Welcome to the Sudden Oak Death Third Science Symposium

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On behalf of the United States Department of Agriculture (USDA)-Forest Service, Pacific Southwest Research Station and the California Oak Mortality Task Force, it is my pleasure to welcome you to the Sudden Oak Death Third Science Symposium. Looking back at the first sudden oak death science symposium held in Monterey in December 2002, it is amazing to see how far we have come in such a short period of time. It is hard to believe that at that point—a little over four years ago—we didn’t even know Camellia was susceptible to Phytophthora ramorum.

It seems like yesterday when P. ramorum was first isolated and identified as the causal agent of sudden oak death. It was the summer of 2000, and Everett Hansen (Oregon State University) was in California for a visit. I remember my feelings of frustration as we drove through Marin County looking at the impacted forests; my mind was reeling with so many questions.

Why were so many coast live oaks (Quercus agrifolia) dying?
Was the same agent killing coast live oak and tanoak (Lithocarpus densiflorus)?
Was the same agent killing both the large and small trees?
Were the beetle attacks primary or secondary?
Why was the pattern of mortality so variable, starting at ridge-lines, mid-slope, or in canyon bottoms?

Everett nodded, smiled and said, “You know Susan, someday it will all make sense.” Well, I am still waiting for that someday, but the unfolding events and research surrounding P. ramorum is an amazing story of discovery. It is fascinating to ponder the current unknowns as well as the key findings that have brought us to where we are today.

Since it is often the questions that drive the discoveries, I challenge you to carefully consider what is presented at this symposium. Throughout this week, ask yourself what doesn’t make sense. Ask yourself what doesn’t fit with your observations. Your questions have been, and will continue to be, the key to our progress.

How many times have you heard, P. ramorum kills trees by inciting girdling cankers? Yet, infected trees often die before they are completely girdled. How can this be? Something about this answer has never seemed quite right. Recognizing this discrepancy, research has recently been conducted to better understand how P. ramorum kills trees. Without giving away too much, I will say that new findings

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1 A version of this paper was presented at the Sudden Oak Death Third Science Symposium, March 5–9, 2007, Santa Rosa, California.
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of *P. ramorum* and its spread in wood are unraveling old theories and introducing new concepts.

So I am on a hunt for new, key questions. Where is the sudden oak death story incomplete? We need your assistance. On Friday we are holding a research needs assessment session to identify and prioritize future research. What problem are you struggling with that needs to be addressed by new or additional research? What pieces of the puzzle do you see not fitting?

As the story unfolds, we face increasingly challenging questions as well as evidence that is sometimes hard to face. How well are sudden oak death treatments working? Can *P. ramorum* be eradicated, or its spread slowed, in nurseries or forests? These are hard problems to solve that require courage, dedication, and the ability to continually adapt to new realities.

Often the greatest opportunity for progress comes in honest dialogue, constructive criticism, and in continuing to wonder and ask good questions. To support a collaborative environment, the format for this symposium has been designed to bring all disciplines of sudden oak death and *P. ramorum* together.

I thank all of you for being here in spite of budget problems and winter weather. It is exciting to see this gathering of the world’s “SOD Squad.”

**Acknowledgments and Thanks**

The conference committee thanks the North American Plant Protection Organization for organizing and hosting Monday’s “Risks to conifers” session.

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