

Tree mortality in the Sumber Pawon protected forest, Kediri, East Java

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Abstract. Tree mortality is a critical, yet episodic, process influencing forest structure, carbon cycling, and ecosystem dynamics. Understanding its causes is essential for effective forest management, particularly in protected areas like the Sumber Pawon Forest in Kediri, East Java, a key water conservation area. This study quantified tree mortality rates and identified causal factors using the ForestGEO Tree Mortality and Damage Protocol. Over a three-month monitoring period (January to March 2022) with monthly intervals, we recorded a high mortality rate of 6.47%. Trees with a diameter at breast height (DBH) of 10-20 cm were the most vulnerable. The primary modes of mortality were uprooting (U) and stem breakage (B), largely attributed to rainstorms. The presence of lianas and fungi further increased mortality risk by physically weakening trees. Additionally, evidence of selective logging by humans was identified as a contributing factor. These findings underscore that external mechanical forces, both natural and anthropogenic, are significant drivers of tree death in this protected forest, providing crucial insights for future conservation and management strategies.

1 Introduction

Forests play a vital role in global and regional ecosystem services, including carbon and water cycling, and protection against natural disasters [1]. Trees are fundamental to these functions, and their mortality directly impacts forest structure and dynamics. Increased tree mortality is linked to atmospheric carbon accumulation, as trees are primary carbon sinks [2]. Even slight changes in mortality rates can alter floristic composition, nutrient cycling, and biodiversity [3, 4, 5].

Despite its importance, the causes and rates of tree mortality, particularly in tropical forests, remain understudied due to its episodic nature and the complex interplay of internal (genetic) and external (environmental) factors [6]. A study in the Amazon found that thunderstorms were a major mortality agent, alongside biotic factors like lianas and fungi [4]. This highlights that drivers can be region-specific, necessitating localized studies. Sumber Pawon Forest in Kediri, East Java, is an essential ecosystem area, partly used for tourism, which introduces human activity. A 2020 census recorded 878 trees from 11 species in this forest [7], but no data exists on its tree mortality. This study aims to: 1) quantify the tree

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mortality rate, and 2) identify the key risk factors causing tree damage and mortality in Sumber Pawon Forest. The results serve as baseline data for predicting future ecosystem conditions and informing appropriate forest management.

2 Methods

2.1 Study Site and Period

The research was conducted from January to March 2022 in Sumber Pawon Forest, Tempurejo Village, Wates District, Kediri, East Java (7°53'15.14"S – 7°53'48.61"S and 112°8'42.90"E – 112°9'18.41"E). In this forest, we divided it into 14 units of sampling plots.

2.2 Data Collection and Analysis

Data was collected following the ForestGEO Tree Mortality and Damage Protocol [8]. For each tree, we recorded: Species and Diameter at Breast Height (DBH), Damage and Mortality Mode: e.g., uprooted (U), broken (B), standing dead (S), Biotic Agents: Liana infestation (categorized as L: >50% crown covered, or S: stem constriction) and presence of wood-degrading fungi, Crown Illumination (CI): Ranked from 1 (low light) to 5 (full light). Lianas: S (Smothered): >50% of tree crown covered by lianas, L (Limited): Lianas restrict the growth of the main axis. Fungi: √ (Present): Fungus affects the inner wood (under the bark), - (Absent): No fungus affecting the inner wood. Data were processed to calculate mortality rates and create simple graphs and tables for analysis.

3 Result and Discussion

3.1 Species Composition and Dominance

The study recorded a total of 170 individual trees across 14 plots, encompassing 11 species. The information regarding this is included in this table. Based on Table 1, a critical finding was the overwhelming dominance of introduced species, which constituted 78.23% of all individuals. Notably, invasive species alone accounted for 75.29% of the total population, primarily represented by *Castilla elastica* (27.06%), *Tabernaemontana macrocarpa* (26.47%), and *Ficus elastica* (15.88%). In contrast, native species were disproportionately low in abundance, making up only 21.77% of individuals, with the highest relative density for a single native species being just 10% (*Alstonia scholaris*). This pronounced dominance by a few invasive species has profound implications for the structure and function of Sumber Pawon Forest. Invasive species are known to threaten native biodiversity by altering competitive hierarchies, disrupting nutrient cycles, and ultimately displacing native populations [9]. The low relative abundance of native species (e.g., *Pterospermum javanicum* at 2.94%, *Swietenia mahagoni* at 1.76%) suggests a potential erosion of the forest's native genetic reservoir and a shift towards a homogenized ecosystem.

