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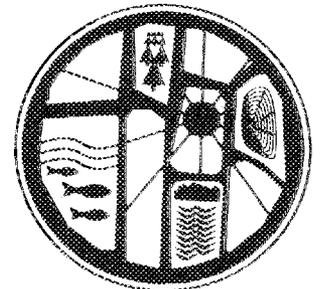
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**Red Spruce Decline -
Winter Injury and Air Pollutants**

T. M. Roberts

Environmental Sciences Division
Publication No. 3269



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ENVIRONMENTAL SCIENCES DIVISION

RED SPRUCE DECLINE - WINTER INJURY AND AIR POLLUTANTS

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Publication No. 3269

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Date of Publication - October 1989

Prepared for the
Electric Power Research Institute under
Interagency Agreement No. TVA 1610-1610-A1
(DOE RP-2799-2)

Prepared by the
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SUMMARY

There has been a widespread decline in growth of red spruce (Picea rubens Sarg.) since 1960 in the eastern United States. There is evidence that this decline is at least partly attributable to age- and density-related growth patterns, particularly at lower elevations. Mortality has been severe at high elevation sites where similar episodes have occasionally occurred in the last 100 years. At these sites, periods of low growth preceding 1960 were related to periods with warm late summers and cold early winters. Since 1960, this relationship no longer holds, although there is an association with unusual deviations from mean temperatures.

There are field reports that one of the main causes of reduced growth and mortality is apical dieback induced by severe winter conditions. Preliminary observations suggest that high elevation red spruce may not be sufficiently hardened to tolerate low autumn temperatures. However, appearance of injury in the spring, association of injury with wind exposure and correlation of provenance susceptibility with cuticular transpiration rates, indicates the importance of desiccation injury.

Sensitivity to both types of winter injury may be increased by air pollutants (particularly ozone and less probably, acid mist or excess nitrogen deposition). Nutrient deficiency (particularly magnesium and to a lesser extent potassium) may also increase cold sensitivity. The nature and extent of these interactions are being actively researched for red spruce.

Progress in understanding the role of climatic extremes and air pollutants will require an improved understanding of the ecophysiology of cold-stressed trees, both in the field and in exposure chambers. Techniques have yet to be devised to manipulate climatic conditions to reproduce the apical dieback symptoms. Methods are in use to reproduce pollutant exposures whereas methods for conditioning air in exposure chambers to reproduce field conditions have not yet been fully developed.

INTRODUCTION

Mortality of red spruce (Picea rubens Sarg.) in high elevation forests in the eastern United States has progressively increased since the mid-1960's (Siccama et al.1982). High elevation sites have short growing seasons, low winter temperatures, severe wind exposure and shallow acidic soils. These sites also have elevated ozone and hydrogen peroxide, high deposition of sulphur and nitrogen compounds, and exposure to acidic cloudwater.

REGIONAL AND HISTORICAL TRENDS

Field surveys have established that mortality occurs above about 800m in New York, Vermont and New Hampshire and at higher elevations in the southern Appalachians (Scott et al.1984; Johnson and McLaughlin 1986). Mortality generally increases with elevation, size class, aspect (i.e., exposure) and in areas where fir (Abies Spp.) mortality following balsam woolly adelgid infestation has provided gaps in forest canopy. Analysis of historical records has shown that a period of severe mortality in red spruce occurred between 1870-90 and, to a lesser extent, in 1935-50 (Weiss and Rizzo, 1987).

Average annual growth of red spruce throughout northern New England and New York showed a regional decline of 13- 40% since 1960 (Hornbeck et al. 1986; Federer and Hornbeck 1987). This was consistent with previous records of age-related growth trends for second-growth, even-aged red spruce stands (Meyer 1929). However, growth trends at high elevation sites with mortality in the Northeast have been anomalous since 1960-65 compared to previous age, density or climatic relationships (McLaughlin et al. 1987). Periods of low growth up to 1960 were most frequently related to unusually warm late summers and cold early winters and weakly correlated with precipitation. Since 1960, the growth decline has been unrelated to mean monthly temperatures although there is an association with unusual departures from summer and winter mean temperatures (Cook et al.1987). The recent breakdown of climate-growth relationships may be due to a unique combination of temperature fluctuations or, air pollutants.

