Pinot Leaf Curl

Rhonda J. Smith and Larry J. Bettiga UC Cooperative Extension Viticulture Farm Advisors, Sonoma and Monterey Counties; and Douglas O. Adams, Department of Viticulture and Enology, UC Davis

In 2015, low temperatures occurred in the second week of April and symptoms known as Pinot leaf curl appeared in Pinot cultivars across many grape growing regions. Symptoms were seen in coastal and northern interior wine grape growing regions. Pinot leaf curl (PLC) is a nitrogen-related disorder that has been observed in the spring in primarily Pinot noir, Pinot blanc and Pinot Meunier grapevines. The cause of PLC is not fully understood. This article is meant to inform growers, and pest control advisers of the wide range of symptoms that may occur in Pinot cultivars affected by the disorder.

The onset of PLC is associated with prolonged periods of low temperatures and brief episodes of sudden temperature depression at the beginning of the growing season through bloom. Periods of relatively low temperatures caused by consistent daily on-shore air movement in coastal regions can usher in a thick marine layer depressing daytime maximum temperatures. Radiational cooling under clear skies – that may or may not result in a frost event - have also been associated with an increase in incidence of PLC. Studies to quantify the relationship between the onset and severity of symptoms of PLC to temperature thresholds or the duration of hours within a range of temperatures have not been conducted.

Pinot leaf curl does not occur in all Pinot vineyards but in others it is chronic under "normal" spring temperatures. In most years, the symptoms are primarily mild leaf curling. The incidence of the disorder varies by clone within Pinot noir but not consistently between years. Symptoms of PLC usually develop prior to bloom; however in 2015, continued cool temperatures in the north coast resulted in sustained and in some cases the reoccurrence of symptoms in vines that had initial onset in April.

Figure 1

Figure 1: Stunted shoot on Pinot noir vine with small leaves that are dying.

Symptom progression

Symptoms occur in expanding blades and in other rapidly growing tissue, thus timing of symptom onset as well as the length of time that conditions are conducive determines severity and degree of crop loss. Low temperatures for three or more weeks during and following bud break often impacts shoot and cluster development resulting in severely stunted shoots (Figure 1). In some vines, "orphaned clusters" may occur on nodes when the shoot tip dies shortly after growth begins and leaves fail to develop or die, thus only the cluster remains (Figure 2). Shelling – flower loss before or during bloom – is common.

If conditions conducive to PLC occur after the basal region of the shoot has finished elongating and leaves on nodes with clusters have fully expanded, then clusters are less likely to be affected if temperatures drop for only a few days. In that case, leaves distal to the clusters will be impacted and the severity is variable. A downward curling of the leaf blades consistently occurs – as described by the name of the disorder – and is considered a mild form of PLC (Figure 3). The undersides of such blades have a necrotic area on a main vein at which point the blade curls or folds downward (Figure 4).

If low temperatures persist

Figure 2

Figure 2: The shoot has been reduced to a single cluster in Pinot noir after the shoot tip has died (seen above necrotic area on cluster stem).

Figure 3

Figure 3: Leaf blades that are not fully expanded curl downward on the terminal end of a Pinot noir shoot.

then symptom severity increases. Initially, petioles become necrotic followed by blades then leaf abscission occurs. Leaf loss can occur on one to several nodes distal to the clusters. Older leaves that are nearly fully expanded as well as small blades that just separated from the growing tip can be killed. From a distance, affected shoots appear to be stripped of leaves above the proximal (top) cluster (Figure 5). When that occurs, the summer buds that formed in the leaf axil may break and lateral shoots develop in the mid and terminal region of the primary shoot. A necrotic area can be seen on nodes that lost leaves (Figure 6). In severe PLC seen in 2011, necrosis continued to develop in the nodes resulting in partial shoot dieback as well as crop loss depending on proximity to the clusters (Figure 7).

Fungicide applications are not advised

It is common for some growers to associate the necrotic area at the nodes where petioles had been attached with symptoms of Botrytis shoot blight. In 2011, there was a significant increase of PLC in coastal growing regions due to below normal temperatures in April and May. At that time, many growers and pest control advisers first became aware of the disorder. Some applied *Botrytis* active fungicides in an attempt to prevent further symptom development however materials were not effective.

Botrytis shoot blight is caused by *Botrytis cinerea* and requires prolonged, warm moist conditions and free moisture often caused by rainfall. Pinot leaf curl occurs when low springtime temperatures occur and is limited to specific cultivars. *Botrytis cinerea* is a weak pathogen; it is primarily a saprophyte thus it colonizes dead tissue which is present at nodes when PLC causes leaves to abscise. However, the fungus does not cause the necrosis observed on the nodes, thus fungicide applications do not reduce the incidence or severity of Pinot leaf curl.