This shift is particularly concerning given the forest's designated role as a water conservation area. Different tree species possess varying root architectures, transpiration rates, and influences on soil hydrology. The replacement of a diverse native community with a monoculture of invasive species could alter the forest's hydrological functions. Key processes like infiltration, groundwater recharge, and the regulation of evapotranspiration may be modified, potentially impacting the long-term stability and quality of the water resources the forest is meant to protect [10].

Table 1. Species composition

No.	Species	Family	Abundance	% domination
1.	<i>Castilla elastica</i>	Moraceae	46	27,06
2.	<i>Ficus elastica</i>	Moraceae	27	15,88
3.	<i>Ficus septica</i>	Moraceae	1	0,59
4.	<i>Tabernaemontana macrocarpa</i>	Apocynaceae	45	26,47
5.	<i>Alstonia scholaris</i>	Apocynaceae	17	10
6.	<i>Tectona grandis</i>	Lamiaceae	1	0,59
7.	<i>Samanea saman</i>	Fabaceae	10	5,88
8.	<i>Hibiscus tiliaceus</i>	Malvaceae	10	5,88
9.	<i>Theobroma cacao</i>	Malvaceae	4	2,35
10.	<i>Pterospermum javanicum</i>	Malvaceae	5	2,94
11.	<i>Swietenia mahagoni</i>	Meliaceae	3	1,76
Total abundance			170	100

3.2 Elevated Tree Mortality Rate and Its Implications

Over the three-month monitoring period, a total of 11 trees were recorded as dead out of the 170 individuals surveyed, resulting in a tree mortality rate of 6.47%. This rate is strikingly high for a short-term study. To provide context, a comprehensive study in the Amazon basin reported an annual mortality rate of 1.15% [5]. While direct comparison requires caution due to differences in forest type, scale, and monitoring duration, the mortality rate observed in Sumber Pawon is notably elevated. This suggests that the forest is undergoing a significant dynamic pulse, potentially driven by acute environmental stressors or inherent ecosystem instability. Such a high rate of loss, if sustained, could have substantial implications for the forest's carbon storage capacity, structural complexity, and long-term viability as a water conservation area [1, 3]

Mortality was not random but showed distinct patterns across tree families. The highest mortality was concentrated in two families: Apocynaceae: Accounted for the highest number of deaths, with 5 individuals of *Tabernaemontana macrocarpa* succumbing. Moraceae: Suffered significant losses, with 4 individuals of *Castilla elastica* and 1 individual of *Ficus elastica* dying. A single individual of *Swietenia mahagoni* (Meliaceae) was also recorded as dead. The concentration of mortality in these families, particularly the invasive *T. macrocarpa* and *C. elastica*, raises critical questions. It is possible that these species, despite their numerical dominance, possess intrinsic traits—such as wood density, root architecture, or high transpiration rates that make them more vulnerable to the prevailing abiotic stresses in the forest, such as strong winds or soil moisture fluctuations [4]. Alternatively, their high mortality could be a result of density-dependent effects, where pathogens or pests spread more easily through a dense population of conspecifics.

The distribution of mortality modes provides crucial insight into the causative agents. The 11 dead trees were categorized as either uprooted (U; 6 trees) or suffering from stem breakage (B; 5 trees). The prevalence of these two modes is a strong indicator that external mechanical forces were the principal direct cause of mortality [6,8]. Uprooting is typically associated with soil saturation and strong winds that overcome the root system's anchorage. Stem breakage can result from the same forces, particularly on trees with pre-existing structural weaknesses.

3.3 Size-Dependent Mortality and Understory Vulnerability

Analysis of mortality distribution across diameter classes revealed a clear pattern of size-dependent vulnerability. The highest number of mortalities (3 individuals) was recorded in

the 10-20 cm Diameter at Breast Height (DBH) class (Fig. 1). This finding aligns with established global patterns in forest ecology, where smaller trees consistently exhibit higher mortality rates than their larger, canopy-forming counterparts [5, 14].