WINTER INJURY AS A PRIMARY STRESS

Field observations of red spruce stands over the past 40 years suggest that winter injury has been the main factor causing crown dieback, growth reductions and mortality.

Curry and Church (1952) recorded unusual and extensive damage to conifer foliage after the 1947-48 winter throughout northern New York State. Injury first appeared as browning of the youngest needles on the south and west of exposed trees in March 1948 and was followed by shedding of necrotic needles. Many species were affected in the following order of sensitivity: red spruce > hemlock > white pine > balsam fir. White cedar, black spruce, white spruce, and red pine showed little damage. Bud mortality on red spruce shoots was proportional to the extent of defoliation. Extensive defoliation was followed by mortality or reduced growth. Observations in subsequent years showed that necrosis and bud mortality occurred frequently but with lower severity.

Friedland et al. (1984) described similar symptoms on red spruce in the Green Mountains of Vermont. Necrosis and defoliation of the youngest needles occurred in all size classes in March-April 1981 and 1984. Evans (1986) also described apical dieback in red spruce with decreasing severity at more southerly sites in the Appalachians (i.e., Whiteface Mountain > Mt. Mitchell > Clingman's Dome).

Joslin and Roberts (unpublished data) observed similar apical dieback on red spruce declining along the ridge of Whitetop Mountain in the Appalachians. Mortality was most severe to the north of a large open compound but symptoms also appeared along the south-facing ridge in areas unaffected by this disturbance. Apical dieback only occurred on exposed branches of trees greater than 4-5 m tall and was less at lower altitude. Red spruce at all altitudes showed tip chlorosis on all year classes (including the current year) of exposed branches. Crown dieback of exposed red spruce also occurs around Clingman's Dome in the GSMNP and on Mt. Mitchell, particularly where Fraser Fir mortality, resulting from balsam woolly adelgid infestation, had opened the canopy.

Field observations point to apical dieback after severe winters as the main factor producing needle loss, crown thinning, reduced growth and mortality. Attention has also been drawn to unusual and widespread winter injury on conifers in Northern Europe (Horntvedt and Venn, 1980; Redfern et al. 1987; Rehfuss 1987).

MECHANISM OF WINTER INJURY

Needle necrosis may be induced in conifers as a result of "freezing/chilling injury" or "desiccation injury". The distinction is important as the metabolic basis of resistance differs (Levitt, 1980) and the interactions with pollutants may also be different (Davison et al. 1987). It is often difficult to distinguish the mechanism of injury in the field and the relative importance of the two types has been the subject of debate (Tranquillini 1979; 1982).

Freezing injury is caused by the formation of extracellular ice which can result in cell dehydration and membrane damage. Intracellular freezing always results in cell death and is avoided in hardened tissue by changes which promote intracellular desiccation. Autumn "hardening" is induced by low temperatures and short days and consists of reduced tissue water content, changes in cell membranes to increase water permeability, increases in soluble sugars, proteins and tannins. Freezing injury generally occurs in the autumn or around budburst in the spring (Cannell and Smith 1984; Cannell et al. 1985). Resistance to freezing injury can be assessed by standard chilling tests in which detached shoots are cooled and allowed to thaw at a fixed rate. The degree of necrosis or rate of cation leakage is then measured at a fixed time (Cannell and Sheppard 1982). Preliminary results of freezing tests on red spruce from Whiteface Mt. and Newfound Gap indicated that hardening was insufficient to prevent autumn frost damage in 4 and 6 of the last 7 years, respectively (Unsworth et al. 1987). However, although winter flecking on understory red spruce develops in the autumn, apical necrosis on mature trees does not develop until the spring in the southern Appalachians.

