INTERNAL & EXTERNAL TREE REACTIONS TO CHLORINE GAS

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The primary tree reactions to chlorine gas, both internally and externally, are reviewed in this publication. This is not a toxicology or environmental dosage review, but is designed to help tree health care professionals understand the potential injuries sustained by trees and other landscape plants when exposed to chlorine gas.

Tree leaf reactions classes to chlorine gas exposures which are most visible include: chlorosis, flecking, glazing and wet spots, and scorching.

Chlorosis: Chlorosis is a yellowing caused by many things which slow or damage the photosynthesis process or the production of chlorophyll. Photosynthesis is extremely sensitive to gas exchange, intercellular acidity and cell water contents. The green pigments of photosynthesis are concentrated in bundles called chloroplasts within leaf cells and are the first to show change. The first ephemeral leaf color change is a gray-green color which most people do not notice due to its quick passage into chlorosis. The chlorotic leaf color change is caused by a decrease in chlorophyll content which exposes some of the auxiliary pigments, many of which are yellowish in color.

The combination of lightening the green leaf color and the exposure of yellows are what is termed chlorosis. General chlorosis can be leaf-wide with some areas between the veins showing more intense chlorosis. With time, chlorosis symptoms can lead to a bleaching (whitening) of the tissue as pigments are destroyed.

The final damage in tissue which was originally chlorotic is necrosis (death), which shows colors ranging from tan to reddish to brown to black, depending upon what is present in the living cells when they were injured and how quickly cell death occurred.

Flecking / Stippling: Flecking, stippling and mottling are words describing the appearance of minute and larger spots on the leaf surfaces. A number of air pollutants and pests can cause flecking. Flecking has three phases on tree leaves. The first is the appearance on the underside of the leaves around the stomates. As the damage progresses to more cells, both the lower and upper side of leaves show flecking.

The second phase of flecking is spots beginning as chlorotic flecks usually progressing to dead spots which are brown or black in color. Bronzing is flecking across a wide area or in larger spots where the tissue color in the leaf spots are light tan to brown in color, sometimes with faint tinges of red or orange.

The final phase of flecking is tissue death in spots of different sizes which can be either brown in color or in higher chlorine exposures can be orange-brown in color.

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Glazing / Wet Spots: One symptom visible after a period of time in leaves is a "silver" or "glazed" appearance. This symptom is a result of increasing air spaces just below the leaf epidermis (surface layer). Light reflectance off the leaf is changed by the air spaces leading to a glazed appearance. These air spaces can become saturated with water generating a symptom known as wet spot or water logging. As tissue is damaged and water is pulled from the cells, cell wall areas and intercellular air spaces become saturated with an acidic and high salt content solution. These spots can continue to develop into reddish-brown to black dead spots.

Scorching: Scorching is the death of large areas of leaf tissues, usually initially concentrated at leaf margins and tips. The more exposure to chlorine, the greater leaf volume damaged and the more scorching visible. Large areas of necrotic tissue (dead areas) may appear to have developed from many flecks and dead spots coalescing into large dead spots, or from massive dead spots developing spontaneously.

Scorching can appear quickly and progress until almost the entire leaf is brown. Whole leaf browning and death can lead to the leaves staying on the tree if the injury was fast, or can initiate a fast senescence and abscission process if the leaf tissue and the twig had a longer time to react. Either result is a loss of leaf area to make food for the tree. Partial scorching can lead to leaf cupping and distortion.

Let's Compare

Chlorine gas is not a rare air pollutant, but is not considered common. As such, many diagnostic guides choose to compare chlorine gas symptoms with other air pollutants. Because trees only have a few reactions to a host of damaging agents, and because the massive number of tree-damaging agents impact the most sensitive and visible portions of tree biology, chlorine gas exposure can be said to mimic other types of pollution.

For example: rapid interveinal leaf chlorosis and death is like sulphur dioxide; marginal and leaf tip dieback (scorching) is like hydrogen fluoride; leaf tissue bleaching is like ozone; and, the overall leaf damage is like acid rain. If good symptomology is developed for these other pollutants, then comparison of damage might be initially helpful in diagnosing chlorine gas damage to tree leaves.

