Novel dendrochronological modelling demonstrates that decades of reduced stem growth predispose trees to Acute Oak Decline

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Decades of reduced stem growth predispose trees to Acute Oak Decline

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Abstract

Acute Oak Decline (AOD) is devastating thousands of native oaks, *Quercus robur* and *Q. petraea,* in the UK and Europe. The syndrome is principally caused by multiple, interacting bacterial species that degrade inner bark tissues. The level of host predisposition required prior to AOD infection is unknown. The two spotted oak buprestid *Agrilus biguttatus* is strongly associated with AOD, although its role

remains unclear. To investigate the nature of predisposition in AOD, to explore the role of the beetle in the syndrome, and to examine growth trends after AOD onset, the stem growth of 243 trees with a range of severity of AOD symptoms was analysed at five sites in England. Novel mixed effects dendrochronological modelling methods were developed. The presence/absence of *A. biguttatus* exit holes in the trees was not specifically linked with reduced stem growth, nor was there evidence of further reductions in growth after the onset of AOD symptoms. Instead, trees with long-term AOD symptoms show significantly reduced growth compared to asymptomatic trees from as far back as the 1930s, following a period of widespread decline in English Oaks. These results suggest that a cohort of oak trees across Britain was permanently damaged in the 1930s and predisposed to develop AOD symptoms decades later. Additionally, correlations with climatic variables suggest that diseased trees are less able to take advantage of good growing conditions in the spring and autumn. This study sheds light on how historical episodes of stress may impact the future resilience of oaks to disturbance, and supports the use of dendrochronological modelling as a technique to study the underlying health status of oak tree populations, and to better understand tree decline episodes.

Keywords: *Quercus robur*, Acute Oak Decline, *Agrilus biguttatus*, Dendrochronology, Predisposition, Ecological modelling

Introduction

Oaks are keystone forest species across the northern hemisphere, and their decline is a subject of increasing international concern [1]. Trees, inherently susceptible due to long-term "predisposing" factors, decline when prolonged, repeated, or severe abiotic and / or biotic stresses reduce their carbon budgets, leaving them unable to mount adequate defences against secondary pests and pathogens which then decrease the vigour of the tree [2–7].

Acute Oak Decline (AOD), a specific type of oak decline, characterised by fluid exudation ('stem bleeding') from necrotic inner bark lesions, was recently described in the UK, and now affects many thousands of native oaks in England (*Quercus robur* L. and *Q. petraea* (Matt.) Liebl.), of which approximately 1.3% die each year [8,9]. Similar decline-diseases of oaks are being investigated in Europe, the Middle East and North America [10]. Multiple, interacting bacterial species damage inner bark tissues, creating necrotic lesions and cavities extending into the sapwood; the damage may be extensive, covering much of the stem [8–11]. Larval galleries of the two-spotted oak buprestid *Agrilus biguttatus* (Fabr.) are almost always found in phloem tissue adjacent to AOD lesions, and larvae appear to be capable of spreading the bacteria along their galleries, potentially initiating new lesions [10,12,13]. AOD does not necessarily result in tree mortality; many affected trees are able to isolate lesions and larval galleries with callus-like tissue and enter a state of remission [12].

The degree of predisposition required before colonisation by the AOD bacteria and *A. biguttatus* remains unknown. At monitored sites, stem bleeds were more common, and more numerous, on trees with poorer crowns [12]. At the same sites, new beetle exit holes, formed by emerging adults, were more likely to appear on previously symptomatic trees, suggesting further weakening was necessary for complete development by the beetle [13]. The lifecycle of *A. biguttatus* is, however, likely to take at least two years in the UK, and eggs may be laid on

77 trees unsuitable for complete larval development; it is thus difficult to determine the
78 chronology of bacterial and beetle attack from external symptoms[13–15].
79
80 Predisposition may occur at the individual tree or stand scale. At the individual scale,
81 predisposing factors may include increasing tree age, severe or successive defoliation by
82 biotic agents, and genotypic variation in resilience [4,5,16,17]. At the stand scale, poor
83 management may play a role, while commonly cited environmental factors inciting oak
84 decline include drought, fluctuating water tables, and late frost [4,6,18,19]. A predisposition model
85 correlating environmental variables with AOD found the syndrome occurs at warmer sites,
86 with low rainfall, high levels of nitrogen and base cation deposition, and low levels of sulphur
87 deposition [20]. The UK distribution of *A. biguttatus* is also limited to warmer areas in England;
88 thus if the beetle is an essential component of AOD, the syndrome must also be limited to
89 these areas [15,21].
90
91 Dendrochronology, or the systematic cross-dating of tree rings, allows the study of forest
92 disturbances that influence tree growth [22]. Tree stem growth may be a useful indicator of
93 vitality because the growth of leaves, roots and buds, and allocations to carbohydrate
94 storage, take priority over stem growth. At times when stress factors reduce a tree's carbon
95 budget, its less important processes are typically limited first [4,23]. Predisposition may be
96 represented by reduced growth. Many studies of oaks have shown reduced annual stem
97 increment in the years before decline onset or tree death, as compared with the growth of
98 healthy trees [19,24–30]. Several studies have also shown greater variability in the annual ring
99 widths of declining oaks, suggesting predisposed trees have decreased stability in response
100 to climatic stress [31,32]. Dendrochronology has also revealed differences in stem growth
101 between oak trees with different levels of colonisation by stem-boring beetles [33,34]. The
102 effects of AOD upon radial growth are unknown.
103

This study aims to examine both recent and long-term stem growth records of trees with and without AOD symptoms, in order to determine whether symptomatic trees show evidence of predisposition to AOD in the form of reduced or declining growth, an important knowledge gap that may influence management of the syndrome. Here we compare the growth of symptomatic trees with and without the exit holes of adult *A. biguttatus,* to analyse the relationship of the beetle with observed stem growth patterns. Finally, by comparing recent growth trends in trees at various stages of decline by AOD, including those in remission or recovery, we determine the impact of AOD on tree growth.

Methods

Study trees were selected from woodlands at five sites with AOD (confirmed through tree symptoms and isolation of AOD bacteria (Table SA1)). Four of the sites, Attingham Park, Langdale Wood, Hatchlands Park, and Richmond Park, had been monitored annually for AOD symptoms since 2009 or 2010. Monitoring included annual counts of active AOD lesions and *A. biguttatus* exit holes visible from ground level (up to approximately 3m), annual classifications of crown condition to an ordinal scale (based on % canopy loss and branch dieback), and measurements of diameter at breast height (DBH, 1.3m) [12]. At Madingley Wood, where AOD symptoms were observed in 2009-2010 (K. Russell, pers. comm.), all oaks were surveyed in 2014 for occluded, active and recent AOD lesions, and *A. biguttatus* exit holes.

