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ARTÍCULO

Anatomical responses in apricot wood structure caused by *Valsaria insitiva*

Oľga Mišíková[1] https://orcid.org/0000-0003-2631-5628
Barbora Slováčková[1] https://orcid.org/0000-0002-7327-6302♠
Pavol Hlaváč[2] https://orcid.org/0000-0002-3730-1873

[1]Technical University of Zvolen. Faculty of Wood Sciences and Technology. Department of Wood Science. Zvolen, Slovakia. https://ror.org/00j75pt62
[2]Technical University of Zvolen. Faculty of Forestry. Department of Integrated Forest and Landscape Protection, Zvolen, Slovakia. https://ror.org/00j75pt62

♠Corresponding author: xslovackova@tuzvo.sk

Abstract:
Understanding anatomical responses in various tree species to fungi causing gummosis is limited. In this article, wood samples from an apricot tree (*Prunus armeniaca*) with severe gummosis and branch dieback were studied macroscopically and microscopically to determine the response of woody tissues to the infection. The fungi species *Valsaria insitiva* was determined visually by the signs of the disease and by cultivating clean cultures. Macroscopical examination found wedge and arch shaped necroses running along the growth rings under the bark necroses and sunken lesions. Microscopical examination found small black fruiting bodies of the fungus on the bark. Vessels and libriform fibers were blocked by gum, thus blocking the water and nutrients flow to the branches and causing the dieback. Sites with fungi infection were compartmentalized and this was discussed according to the CODIT model. Oxidized ray parenchyma was visible in tangential and cross sections of the wood; calluses were covering the infected areas. Compartmentalizing the infections by releasing gum into vessel lumina, restricting the flow of nutrients and water in the tree thus drying it out is a way of protection of the tree against the pathogens. Apoplexy of apricots is an acute disease and can be very extreme in some cases. The studied tree had signs of compartmentalization of the infection, but the infection was spreading faster than new tissues were formed.

Keywords: Apoplexy, gummosis, *Prunus armeniaca*, *Valsaria insitiva*, wood anatomy.

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Introduction

Trees are in general always at risk of biological degradation. During recent years, extreme weather events have been the cause of damage to trees. For fruit bearing trees especially, frost during blooming, strong winds, heavy rain and hail can be very damaging. Strong wind and rain splashes are one of the many elements responsible for spreading bacteria and fungi among the trees (Boothby 1983, Hričovský *et al.* 1990, Pokharel 2013). Freezing temperatures increase the severity of *Pseudomonas syringae* pathovar *syringae* infection of leaves of sour cherry and branches of apricot and peach (Weaver 1978, Klement *et al.* 1984, Süle and Seemüller 1987,). The disease name is attached to the fungus name, and the fungal names change with new taxonomic research, thus there are different names for the same disease such as Leucostoma canker, Cytospora canker, Valsa canker or Perennial canker (Pokharel 2013). We will refer to the disease according to the cited references.

Most common diseases in apricot trees caused by fungi and bacteria are sharka (plum pox virus), necrotic ringspot, browning of the leaves caused by *Gnomonia erythrostoma*, moniliosa, early dieback caused by *Cytospora cincta* and *Pseudomonas syringae* and many more. Symptoms of bacterial canker caused by *Pseudomonas syringae* are very similar to symptoms of a *Cytospora* infection (Hričovský *et al.* 1990). According to Ivanová (2010), withering and dieback of branches can be caused by *Monilinia laxa* (Aderh. & Ruhland) Honey or Verticillium fungi.

Cytospora canker caused by *Cytospora cincta* (teleomorph *Leucostoma cincta*) and *Cytospora leucostoma* (teleomorph *Leucostoma persooni*) and bacterial canker caused by *Pseudomonas syringae* reduce the vigor and longevity of numerous *Prunus* spp. (English *et al.* 1982, Luepschen 1981, Rozsnyay and Klement 1973, Wensley 1971, Willison 1936). *Cytospora* spp. can be highly virulent and destructive pathogens on cultivated *Prunus* (Biggs 1989) in commercial orchards, *Populus* species (Kepley and Jacobi 2000) in forestry and *Acer* spp. in the landscape (Adams *et al.* 2006). *Leucostoma persoonii* has been reported on *Cydonia*, *Malus*,

Pyrus, *Sorbus* species, and *Leucostoma cinctum* has been reported on *Cratageus* and *Robinia* (Biggs and Grove 2006). *Cytospora pruinosa* was reported even on olive trees (Petrović *et al.* 2023) and various species of Cytospora have also been reported on walnut trees (Fan *et al.* 2015).

