Visible symptoms of fluoride injury [to vegetation]

Alan Davison,
AES, Ridley Building,
University of Newcastle,
Newcastle upon Tyne NE1 7RU
England

Leonard Weinstein,
Boyce Thompson Institute,
Tower Road, Cornell University,
Ithaca NY

Fluoride released in the environment due to water fluoridation or other pollution, damages vegetation.

Fluoride-induced symptoms have been described in many reviews (Weinstein and McCune 1970; Weinstein and McCune, 1971; Thomas and Alther 1966; Brandt and Heck 1977; Treshow and Pack 1970; Guderian et al. 1969; Hindawi 1970; Thomas 1961). The most recent account, with illustrations is: Weinstein, Davison & Arndt, 1998 (Recognition of Air Pollution Injury to Vegetation, Ed. Richard B Flagler). The basis for the following description is Weinstein and McCune (1971) plus our own experience in the field in several countries.

Gaseous fluoride enters the leaf through the stomata (=pores) then it dissolves in the water permeating the cell walls. The natural flow of water in a leaf is towards the sites of greatest evaporation, which are the margins and tip. Carried by the water, the fluoride concentrates in the margins and tip so it is these areas that generally are the first to show visible injury. Clearly this concentration mechanism is one reason why fluoride is so toxic to plants but there is an important corollary; most of the leaf may have very little fluoride present and may function normally in terms of assimilation.

Generally, leaves are most sensitive when they are young and still expanding. Once fully developed they may be many times more resistant. Therefore symptoms are more often seen in young, expanding leaves. Where fumigation is periodic, symptoms may reflect this as only those leaves that are at the sensitive stage of development when the fumigation occurs will develop injury. The rate at which symptoms appear depends on the weather. There can be a considerable lag

between the time of exposure to the fluoride and the development of the symptoms.

Exposure to a high concentration causes necrosis of part or even the whole of the leaf. The term necrosis comes form the Greek nekros meaning a dead body. The tissues die. The initial stages vary with species and both the speed of development of the symptoms and their appearance depend on the weather.

In most monocotyledonous (narrow-leaved species including grasses and lilies) plants, the initial symptom is the development of chlorosis (= yellowing) at the tips and margins of elongating leaves. In some the tissues take on a "water-soaked" appearance that looks very like early frost injury, then the tissues desiccate and change colour. In some species the dead, necrotic areas are pale white to tan, in others they are brown and they may be black (eg in Populus spp.) or have reddish tinges. Characteristically, there is a dark brown margin along the basal part of the necrotic area. This line of demarcation is very useful in identifying multiple exposures.

The necrotic area is sharply delineated from the healthy portion of the leaf blade by a narrow band of chlorotic tissue sometimes streaked with red as in some varieties of Sorghum. Dead, dry pieces of leaf may become brittle and fall off, giving the leaf a tattered appearance. This is common in Chinese apricot and Italian prune and many Populus varieties. When very young leaves are injured in this way the resulting leaf may only be a fraction of the normal size and completely mis-shaped.

Pine species (Pinus) vary greatly in sensitivity. For example, young ponderosa pine (Pinus ponderosa) needles first exhibit a lightening in color which turns light brown to reddish-brown at the tip and progresses basipetally along the needle. The discoloration is often accompanied by narrow, dark banded zones, which may be the result of intermittent exposures to fluoride spaced at different periods. Dark bands may also occur at the interface of necrotic and healthy tissues. Needles are born in groups (2, 3, 5 depending on species). They tend to be marked to the same extent.

Although necrosis is the symptom most frequently referred to in texts, often being called tip burn, other symptoms are at least as common or, in some areas, more common. In dicotyledonous ("broad-leaved") species the initial symptom of fluoride effects on leaves is usually chlorosis of the tip, which later extends downward along the margins and inward toward the midrib. This chlorosis becomes more intense and extensive with prolonged exposure until the midrib and some veins appear as a green arborescent pattern on a chlorotic background. Continued exposure may lead to the tip becoming necrotic and falling off, leaving the leaf notched.

The symptoms produced in corn (Zea mays), Sorghum, and some other grasses begin as scattered chlorotic flecks at the tips and upper margins of middle-aged leaves. As the symptoms progress, the flecking becomes more intense and extends downward, especially along the margins. The amount of chlorosis diminishes from the tip downward and from the margins toward the midrib. A greater degree of chlorosis is usually present at the arch of the leaf and wavy areas of the margin. At high fluoride concentrations, there is less chlorotic flecking and a greater tendency for tip, marginal, and interveinal necrosis, or a transverse necrotic band at the arch of the leaf.

In young, developing leaves of broad-leaved species, and occasionally in petals, the translocation of fluoride to the margins and tips leads to a distorted shape. This may be accompanied by chlorosis at the margins and/or necrosis . This occurs because cells in the mid parts of the leaf have low fluoride and expand normally but those on the margins are slower-growing so the leaf buckles and distorts, becoming cupped and concave or convoluted like a savoy cabbage.

There is little information about the effects of fluoride on fruits but there are two important examples. Bonte & Garrec described fluoride-induced distortion of strawberry fruits (Fragaria). It was cuased by lack of fertilisation of some of the seeds, wich are responsible for hormonal-induced swelling of the fruit. Peach also shows an unusual disorder induced by fluoride called "suture red spot" or "soft

suture" of the fruit. It is characterized by premature ripening of the flesh on one or both sides of the suture toward the stylar (blossom) end of the fruit (Benson 1959; MacLean et al. 1984). The ripening of this tissue considerably precedes that of the normal fruit and is often accompanied by splitting of the flesh along the suture. At harvest, the affected areas are soft and often decomposing.

Finally, although the economic value of injury to a peach crop can be claculated, it is almost impossible to calcualte or predict the effects of injury on other plants. If fluoride kills all of the leaves on a tree then there will, of course, be an effect on subsequent growth. However, apart from this very rare occurrence, there is little or no relationship between visible injury and either growth or longevity. A plant that is visibly injured is not necessarily dying and there have been some cases of spectacular recovery of trees after severe injury. Many that show a significant degree of injury (such as Populus) continue to grow at normal rates. Conversely, just because a plant does not show visible injury it does not mean that there is no effect of fluoride on assimilation or growth. Predicting the effects of fluoride is not a job to be undertaken lightly!

(Weinstein's and Davison's website at *http://www.ncl.ac.uk/airweb/* which contains a number of photos of fluoride induced damage to vegetation.)