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ECOSYSTEM ECOLOGY – ORIGINAL RESEARCH

Wildfire and forest disease interaction lead to greater loss of soil nutrients and carbon

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Abstract Fire and forest disease have significant ecological impacts, but the interactions of these two disturbances are rarely studied. We measured soil C, N, Ca, P, and pH in forests of the Big Sur region of California impacted by the exotic pathogen *Phytophthora ramorum*, cause of sudden oak death, and the 2008 Basin wildfire complex. In Big Sur, overstory tree mortality following *P. ramorum* invasion has been extensive in redwood and mixed evergreen forests, where the pathogen kills true oaks and tanoak (*Notholithocarpus densiflorus*). Sampling was conducted across a full-factorial combination of disease/no disease and burned/unburned conditions in both forest types. Forest floor organic matter and associated nutrients were greater in unburned redwood compared to unburned mixed evergreen forests. Post-fire element pools were similar between forest types, but lower in burned-invaded compared to burned-uninvaded plots. We found evidence disease-generated fuels led to increased loss of forest floor C, N, Ca, and P. The same effects were associated with lower %C and higher PO_4–P in the mineral soil. Fire–disease interactions were linear functions of pre-fire host mortality which was similar between the forest types. Our analysis suggests that these effects increased forest floor C loss by as much as 24.4 and 21.3 % in redwood and mixed evergreen forests, respectively, with similar maximum losses for the other forest floor elements. Accumulation of sudden oak death generated fuels has potential to increase fire-related loss of soil nutrients at the region-scale of this disease and similar patterns are likely in other forests, where fire and disease overlap.

Keywords *Phytophthora ramorum* · Sudden oak death · Tanoak · Nitrogen · Invasive pathogens · Ecosystem disease impacts

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Introduction

Disturbances affected by global change, such as fire and the introduction of invasive species, have powerful influences on resources associated with temperate forests, including soil carbon storage and nutrient cycling (Mack and D'Antonio 1998; Ehrenfeld 2010; Mascaro et al. 2012). A detailed and growing body of research has quantified the ecosystem impacts of fire (Bormann et al. 2008; Nave et al. 2011; Dooley and Treseder 2012), but far fewer studies have investigated the impacts of outbreak, either by insects or by pathogens, on soil properties and ecosystem functions, such as carbon sequestration (Gandhi and Herms 2010; Hicke et al. 2012). Insect and disease outbreaks are often long-term, persistent forces that structure ecosystems in spatially heterogeneous patterns driven by host distribution, environment, and dispersal processes (Lynch and Moorcroft 2008; Garnas et al. 2012; Orwig et al. 2012). Epidemiological drivers are distinct from the drivers of fire, yet almost no studies have examined these events in

combination, meaning that their interactions are poorly understood (Simard et al. 2010; Metz et al. 2013; Buma 2015). At the same time, forests are increasingly impacted by outbreak suggesting the interaction of these events will drive mortality, shifts in species composition, and critical ecosystem processes.

Both fire and outbreak (hereafter referring to either insects or pathogens) are spatially and temporally heterogeneous disturbances. Fire can vary greatly within forests from relatively low intensity burns to intense crown fire on scales ranging from tens to hundreds of hectares (Vose et al. 1999; Odion et al. 2004). Outbreaks can also have substantial spatial heterogeneity driven by environment and the distribution of hosts with variable capacity to transmit a pathogen or support insect reproduction (Meentemeyer et al. 2008; Fitzpatrick et al. 2011). Temporal differences are even more fundamental between these disturbances. Fire is a relatively discrete disturbance albeit one that occurs on intervals which vary greatly across ecosystems (Lorimer et al. 2009). Outbreaks of exotic pathogens and insects are often chronic stresses that typically span years to decades (Albani et al. 2010; Cobb et al. 2012b) and result in slower, and relatively lower magnitude ecosystem changes (Griffin and Turner 2012; Cobb et al. 2013; Orwig et al. 2013). The spatial–temporal heterogeneity of outbreaks can moderate ecosystem change by creating partial canopy disturbances that are initiated at different times within stands (Orwig et al. 2008; Cobb et al. 2013). However, this mortality also creates spatially and temporally heterogeneous fuel conditions that may alter the impacts of fire (Simard et al. 2010; Metz et al. 2011, 2013).

The goal of this study is to assess interactive effects of fire and forest disease on soil carbon and nutrient pools by focusing on the impacts of *Phytophthora ramorum*, the cause of sudden oak death, as a case study of a regional-scale outbreak of an exotic forest pathogen. *P. ramorum* kills several overstory tree species common in the redwood (*Sequoia sempervirens*) and mixed evergreen forest types of coastal California and Oregon: tanoak (*Notholithocarpus densiflorus*), coast live oak (*Quercus agrifolia*), and Shreve's oak (*Q. parvula*; Rizzo et al. 2005). The name 'sudden oak death' is somewhat misleading, as the most greatly impacted tree, tanoak, is not a true oak. Furthermore, the disease causes extensive overstory tree mortality of true oaks and tanoak within redwood and mixed evergreen forests. Both forest types have moderate canopy-tree diversity, where many trees, such as redwood, are infrequently infected and suffer no known negative effects (Davidson et al. 2005; Cobb et al. 2010). Sudden oak death causes disease at the ecosystem-level through selective mortality of canopy trees that in turn alters elemental cycling processes and redistributes elements among stand-level pools (Cobb et al. 2012a, 2013; Metz et al. 2012). Redwood and mixed evergreen forest types are important components of California Mediterranean forests, where wildfire is frequent and often intense. The extensive area invaded by *P. ramorum* combined with the common occurrence of fire implies that these disturbances often overlap (Ramage et al. 2010; Meentemeyer et al. 2011; Metz et al. 2012). Disease severity is variable among stands, but is strongly associated with the distribution of hosts that support sporulation: tanoak and bay laurel (*Umbellularia californica*), a common coastal-forest overstory tree which does not suffer deleterious impacts following infection (Davidson et al. 2005; Meentemeyer et al. 2011; Cobb et al. 2012b). *P. ramorum* is broadly representative of a rapidly spreading exotic organism that creates novel forest structure and fuel conditions by causing rapid and selective canopy mortality.