Internally Yours

Internally chlorine gas initiates a variety of changes within tree leaves. These internal changes are concentrated around photosynthesis and physical changes to leaf surfaces which impact water content. The short term changes in leaves arise from the lowering of pH as cell spaces become more acidic. Long term leaf changes arise from the transformation and accumulation of chlorides. Note that chloride concentrations in tissues are NOT a proxy to determine damage extent or exposure concentrations. The primary impact of chlorine gas is the entrance into the stomate and its dissolving in the cell wall water, allowing chlorine (in several damaging forms) to reach living cell membranes. Membrane permeability is altered and osmotic changes occur disrupting both chloroplasts and mitochondria. Cell regulatory links become severed and fail leading to cell death.

Internal impacts of chlorine gas on tree tissues are concentrated around the photosynthetic systems and tissue water control.

Photosynthesis: Photosynthesis is the first process to be impacted with exposure to chlorine gas. Photosynthesis is extremely sensitive to even minor water content changes, and the water context changes from chlorine damage can be massive. The efficiency of the entire process falls rapidly as compounding damage accumulates. As water contents change, stomate control is lost and acidification of cell wall water disrupts carbon-dioxide uptake and cell wall permeability in both directions (out and in

of the cell). The photosynthesis processes are reduced by the loss of materials leaking out of the cell, the lack of chloroplasts generating a gradient of protons, and the disruption of chlorophyll maintenance and production sequences.

Magnesium atoms, used as the centerpiece of working chlorophyll molecules are replaced (bumped) by hydrogen atoms in the acidic environment, rendering chlorophyll inoperative. The repair enzymes which would normally correct structural problems in the chlorophyll are also shut-down. The primary full sunlight chlorophyll molecule (chlorophyll a) breaks down four times more quickly in acidic conditions than the shade dominant chlorophyll (chlorophyll b). Other pigments which protect chlorophyll from sunlight or chemical damage are themselves destroyed. The blue colored pigments are especially vulnerable to chlorine gas exposures.

The amount of light during and immediately after exposure modifies damage levels seen in leaves. Bright light tends to allow more leaf damage to be visible, while a dark period reduces damage potential. Figure 1. Most light effects are due to stomatal opening and closing which affects leaf access by chlorine gas.

Leaf Surfaces & Water Content: As the acidity increases in the leaf (pH falls), the living cell membranes are damaged. The surface structures on the membranes are disengaged or destroyed. As membranes become more disabled, more materials hoarded for essential processes leak from the cells. As more materials build-up outside the cells, more water flows out of the cells and builds-up in the intercellular spaces. Without positive water pressure, the cells begin to pull away from the cell walls and collapse. Water loss from cells, and from the leaves in general, cause stomates to close.

Turgor loss allows the wilting, collapse, cupping and shrinking of leaves. Water loss from the leaves generates a severe drought-like desiccation. The epidermis cells separate and lose continuity with each other from both internal and external acidification. Desiccation from damaged leaf surfaces (epidermis, tricombs, and cuticle) can influence growth for more than a year after chlorine gas exposure. A final interaction occurs as the water loss accelerates from leaf surface damage, the leaf surface wettability increases, allowing for longer moisture residence times on the leaf which increases leaking of materials and increases the potential for successful pest entrance.

Whole Tree Growth

On a whole tree basis, the precipitous drop in photosynthesis and continued desiccation leads to food allocation problems and measurable growth changes lasting as much as five years. The most noticeable impact with trees not killed outright is an early on-set of senescence in the year of exposure. A shortened growing season leads to less food produced and stored, as well as modified defensive processes during a longer warm period without active crown productivity. On evergreen and deciduous trees, leaf longevity is compromised and decreased. On otherwise stressed trees with evergreen leaves, the loss of additional perennial leaves is a tremendous resource loss. See Figure 2.

Stem growth is greatly reduced from chlorine gas exposure. In one study, tree crowns initially reacting with mostly chlorotic symptoms had 25% less stem growth after three years, while trees initially with mostly necrotic crowns had 55% less stem growth after three years. In trees with greater than 85% initial defoliation, death was the usual result. Reproductive tissues are also severely damaged by chlorine gas exposure. In pines, cone production has been cited as greatly reduced (60% less) following chlorine gas exposure. Tree fruiting may be impacted through premature fruit abortion upon exposure.