The first sign of an infection by the mentioned fungi and bacteria is gummosis, other symptoms include leaf wilting, bark lesions with dead phloem and cambium, discoloration of the xylem, wood necrosis, sunken discolored areas and lesions (Biggs and Grove 2006, Lawrence *et al.* 2018, Petrović *et al.* 2023, Hričovský *et al.* 1990). Bark above the infected cambium may appear yellow, brown, reddish brown, gray or black, becoming watery and odorous as the tissues deteriorate (Fan *et al.* 2019, Pan *et al.* 2020). Over the spot with bark necrosis, the leaves lighten in colour, get smaller or wilt. Under the bark periderm, in the browned bark tissue, there are small, brown-black fruiting bodies of the Cytospora fungus (Pokharel 2013). The symptoms vary with host species and stage of the disease (Fan *et al.* 2019). The external symptoms of the disease are very well documented, however, the observation of the anatomical response and signs of the disease in the wood is scarce and needs to be studied.

The fungus releases toxins into the tissue; these toxins have presumably the ability to express the water from the tissues which explains noticeable branch drying out (Hričovský *et al.* 1990). Severely affected trees exude large amounts of gum from these lesions, especially following extended periods of rainfall (Daniell and Chandler 1982, Weaver 1974). Gum production is a natural host response to irritation, but gum production due to infection by *Leucostoma* spp. is excessive (Biggs and Grove 2006). The impact of excessive gum production on the hydraulic processes in the tree and its appearance within the wood anatomy remains uncharacterized.

As it was pointed out, observation of the infected wood and a description of the wound occlusion and compartmentalization processes on a microscopical level is missing in the research of the disease. It could be a key part in devising protective measures. The aim of this

article is the macroscopical and microscopical observation of apricot (*Prunus armeniaca*) wood samples with signs of branch dieback and gummosis. A determination of the specific fungus causing the symptoms was performed. Compartmentalization of the infected areas was discussed according to the CODIT model (Shigo 1984).

Materials and methods

This study is designed as a case-based observational analysis of a single apricot tree exhibiting symptoms of gummosis and branch dieback. Due to the nature of the study, the findings are intended to provide anatomical insight into host–pathogen interactions rather than statistically generalizable conclusions.

Description of the tree

The apricot (*Prunus armeniaca* L.) tree is growing in Banskobystrický region, Slovakia and it is planted next to the east side of a detached family house. The tree was planted in 2015 as a 3-year-old sapling, it is standing alone. The owner formed the branches of the crown into espalier palmetto shape. The apricot tree was heavily damaged during early onset blooming by early morning frosts in March and April 2023 and according to the owner, this happened almost every

year since the planting of the tree. The frost damage was so extensive in some years, that nearly all flowers withered, and the tree bore very few fruits, or it did not bear any fruit.

Gummosis on the tree was first observed in 2021, but the oozing gum had a light colour and it occurred only in few spots. In 2023, it rained or drizzled often during March and April, and it was also very windy. In that year, the oozing gum appeared on most of the branches and had a darker amber or dark brown colour. The gum appeared first on the flower buds damaged by frost (Figure 2a), and it soon appeared on the branches all over the tree as well. The places where the gum was observed were flower buds damaged by frost (Figure 2a), existing bark necroses, pruning wounds (Figure 2b), and small bark wounds from frost damage. The gummosis worsened every time it rained.

Sample material collection

The sample material was collected from the lower layers of the branch system. Sites with bark necroses and dark brown oozing gum were selected as sample material in March and April 2023. At first, young twigs with dark brown gum were collectedand later, as the owner pruned the infected branches, more sample material with older wood was collected. Sample material is shown is Figure 1. The sample material branches were collected right after they were cut and the macroscopical examination was performed on selected samples on the following day.

Figure 1: Sample material.