In June 2008, the Basin Complex wildfire was ignited by dry-lightning storms that resulted in widespread wildfires throughout coastal California (Ramage et al. 2010; Metz et al. 2011). A second wildfire, the Chalk Fire, was ignited in late September 2008 and enlarged the contiguous area of burned and *P. ramorum*-invaded forests. The 2008 wildfires burned ~70,000 ha of the Big Sur landscape, including 121 of 280 long-term plots established to examine environmental and epidemiological factors, influencing the spread and impacts of *P. ramorum* (Metz et al. 2011; Beh et al. 2012). Similar to many fire-prone coastal California redwood and mixed evergreen forests, the Big Sur landscape had been greatly impacted by sudden oak death at the time of the fire and was characterized by extensive, but spatially heterogeneous mortality of tanoak and true oaks (Meentemeyer et al. 2008; Metz et al. 2012). This mosaic of disease impacts ranged from recent-to-older mortality in addition to a broad range of cumulative biomass killed by the pathogen (Metz et al. 2011; Cobb et al. 2012a).

Soils are critical forest resources that store carbon and provision nutrients for plant and microbial growth. Previous analysis of the 2008 Big Sur wildfires demonstrated that host mortality and fuel accumulation was associated with greater post-fire mortality and soil-burn severity (Metz et al. 2011, 2013). Here, we determine if disease driven mortality also increased loss of forest floor and mineral soil (<15 cm depth) carbon and nutrients, a potentially important ecological interaction between fire and disease. We also investigated changes in other forest floor and mineral soil characteristics, including mineral soil pH, forest floor mass, and forest floor depth. We leveraged extensive pre-fire information on pathogen populations and disease-caused mortality to understand forest floor and mineral soil differences between pathogen and/or fire-impacted plots.

Although fire impacts to soil nutrients and structure have been studied in several Mediterranean forests (Rashid 1987; Kaye et al. 2010; Certini et al. 2011), no assessment

of forest floor and mineral soil nutrient or carbon changes has been conducted in the biologically and environmentally complex Mediterranean forests of the Big Sur region. Intensity of the 2008 Big Sur wildfires was high in many disease-impacted forests (Metz et al. 2013); a body of previous studies suggests fire alone would reduce forest floor and mineral soil nitrogen and carbon (Kaye et al. 2010; Nave et al. 2011; Certini et al. 2011). Sudden oak death has been shown to decrease tanoak litterfall C and N while modestly increasing soil NO_3–N availability without changes to total soil C, N, or N mineralization in the redwood forest type (Cobb et al. 2013); this suggests disease alone will not affect total soil elemental pools, including C, N, P, and Ca in the forests of Big Sur.

Our study was motivated by two linked questions, reflecting the need to understand fire–disease interactions and the unique opportunity to test for these effects created by pre- and post-fire measurements in a landscape with spatially heterogeneous disease impacts: (1) what is the magnitude of differences in forest floor and mineral soil nutrient and carbon amounts in a full-factorial combination of fire and disease; are the magnitude of these differences dependent on forest community? (2) Are differences in forest floor and mineral soil element concentration and/or mass driven by amounts of pre-fire disease-caused mortality? The body of previous fire and outbreak studies suggests that intense fire will result in substantial differences in forest floor and mineral soil carbon and nutrients between burned and unburned plots, but because disease impacts occur over a longer-term and are heterogeneous, no differences will occur between outbreak-impacted/non-impacted plots within unburned areas. Given the full-factorial study design, we expected interactive effects of fire and disease would be reflected in significant fire x pathogen–invasion interaction terms for statistical models that contrasted both burned and unburned plots. Within burned plots only, we expected fire–disease interactions to be reflected as statistically significant relationships between pre-fire dead host basal area—a continuous measure of disease—and post-fire forest floor and mineral soil element pools.

Methods

Study area

The Big Sur region of California is notable for steep terrain, substantial environmental and biological diversity, and frequent, sometimes catastrophic, wildfires (Meentemeyer et al. 2008; Davis et al. 2010; Metz et al. 2012). Our plot network was established to understand the patterns of pathogen–invasion interactions between host community composition and disease emergence, and the consequences of disease outbreak on ecosystem processes. As a consequence of this aim, our study plots span diverse mapped soil types dominated by excessively well-drained, course textured sandy loams on steep slopes typified by the McMullin-Plaskett, Gamboa-Sur, and Sur-Junipero complex soil types which originate from volcanic, limestone, and metamorphic parent materials. Redwood and mixed evergreen forest types dominate the forest ecosystems of this landscape with redwood forests occurring closer to the ocean and in deeper inland valleys. Mixed evergreen forests occur in a wide range of landscape positions, including valleys and uplands, but generally at greater distance from ocean relative to redwood forests (Meentemeyer et al. 2008; Davis et al. 2010). The Esselen and Rumsien peoples have occupied the region for several thousand years with cultural practices, including frequent burning, accumulation of shellfish middens, and use of oak forests for acorn gathering and processing (Henson 1996; Keeley 2002). The vegetation covered by our study plots has almost certainly been shaped by land use of indigenous people as well as between three and nine fires during the historical period, depending on the location. The overall study area is approximately 80,000 ha in extent with land ownership distributed in a patchwork of private and public holdings ranging in size from a few ha to the extensive Los Padres National Forest. The Big Sur region was one of the first landscapes to be invaded by *P. ramorum* circa 1998 and has since developed some of the most severe disease-caused forest mortality (Meentemeyer et al. 2008; Metz et al. 2012; Garbelotto and Hayden 2012).