Nitrogen-related disorders

Pinot leaf curl has similarities to a nitrogen-related disorder in grape cultivars known as false potassium or "spring fever" that may occur when cool weather follows a warm period in the spring (Christensen et al. 1990). False potassium (K) symptoms in leaf blades appear similar to those caused by true K deficiency in that leaf margins become lighter green followed by interveinal chlorosis. As severity increases, the margins become necrotic and leaves may abscise. "Spring fever" symptoms, particularly in Flame Seedless, include shelling of flowers and necrosis of the cluster stem.

Tissue analyzed from symptomatic and non-symptomatic shoots showed that at symptom development, K levels were temporarily deficient in vines with "spring fever" yet returned to normal in later maturing leaves when symptom developed ceased (Christensen et al. 1990). Potassium levels in true K deficient vines continued to decline. False potassium tissues had higher total N and ammonium levels at the time of symptom development and once again, levels returned to normal when symptom development ceased (Christensen et al. 1990). This dynamic indicated that "spring fever" was not a true K deficiency. Nitrogen metabolism problems were suspected to be involved in symptoms development given that ammonia and other nitrogen compounds were associated with symptoms.

Elevated levels of the polyamine putrescine (1,4-diaminobutane) have been associated with several conditions that lead to death of plant tissue. Thompson Seedless leaf blades with symptoms of true K deficiency or with "spring fever" contained several-fold more putrescine than non-symptom leaves (Adams et al. 1990). When "spring fever" symptom development had ceased, the concentration of putrescine in older, symptomatic blades was far greater than in recently matured blades more distal to the clusters (Adams et al. 1990).

Figure 4

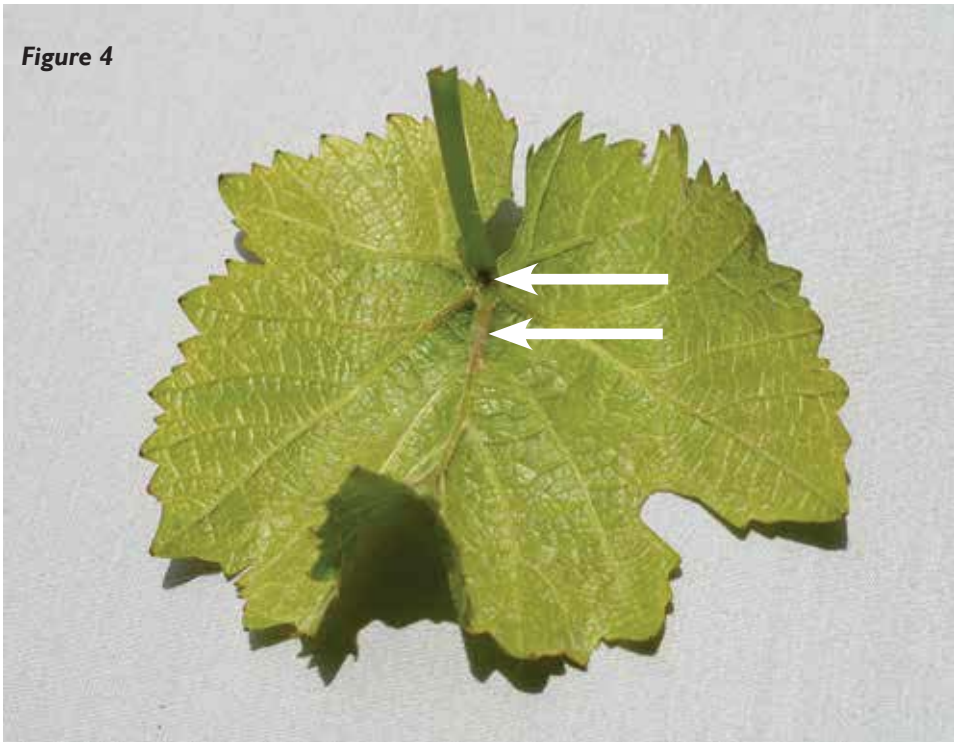

Figure 4: Arrows indicated necrotic areas in a vein on the underside of a Pinot noir leaf blade as well as at the point of attachment of the petiole.