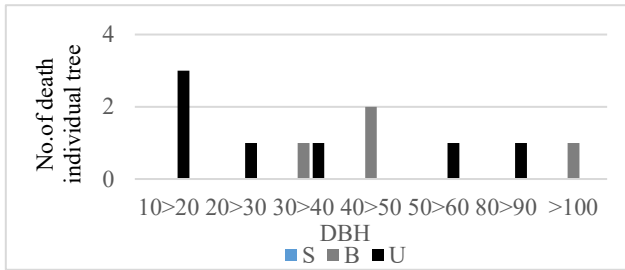


Fig. 1. Proportion of Dead Tree Individuals Categorized by DBH Class and Stand Mode

This disparity in survival can be attributed to a combination of physiological and ecological constraints. Smaller trees, typically residing in the forest understory, operate under a significant competitive disadvantage. They are often suppressed by larger trees, resulting in chronic light limitation (as supported by our Crown Illumination data) and reduced access to below-ground resources like water and nutrients [11]. This "sub-canopy suppression" leads to lower carbon assimilation and growth, making them more susceptible to resource stress.

Furthermore, their smaller structural dimensions render them more vulnerable to a wider range of mortality agents. They are more likely to be physically damaged by falling debris from larger trees, trampled by fauna, or succumb to abiotic stresses like short-term droughts or waterlogging that a larger, more established root system could withstand. This aligns with the concept of "stochastic mortality," where a multitude of small-scale, random events disproportionately affect smaller individuals [4]. The high mortality in this critical recruitment size class poses a threat to the forest's long-term regenerative capacity, potentially creating a demographic bottleneck that could alter future forest structure.

Furthermore, light is a fundamental abiotic factor limiting the physiological processes of trees, particularly in dense tropical forests where competition for sunlight is intense [6, 12]. The Crown Illumination (CI) index quantifies the proportion of a tree's crown exposed to direct sunlight, serving as a key indicator of its competitive position for this critical resource.

Our results revealed a dynamic shift in light availability over the three-month study period. The majority of trees were consistently categorized in the high-illumination classes (CI 4 and CI 5). A notable temporal pattern was observed: the proportion of trees in CI 4 and CI 5 increased in February, coinciding with a decrease in CI 3. This was followed by a recovery in CI 3 and a drop in CI 2 in March (Fig.2).

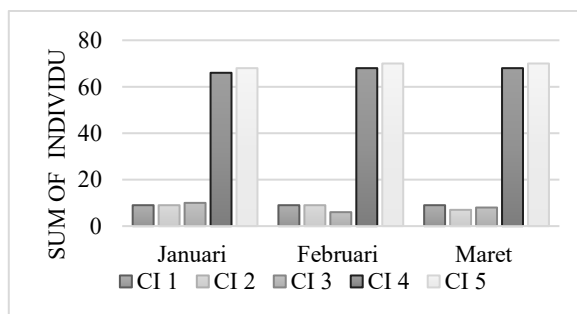


Fig. 2. Number of Individual Trees in Each Crown Illumination (CI) Category

These fluctuations are not random but can be directly linked to the mortality-induced formation of canopy gaps. The death and subsequent falling of 11 trees, particularly those that were uprooted or suffered stem breakage, opened gaps in the forest canopy [13]. This sudden increase in light penetration initially boosted the illumination for surrounding trees, shifting many from CI 3 to higher categories (CI 4/5) in February. The subsequent changes in March may reflect a more complex redistribution of light or the initial growth responses of surviving trees and understory vegetation to the new light regime.

This finding underscores that tree mortality is not merely an endpoint but a catalyst for forest regeneration and structural change. The creation of these gaps is a critical ecological process. The increased light availability can enhance the photosynthetic rates and biomass accumulation of suppressed trees, potentially releasing them from light limitation and altering the competitive hierarchy within the forest [13]. This dynamic interplay between mortality and light environment highlights the natural cycle of disturbance and recovery that shapes the structure and composition of tropical forests.

3.4 The Interplay of Biotic and Abiotic Factors in Mortality Modes

The mortality events were categorized into two primary mechanical modes: uprooting (U), accounting for 6 trees, and stem breakage (B), accounting for 5 trees. The prevalence of these modes points to strong external mechanical forces as the immediate cause of death. However, a deeper analysis reveals a complex interplay between predisposing biotic factors and triggering abiotic events.

3.4.1 Biotic Factors as Predisposing Agents

Biotic agents, such as lianas and fungi, are significant contributors to tree stress and mortality in tropical forests. Our assessment of these factors followed a standardized protocol where liana infestation was categorized as either Code L (liana coverage >50% of the tree crown) or Code S (liana constricting the main stem), and fungal presence was noted if it affected the inner wood [14].