Pre-fire measurements

Our full plot network consists of 280,500-m^2 circular plots randomly selected in a GIS with stratification by mapped forest type, public/private ownership, fire history, and remotely sensed levels of pathogen-caused mortality; this process was conditioned on a minimum of 300-m distance between plots although in the final network, plots averaged ~650-m spacing (Meentemeyer et al. 2008; Metz et al. 2011; Beh et al. 2012). During plot setup (2006–2007), each stem >1-cm diameter at breast height (dbh) was mapped, measured for dbh, and assessed for health and symptoms of *Phytophthora* infection. *Phytophthora* infections are limited to minor leaf blight in bay laurel, typically small lesions with necrotic or discolored tissue that cover less than 15 % of the total leaf surface. On tanoak, *Phytophthora* infections can be minor leaf lesions, but more commonly occur as twig or bole cankers which cause mortality by disrupting vascular function and girdling the tree. On true oaks, infections are limited to bole cankers which can result in mortality, but which do not support any transmission. During the plot surveys, symptomatic

Table 1 Mean and standard deviations of forest floor and mineral soil C %, N %, and pH of soil damage classes immediately following the 2008 Basin Fire in Big Sur, CA

Parameter	Unburned mineral soil	Charred forest floor	Charred mineral soil	ash	Heat-oxidized mineral soil
C %	4.16 (1.21)^{ab}	23.13 (7.75)^{d}	4.9 (2.46)^{bc}	8.59 (2.99)^{c}	0.899 (0.77)^{a}
N %	0.25 (0.779)^{bc}	1.018 (0.33)^{d}	0.322 (0.124)^{c}	0.117 (0.093)^{ab}	0.042 (0.044)^{a}
pH	6.61 (0.62)^{a}	7.55 (0.57)^{a}	7.42 (0.49)^{a}	10.32 (0.82)^{b}	9.65 (1.12)^{b}
N	11	8	11	17	13

Prevalence and depth of each damage class were assessed on 61 plots, while chemistry of these damage classes were assessed on a subset of plots (*N*). Not all damage classes were found in each plot

Different letters indicate statistically significant differences with a one-way analysis of variance and Tukey's HSD means comparison

tissue tanoak, oak, or bay laurel were returned to the lab for the confirmation of infection by direct pathogen culturing on a *Phytophthora* selective media (PARP, see Davidson et al. 2005). Plots were only considered *P. ramorum*-invaded when the pathogen had been directly cultured from a symptomatic host tree. A full census wood debris volume (>20-cm diameter and >0.5-m length) was conducted during plot establishment with each piece of woody debris also assessed for decomposition state using a methodology similar to Cobb et al. (2012a).

Post-fire soil sampling

Soil sampling in burned areas was conducted in two stages. In 2008, within weeks of containment of the Basin Fire, 61 plots within the burn perimeter were resurveyed to assess fire intensity and severity of impact to the soil (Metz et al. 2011). Two and three years following the fire, a quantitative assessment of forest floor and mineral soil nutrients and carbon was conducted in redwood (2010) and mixed evergreen (2011) dominated stands. Unlike the immediate post-fire survey in 2008, these surveys spanned burned/unburned plots and pathogen-invaded/uninvaded plots for both burn categories at locations within the contiguous Basin and Chalk fire perimeter. Our more systematic soil sampling occurred in 2010 (redwood—2-years post-fire) and 2011 (mixed evergreen—3-years post-fire) and included most of the burned plots measured in 2008. Big Sur is a landscape widely invaded by *P. ramorum*, and as a result, uninvaded plots (burned or unburned) were less well represented in our data set (31 uninvaded vs. 58 invaded plots). Unburned and uninvaded plots were the least common of each category and resulted in only seven suitable plots within each forest type. Within the burned-uninvaded category, we located and surveyed nine and eight plots in mixed evergreen and redwood forest types, respectively. For invaded plots, we measured more than ten plots in each burn category and forest type. During 2008, the depths of several soil damage classes were measured at eight random locations 2–11 m from the center of the plot on radii with 45° angles for the purpose of rating soil-burn severity among plots (Metz et al. 2011). We collected at least eight representative samples of each soil-burn class (Table 1) for the later chemical analysis. All soil chemistry data included in Table 1 were collected prior to a small rain event on 4th October, 2008, because of concern that rain-caused intermixing of overlaid layers with different soil-burn severities and ash layer compaction could render these samples incomparable. During the 2010/2011 surveys 2 and 3 years later, we established three evenly spaced 15-m transects within each plot emanating from the plot center by randomly selecting a starting azimuth. Litter (O_i layer) and humic ($O_e + O_a$ layers) depths were measured, and mineral soil was sampled to 15-cm depth using a ~5.7-cm diameter soil core at 5-m interval on each of the three transects (nine samples per plot). Forest floor was quantitatively sampled (O_i, O_e, and O_a) at three locations in each plot (at the 5-m transect sampling location) using a 20 × 20-cm frame. Both forest floor and mineral soil samples were composited into a single sample per plot in the field.

Field and laboratory methods

All soil samples were air dried for 4–8 weeks prior to processing. Air-dry samples were weighed and, subsequently, mineral soils were passed through a 2-mm mesh size sieve, while forest floor samples were hand-sorted for rocks and passed through a 4-mm mesh size sieve. During sieving of the forest floor, woody material (>2-mm diameter) was removed, and all materials were again weighed and subsampled for the determination of moisture content (mass after 48 h at 105 C). This procedure yielded estimates of mass and rock content for mineral soil and forest floor soil layers as well as the woody material mass within the forest floor. Sieved bulk samples (mineral soil and forest floor without woody biomass) were assessed for total C, N, P, and Ca concentrations. Mineral soil samples were also assessed for Olsen PO_4–P, NH_4–N, and NO_3–N concentrations; mineral soil pH was measured using a saturated paste method. All chemical analyses were conducted

at the UC Davis Analytical Lab (http://anlab.ucdavis.edu/), where total CN was determined by combustion on a LECO FP-528 and TruSpec CN Analyzer, and total P and Ca were determined by nitric acid/hydrogen peroxide closed-vessel microwave digestion followed by inductively coupled plasma atomic emission spectrometry (ICP-AES). Soil NO_3–N and NH_4–N were measured in 2-N KCl soil extracts, while Olsen PO_4–P was measured in 0.5-N sodium bicarbonate soil extracts using cadmium reduction-sulfanilamide, indophenol blue, and ascorbic acid-ammonium molybdate colorimetric methods, respectively.

