



**Figure 3.** Wood discoloration around hole three. Image courtesy of Don Hodel.

that period, the tree deposited new callus wood over the entry holes of the observed galleries, so the depth measurements included the additional 0.625 cm new wood that was deposited since they were created.

At the time of dissection, the galleries for holes 1 to 3 were covered by an average of 2.2 cm (+ 0.7 cm) of new wood. Hole 4 was 2.9 cm from the surface, and hole 5 was 2.2 cm from the surface (**Figure 3**). The galleries connecting with holes 1 to 3 were on average 1.4 cm (+ 0.9 cm) in length.

We measured the extent of wood discoloration, which is an indication of disease spread, around holes 4 and 5 with respect to the CODIT walls comprising the three reaction zones (Mason, 2020; Shortle, 2000):

- Wall 1, which resists the vertical spread of infection,
- Wall 2 which resists the inward spread, and
- Wall 3 which resists the lateral spread.

Discoloration in hole 4 extended the farthest through wall 1 along the trunk axis (on average 6.3 cm + 3.0 cm), followed by wall 3 along the rings (on average 0.7 cm + 0.3 cm). Discoloration in hole 5 extended the farthest through Wall 1 along the trunk axis (on average 2.6 cm + 1.89 cm), followed by wall 2 towards the center of the tree (on average 1.0 cm + 0.9 cm).

### Discussion

Dissection of one tree revealed the extent of the galleries and the resulting wood discoloration

*Fusarium* dieback disease. On average, the length of a gallery within a cross sectional layer was 1.4 cm. The galleries of holes 1 through 3 extended an average of 7.8 cm along the vertical axis of the stem, none of which was deeper than 7 cm from the surface, which was approximately 35% of the way to the center of the stem.

Wood discoloration surrounding the galleries was significantly more extensive. The spread of the discoloration in each dimension behaved as the CODIT model predicted (Shortle, 2000). Wall 1 resisted the spread of disease decay along the stem axis and is the weakest wall. Wood discoloration extended the farthest from the gallery along this dimension (on average 6.3 cm + 3.0 cm). Wall 2, comprised of the latewood cells within each annual ring, resisted the spread of disease decay towards the center of the tree. As expected, the discoloration extended the next farthest distance in this dimension (on average 1.0 cm + 0.9 cm). Finally, wall 3, comprised of ray cells, resisted the spread of disease decay around the circumference of the annular rings. The least spread of disease decay was along this dimension (on average 0.7 cm + 0.3 cm)

Our observations and measurements of the extent of *Fusarium* dieback disease decay in the ISHB galleries in this London plane tree are consistent with the CODIT model. Wall 1 was the least effective, wall 2 was intermediate, and wall 3 was the most effective in resisting disease decay spread.

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