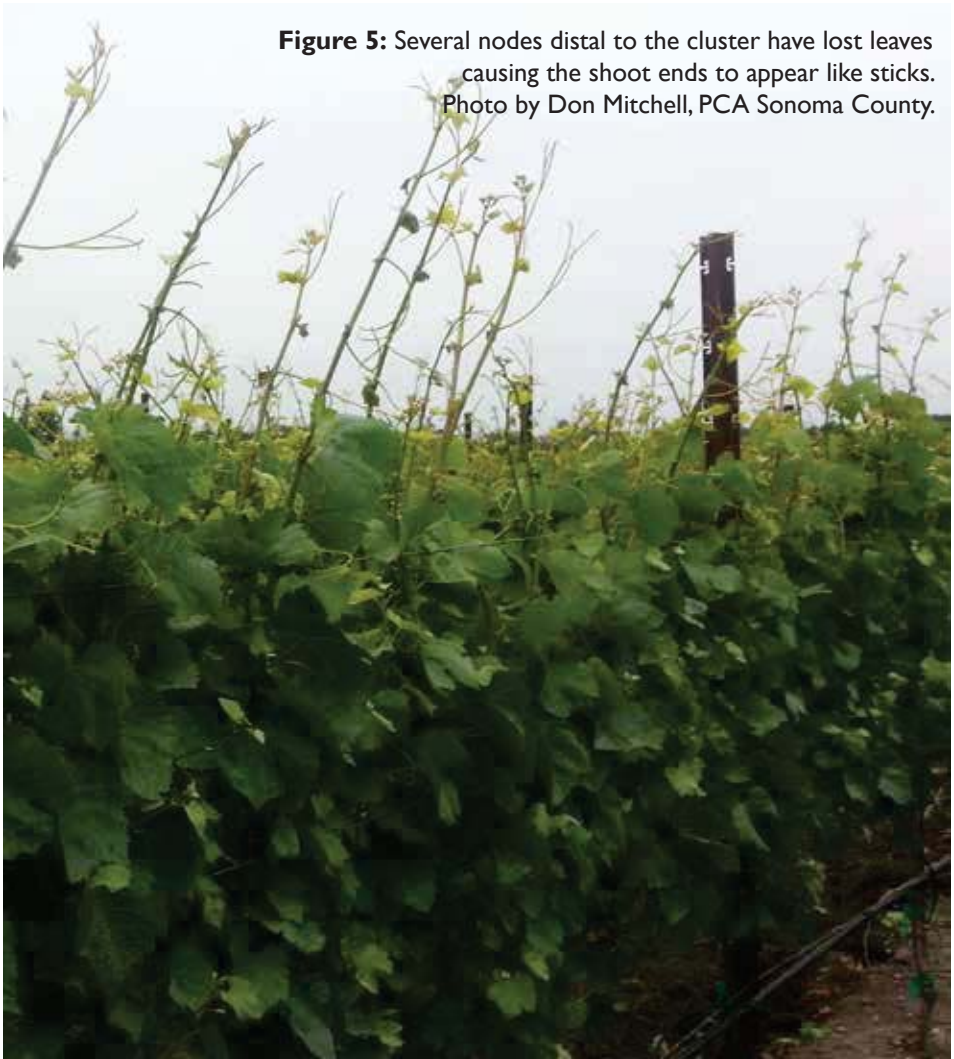

Figure 5: Several nodes distal to the cluster have lost leaves causing the shoot ends to appear like sticks. Photo by Don Mitchell, PCA Sonoma County.

Given those findings, putrescine was suspected of being related to PLC. Early in the season when PLC occurs, ammonium and glutamine are sourced from stored nitrogen and carbon in the permanent vine parts. The amino acid arginine is a major form of soluble nitrogen in *Vitis vinifera* roots. It is converted to glutamine which is found in both xylem and phloem exudate at bud break. After dormancy, bud development and shoot growth rely on remobilization of nutrient reserves in the xylem from starch, amino acids (primarily glutamine) and sugars until a net gain in photosynthate occurs around bloom (Roubelakis-Angelakis et al. 1979). Arginine can be converted to putrescine if arginine decarboxylase is induced by stress or disease conditions (Adams et al. 1990).

In 2012 we investigated the relationship between elevated ammonium and glutamine levels in grape leaf tissue and PLC and we compared the levels of putrescine in symptomatic and non-symptomatic leaves. A total of 29 paired samples were collected in Pinot noir, Pinot Meunier, Pinot gris and Pinot blanc vineyards; a single pair came from the susceptible cultivar Schioppettino. Each sample consisted of 5 blades with attached petioles.

No correlation could be seen between the amount of ammonia or glutamine in symptom versus non-symptom leaves (data not shown). Putrescine was elevated 2 to 25 fold in symptomatic blades compared to non-symptom blades with the exception of two pairs, where the putrescine levels were equal (Figure 8).