Desiccation injury is caused by large water deficits in needles induced by transpiration losses during periods of radiative warming, high vapour pressure deficits and/or high winds in winter (Tranquillini 1982). Injury appears as necrosis of youngest needles on wind-exposed shoots and often results in shaping or "flagging" of the tree crown. As the temperature drops in the autumn and water conductivity in the soil, root and trunk decrease, resistance to water loss increases through stomatal closure and formation of wax platelets over the stomatal pore. The cuticular resistance to water loss then depends on the thickness of the cutinized layers and epicuticular waxes (as well as the chemical composition). Thickness of the cuticle may be reduced by cool summers or a short growing season. Abrasion of the cuticle may also result from exposure to high winds or ice (Hadley and Smith 1986). Consequently, although seedlings from populations of high-altitude origin grown at the same site as low-altitude populations have higher desiccation resistance, increased cuticular transpiration often occurs at higher altitudes. If the water deficit drops below a critical level then the needles become necrotic during spring warming. Hadley and Smith (1983) developed a test for needles viability and found it was correlated with relative water content and cuticular resistance of wind-exposed timberline conifers in Wyoming. Sheltered needles developed lower night-time temperatures under clear skies than exposed needles but had higher relative water contents due to lower evaporative demand and thicker cuticles.

The appearance of apical needle necrosis and bud mortality of red spruce in later winter in the northeastern USA appears to be related to desiccation injury rather than freezing injury. Wilkinson (1988) studied the susceptibility of 12 red spruce provenances to winter injury at the Coleman State Forest in New Hampshire. The difference between provenances in apical necrosis and subsequent needle loss after the severe 1985-86 winter, was related to the cuticular transpiration rate, the amount of epicuticular wax, stomatal density and morphological features that may also affect water loss. Winter injury was greatest in shorter provenances reflecting the reduced crown density following

repeated apical dieback and needle loss in sensitive provenances. Winter injury was also related to differences in mortality between provenances. The winter injury score was correlated with the "desiccation index" developed for the same provenances in Ontario after the 1966-67 winter (Morgenstern 1969). Contrary to expectation, the least amount of wax was found on provenances from the high elevations. This may contribute to the sensitivity of high-altitude red spruce to winter injury. It is interesting to note that preliminary measurements of foliar isozymes showed a reduced genetic diversity in red spruce from high-mortality sites (Eckert et al. 1988; DeHayes 1988).

Whilst large water deficits have been correlated with wind exposure of alpine timberline conifers in the Rocky Mts., physiological measurements relating apical dieback of red spruce to desiccation are less conclusive. Hadley and Smith (1986) showed that decreases in needle viability of timberline conifers in Wyoming occurred when either xylem pressure potential or needle water potential fell below -3 MPa. Viability declined rapidly below -4.5 MPa and a corresponding relative water content of 60%. High rates of water loss from wind-exposed needles was ascribed more to ice abrasion than inadequate cuticle development in summer. The development of winter injury and changes in water potentials of red spruce at four altitudes on Mt. Monadnock (NH.) were monitored over two winters (1978-79) by Kincaid and Lyons (1981). At the tree line (>750m), death of new shoots and needles increased during the winter. The water potential of green shoots averaged only -1.14 MPa indicating that large deficits did not generally occur even at the tree-line. Microclimatic severity and needle necrosis increased with altitude but no trend was detected for water potential. Air temperatures reached -35° C and frequently rose above freezing thereby promoting water loss. However, trunk temperature followed air temperature so bulk water flow may have occurred during mild conditions. In addition, detached branches did not develop severe water deficits so green needles were able to maintain low cuticular transpiration rates. The cuticle did not appear to be damaged and the stomatal pores were plugged with wax thereby reducing transpiration. However, the authors

noted considerable variation in water potential within single trees and concluded that water deficits vary from shoot to shoot so the status of necrotic needles may not be reflected in measurements of green shoots. Indeed the necrotic needles had a high frequency of porous mesh-like stomatal plugs.

Marchand and Chabot (1978) studied the winter water relations of tree-line species at Mt. Washington (NH.) and noted that wind may decrease cuticular transpiration of sunlit needles due to cooling. However, water loss was accelerated where abrasion of the cuticle had occurred but this was usually localized. McLaughlin et al (1988) also found small seasonal differences in saturated osmotic potential of understory saplings in 1987 at two sites in the GSMNP. However, water potentials in shoots were close to the turgor loss point (estimated from pressure-volume analyses) particularly at the higher site.