The scale bars in the lower right corners of the figures represent 1 cm.

Determination of the fungus species

The fungus was determined visually and microscopically. Visual determination was performed based on the characteristic symptoms of apoplexy (sudden withering, necrosis of the bark accompanied by typical gummosis, presence of black lentil shaped fruiting bodies). Microscopic determination was performed by cultivation of clean cultures according to Jaklitsch *et al.* (2015).

Small pieces of bark and wood were cut from the wounds and their vicinity with a transplantation knife. These were partially pressed into Malt Extract Agar poured into Petri dishes. After inoculation, the samples were incubated at a constant temperature of 23 °C and at an alternating light regime (12 hours or cold light and 12 hours of darkness). Grown clean

cultures were detected in deionized water, Congo red was used as the stain. The samples of the cultures were examined by Olympus SZ 60 microscope and NLCD - 120 E digital microscope.

Macro- and microscopic examination

The selected material was examined macroscopically with a Leica stereoscope. During the macroscopical examination, parts of the sample material were selected for microscopical observations. The selected parts were carefully cut out of the samples and embedded in epoxy resin. After the epoxy resin was fully cured, the samples were cut on a sledge microtome into 15 - 20 μm thick microsections. The microsections were stained with Safranin and a combination of Astra Blue and Safranin. Some microsections were left unstained in their native state. These stains are among the most important for wood anatomists; they are widely used and call for minimum reagents and techniques (Tardif and Conciatori 2015).

A combination of Safranin and Astra Blue is used to detect early stages of white rot, i.e., selective delignification. Safranin stains lignin regardless of whether cellulose is present, whereas Astra Blue stains cellulose only in the absence of lignin (Srebotnik and Messner 1994, Schwarze and Engels 1998). The microscopical observation was performed on a light microscope (Zeiss AxioLab).

During microscopic observation, vessels of unusual shape and size were discovered in one sample. These “unusual vessels” had an irregular lumen and were later identified as traumatic gum ducts according to Setia (1983) and Carolina and Kusumoto (2020). Lumina of these traumatic gum ducts and normal vessels was measured on transverse sections in both tangential and radial directions. According to IAWA List of Microscopic Features for Hardwood

Identification (Alfonso *et al.* 1989), 25 vessels should be measured at their widest part of the opening. Apricot (*Prunus armeniaca* L.) is a ring-porous wood, so only the larger size class of vessels was measured.

Results and discussion

Determination of the fungus and infection

The fungus causing the apoplexy symptoms on the apricot tree was determined as *Valsaria insitiva* (Tode) Ces. & De Not, syn. *Leucostoma cinctum* (Fr.) Höhn. (syn. *Cytospora cincta* Sacc.). This ascomycete fungus is a common causal agent of apricot apoplexy with gummosis bark and branch dieback, it lives parasitically in the infected tissues. A slower progression of the disease is characteristic of these pathogens.

Apoplexy or sudden early drying, a so-called stroke is a serious disease of apricot trees. The main symptom is sudden withering of infected branches, sometimes even whole trees during spring (while the trees are in bloom, or after blooming, even during ripening of fruit). Predisposition to apoplexy is given primarily by the tree variety and growth conditions of the tree. The development of the disease is often acute; the whole tree or its parts dry out very fast, sometimes even within several days. A rare chronic form of apoplexy causes the apricot trees to die slowly in the span of several years. It is manifested by a weak chlorosis (yellowing) of the leaves, reduction of the leaf blade, lenticel enlarging, early leaf fall, cessation of the

vegetative growth, water sprout growth, and typical gummosis. Gummosis is a symptom of wounding of the tree. It can appear on branches and on the trunk. Infected branches and trunks can develop bark peeling from the wood and tumor wounds. The transition from the necrotic to the healthy part might not be visible. The latter cause phloem necrosis and cambium dieback.

Valsaria insitiva attacks mainly the bark of apricot trees. The entrance of the infection are dead cells of the bark. These cells die because of damage to the tree by strong frost or other mechanical damage. Mycelium of the fungal infection spreads from the damaged bark tissues to the healthy bark. The symptom accompanying the disease is releasing gum - at first clear and later yellow orange to orange, brown in color. The infected bark dies and subsequently typical cancer like wounds are created (Zacha *et al*. 1989). The progression of the gum discoloration as well as the cancer like wounds on the bark were found on the researched apricot tree. The gum discoloration is documented in Figure 2a and Figure 2b (clear gum) and Figure 1 (orange gum).