Trees were assigned to one of four AOD categories according to their history of lesion symptoms (Table SA2): ‘long-term symptoms’, ‘new symptoms’, ‘remission’, and ‘asymptomatic control’ (= ‘four level AOD category’), and further sub-categorised according to the presence/absence of *A. biguttatus* exit holes on the symptomatic trees (= ‘seven level AOD category’). Trees with ‘long-term symptoms’ had at least one active lesion at the start of the monitoring period. Trees with ‘new symptoms’ developed at least one active lesion

after monitoring began. Trees with long-term and new symptoms had active symptoms in the year during which they were cored, although they may have been in remission (e.g. no active lesions seen) for one or more years during the monitoring period. Trees in 'remission' had at least one active lesion during the monitoring period, but showed no active lesions for at least two years before coring. At Madingley Wood, due to insufficient monitoring history, all trees with active lesions were assigned to the long-term symptoms category because they appeared to be in an advanced state of decline. At each site, up to 14 trees were selected within each of the four-level AOD categories, often comprising almost all available trees. Trees with potentially confounding conditions (unusual growth, extensive mechanical or structural damage, symptoms of infection by root pathogens) were excluded from the sample. Where a choice of trees was available, asymptomatic trees were selected by proximity to symptomatic trees, and similarity in age. Due to the preferences of site managers, the largest asymptomatic trees were avoided. Before coring, diameter at breast height (DBH) was re-measured for each tree.

Trees were cored in May 2015 (Hatchlands Park, Richmond Park, and Madingley Wood) and January 2017 (Attingham Park and Langdale Wood). Increment cores were collected using 5.15mm Haglof increment borers, 400mm in length. Two cores were taken from each tree at breast height (1.3m), preferentially from the east and west aspects, except where the tree was leaning, on a slope, or adjacent to a footpath, in which case aspects were chosen to minimise these influences. If deformation was present at breast height, cores were taken up to 50cm below or above. AOD lesions were avoided. To minimise the risk of between-tree spread of AOD bacteria, increment borers were thoroughly cleaned with 70% IMS after each use.

Cores were transported to the laboratory in labelled paper straws and air-dried between sheets of corrugated cardboard. One core per tree was prepared for measurement, with the easterly or northerly cores chosen preferentially. Cores were mounted on wooden blocks

and sanded using progressively finer papers, up to 1000 grit, with a handheld orbital Sander (Makita UK, Ltd.). Cores were scanned at 2,400 dpi using an Epson Perfection 4990 photo scanner (Seiko Epson Corporation, Japan) and annual ring widths were digitally measured to 0.01mm in CooRecorder 7.8 [35]. Incomplete rings from the current year were not measured. If the first core was damaged, or its most recent rings comprised irregular callus-like tissue, or proved difficult to resolve due to very narrow rings, then the second core was prepared. Ring width series were cross-dated using a combination of visual assessments and comparisons of correlation coefficients (a measure of the covariance between the normalised, overlapping portion of the chronologies) and T-scores (based on the correlation coefficient, but weighted to account for the length of the overlapping segment) in CDendro 7.8 [35]. To determine and verify the year of each annual ring [22], trees within individual sites were cross-dated with each other first. Mean chronologies from the five study sites were then compared to each other, and to published ring width series from 18 sites in England[36,37].

Statistical Analysis

Analysis of long-term growth trends

All analyses were carried out in R [38], with graphics produced using ggplot2 [39]. Ring widths were natural-log transformed, as initial visualisations showed an increase in variance with increasing ring width at all sites. Data were combined across the five sites, and year was centred, based on the range across all individual trees (the year 1960 was used as the zero point, as this fell within the time series of all trees); this aided model fitting and reduced correlation between slopes/intercepts.

Statistical analysis focussed on producing a single model to replace the multiple stage process frequently used in dendrochronology [40] by using linear mixed effects modelling [41]

with the basic structure of tree ring width being determined by the fixed effects of final tree age, aspect and the interaction of year and location, with a tree-specific random effect applied to year.

Appropriate detrending was conducted using a range of n-order polynomials applied to year data (both fixed and random components) to allow for location-level and tree-level turning points; biologically, these turning points were likely to coincide with active management within each compartment/site (e.g. thinning); as such, the model included compartment-specific location fixed effects to account for these changes. The most efficient n-order polynomial was determined using Akaike Information Criteria applied to the maximum likelihood fit.

Temporal autocorrelation was accounted for in the model using a range of corARMA (autoregressive moving average) models (AR regresses the current ring widths with its previous n values; MA models the error term of the current ring widths based on previous n error terms). Analysis of the (partial) autocorrelation function applied to the model fit indicated that up to two previous years needed to be included within the corARMA structure. All combinations of corARMA structure (up to 2 time points) were therefore applied to the appropriate n- order polynomial and the most efficient model again determined using Akaike's Information Criteria (AIC) applied to the maximum likelihood fits.

The potential effects of AOD type and beetle exit holes were included within the model. Firstly, models were fit using the seven treatment definition (long-term symptoms ± exit holes, new symptoms ± exit holes, remission ± exit holes, asymptomatic) as a main effect, interaction with year (linear coefficient only) and interaction with the n-order polynomial applied to year. The most efficient model was then further broken down by simplifying the seven treatments to the four treatment model, *Agrilus*-only model (with/without beetle exit

holes) and four treatment plus *Agrilus* with most efficient model determined using AIC applied to the maximum likelihood fits.

Having determined most efficient model , predicted tree-level outputs were used to visualise differences across treatments, with further post-hoc tests conducted on fixed effects using marginal means to explore significant differences, using Tukey's HSD for multiple comparison contrast p value estimates; appropriate weighting was applied at the compartment level such that the marginal means were weighted equally by site [42].

Analysis of recent growth trends

Having determined the most efficient model for long-term growth trends, the same model was re-run but restricting the data sets to time points earlier than, and up to 2004 only. This model was then used to forecast tree-specific ring widths up to 2017, to compare with the actual ring width data. To meet model normality assumptions, differences were calculated as ln ((actual ring widths - 2004 forecast ring widths)), baselined on the minimum difference, then back-transformed to arithmetic ring width differences for presentation.