Data analysis

Dependencies among mineral soil and forest floor chemical and physical characteristics were examined by calculating Pearson's correlations for each variable across the entire data set and within unburned plots for each forest type. We calculated correlation coefficients (r) and adjusted p values for standard Pearson's correlations, a less conservative test of statistically significant correlation, to identify potential sources of collinearity as opposed to testing specific hypotheses. A series of linear models were constructed that fully-crossed the independent variables of burn status, pathogen invasion status, and forest type. Identical linear models were implemented for each individual elemental analysis in the forest floor and mineral soil. A second set of identical linear models were constructed for a subset of data that only included the burned plots. Here, the nominal burn status variable (burned, unburned) was substituted with pre-fire disease-caused dead host basal area to assess the direct effects of disease on each variable. We calculated partial residuals, the parameter effect given all other factors in the model, for dead host basal area when this variable was statistically significant. Dead host basal area was the sum of tanoak, coast live oak, and Shreve oak—collectively the overstory species which develop lethal *P. ramorum* caused bole cankers—documented as killed by 2006 and 2007, the survey prior to the fire. A summary of this forest structure information gathered prior to the wildfires can be found in the supplementary information (Tables S.1, S.2). In both sets of linear models, forest floor elemental masses, mineral soil %C, and %N were log-transformed to meet the model assumptions of normal distribution of error and constant variance over the range of the predictors. Olsen PO_4–P data were square root-transformed to meet the same model assumptions. The linear models and correlation analyses revealed the common patterns of soil nutrient and carbon across fire, disease, and forest types (see also supplemental information Table S.3). This characteristic allowed us to reduce the dimensionality of the data set with a nonmetric multidimensional scaling (NMDS) of the data. Preliminary analyses consisted of constructing NMDS distance matrices with six variables (forest floor and mineral soil C, N, and P) and gradually adding additional variables representing soil structure and chemistry. Each NMDS resulted in stress levels <0.15 and consistent qualitative patterns and statistical significance of the environmental variables in a permutational multivariate analysis of variance of the NMDS on pre-fire dead host basal area, our hypothesized driver of fire–disease interactions. The final NMDS analysis consisted of 15 variables, including mass of C and nutrients, inorganic nutrient concentration, humic layer ($O_e + O_a$) depth, forest floor mass, and forest floor woody material mass and had stress of 0.113. Statistical significance was considered when $p \leq 0.05$ for all analyses; all data analyses were conducted in R version 3.0 with multivariate analysis using the package Vegan version 2.3-1 and partial residuals of the linear models calculated with the package visreg.

Results

Soil-burn categories measured in 2008 were notable for very distinct chemistries (Table 1); heat-oxidized mineral soil had the lowest C and N concentrations and the highest pH values relative to all other damage classes. Forest floor ash had nitrogen concentration similar to unburned soil, but much higher pH and carbon concentration, a pattern consistent with intermixing of charcoal. Heat-oxidized mineral soil was characterized by a reddened color and had the low CN and high pH characteristic of ashed mineral soil. Unburned mineral soil collected immediately following the Basin Fire had C and N concentrations and pH similar to values measured 2 years later in the more systematic soil survey of unburned plots.

During the 2010–2011 survey, substantial differences in forest floor C, N, P, and Ca masses were found between forest types and burned vs. unburned plots (Fig. 1). Forest floor mass, total forest floor depth, and depth of the humus layer ($O_i + O_e$) were significantly greater in redwood vs. mixed evergreen forests and significantly greater in unburned vs. burned forests. However, on a concentration basis, none of these forest floor elements were significantly different between burn statuses, indicating that combustion of forest floor material, rather than changes in concentration, was the cause of these differences. This inference is supported by significant Pearson's correlations between forest floor mass and forest floor C, N, Ca, and P masses ($r > 0.92$ and $p < 0.001$ each correlation; Table S.3 supplemental information). Variation in mineral soil and forest floor element pools within unburned plots was not significantly correlated with forest structure with two exceptions. In unburned plots, redwood basal area and forest floor C were positively correlated in redwood forests ($r = 0.47$;