The first symptom of the fungal infection is dying of the bark in winter, in the vicinity of buds on sprouts growing in summer (Zacha *et al.* 1989). Small, black lentil shaped fruiting bodies (pycnidia, shown is Figure 3c) grow from beneath the bark on the damaged bark (conidial state). Perithecia grow during spring. The infection itself spreads further by ascospores and conidia. When the mycelium grows through the whole circumference of the infected branch, this branch dies quickly above the infection site (Zacha *et al.* 1989). As it was stated by Hluchý *et al.* (1997) growing necrotic wounds causes damage to conducting tissues.

Macro- and microscopical examination of the wood

Macroscopic examination of the collected samples revealed gum oozing from beneath the bark on freshly cut twigs and branches, as displayed in Figure 2c. Branches which were already dried out but still had hardened gum on the bark, revealed bark detaching from the wood. The detached zone ran along the darkened areas of the wood (Figure 2d and Figure 2e, black arrow heads). Black arrow in the Figure 2d points at a sunken lesion on the branch. A very distinct callus looking like a growth ring is walling the sunken lesion off (white arrow with a black outline in Figure 2d). Soltaninejad *et al.* (2017) described multiple shapes of necroses in peach and apricot trees with gummosis, defoliation, and dieback caused by *Botryosphaeriaceae* and *Phaeoacremonium* species: wedge-shaped, irregular-shaped, black spots, brown to black wood streaking and arch shaped necrosis.