Analysis of climatic drivers of growth differences

In order to determine whether climatic variables were driving growth differences between asymptomatic and symptomatic trees, correlations were examined between the ring widths of asymptomatic and long-term symptomatic trees; median tree ring width differences (long-term symptomatic minus asymptomatic) were determined for these two groups first by compartment, then by site and finally by year (i.e. equal weighting to each site). Year-specific median tree ring width differences were then correlated against historical (post 1873) daily precipitation and temperature data for the central England area (defined as the geographic area bounded by the cities of Lancashire, London, and Bristol), averaged by

season [43]. The influence of very warm years on growth within the four AOD categories was also tested by comparing growth in the year before and during sudden changes in temperature, defined by an increase of > 1˚C across at least three seasons (1880, 1889, 1957, 1989, 2014), versus 20 randomly selected years post 1873.

Results

The full dataset, comprising annual ring data from 243 trees, gives a robust view of the historical growth of trees affected by AOD at five sites in England. Table SA3 summarises the ring width data by four-level AOD category and site. Table SA4 shows between-site chronology comparisons (correlation coefficients (CorrC) and T-scores (TT)).

Analysis of long-term growth trends

The most efficient model was a fourth order polynomial (see Figure SA1), with a corARMA(2,1) temporal autocorrelation structure and an interaction between the linear year coefficient and the AOD category. The fit of the seven treatment model (AIC = 21,026) was similar to the four treatment model (AIC =21,026), and the addition of an *Agrilus* main effect with the four treatment model led to a less efficient model (AIC = 21,037), therefore the weight of evidence did not support the inclusion of *Agrilus* as a factor within the model, and the simpler four treatment model was chosen as best fit (see Table SA5 for full results). ANOVA applied to fixed effects supports the AIC results, with significant main effect and interaction for the four treatment model (see Table SA6).

The significant interaction between four factor AOD type and the linear temperature coefficient indicates that the rate of decline in ring width size was significantly different across these four groups. Plotting the tree-level predictions against the raw data across site

and AOD type (see Figure SA2, SA3) shows the predictions to provide a reasonable fit to the data, with the largest differences across types being observed between asymptomatic and long-term symptomatic trees (see Figure 1).

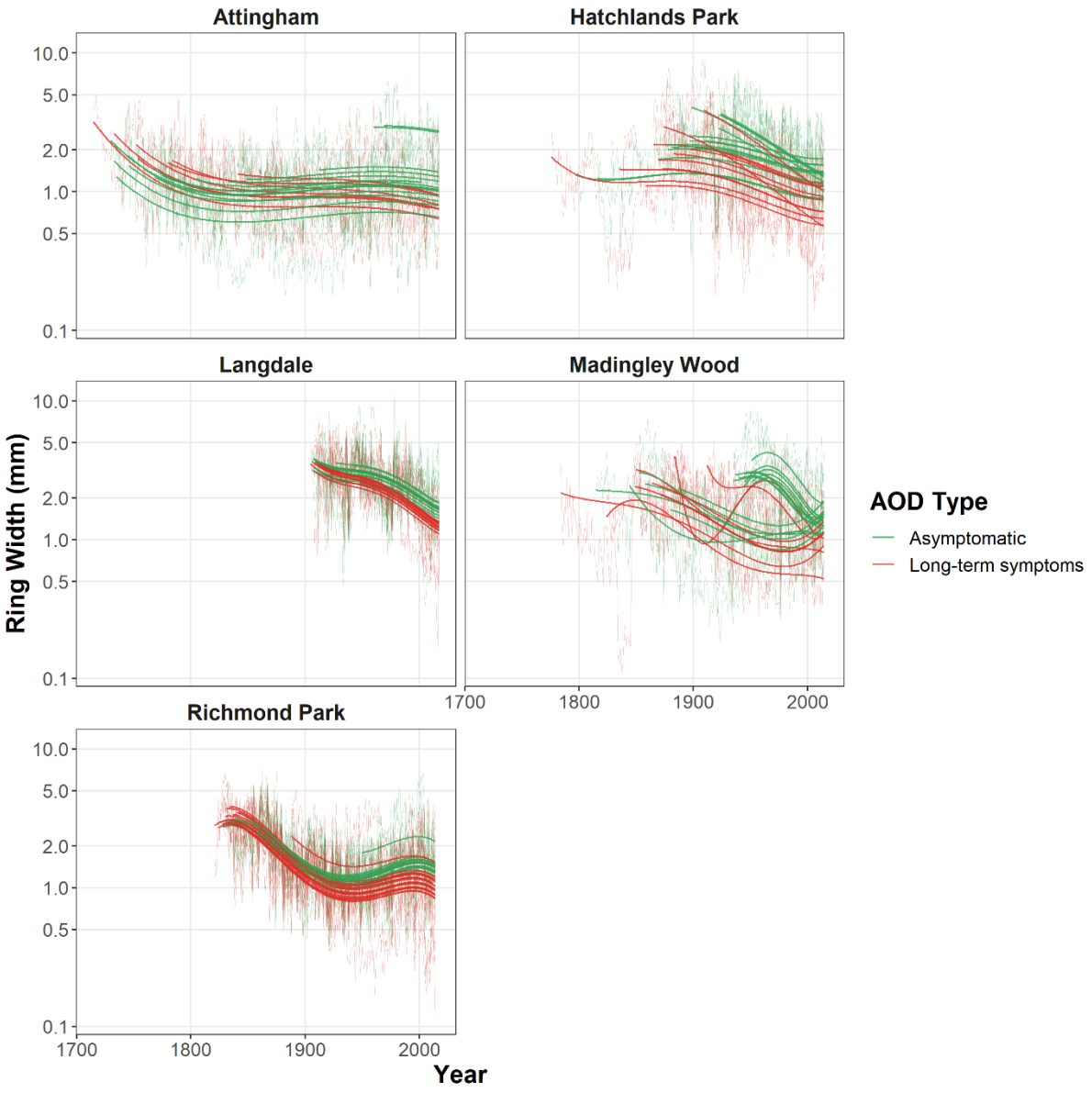

Figure 1. Raw tree ring width data (thin lines) overlaid with most efficient model by tree (thick lines; asymptomatic and long-term symptomatic only). Note log10 scaling on y axis.

Figure 1 shows variability across sites and compartments, but a generally consistent pattern can be seen with trees showing similar ring widths early in their lives and greater divergence between symptom types developing over time. This can best be displayed by analysing contrasts between marginal means, corrected for age, site and aspect. Figure 2 shows these

contrasts for remission, new symptom and long-term symptomatic trees, with the dashed horizontal line being equivalent to the predicted growth of asymptomatic trees. The general trend in all three categories in Figure 2 is one of decline versus asymptomatic trees, but only long-term symptomatic trees show a significant difference in ring widths versus asymptomatic trees - from 1931 onwards.