The role putrescine has in symptom development of PLC is not clear. Elevated putrescine was not observed in every case of symptom versus non-symptom leaves as is seen in cases of true potassium deficiency and "spring fever". Secondly, in some sites, the level of putrescine seen in many of the symptom leaves was well below the level observed in non-symptom leaves from other vineyards.

Figure 6: A necrotic region remains at the node from which a leaf detaches from the shoot.

Figure 7: A dead leaf hangs from each shoot on which the tips died back to the cluster-bearing node.

Nitrogen metabolism in leaf blades with symptoms of PLC is different than in blades that do not have symptoms. However, there does not appear to be a relationship with applied nitrogen (fertilizer) to the incidence of symptoms or putrescine levels. Leaf pairs collected from sites that had received annual applications of N did not consistently show elevated levels of putrescine in symptomatic blades. In some sites which had not received N fertilizer, putrescine was greater in symptomatic blades than in non-symptomatic blades (data not shown).

The polyamine putrescine has been implicated as a toxic metabolite leading to leaf chlorosis and necrosis in potassium deficiency and "spring fever." Pinot leaf curl is also associated with putrescine. However, given the range of putrescine observed in symptom and non-symptom leaves from various Pinot cultivars and clones from different sites, its presence remains correlative relative to symptoms and a causal role remains to be established.

Figure 8

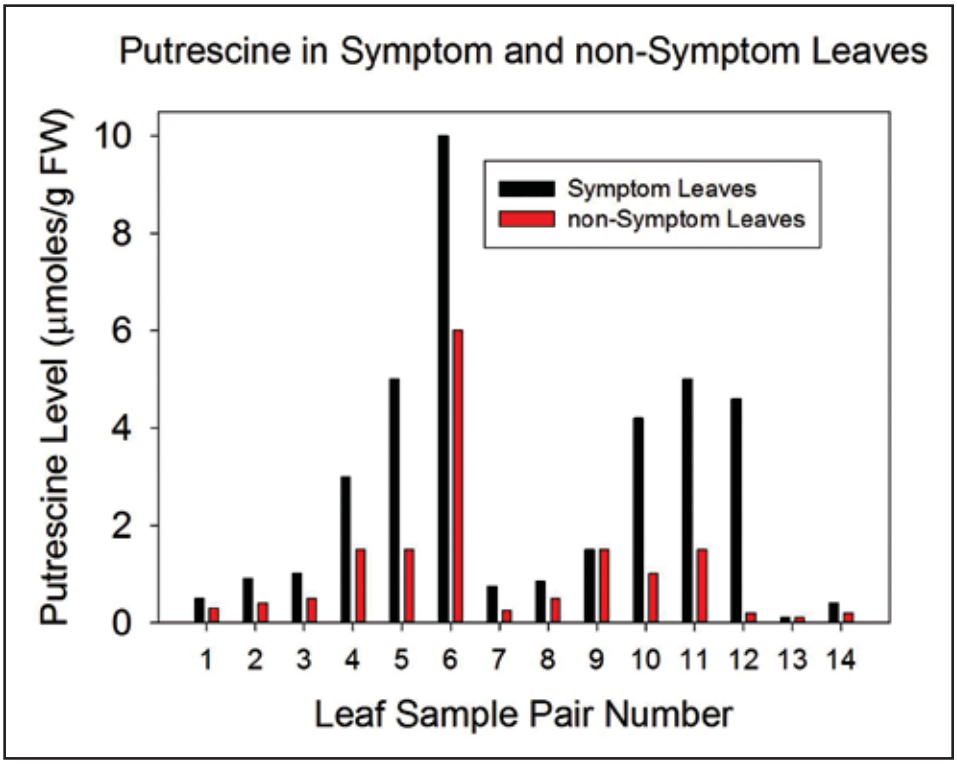

Figure 8: Putrescine is elevated in most but not all of the symptomatic blades compared to non-symptomatic blades collected from 14 vineyards with Pinot leaf curl.

Literature Cited:

Adams, D.O., K.E. Franke and L.P. Christensen, 1990. Elevated putrescine levels in grapevine leaves that display symptoms of potassium deficiency. Am. J. Enol. Vitic. 41:121-125.

Christensen, L.P., J. Boggero, and M. Bianchi. 1990. Comparative leaf tissue analyses of potassium deficiency and a disorder resembling potassium deficiency in Thompson Seedless grapevines. Am. J. Enol. Vitic. 41:77-83.

Roubelakis-Angelakis, K.A., and W.M. Kliewer, 1979. The composition of bleeding sap from Thompson Seedless grapevines as affected by nitrogen fertilization. Am. J. Enol. Vitic. 30:14-18.