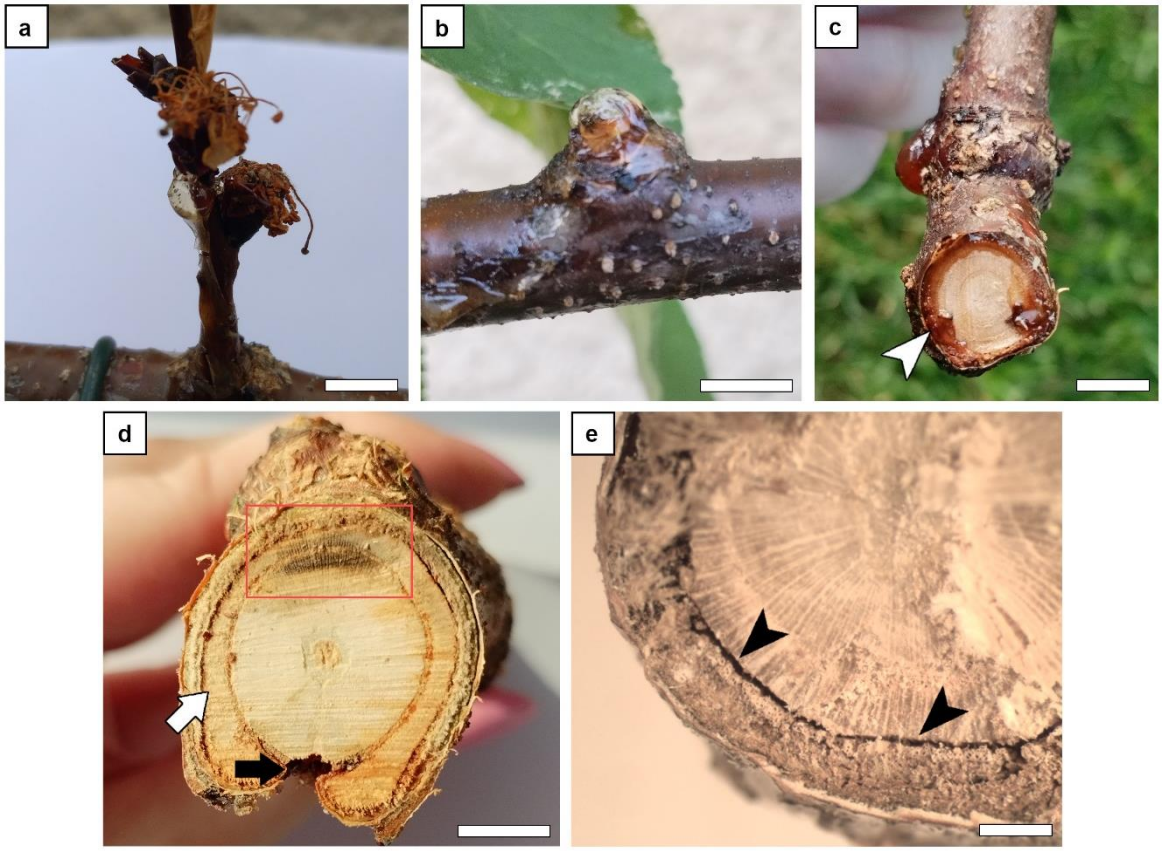

Figure 2: Image (a) shows light coloured gum on a flower bud damaged by frost, image (b) shows light coloured gum covering a pruning wound, image (c) shows dark coloured gum on

the side of the branch and gum flowing out from beneath the bark. Image (d) shows a healing necrosis and image (e) shows bark detached from wood in a branch. The scale bars in A - D represent 1 cm and 3 mm in E.

In the infected apricot tree observed in this paper, wedge shaped and arched necroses were observed on cross sections of the infected branches. The arched necrosis is showed in Figure 2d (red rectangle) and a part of a wedge-shaped necrosis is visible in Figure 3a.

A bigger magnification of the callus covering a necrotic area of wood as well as a microsection of this area are shown in Figure 3a and Figure 3b. The callus wood has a pale beige colour, whereas the infected wood being walled off has a very dark brown colour. The microscopic section of this area is displayed in the Figure 3b. On the left-hand side of the is the callus and the infected dark brown wood in on the right-hand side of the image. This microsection was not stained with dyes, the natural colours of the microsection are visible. A gap between the callus and infected wood is clearly visible, it is denoted with a black arrowhead. Multiple black spots are visible in the callus wood (denoted with black arrows). White arrowheads with a black outline point at an area of the wood overflowed with the gum. The gum is clearly visible as a reddish-brown substance filling out the lumina of vessels.