The results of the model are matched by DBH measurements; when DBH was corrected for age, as estimated from the core ring width counts and percentage of the tree's radius sampled by the core, there was a significant effect of the four level AOD type on tree size ($F = 4.8$, $df = 3$, $p = 0.003$), but not of site ($F = 1.6$, $df = 2$, $p = 0.20$), with post hoc tests indicating that trees with long-term symptoms were smaller in girth than asymptomatic control trees, and trees with new symptoms and in remission were intermediate, but not significantly different from either (least-square mean ± SE for long-term symptoms, new symptoms, remission, asymptomatic control = 0.32 ± 0.01, 0.33 ± 0.02, 0.35 ± 0.01, 0.39 ± 0.02, respectively).

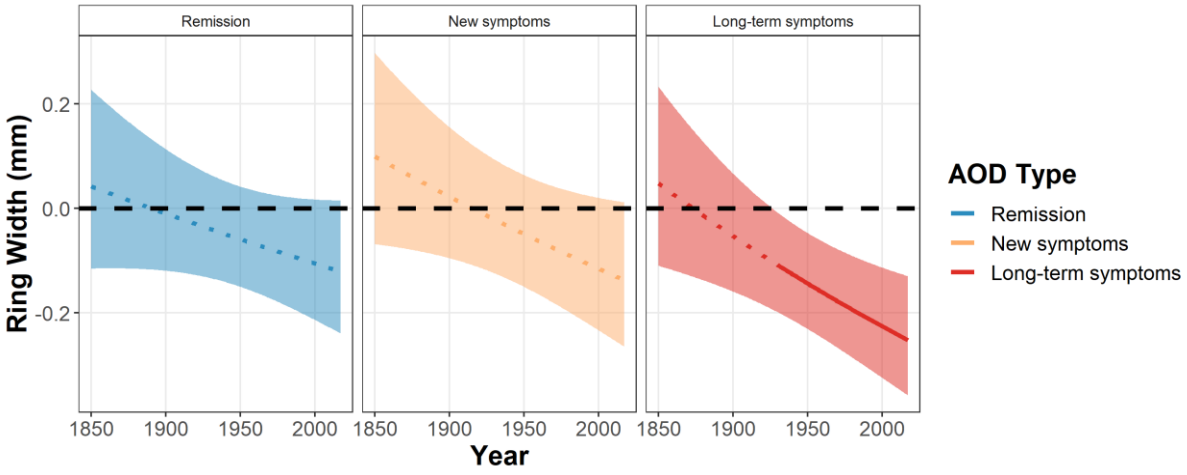

Figure 2. Estimated marginal mean contrasts between asymptomatic trees (dashed horizontal line) and other tree categories. Dotted lines indicate non-significant differences (corrected for multiple comparisons); solid lines indicate significant difference ($p<0.05$). Error bands show 95% confidence intervals for contrasts, corrected for multiple comparisons.

Analysis of recent growth trends

Figure 3 shows the comparison between the actual ring widths and those predicted using the projected linear mixed effects model limited to data from 2004 and earlier. Figure 3 did not indicate any marked deviation between actual ring widths and those predicted from 2004 and earlier. This was supported by tree-level analysis of these absolute differences, which showed no difference by AOD type ($F_{3,199}$ = 0.73, p = 0.54). This provides further evidence for the driver of differences in ring widths not only having occurred over the recent past (i.e. ~10 years) but over a longer time period.

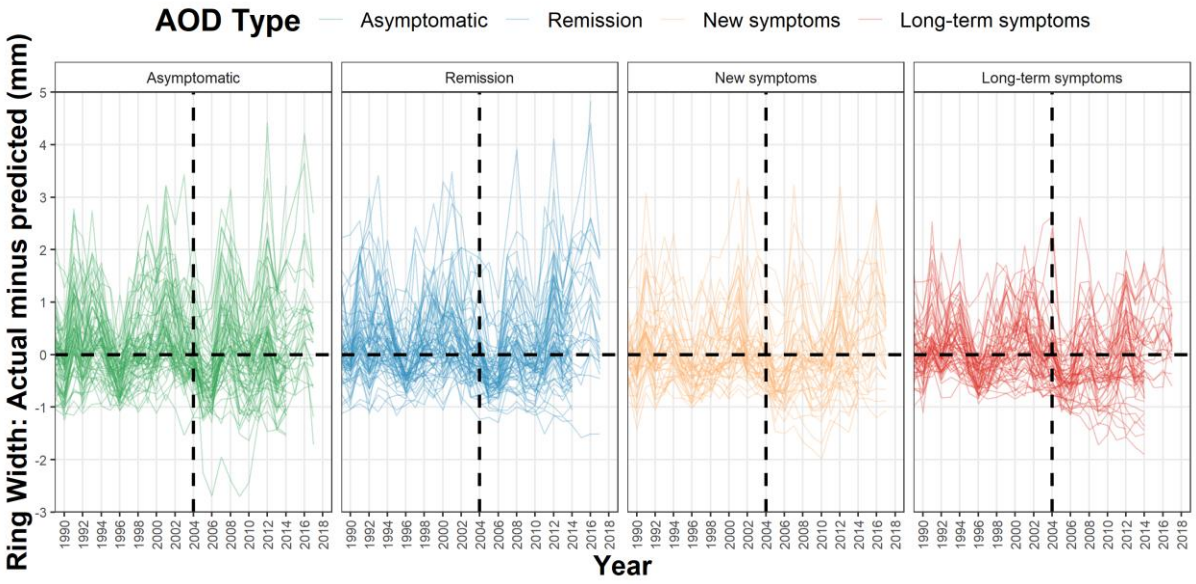

Figure 3. Comparison between the projected vs actual ring width data from 2004, by AOD category.

Analysis of climatic drivers of growth differences

Figure 4 shows the correlation plots of the average difference between long-term symptomatic trees and asymptomatic trees (yearly data from 1873 onwards) and climatic variables by season. There was a significant correlation between the difference in ring widths and seasonal temperatures in Central England for spring and autumn, with larger

negative divergences observed at warmer temperatures, suggesting tree responses to warmer temperatures were contributing more to growth differences between asymptomatic and long-term symptomatic trees. Analysis of very warm years on tree growth indicated that ring width size was reduced in unusually warm years (ring widths were smaller in the year following unusual warming as compared to the previous year) across all tree types ($F_{23,4838}$ = 56.9, $p < 0.001$), suggested acute periods of unusually warm weather are detrimental to all trees.