Decline mortality of red spruce is most severe in exposed sites particularly where disturbance has opened the canopy or balsam fir have been affected by the balsam woolly adelgid Harrington (1986) observed that red spruce and balsam fir suddenly exposed to winds along a ski trail cut in 1962 in New Hampshire also showed crown dieback, reduced growth and mortality. Mortality at Whitetop Mt. was greatest in 1987 along edges of stands exposed by felling to create an open compound (Devlin and Roberts unpubl.). Marchand et al. (1986) concluded that wave mortality of balsam fir is due primarily to exposure to high winds along the leading edge of the dieback front. Battles et al. (1988) reported that mortality of red spruce balsam fir on Whiteface Mountain was more pronounced for large trees on wind-exposed faces especially at higher elevations.

Preliminary measurements of freezing resistance indicate that autumn hardening of red spruce can be insufficient to prevent freezing injury. However, the late winter development of apical dieback, the association with exposure and the correlation between susceptibility and cuticular transpiration rate is consistent with desiccation injury. The field measurements of changes in water relations overwinter are less clear and require further investigation.

NUTRITION AND WINTER INJURY

Although mortality and growth declines have historical precedents and freezing and/or desiccation injury are the main causal factors, there is still the possibility that other stresses have functioned as predisposing factors.

The nutrition of conifers must be optimal for maximal hardening. Full hardening may not occur in the presence of elevated needle nitrogen or deficiencies of other elements (Levitt 1980). Friedland et al. (1984) proposed that excess nitrogen could predispose red spruce to winter injury. However, Johnson (DW) et al. (1988) have observed that the foliar nitrogen concentration of declining red spruce was moderate to low in the GSMNP, despite elevated nitrate levels in soils and leachate water. In addition, Weinstein et al. (1987) found that four year-old red spruce seedlings were not affected by sprays of nitric acid down to pH 2.5 on 32 occasions over a 57-day period.

Johnson (A) et al. (1988) reported upper-surface chlorosis of older needles of red spruce at Whiteface Mt. which could have resulted from photo-oxidation due to chlorophyll breakdown by free radicals induced by high irradiance overwinter (Oquist 1983). However, on Whitetop Mountain, Joslin and Roberts (unpublished) observed tip chlorosis on exposed needles of all year classes in early Nov. 1987 which increased with elevation. Even the current year's needles were affected which could not be due to photooxidation overwinter.

Photoinhibition and photooxidation can occur in plants under nutrient stress as well as low temperature stress (Powles 1984; Hendry et al. 1987). Irradiance can inhibit electron transport in Photosystem I and induce free radical formation in nutrient deficient plants. Indeed Osswald et al. (1987) have measured increased levels of ascorbate and glutathione, which deactivate reactive oxygen species, in Mg-deficient Norway spruce. It should also be noted that ozone also causes free radical formation which means that specific biochemical indicators of stress types are unlikely (Tingey and Taylor 1982).

Friedland et al. (1988) reported that foliar Mg levels were lower at high elevation sites in the Green and Adirondack Mountains and in the range of moderate deficiency. Johnson et al. (1988) made similar observations at Whiteface and the Smoky Mountains. Stone (1953) observed same time ago that Mg-deficient pines and balsam fir on sandy glacial outwash soils bordering the Adirondacks, showed tip chlorosis (compared to chlorosis of older needles on Norway spruce which has also been reported on Mg-deficient sites in Central Europe).

Soils at high-elevation sites with red spruce mortality have elevated concentrations of heavy metals (Friedland et al. 1984), but these are presently below phytotoxic levels determined experimentally. Similarly, monomeric aluminum is elevated in soil solution at high-altitude sites and can reach 15-80 μM (Cronan et al. 1987; Johnson et al. 1988). Growth reductions occur in solution culture experiments at 185 μM Al. However, reduced uptake of Ca, Mg, K and P may occur at lower levels (Thornton et al. 1987; Joslin 1987).

Johnson et al. (1988) reported that only foliar K decreased with crown thinning at Whiteface despite an increase in soil K. In addition, the same authors reported that no change in pH or extractable Ca had occurred in acid organic or mineral horizons throughout the Adirondacks between 1934 and 1984. The possibility of marginal Mg deficiency, through naturally low soil Mg, acid leaching or restricted uptake by Al in the soil solution, is potentially important in that there is anecdotal evidence that Mg-deficient Norway spruce in the Bavarian Forest may be more susceptible to winter injury (Rehfuess 1987).