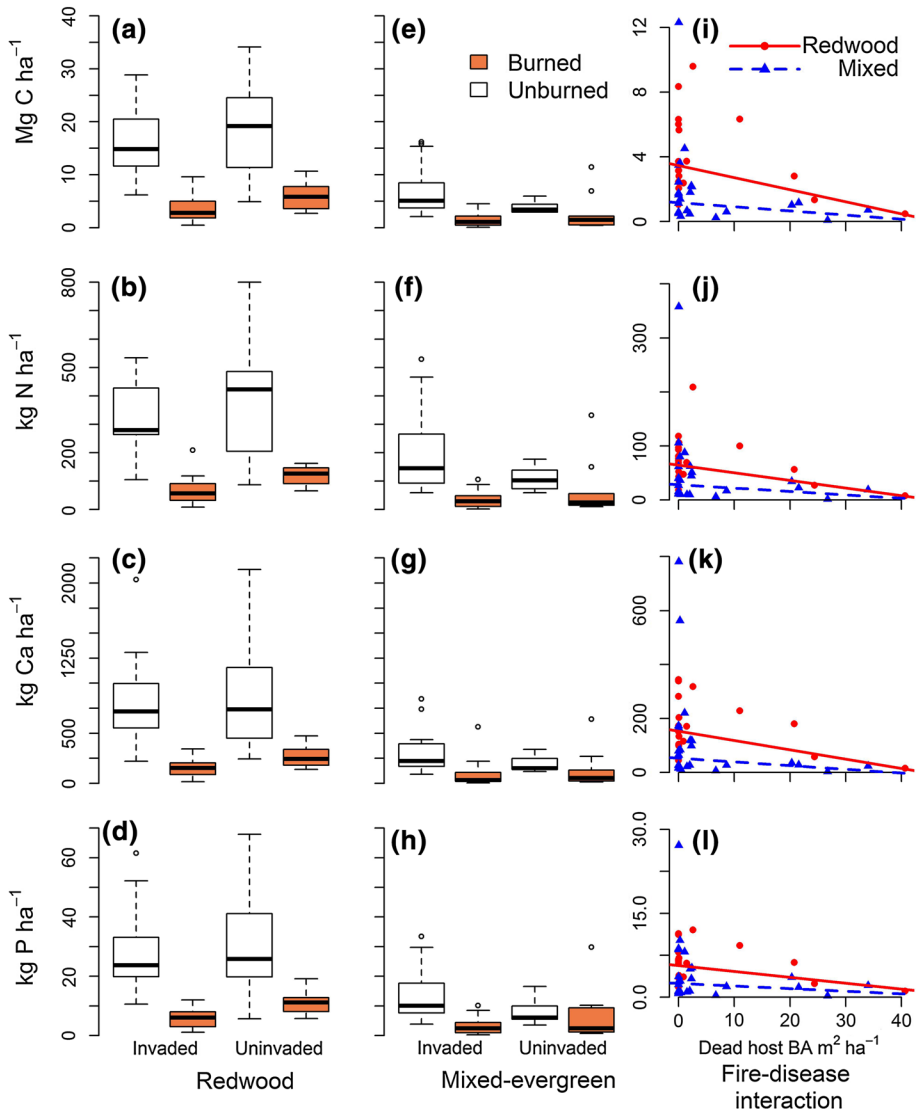

Fig. 1 Forest floor element masses in unburned (*no fill*) and burned (*orange fill*) redwood and mixed evergreen forests in Big Sur, California, impacted by sudden oak death. **a**–**d** Element masses in redwood forests; **e**–**h** respective masses in mixed evergreen forests. **i**–**l** Back-transformed linear predictors of pre-fire dead host basal area for post-fire element masses when the main effects of dead basal area and forest type were significant within each respective model (*burned plots only*). The respective back-transformed partial residuals are shown for redwood (*red*) and mixed evergreen forests (*blue*) (color figure online)

$p = 0.037$), and tanoak basal area and forest floor Ca mass were positively correlated in mixed evergreen forests ($r = 0.41$; $p = 0.042$).

Burned plots had consistently higher mineral soil Olsen PO_4–P concentration and pH compared to unburned plots (Fig. 2). In addition, mineral soil Olsen PO_4–P, C, and N concentrations were significantly greater in redwood compared to mixed evergreen forests, but pH was not significantly different between forest types. Mineral soil extractable NH_4–N was positively associated with mineral soil C within burned and unburned plots ($r = 0.76$ and $p < 0.0001$, both soil types). In accordance with this pattern, NH_4–N concentration was significantly lower in burned vs. unburned plots in both forest types. Mineral soil NO_3–N concentrations were low across all study plots with a mean concentration of 12.97 μg g^{-1} (22.44 standard deviation). Measured NO_3–N levels did not significantly differ between burn status or forest type.

Invasion status alone had no significant effect on any of our measured soil parameters. However, burn-invasion status interaction terms were significant ($p < 0.05$) for all of the forest floor mass variables except Ca ($p = 0.07$), indicating that amounts of forest floor C, N, and P were significantly greater in uninvaded compared to pathogen-invaded plots within in the burned area. Analogous results were found within burned plots; our continuous measure of disease severity—pre-fire dead host basal area—was negatively associated with forest floor elemental masses (Fig. 1) and mineral soil %C (Fig. 2). These patterns were consistent between forest types although the magnitudes were greater in redwood compared to mixed evergreen forests (Table 2). A positive association also occurred between dead host basal area and Olsen PO_4–P although this relationship was much stronger in redwood compared to mixed evergreen forests (see Fig. 2k; Table 2). In contrast, dead host basal did not have a significant effect on