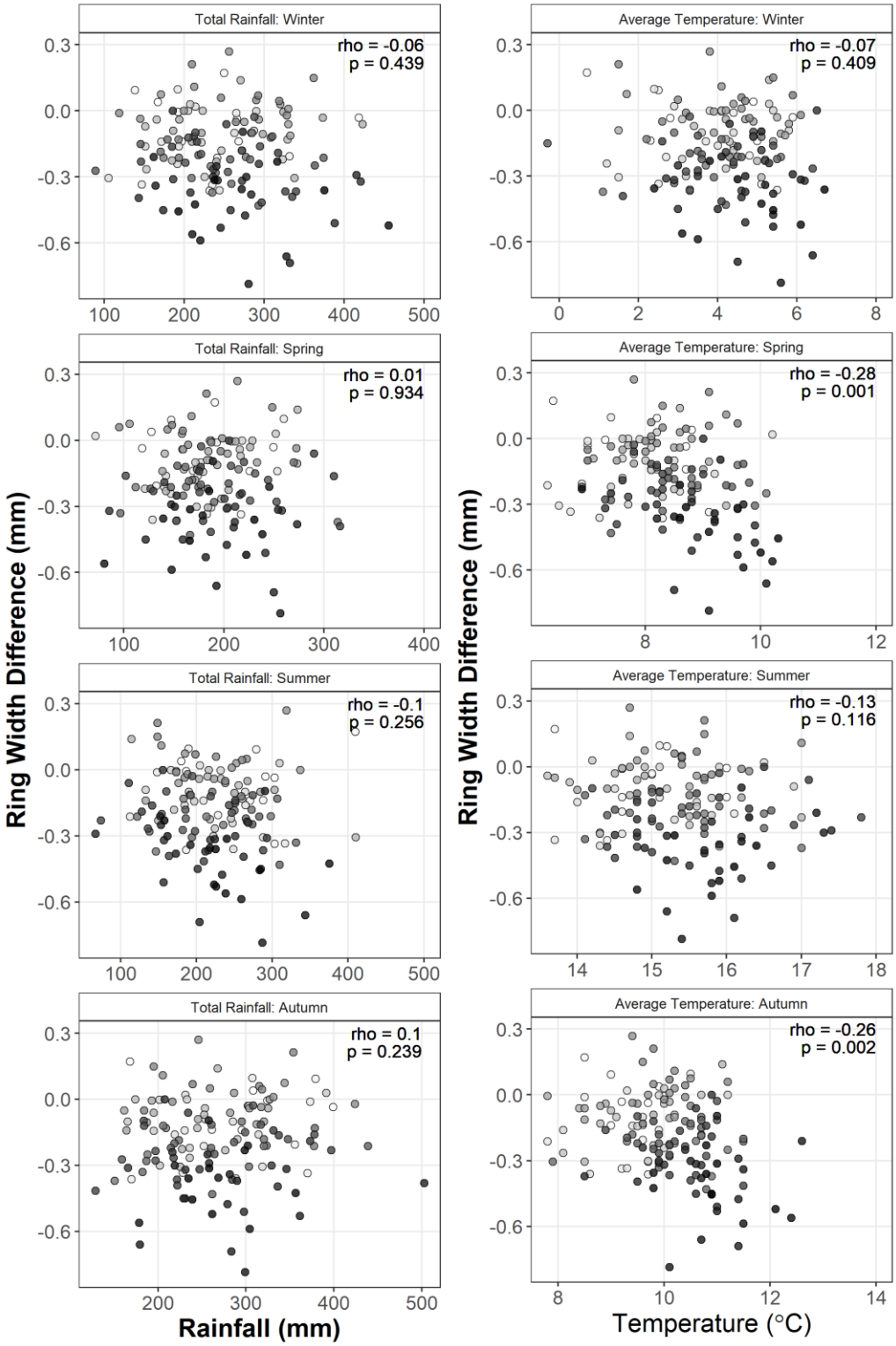

Figure 4. Pairwise contrast of ring widths for asymptomatic and long-term symptomatic trees (median for long-term symptomatic minus median for asymptomatic: more negative values shows poorer growth in long-term symptomatic trees) weighted across sites/compartments versus precipitation and temperature data (Central England data: https://www.metoffice.gov.uk/hadobs/hadcet/data/download.html). Point fill shows year (1874-2014) from white to black. Text shows correlations (Spearman's rho and p values).

Discussion

Trees with long-term AOD symptoms appear to be predisposed to the syndrome, exhibiting reduced vigour in the growth record for many decades before the onset of symptoms. Analysis of long-term growth trends through mixed effects modelling reveals significant growth differences between asymptomatic and long-term symptomatic trees from the early 1930s. Trees that only developed AOD symptoms in the last few years (new symptoms), and trees that were able to successfully occlude lesions and enter a state of remission or recovery, had mean stem growth that was intermediate between that of the asymptomatic controls and long-term symptomatic trees, but was not significantly different from either.

In England, the period from the end of WWI until 1925-6 was marked by widespread oak decline and a high oak death rate [44–46]. Oaks, potentially also impacted by widespread conifer clearing that altered water tables locally, are thought to have been weakened by successive severe defoliation of spring and lammas growth by lepidopteran larvae and recently-arrived powdery mildew, sequentially, with *Armillaria mellea*, a honey fungus, acting as a key secondary agent contributing to tree death [44,45]. While a single defoliation event is not usually sufficient to kill oaks, which are highly tolerant of herbivory, the loss of photosynthate production due to successive severe defoliation may result in exhausted carbohydrate stores, reduced fine root production and impaired hydraulic efficiency [18,47]. In this study, it appears that the trees currently exhibiting long-term AOD symptoms suffered permanent physiological damage in the 1920s, their annual growth correspondingly dropping below previous levels [26,48]. Other studies of declining European oaks have shown a similar pattern, wherein after severe defoliation events weakened cohorts continue to grow at a reduced annual growth increment. Such trees remain vulnerable, and may be killed when a woodland is affected by secondary pests and pathogens decades later [19,24,27,49].

Intense competition for resources in over-stocked stands may also have predisposed some of the study trees to AOD through growth suppression. For some sites, a number of the long-term symptomatic trees exhibit relatively poor growth from near the beginning of the stem growth record (Figure 1). Genotypic or microhabitat differences may have resulted in poorer initial growth in some trees that continued under dense management regimes. Physiological differences and lower carbohydrate reserves in suppressed trees may increase their susceptibility to stress [4]. Development of decline in suppressed trees is consistent with a recent study examining the ring widths of mature *Quercus robur* diagnosed with Chronic Oak Decline (COD), which showed reduced growth in the declining trees throughout most of their lifespans, before the development of COD symptoms [29]. Several other studies have also found that smaller, more slowly-growing oaks are prone to decline [30,50].