Liana Infestation: The role of biotic agents was significant, with 13 trees (7.6% of the surveyed population) supporting liana infestations. A critical observation was that the vast majority of these infested trees (12 out of 13) were still alive at the study's conclusion. This indicates that lianas rarely act as a sole, immediate killer but rather as a chronic stressor that predisposes trees to mortality.

The nature of this stress was twofold, as defined by our infestation codes: **Crown Competition (Code L - 77% of cases):** The heavy coverage of tree crowns by lianas (>50%) directly intercepts light, reducing the host's photosynthetic capacity and carbon gain. This places the tree in a state of energy deficit. **Structural and Vascular Damage (Code S - 23% of cases):** Lianas constricting the main stem can cause direct physical damage to the xylem, impairing water transport and mechanically weakening the trunk, which can lead to reduced growth and increased vulnerability to breakage [14].

Below ground, lianas are equally potent competitors, often outperforming trees in the uptake of water and soil nutrients due to their highly efficient root and vascular systems [14]. This combination of above- and below-ground competition creates a "slow-acting stress," gradually reducing the tree's vigor, growth, and mechanical strength. Therefore, while a liana-infested tree may appear healthy, it is often critically weakened. When an external abiotic "pulse" event occurs—such as the strong winds and saturated soils identified as primary mortality drivers—these pre-stressed trees are the most likely to succumb, explaining the observed modes of uprooting (U) and stem breakage (B). This establishes liana

infestation not as the proximate cause of death, but as a major underlying risk factor that significantly increases tree vulnerability.

Fungal Pathogens: The assessment of fungal infestation was strictly limited to wood-degrading fungi that visibly affected the inner wood of the tree, as superficial growth on the bark was excluded from the dataset. This focused approach allowed us to identify pathogens that directly compromise tree structural integrity.

Our survey identified the presence of the pathogenic fungus *Ganoderma* sp. on a single individual of *Ficus elastica*, which was recorded as dead. While only one case was confirmed, the role of such pathogens is disproportionately significant. *Ganoderma* is a white-rot fungus that secretes enzymes to break down lignin and cellulose, the fundamental structural components of wood [15]. This process causes internal decay, hollowing the trunk and severely reducing its mechanical strength without necessarily producing immediate external symptoms.

The tree infected with *Ganoderma* sp. ultimately died from stem breakage (B), a finding that strongly suggests a causal relationship. The fungus acted as a chronic, internal "press" that progressively weakened the stem's core. This latent deterioration likely primed the tree for failure, meaning that the abiotic trigger required to cause breakage—such as a moderate wind gust or the added weight of a rain-saturated crown—would be far less severe than for a healthy tree. This case provides a clear example of how a biotic agent can interact with physical forces to determine the specific mode of tree death. Although not widespread in our dataset, the presence of such a virulent pathogen highlights an important mechanism of mortality, particularly for explaining the death of otherwise large-stemmed individuals that succumb to stem snap.

3.4.2 Abiotic Factors as Triggering Events

The distribution of mortality modes, with 6 trees uprooted (U) and 5 trees broken (B), provides critical insight into the causative mechanisms. Further analysis of the broken trees revealed two distinct pathways: a gradual S→B pathway (standing dead to broken) and an acute B→B pathway (direct breakage) [14]. This distinction allows us to propose a coherent "press-pulse" model to explain tree mortality in Sumber Pawon Forest.

The S→B Pathway: Chronic Press Leading to Collapse. Five trees, including *Ficus elastica* and *Tabernaemontana macrocarpa*, followed the S→B pathway. This trajectory indicates that mortality was initiated by a gradual, internal "press." These trees likely experienced a prolonged period of decline, transitioning to a standing dead state (S) before their eventual collapse (B). This decline can be attributed to the cumulative impact of the biotic stressors identified earlier. For instance, the *F. elastica* (Plot 7) was co-infected with the pathogenic fungus *Ganoderma* sp., which causes internal wood decay [15], and suffered from liana stem constriction (Code S), which damages vascular tissues [14]. These factors, combined with light competition suppressing photosynthesis, progressively weakened the structural integrity and physiological health of these trees. The final breakage was merely the culmination of this chronic weakening; the trees were already dead or dying and eventually succumbed to their own weight or a minor physical trigger.

The B→B and Uprooting (U) Pathways: Acute Pulses as Proximate Causes. In contrast, the B→B pathway and all cases of uprooting (U) represent mortality driven by an acute external "pulse." The B→B pathway suggests a single, catastrophic event like a windstorm or lightning strike that instantly broke a previously living tree [14]. The high incidence of uprooting (U) strongly implicates severe weather, particularly the intense rainstorms common during the study period. Saturated soil loses its anchoring strength, and when combined with strong winds, it can lead to the mechanical failure of the root-soil plate, toppling even apparently healthy trees [8].