AIR POLLUTANTS AND WINTER INJURY

Following the early reports of red spruce mortality in the early 1980's, air pollutants were put forward as the primary stress factor on the grounds of the high concentrations of ozone and acids in cloudwater and the simultaneous appearance of forest decline in Central Europe (Schutt and Cowling 1985). However, it is now clear that forest decline in Europe consists of a number of damage "types" (Rehfuess 1987). The

apical necrosis/bud mortality of red spruce does not occur in any of the three widespread damage "types" of Norway spruce in Europe (ie. Mg-deficient chlorosis of older needles on acid soils; K/Mn chlorosis of youngest needles on calcareous soils; needle-cast fungal damage). Necrosis of youngest needles does, however, occur in localized areas close to the East European border and has been ascribed to "acute" SO₂ injury or predisposition to "frost shocks" (Materna 1984). The annual mean SO₂ concentration in this area is 15-30 ppb with peaks in excess of 100 ppb compared to mean values around 5 ppb at high-elevation red spruce sites. Davison et al. (1987) reviewed the evidence for predisposition of plants to freezing or desiccation injury by air pollutants. SO₂, NO₂ and particularly ozone may modify increases in unsaturated lipids of membranes that are associated with cold hardening. Reduced net photosynthesis in the growing season could affect the solute changes associated with hardening or delay cuticle development. Both SO₂ and acid mist have been shown to alter the surface structure of the epicuticular wax on conifer needles.

Progress has been slow in the evaluation of the possible interaction between air pollutants and winter injury. Friedland et al. (1984) proposal that excess nitrogen deposition could increase cold sensitivity of red spruce has been discussed in the previous section. In addition, Freer-Smith and Mansfield (1987), Keller (1978) and Huttunen et al. (1981) have all shown that elevated SO₂ can increase winter injury in conifers. However, the SO₂ concentrations at high-elevation sites in the northeastern United States are very low.

Two studies have now been carried out to assess the effects of acid deposition on red spruce. Taylor et al. (1986) found that exposure of red spruce seedlings to pH 4.1 rain + 3.6 mist and 120 ppb O₃ over 4 months did not reduce growth nor produce the apical necrosis symptoms. Weinstein et al. (1987) sprayed 4-year old red spruce with mist at pH 4.5, 3.5 and 2.5 for 70% of nighttime hours over a 57 day period in late summer 1985. Significant needle injury occurred at pH 2.5 in September but there were no effects on bud condition or needle loss by the following spring. Nevertheless, given the importance of cuticular

transpiration in controlling water deficits, observations of changes in cuticle morphology induced by acid mist (Percy and Baker, 1987, Schmidt et al. 1987) need further consideration.

The possibility that ozone may predispose red spruce to winter injury developed from the studies of Brown et al. (1987) which showed that >100 ppb O₃ for about 60 days in summer subsequently increased the sensitivity of some Norway spruce clones to cold stress. These results were later confirmed by Barnes and Davison (1988) for Norway spruce and Cottam et al. (1988) for Sitka spruce. This concentration is only two/three times greater than mean O₃ levels at high-altitude in the NE States. However, it should be noted that the necrotic symptoms appeared only on the older needles of Norway spruce whereas cold stress in the field normally affects the youngest needles (i.e., as older needles are often more ozone-sensitive, the cold stress may have exacerbated ozone stress rather than vice versa). Weinstein et al. (1987) exposed 3-year-old red spruce seedlings to O₃ up to 2x ambient levels in northern New York for 16 weeks from June-October 1985. Ozone did not affect growth nor induce apical necrosis but chlorophyll and carotenoid levels were reduced by about 10%. However, O₃ may have affected hardening by delaying the autumn decrease in photosynthesis and decreasing tannin formation.

CONCLUSIONS

The recent decline of red spruce in the eastern United States has historical precedents. However, there is evidence that climate-growth relationships could have been modified since 1960. There is field evidence that recent decline of red spruce at high altitudes in the eastern United States is primarily the result of winter injury. Circumstantial information suggests that "desiccation injury" is more important than "freezing injury". Preliminary studies indicate it is more probable that O₃ rather than excess nitrogen or acid deposition predisposes spruce to winter injury. Marginal supply of Mg (and to a lesser degree K) may also increase cold sensitivity. However, none of

these predisposing factors has been confirmed as exacerbating winter injury of red spruce but this is an area of intense research.