Since inclusion of the seven level AOD category, incorporating presence/absence of beetle exit holes with AOD type, did not markedly improve the mixed effects model fit, beetles are not simply completing development on the weakest trees, furthest along in the decline syndrome, as might be expected. Other factors than host vigour must influence beetle breeding success, such as bark moisture and individual trees' physical and chemical responses to wounding [13,21,51]. Under-bark temperature may also be very important; many parts of England are sub-optimal for beetle development, particularly in cooler years, and at some sites the beetles will have a better chance of developing on more open-grown trees where sunlight hits the stem [15,21]. Host selection by the adults is also poorly understood.

In this study, the mixed effect model forecasts from 2004-2017 provide very little evidence of recently-induced predisposition directly before AOD onset. The relatively similar growth patterns across categories, within each site, do not suggest varying resilience to short-term, inciting factors such as climatic (e.g. drought) stress. This provides further support that differences in long-term vigour, rather than varying responses to recent stress events, predisposes trees to AOD.

Surprisingly, the model forecasting also does not indicate a divergence between the growth of asymptomatic and symptomatic trees, or trees in remission, after AOD symptom onset and beetle colonisation. At least some trees with AOD symptoms continued to produce broad annual rings. Some of the symptomatic trees may have benefited from a reduction in competition due to the death of other trees. Growth declines may have been masked by the removal of the worst-affected trees through death or as part of woodland management [48]. Oaks also often do not exhibit sharply reduced stem growth until the years just prior to tree death [52].

The correlation between warm spring and autumn temperatures and larger growth differences between asymptomatic and long-term symptomatic trees suggests that diseased trees are less able to take advantage of favourable growing conditions. In the UK, warm temperatures in early summer have been linked to enhanced oak growth, when moisture is sufficient; in contrast, warm temperatures in winter negatively influence growth, because elevated winter respiration rates increase carbohydrate use [53]. Diseased trees may be hampered by changes in their photosynthetic capacity, carbohydrate storage, and fine root anatomy [26]. There was no relationship between seasonal precipitation and the growth difference between asymptomatic and long-term symptomatic trees. Interestingly, both asymptomatic and long-term symptomatic trees showed equally reduced growth in years of sudden, unusual warming, suggesting the presence of an upper threshold temperature of optimal growth and a limited adaptability to climatic change, regardless of tree vigour.

Conclusions

This study gives an insight into the long-term health trends of oak trees, keystone tree species worldwide which are increasingly threatened by pests, pathogens and climatic change [6]. The novel statistical techniques developed to analyse the dendrochronological

data simplify the analysis of stem growth, facilitating its use as a tool to determine the underlying health status of tree populations and to better understand episodes of forest decline. This study provides a valuable insight into how historical episodes of stress may impact trees' future susceptibility to disturbance, and resilience to a changing climate. Despite differing site histories at the five study sites, trees with long-term AOD symptoms appear to have been predisposed to the condition many decades before symptom onset. These trees are likely to have suffered permanent physiological alterations that render them vulnerable to AOD, as well as future potential impacts from other pests and diseases and climatic extremes. Some affected trees also appear to have been suppressed from the beginning of their stem growth record. The best-fit models of stem growth reveal stand-wide growth releases which are probably related to thinning. Opening up stands by removing trees with long-term AOD symptoms and suppressed trees which are predisposed to the syndrome, may increase the resources available to the remaining trees. Whilst the role of *A. biguttatus* in AOD is not fully resolved, thinning out suppressed trees may increase beetle developmental rates and breeding success, by opening up a stand and raising under-bark temperatures. However, thinning should reduce host availability if the remaining trees benefit from such management, as well as removing some developing insects. The lack of divergence in symptomatic vs asymptomatic tree growth after AOD onset was surprising and may be due to site-specific conditions influencing growth more strongly than AOD. An examination of the last few years of stem growth of AOD-killed trees might clarify the patterns that lead to ultimate tree mortality.

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586 Supplementary Appendix

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	Hatchlands Park	Langdale Wood	Rookery Wood, Attingham Park	Sheen Wood, Richmond Park	Madingley Wood
Coring date	May, 2015	January, 2018	January, 2018	May, 2015	May, 2015
Location	Guildford, Surrey	Malvern, Worcestershire	Attingham, Shropshire	Richmond Upon Thames, Greater London	Cambridge, Cambridgeshire
Latitude / Longitude	51.263098 / -0.46337962	52.083988 / -2.308511	52.68521325 / -2.66878724	51.455636 / -0.27052781	51.263098 / -0.46337962
Number of oaks within AOD monitoring plot	140	250	100	150	N/A
Elevation (m/asl)	80m	60m	60m	20m	60m
Site description	Discrete *Quercus robur* shelter belt within parkland	*Quercus robur* high forest over mowed grass	Discrete *Quercus robur* plantation within parkland	Discrete *Quercus robur* plantation within urban parkland	Mixed-age stands of *Quercus robur* within ancient and secondary ash-maple woodland
Site oak stocking density (stems / ha.)	6.14	30.95	78.50	38.32	24.43
Uniformity	Relatively uniform stand	Relatively uniform stand	Relatively uniform stand	Relatively uniform stand	Trees drawn from 5 compartments; variability in tree age and stand environment
AOD symptoms first recorded	After 2001	Before 2006	2007	1990	2009-2010
Current monitoring began	2009	2009	2010	2010	2014
DBH of study trees (mean ± SE)	66.0 ± 2.1	72.4 ± 1.3	88.8 ± 3.8	81.6 ± 2.0	63.8 ± 2.9
Human disturbance level	Medium	Medium	Medium	High	Low
Other disturbance factors	Chronic Oak Decline	Chronic Oak Decline	Chronic Oak Decline	Chronic Oak Decline; oak processionary moth; deer	Chronic Oak Decline

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589 **Table SA1.** Description of the AOD study sites at Hatchlands Park, Langdale Wood, Attingham Park, and
590 Richmond Park (Brown *et al*., 2016, 2017); and Madingley Wood (Natural England, 2017; Karen Russell,
591 unpublished).

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Four level AOD category	Seven level AOD category	Hatchlands Park boundary belt (HP)	Langdale Wood	Rookery Wood, Attingham (RW)	Sheen Wood, Richmond Park (RP)	Madingley Wood (MW)
Long-term symptoms	Long-term symptoms with exit holes	4	1	3	12	3
	Long-term symptoms without exit holes	7	12	4	5	5
New symptoms	New symptoms with exit holes	2	3	5	5	-
	New symptoms without exit holes	6	11	8	6	-
Remission	Remission with exit holes	0	7	1	4	2
	Remission without exit holes	11	8	8	7	21
Asymptomatic control	Asymptomatic of AOD or exit holes	14	14	15	14	15

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600 **Table SA2**. Description of AOD symptom and *Agrilus biguttatus* exit hole categories, and numbers of trees within

601 the categories, at each site.