Statistical analysis revealed an exceptionally strong and significant positive correlation between the occurrence of rainstorms and tree mortality in the B (broken) and U (uprooted) modes. The Pearson correlation coefficient was $r = 0.999$, with a significance value of $p < 0.05$. This result provides robust quantitative evidence that rainstorms are a primary and dominant abiotic driver of the observed tree mortality in Sumber Pawon Forest. The near-perfect correlation underscores that the episodic mortality event recorded during the three-month study was overwhelmingly linked to severe weather conditions.

3.5 Synthesis of Mortality Pathways: A Press-Pulse Model of Tree Death in a Degraded Forest

The elevated tree mortality observed in Sumber Pawon Forest is most coherently explained by a "press-pulse" model, which integrates the chronic, underlying stresses with the acute, triggering events that culminated in tree death. This model provides a robust framework for understanding the interplay of multiple factors that led to the recorded 6.47% mortality rate over a mere three-month period.

The chronic "press" in this system was constituted by a suite of biotic and competitive stressors that progressively eroded tree health and structural integrity over time. This press is clearly evidenced by our data: a significant portion of the stand (7.6% of surveyed trees) was afflicted by liana infestation. For the majority of these, the infestation was severe (Code L), leading to significant crown coverage that directly reduced photosynthetic capacity and carbon assimilation. In other cases (Code S), lianas physically constrict stems, damaging vascular tissues and impeding growth. Simultaneously, the presence of aggressive wood-decaying pathogens like *Ganoderma* sp. functioned as a potent internal press. This fungus, identified on a dead *Ficus elastica*, secretes enzymes that break down the structural polymers lignin and cellulose, leading to internal rot that critically weakens the trunk long before external failure is apparent. Furthermore, the stand's composition, dominated by invasive species and characterized by intense competition for light, created a background of physiological stress that left many trees, particularly smaller individuals in the 10-20 cm DBH class, in a perpetually weakened state. This chronic press did not immediately kill most trees, but it placed them in a precarious state of low vigor and high vulnerability.

The acute "pulse" was delivered by the abiotic forces of the seasonal environment, specifically the intense rainstorms that occurred during the January-March monitoring window. This pulse is directly reflected in the mortality modes: uprooting (U) and stem breakage (B). The uprooting of six trees is a classic signature of soil saturation and high winds, where the root-soil plate fails. The stem breakage observed in five trees represents a direct mechanical failure of the trunk. This pulse was the proximate, final cause of death.

Crucially, these pathways were not mutually exclusive; their interaction is key to explaining the high mortality. A tree that was already critically weakened by the chronic press of liana competition and internal fungal decay—essentially a candidate for the gradual S→B pathway—would have a dramatically lowered threshold for failure. What would be a non-lethal gust of wind for a healthy tree could become the final, catastrophic force that snaps the pre-weakened stem (B→B pathway) or tears its compromised root system from the ground (U pathway). Therefore, the observed mortality was not solely due to an unusually severe storm, nor was it purely a result of internal decay. Instead, it was the consequence of a pre-existing "cohort of vulnerable trees," created by chronic biotic stresses, being exposed to a perfectly timed abiotic pulse. This synergy between the gradual press and the instantaneous pulse efficiently culled these vulnerable individuals, leading to the significant short-term mortality recorded in this study and highlighting the compounded vulnerability of forests facing multiple stressors.

4 Conclusion

This study recorded a high tree mortality rate (6.47%) over three months in Sumber Pawon Forest. Small-diameter trees (DBH 10-20 cm) were the most vulnerable. Mortality was primarily driven by external mechanical forces (uprooting and stem breakage), likely associated with episodic storms. Biotic agents, specifically liana infestation and fungal pathogens, acted as significant contributing factors by physically weakening trees and compromising their health. These findings highlight the interaction between natural disturbances and biological stressors in driving tree mortality. For effective conservation of this vital water catchment area, management strategies should consider these risk factors to mitigate future tree loss.

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Lastly, I realize that this research is far from perfect. Therefore, constructive criticism and suggestions are highly appreciated for future improvements. I hope this work can provide useful insights and serve as a reference for future research related to tree mortality and risk factor for forest conservation.

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