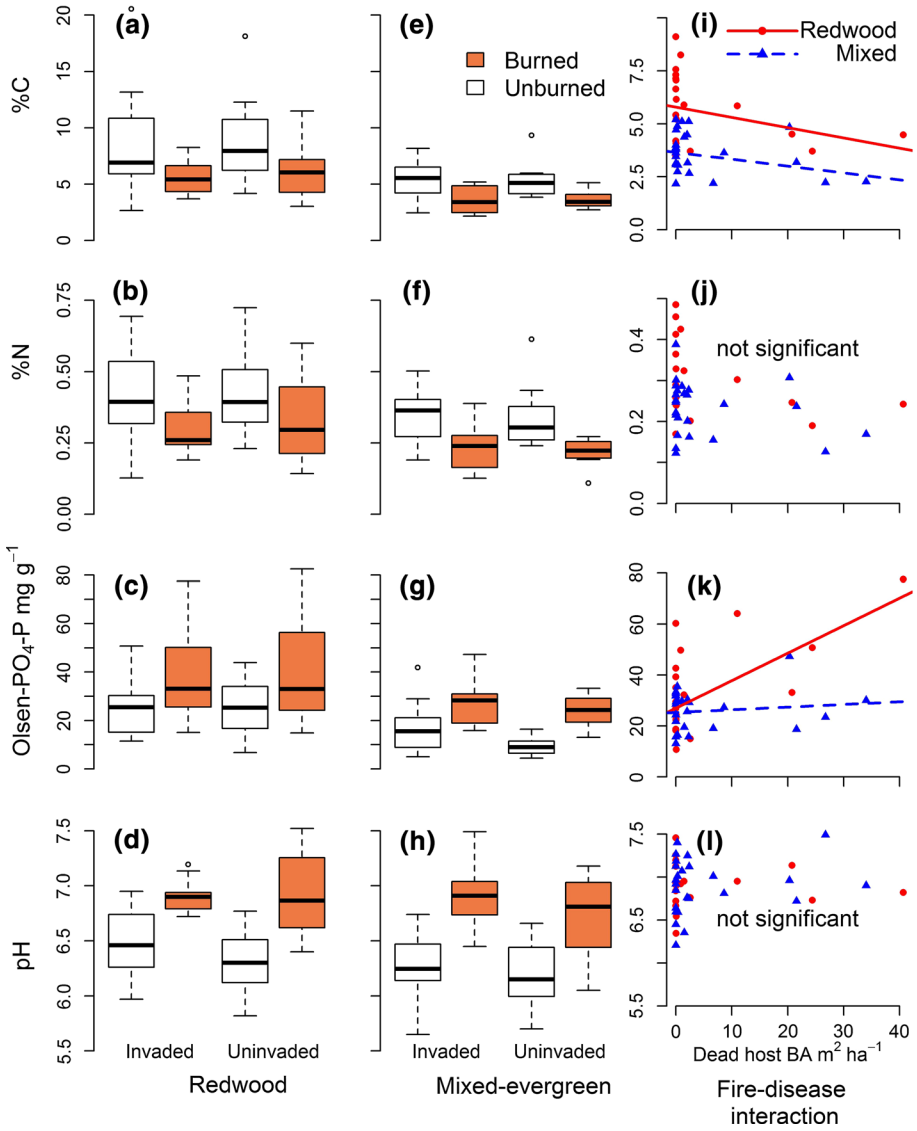

Fig. 2 Mineral soil elemental concentrations and pH in unburned (*no fill*) and burned (*orange fill*) redwood and mixed evergreen forests in Big Sur, California, impacted by sudden oak death. **a–d** Data for redwood forests; **e–h** respective data for mixed evergreen forests. **i–l** Back-transformed linear predictors of pre-fire dead host basal area for post-fire element masses when the main effects of dead basal area and forest type were significant within each respective model (*burned plots only*). The respective back-transformed partial residuals are shown for redwood (*red*) and mixed evergreen forests (*blue*) (color figure online)

mineral soil %N and pH (Fig. 2). Most second and third–order interaction terms were not significant; however, dead host basal area * invasion status and the third–order interaction of dead basal area, invasion status, and forest type were significant for mineral soil %C and Olsen PO_4–P, respectively. In both the cases, responses of burned-uninvaded plots were less sensitive to dead host basal area compared to burned-invaded plots. In addition, Olsen PO_4–P increased at a greater magnitude in redwood vs. mixed evergreen forests with increasing dead host basal area (Fig. 2). The summary tables for both sets of linear models can be found in the supplemental information (Tables S.4, S.5).

In agreement with the univariate models, our NMDS analysis of soil chemical composition demonstrated most variation between plots was associated with burn status ($r^2 = 0.24$) followed by forest type ($r^2 = 0.18$; Fig. 3). Similar to the univariate analyses when the effect of *P. ramorum* was represented as a categorical variable, invasion status alone did not have a significant effect on the multivariate differences in soil parameters. However, pre-fire dead host basal area (Fig. 3) was significantly associated with multivariate differences in soil characteristics among plots ($r^2 = 0.16$; $p < 0.001$), suggesting pre-fire patterns of disease influenced overall differences in soil parameters in the forest floor and mineral soil. Both the individual analyses of forest floor and mineral soil chemical characteristics as well as the multivariate analysis suggest that the degree of differences within burned plots as well as the differences between burned and unburned plots was affected by the extent of disease-caused mortality and support the hypothesis that fire and disease were interactive drivers of post-fire soil chemical characteristics, including soil C, N, P, and Ca.

Table 2 Estimates of changes in soil parameters due to burning (Fire effect; unburned vs. burned plots), and the additional estimated fire-caused changes in soil parameters due to the accumulation of dead host basal area (fire-disease effect; burned plots only)

Forest type Redwood	Fire effect	Fire-disease effect (m^2 ha^{-1})	Change at mean mortality (5.28 m^2 ha^{-1})	Change at maximum mortality (40.6 m^2 ha^{-1})
Forest floor				
C (Mg ha^{-1})	−12.4 (8.6)	−0.0746	−0.394	−3.029
N (kg ha^{-1})	−257.4 (182)	−1.407	−7.43	−57.124
Ca (kg ha^{-1})	−651 (508)	−3.454	−18.24	−140.23
P (kg ha^{-1})	−21.7 (16.6)	−0.106	−0.56	−4.304
Mineral soil				
C (%)	−3.05 (3.86)	−0.0484	−0.256	−1.965
PO_4–P (mg g^{-1})[a]	15.08 (18.18)	1.08	5.7	43.85
N (%)	−0.14 (0.2)	NA	NA	NA
pH	0.48 (0.38)	NA	NA	NA
Forest type Mixed evergreen	Fire effect	Fire-disease effect (m^2 ha^{-1})	Change at mean mortality (5.21 m^2 ha^{-1})	Change at maximum mortality (34.0 m^2 ha^{-1})
Forest floor				
C (Mg ha^{-1})	−4.09 (4.0)	−0.0256	−0.133	−0.87
N (kg ha^{-1})	−127.4 (121.1)	−0.6288	−3.28	−21.38
Ca (kg ha^{-1})	−159.8 (190.5)	−1.337	−6.97	−45.46
P (kg ha^{-1})	−7.74 (8.2)	−0.0468	−0.244	−1.59
Mineral soil				
C (%)	−1.93 (1.7)	−0.0325	−0.169	−1.105
PO_4–P (mg g^{-1})[†]	11.2 (10.1)	0.1046	0.545	3.56
N (%)	−0.12 (0.1)	NA	NA	NA
pH	0.589 (0.43)	NA	NA	NA

Fire effects are reported with the combined standard deviation from burned and unburned forests. Fire-disease effects were estimated from the analyses of burned plots only, where accumulated dead host basal area—killed prior to the 2008 fire—was a statistically significant factor in a multiple linear model. Fire-disease effects are reported in units of pre-fire dead host basal area (m^2 ha^{-1}) which is the total amount of oak and tanoak mortality observed within each plot in 2006 or 2007; these species develop lethal bole cankers following infection by *Phytophthora ramorum*. The linear Fire-disease effect parameter was used to predict changes for the mean amount of mortality (change at mean mortality) as well as the maximum mortality observed for the respective forest type (change at maximum mortality). Forest type and/or pre-fire dead host basal area were not significant in the respective model