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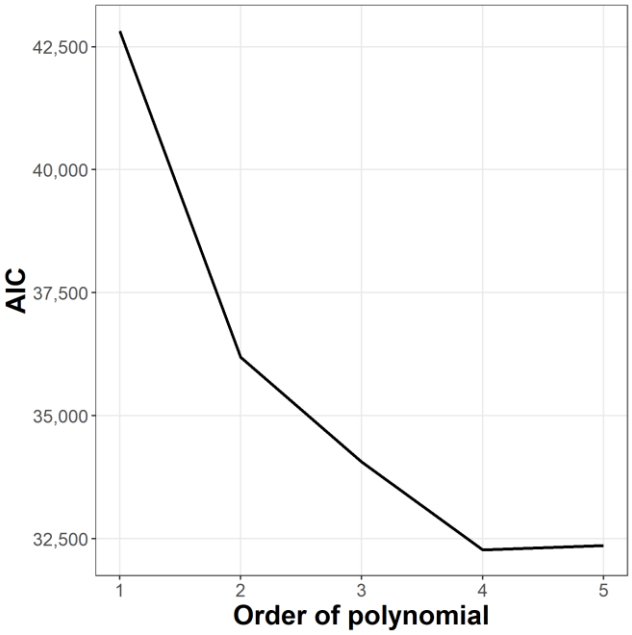


Figure SA1. Akaike Information Criteria (AIC) values for mixed effects with the basic fixed effects structure (Year+…)*Compartment+Tree Age+Aspect and random effects of (Year+…) per tree, where "…" represents additional n order Year polynomial to allow turning points for each individual compartment (fixed effect) and tree (random effect) through time. Change in AIC with increasing number of turning points (1 = linear (no turning points); 5 = 4 turning points) indicates a fourth order polynomial to be the most efficient structure.

	Category	Attingham Park	Langdale Wood	Hatchlands Park	Madingley Wood	Richmond Park
Average number of annual rings	LS	251	103	147	160	175
	NS	239	105	152	-	170
	R	238	95	126	138	166
	C	208	100	129	108	160
Minumum - maximum number of annual rings	LS	177-301	70-113	106-239	103-231	127-194
	NS	163-293	89-115	111-218	-	145-189
	R	55-299	71-115	74-202	76-219	118-194
	C	49-288	73-112	91-218	63-200	65-187
Average annual ring width (mm) (± SD)	LS	1.2 ± 0.2	2.6 ± 0.3	1.5 ± 0.2	1.8 ± 0.2	1.6 ± 0.1
	NS	1.3 ± 0.3	2.7 ± 0.3	1.7 ± 0.1	-	1.7 ± 0.1
	R	1.3 ± 0.3	2.7 ± 0.7	2.0 ± 0.1	1.8 ± 0.1	1.5 ± 0.1
	C	1.4 ± 0.7	2.8 ± 0.3	2.2 ± 0.2	2.1 ± 0.2	1.8 ± 0.1

Table SA3. Ring count and ring width summary data by site and four level AOD category (LS = long-term symptoms, NS = new symptoms, R = remission, C = asymptomatic control)

Site	Hatchlands Park		Attingham Park		Langdale Wood		Richmond Park		Madingley Wood		First and last year	
	CorrC	TT	CorrC	TT	CorrC	TT	CorrC	TT	CorrC	TT	start	end
Bath	0.52	8.6	0.32	5.1	0.57	5.9	0.33	4.4	0.36	5.4	1754	1979
Old Park Wood, Hillingdon, Greater London	0.59	10.5	0.39	6.1	0.61	7.2	0.59	9.5	0.44	7	1786	1994
Scotney Castle, Tunbridge Wells, Kent	0.47	8	0.38	6.4	0.46	5.1	0.52	8.2	0.34	5.3	1752	2003
Hatchlands Park	-	-	0.36	5.9	0.6	7.8	0.57	9.6	0.57	10.5	1776	2014
Attingham Park	0.36	5.9	-	-	0.58	7.6	0.46	7.2	0.52	9.2	1715	2017
Langdale Wood	0.6	7.8	0.58	7.6	-	-	0.55	6.8	0.58	7.5	1903	2017
Richmond Park	0.57	9.6	0.46	7.2	0.55	6.8	-	-	0.63	11.3	1821	2014
Madingley Wood	0.57	10.5	0.52	9.2	0.58	7.5	0.63	11.3	-	-	1797	2014