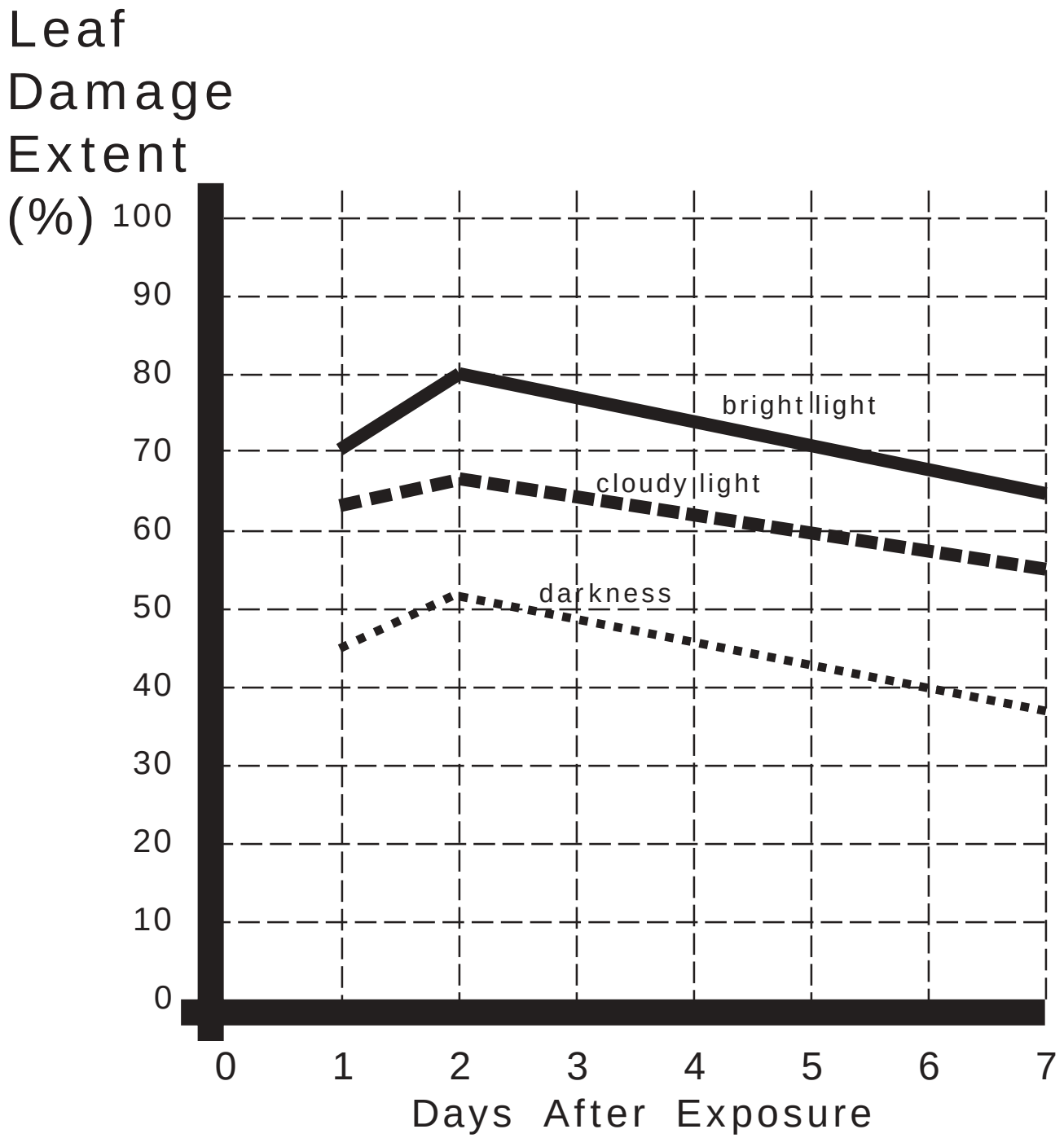

Figure 1: Laboratory chlorine exposure experiment examining plant leaf damage (100% is complete leaf damage and 0% is no leaf damage) over a one week period. Chlorine gas exposure was 20 ppm for 10 minutes under three light levels.
(after Griffiths & Smith, 1990).