NA not applicable

[a] Olsen PO_4–P

Discussion

This study demonstrates the interactive effects of fire and disease on critical soil element amounts and concentration, including C, N, P, and Ca. The 2008 wildfires and sudden oak death interacted in the Big Sur region to significantly reduce these elements compared to levels expected from fire alone. Disease intensity determined the magnitude of these effects on forest floor and mineral soil element pools, a pattern consistent with previous ecosystem studies of sudden oak death (Cobb et al. 2013). Meta-analysis of fire and forest harvest has also shown that disturbance intensity is a critical driver of bulk soil C or nutrient change (Nave et al. 2010, 2011) and this study suggests that forest disease impacts are broadly consistent with these disturbances. Increased ground-fuel accumulation is a common pattern in stands with a significant overstory mortality from sudden oak death (Meentemeyer et al. 2008; Metz et al. 2011; Cobb et al. 2012a), suggesting that interactive impacts of fire and disease on soil nutrients are likely to occur across the regional extent of *P. ramorum* invasion. However, these impacts should also occur with substantial spatiotemporal variation driven by heterogeneous patterns of pathogen establishment and tree mortality occurring over the course of decades (Davis et al. 2010; Cobb et al. 2012b; Metz et al. 2012). Several insect and disease outbreaks have been shown to increase ground fuels (Hoffman et al. 2007; Simard et al. 2010; Valachovic et al. 2011); understanding

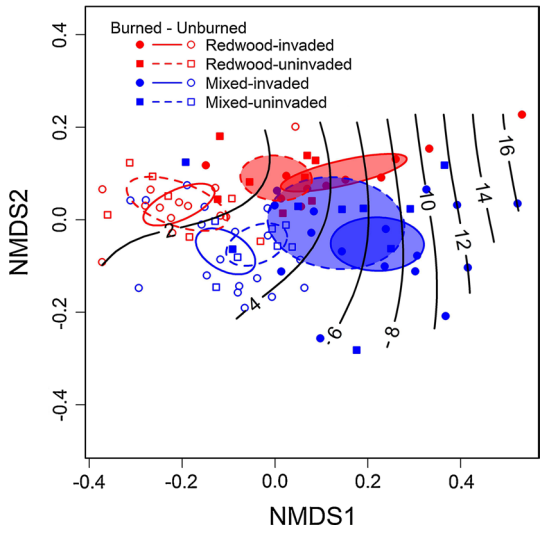

Fig. 3 Multivariate nonmetric multidimensional scaling (NMDS) of forest floor and mineral soil characteristics in burned (*solid symbols*) and unburned (*open symbols*) redwood (*red points* and *lines*) and mixed evergreen forests (*blue points* and *lines*). Multivariate plot-level data are indicated by points, while the 95 % confidence interval of the multivariate means for each forest type and burn status are indicated by the *ellipses* with *solid lines* denoting invaded plots and *dashed lines* indicating uninvaded plots. Contours demonstrate the multivariate influence of pre-fire dead host basal area (m^2 ha^{-1}), our hypothesized mechanistic driver of fire-disease interactive effects on forest floor and mineral soil C and nutrients (color figure online)

the degree and spatial patterns of these changes may hold broad and qualitative potential to predict fire impacts to soils in outbreak-impacted forests.

Previous research on interacting disturbances has focused on changes in the probability of one event, such as fire, due to historical occurrence of a second event, such as outbreak (Bebi et al. 2003; Kulakowski and Veblen 2007; Lynch and Moorcroft 2008). An associated line of work has focused on changes in disturbance impacts, such as post-fire mortality in outbreak-impacted forests (Simard et al. 2010; Metz et al. 2013). In the fire-prone Mediterranean forests of Big Sur, large fires are relatively common and their extent can be greater than that of *P. ramorum* host communities (Oneal et al. 2006; Davis et al. 2010). In this system, interactions between fire and disease are unlikely to alter the extent of fire; rather fire–disease interactions will have a greater influence on within-stand impacts. For the 2008 wildfires, pre-fire fuel accumulation and changes in aerial fuel distribution have been implicated in increasing tree mortality as well as fire intensity under a narrow range of conditions (Metz et al. 2011, 2013). The present study extends these findings by demonstrating that soil chemical differences between burned and unburned plots are influenced by pathogen-caused mortality, suggesting that forest disease can increase fire impacts to soil carbon and nutrients. Beyond our case study of sudden oak death, we hypothesize that these interactive fire-disease impacts are most likely to occur when outbreak results in greater accumulation of ground fuels that in turn transmit significant amounts of energy to soils via radiative heating. Fuels accumulation in root disease centers and diseases caused by native mistletoes are likely to result in similar effects even though pathogen dispersal and disease progression substantially differ (Rizzo et al. 2000; Hoffman et al. 2007). Depleted C and N concentrations in the most severely burned soils (heat-oxidized mineral soil; Table 1) is consistent with this expectation given that these soil-burn conditions were more frequent in stands with greater pre-fire ground fuels (Metz et al. 2011). Thus, even when ecological interactions between outbreak and fire result in decreased fire intensity because of changes in canopy fuels and other factors (Kulakowski and Veblen 2007; Simard et al. 2010); fire impacts to soil nutrient and carbon pools are likely to intensify when disease-associated accumulations of woody debris increase the cumulative energy flux to soils.

The lack of pre-fire soil analysis limits our inference to differences in soil characteristics between burned and unburned plots. However, additional data suggest the unburned soil characteristics reported here are accurate indicators of average pre-fire soil conditions in the burned study plots: (1) litter cover (assessed at plot establishment) was similar between burned and unburned plots prior to the fire; and (2) the correlation analysis of forest structure and soil elemental pools in unburned plots revealed very few significant relationships. This suggests that although forest type has a strong influence on soil elemental pools prior to the fire, differences in basal area or species composition within forest type would not confound our interpretations fire–disease interactions. In addition, volatilization of C and N from tanoak, bay laurel, and redwood litter—the dominant overstory species in these forests—has been measured in excess of 95 % in laboratory conditions (M. Varner unpublished data; see also Kuljian and Varner 2013), a magnitude similar to our observed differences between burned and unburned plots (Fig. 1). During the 2- and 3-year lag between the fire and our soil sampling, C and nutrients likely recovered to a small degree relative to unburned conditions, particularly in the forest floor which accumulated some leaf litter from trees which died following the fire as well as litterfall from surviving trees (see Beh et al. 2012; Metz et al. 2013). However, sampling immediately following the fire (Table 1) indicates burn impacts were of sufficient magnitude to drive the patterns in soil chemistry observed 2 and 3 years post-fire. Follow-up sampling could give insight into the rates of element accumulation as well as evaluate the duration of interactive fire-disease impacts on forest floor and mineral soil element pools.