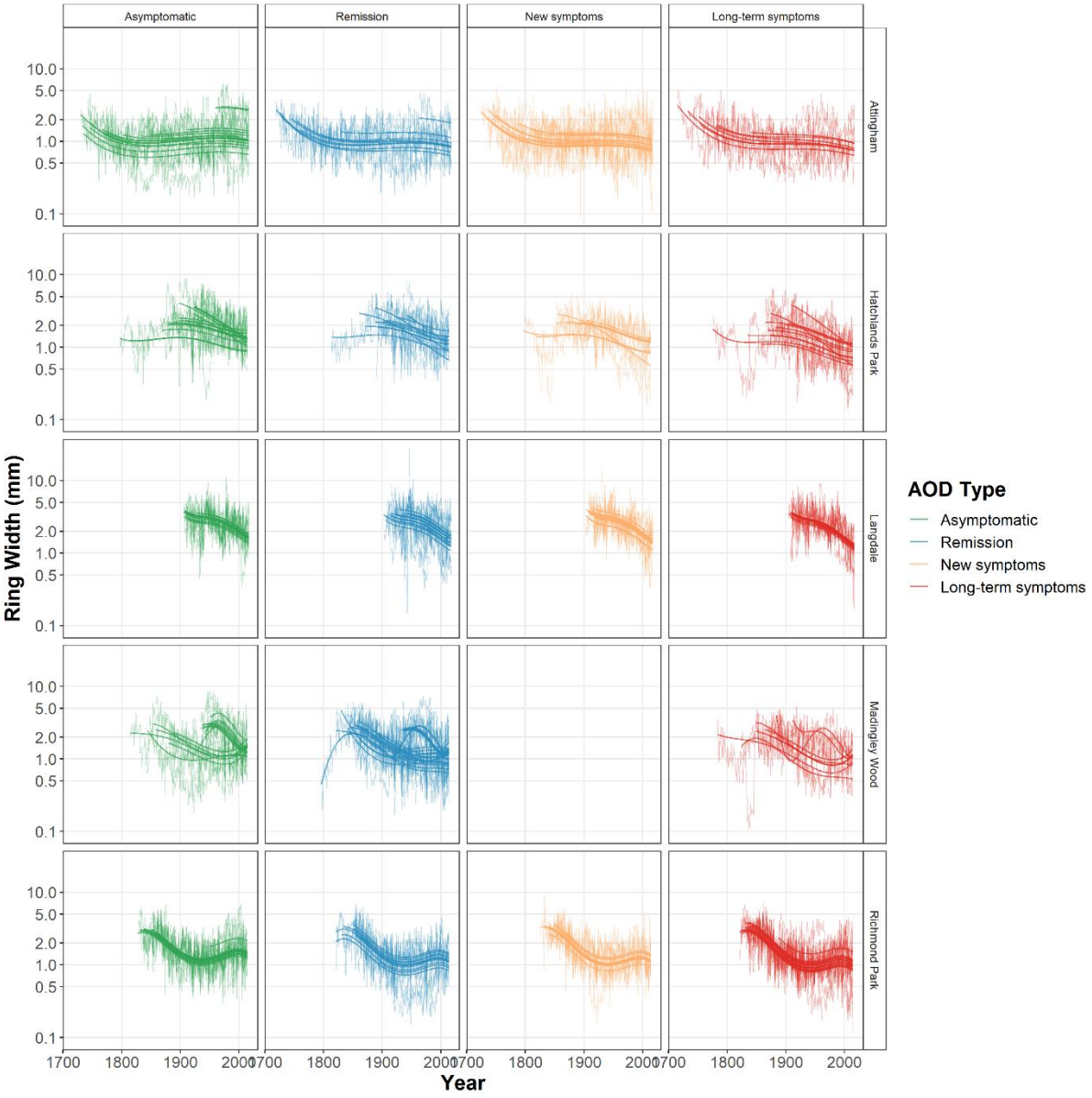
Table SA4. Correlation coefficients (CorrC) and T-scores (TT) calculated in C-Dendro (Larsson, 2014), between two recent reference chronologies and the five study woodlands (Arnold, Howard, & Litton, 2005; Bridge & Winchester, 2000; Jon R Pilcher & Baillie, 1980)

Model	DF	AIC
Fourth order polynomial	72	32,274
Fourth order polynomial, corARMA(0,1)	73	25,317
Fourth order polynomial, corARMA(0,2)	74	22,905
Fourth order polynomial, corARMA(1,0)	73	22,228
Fourth order polynomial, corARMA(2,0)	74	21,614
Fourth order polynomial, corARMA(1,1)	74	21,475
Fourth order polynomial, corARMA(1,2)	75	21,233
Fourth order polynomial, corARMA(2,1),	105	21,051
Fourth order polynomial, corARMA(2,1)	75	21,050
Fourth order polynomial, corARMA(2,1),	77	21,042
Fourth order polynomial, corARMA(2,1),	83	21,037
Fourth order polynomial, corARMA(2,1),	81	21,031
Fourth order polynomial, corARMA(2,1),	**87**	**21,026**
Fourth order polynomial, corARMA(2,1),	**81**	**21,026**

Table SA5. AIC values for range of corARMA and fixed effects models applied to data.

Fixed Effect	numDF	denDF	F-value	p-value
(Intercept)	1	36,281	764.8	<.0001
Year	1	36,281	174.2	<.0001
I(Year^2)	1	36,281	6.0	0.0142
I(Year^3)	1	36,281	14.9	0.0001
I(Year^4)	1	36,281	4.1	0.0428
Compartment	9	223	50.5	<.0001
Type (4 Treatment)	3	223	6.5	0.0003
final_age	1	223	106.0	<.0001
Aspect	5	223	6.7	<.0001
Year:Compartment	9	36,281	19.6	<.0001
I(Year^2):Compartment	9	36,281	13.0	<.0001
I(Year^3):Compartment	9	36,281	6.3	<.0001
I(Year^4):Compartment	9	36,281	8.5	<.0001
Year:Type (4 Treatment)	3	36,281	4.3	0.0048

Table SA6. ANOVA output applied to final model.



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631 **Figure SA2**. Raw tree ring width data, overlaid with best fit model by tree. Note log10 scaling on y axis.

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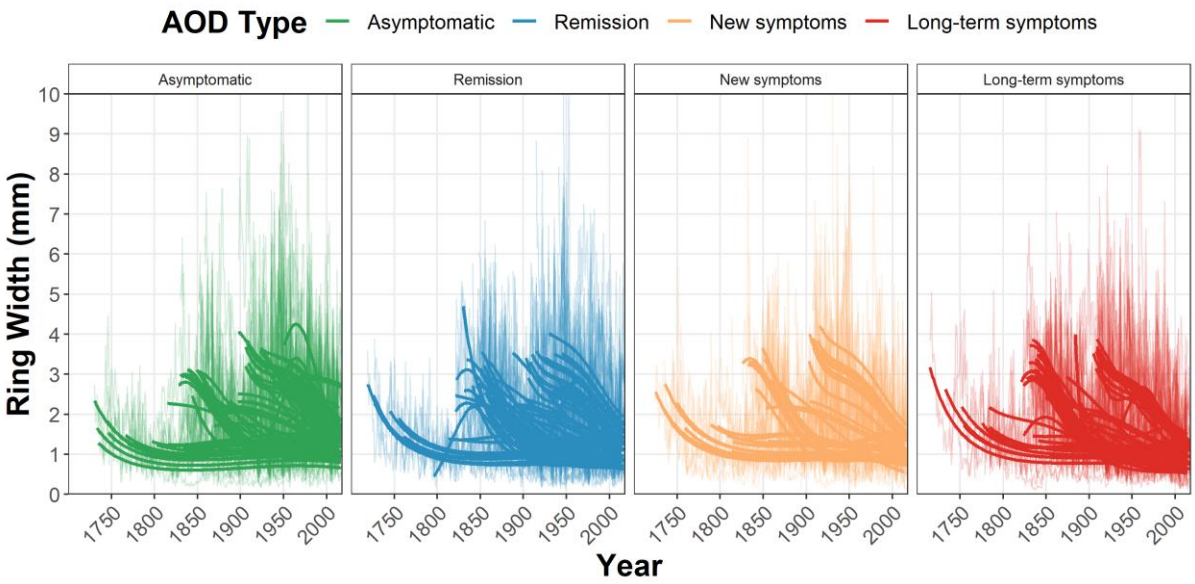

Figure SA3. Raw tree ring width data, overlaid with best fit model by tree. Note log10 scaling on y axis.