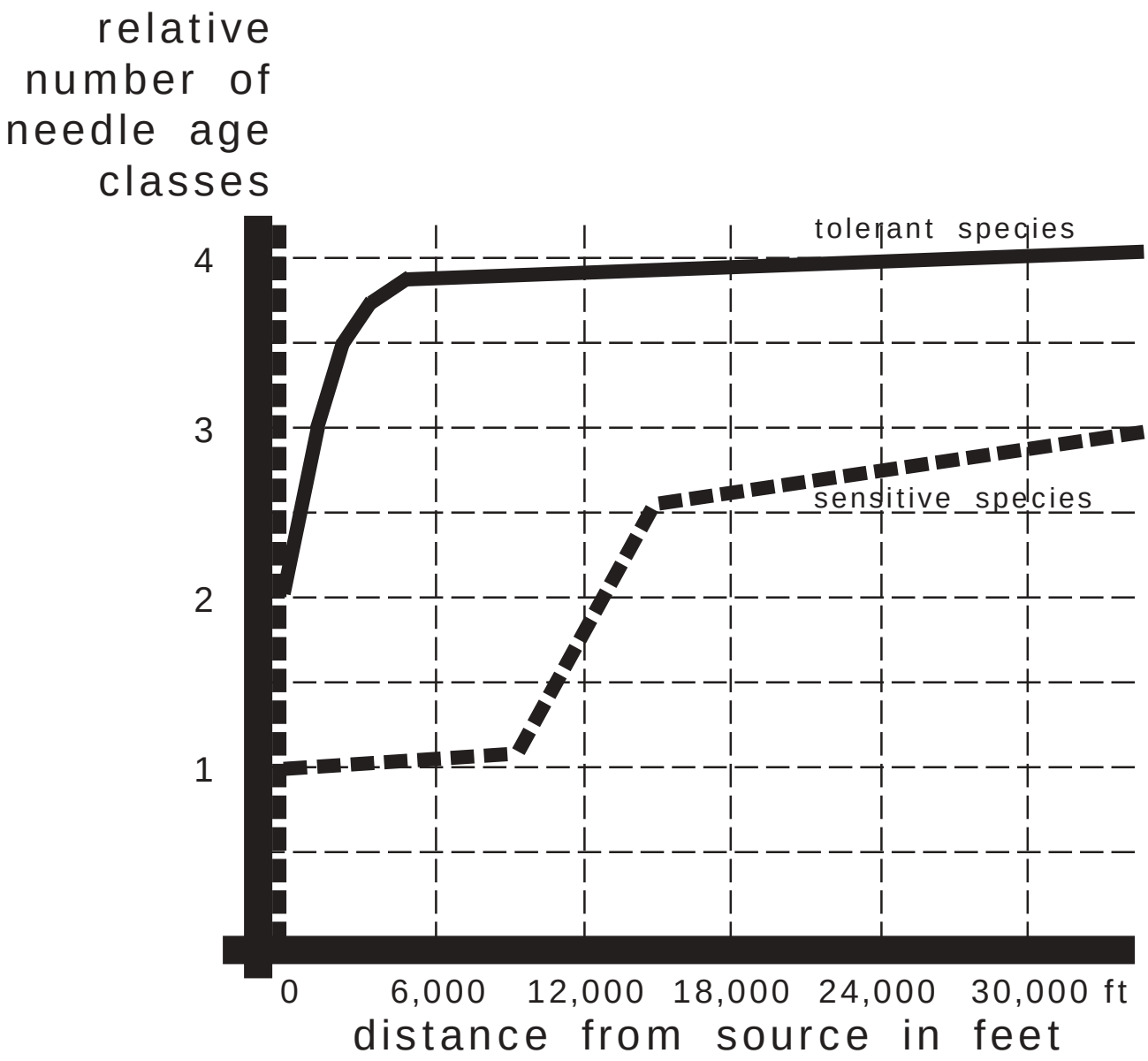

Figure 2: Historic needle age classes present for a sensitive and tolerant gymnosperm species downwind of chlorine gas source after 2 years from exposure. The fewer age classes, the more damage from exposure.
(after Schreuder & Brewer, 2001)