RESEARCH NEEDS

(1) General Principles

The concept that air pollutants may increase the susceptibility of plants to other stress factors has been on the fringe of mainstream dose-response research for some time. However, the concept that a species severely affected by edaphic or climatic stress may become even more susceptible when exposed to air pollutants requires a new approach. Relevant experiments cannot be carried out by exposing plants to air pollutants in laboratory or field chambers unless stress conditions are created similar to those observed at damaged sites. Pollutant exposure technology has developed towards systems which allow control of pollutant profiles and maximise dose to plants under optimal growth conditions. The current challenge is to develop systems which reproduce stress conditions. This will require much greater attention to the ecophysiology of stressed plants as the basis on which to design experimental investigation of interactions between the stress factor and air pollutants.

A particular problem with trees is that stresses and air pollutants may act over several seasons. Short-term experiments within a single growing season have been able to provide only the most general insight into the effects of air pollutants on trees. Many exposure chambers are removed during the winter months thereby eliminating the possibility of manipulating the microclimate and physiological condition of the tree to approximate stresses at high-elevation sites (eg. by changing airflow rates to induce cuticle abrasion or modifying the air temperature).

Attempts in Europe to induce Mg-deficiency in healthy Norway spruce by air pollutant treatments have been unsuccessful. However, the studies should have been designed to determine whether pollutants could exacerbate nutrient deficiencies. Similarly, studies on red spruce should focus on methods for reproducing the winter injury symptoms so

that interaction with air pollutants can be assessed on stressed plants. Apical dieback is often less on young trees or sheltered branches and does not occur on trees protected by snow.

Methods for assessing freezing resistance after exposure to pollutants are currently in use. Methods for manipulating water deficits and estimating needle viability should also be included.

(2) Red Spruce and Winter Injury

(a) Ecophysiological investigations are required at high-elevation sites with red spruce mortality to determine the relative importance of "freezing injury" and "desiccation injury". This involves regular measurement of needle necrosis, pigment and nutrient content, starch and sugar turnover. Water relations should be investigated by measurement of needle, stem and soil temperatures, cuticle morphology and resistance to water loss, relative water content, xylem pressure potential and tissue water potential.

(b) Field experiments to determine the nature of the winter injury should include protection from wind and/or radiation, thawing of needles, sprayed with nutrients and cryoprotectants. Methods should be developed for excluding gaseous pollutants and/or cloudwater.

(c) Assessment of resistance to freezing injury should be made by the chilling method of Cannell and Sheppard (1982). Assessment of resistance to desiccation injury should be made using the viability and water relations measurements of Hadley and Smith (1983).

(d) The standard chilling and desiccation tests should be compared with conditions at high altitude red spruce sites with a view to perfecting assays which reflect the field stress (eg. rate of temperature change).

(e) Controlled-environment or field chamber methods should be developed to create the degree of exposure monitored at the damaged sites so that apical dieback symptoms can be reproduced and manipulated with confidence.

(f) Exposure chamber studies should at least monitor air, soil and tissue temperatures, vapour pressure deficit and tissue water

potentials. Consideration should be given to methods of developing water deficits in exposure chambers similar to values recorded at high-altitude sites (eg. by manipulation of air flow or conditioning air to reproduce periods of wide temperature fluctuation).

A number of these field measurements and experimental approaches are already underway. However, there should be an improvement in experimental approaches to reproduce stress symptom and facilitate studies on interactions with pollutants.

ACKNOWLEDGMENTS

This review was prepared while the author was participating in the project "Response of Plants to Interacting Stresses (ROPIS)" at the Oak Ridge National Laboratory, funded by the Electric Power Research Institute and the Tennessee Valley Authority. The author is indebted to G. E. Taylor, M. Kelly, D. W. Johnson, D. Joslin, R. Norby, C. Andersen, P. Hanson, W. Winner, and F. C. Thornton for assistance and J. Huckabee, L. Pitelka, and R. Goldstein for encouragement.

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