Assuming that differences between burned and unburned plots are reflective of the actual changes in the soil parameters we measured, across both forest types our data suggest an average fire-caused loss of 7.8 Mg ha^{-1} C, 186.2 kg ha^{-1} N, 14.1 kg ha^{-1} P, and 380.3 kg ha^{-1} Ca from the forest floor and a reduction of mineral soil C from 7.0 to 4.55 %. By the same estimate, burning led to an increase of 12.4 kg ha^{-1} Olsen PO_4–P and pH of 0.53 with no change in C or N mass in the mineral soil. These estimated changes are similar to those reported by Nave et al. (2011) in a meta-analysis of fire impacts to soil CN in temperate forests, where fire resulted in a reduction of forest floor C and N masses and mineral soil C and N concentration but not mineral soil nutrient or C amounts. The magnitude of our estimated forest floor losses are similar to those reported by Bormann et al. (2008) in a detailed pre–post-fire assessment of soil C and N in Oregon forests that share species and forest structure with our study plots in the Big Sur region. However, Bormann et al. (2008) used a comparable-layers approach to sample mineral soil which revealed a significant loss of fine soil material and produced more accurate estimates of mineral soil C and N change. Fixed-depth sampling likely reduced our capacity to detect differences in the mineral soil and render our estimates fire-caused losses conservative.

Our data do not inform how long reductions of element pools will persist, but several published studies provide initial expectations of their duration. Using a meta-analysis approach, Dooley and Treseder (2012) estimated recovery of soil microbial community biomass to pre-fire levels in ~15 years and suggested recovery time may be determined in part by C limitation to microbial communities. Similarly, Kaye et al. (2010) found recovery to pre-fire mineral soil C levels within 10 years in a Mediterranean oak-pine forest (Catalonia, Spain). In contrast to our study, Kaye et al. (2010) found increased mineral soil C immediately following the fire, possibly due to increased deposition of charcoal. High C concentrations in forest floor ash and charred forest floor (Table 1) suggest charcoal formation occurred in our plots as well, but these soil-burn layers were rapidly integrated into the mineral soil. Our more rigorous sampling of mineral soil in 2010–2011 supports the inference that fire reduced %C between 0 and 15 cm depth.

We report a partial estimate of fire-caused loss of ecosystem element pools given that our data do not include estimates of volatilization from living vegetation. Although the fire resulted in substantial overstory tree mortality, very little of the canopy was directly volatilized (Metz et al. 2011, 2013). Recently killed but standing tanoak with full canopies of dead leaves were the exceptions. Prevalence of recently killed trees was associated with increased fire intensity presumably, because these trees held canopies of low-moisture dead leaves that effectively carried flames into the overstory (Metz et al. 2011, Kuljian and Varner 2013). Sudden oak death does not cause spatially or temporally uniform mortality meaning that in any given disease-impacted stand, disease-killed trees will be at different stages of decomposition and collapse (Metz et al. 2011; Cobb et al. 2012a; Kuljian and Varner 2013). Dead tanoak loose most of their leaves within a few years of mortality; most of the leaf mass from trees killed prior to our plot establishment (1998–2005) would have become integrated into the forest floor litter layer (O_i) at the time of the fire. Fire–disease interactions which decrease tree survival are likely to have a greater influence on soil elemental pools by reducing organic matter accumulation rates following the fire compared to direct volatilization of living biomass by the fire.

Although several recent studies have shown complex- or context-dependent interactions between outbreak and fire intensity (Bebi et al. 2003; Simard et al. 2010; Metz et al. 2011), our study shows relatively straightforward impacts of tree mortality on mineral soil and forest floor element pools. While these results are important from the perspective of basic ecological understanding, their relevance to policy and management is dependent on the implications for post-fire vegetation recovery and soil erosion, two critical post-fire management priorities. In Big Sur, high concentrations of mortality were common in mixed evergreen and redwood forests that burned during the 2008 wildfires (Meentemeyer et al. 2008; Metz et al. 2011). Fuels reduction treatments in these highly impacted stands—for example woody debris mastication or pile-and-burn treatments—may protect soil elemental pools, reduce fire-caused mortality, and improve recovery of forest floor elemental pools following fire. Given that the most significant losses occurred in stands with high levels of overstory mortality (Table 2), fuels treatments focused on the ground fuels generated by disease could reduce loss of nutrient pools and improve tree survival in the event of future wildfire. More broadly, the body of work on fire and sudden oak death interactions imply that spatial and temporal patterns of ground-fuel accumulation will influence fire impacts to soil element pools and fire-caused mortality in other insect or disease outbreaks, including those where outbreak does not directly alter fire intensity.

Redistribution of biomass by *P. ramorum*, such as fuels accumulation, is another example of ecological changes driven by invasive organisms (Ehrenfeld 2010). While it is recognized that the extent of invasion and degree of impact by invasive microbes has strong and potentially cryptic potential to shape ecosystem structure and function (Eviner and Likens 2008; Gandhi and Herms 2010), these invasions can produce disturbance interactions that compound the resulting changes and management challenges. It is unclear if the increased loss of soil element pools resulting from

fire-sudden oak death interactions will slow post-fire vegetation recovery, but this kind of effect could limit future forest carbon sequestration following wildfire. Across California, increasing forest carbon sequestration has been a major policy initiative (Stephens et al. 2009). Understanding if fire and outbreak influence fire-emissions and post-fire carbon sequestration at regional scales has implications for these policies, given that impacts of insects and pathogens are widespread in California forests. The straightforward fire–disease interactions to soil C and nutrients documented in this study suggest that these interactive effects are likely to occur for other outbreaks and at broad spatial scales.

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Author contribution statement RKM and DMR conceived and designed the plot monitoring network and long-term data acquisition. RCC designed the soil sampling and collected the data; RCC, RKM, and DMR analyzed the data. RCC, RKM, and DMR wrote the manuscript.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

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