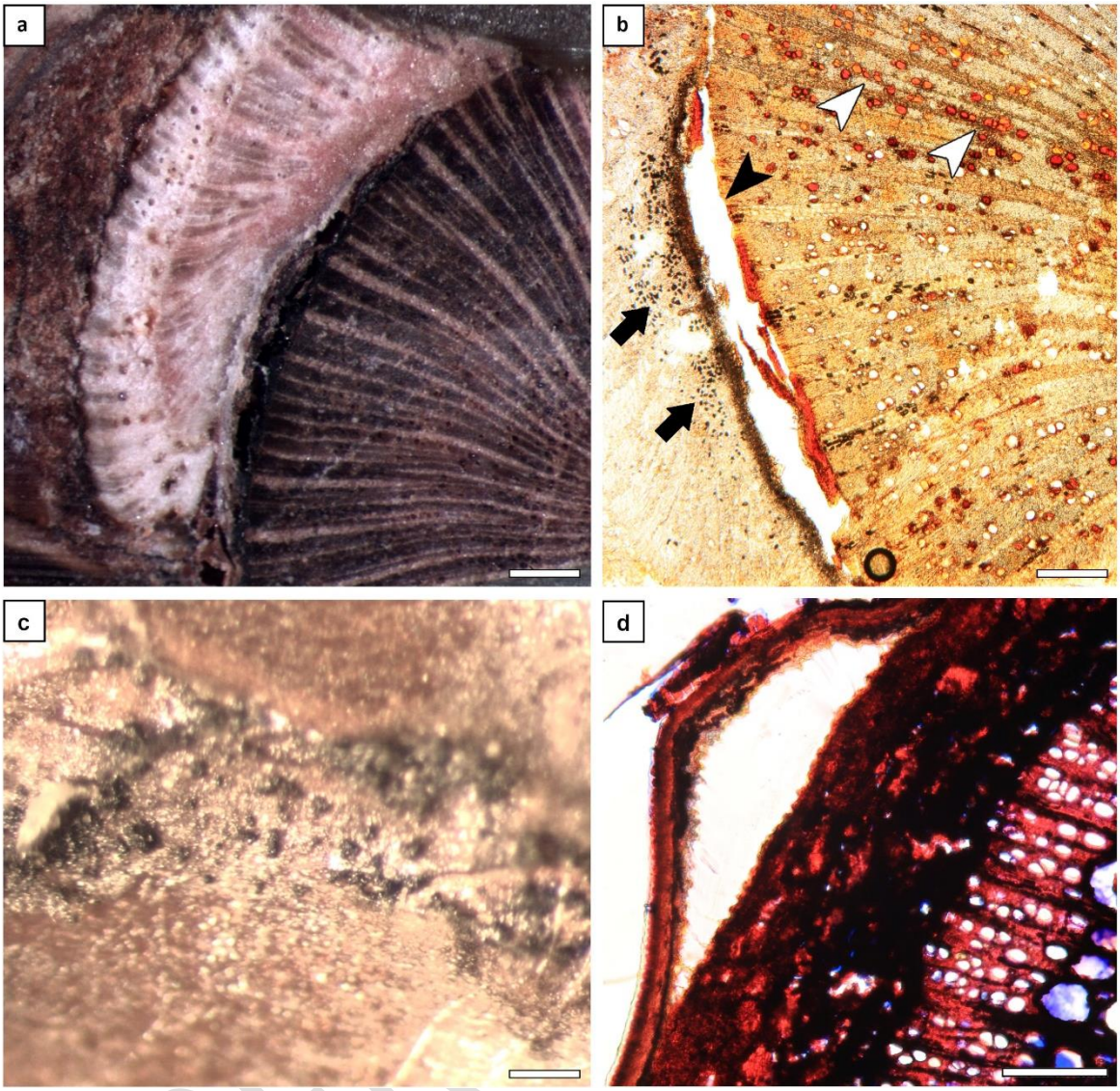

Figure 3: Image (a) shows a pale beige callus covering a dark brown area of infected wood, (b) shows a microsection of the two zones in a. (c) shows small black fruiting bodies under hardened gum and (d) shows a cross section through a fruiting body. The scale bars in a are 100 µm and 200 µm in b, c and d.

By further macroscopical examination of the collected samples with the naked eye no fruiting bodies of the fungus were found. Lawrence *et al.* (2018) reported that pycnidia occurred just beneath the periderm of the collected fruit and nut tree samples infected by *Cytospora*. According to (Biggs and Grove 2006) dead twigs and branches are usually covered with a multitude of pin-head-sized black structures erupting through the dead bark. The authors (Biggs

and Grove 2006) also stated that fruiting bodies found on dead twigs and branches contain asexual reproductive structures of the pathogen.

In the apricot tree samples selected in this article, fruiting bodies were found under the hardened gum (Figure 3c), and they were visible only under the stereoscope. They appear as very small black dots (Figure 3c). A cross section of the fruiting body in shown in Figure 3d. Cross sections of multiple fruiting bodies were cut and examined. Conidia were not found inside the fruiting bodies, which corresponds with the fact that sexual reproductive stage of Cytospora canker is hard to find in orchards (Pokharel 2013).

Figure 4 presents tangential microsections of the infected wood and cross sections with unusual and normal vessels. The tangential microsections show the extent of the gum flowing through the wood structure and filling out entire vessel lumina. Figure 4a shows that the gum flowed even into libriform fibre lumina. Both 4a and 4b microsections were stained with Astra Blue and Safranin stain combination. The gum did not absorb any dye from the stains, and it is visible as a light-yellow orange to reddish-brown substance. This is denoted with black arrows. The observed gum filling the vessel lumina may restrict the function of vessels in water and nutrient transport.. Among the collected and selected samples, usually only a part of the branch was overflowed with the gum. Even though only a part of the vessels had restricted flow, the tree cannot distribute enough water and nutrients to these areas. Because of this, the branches gradually dry out. The observed release of gum into vessel lumina, and the associated restriction of nutrient flow in the infected twigs and branches, is consistent with defense mechanisms described in the literature and may represent part of the wound and infection compartmentalization process in the studied tree.”

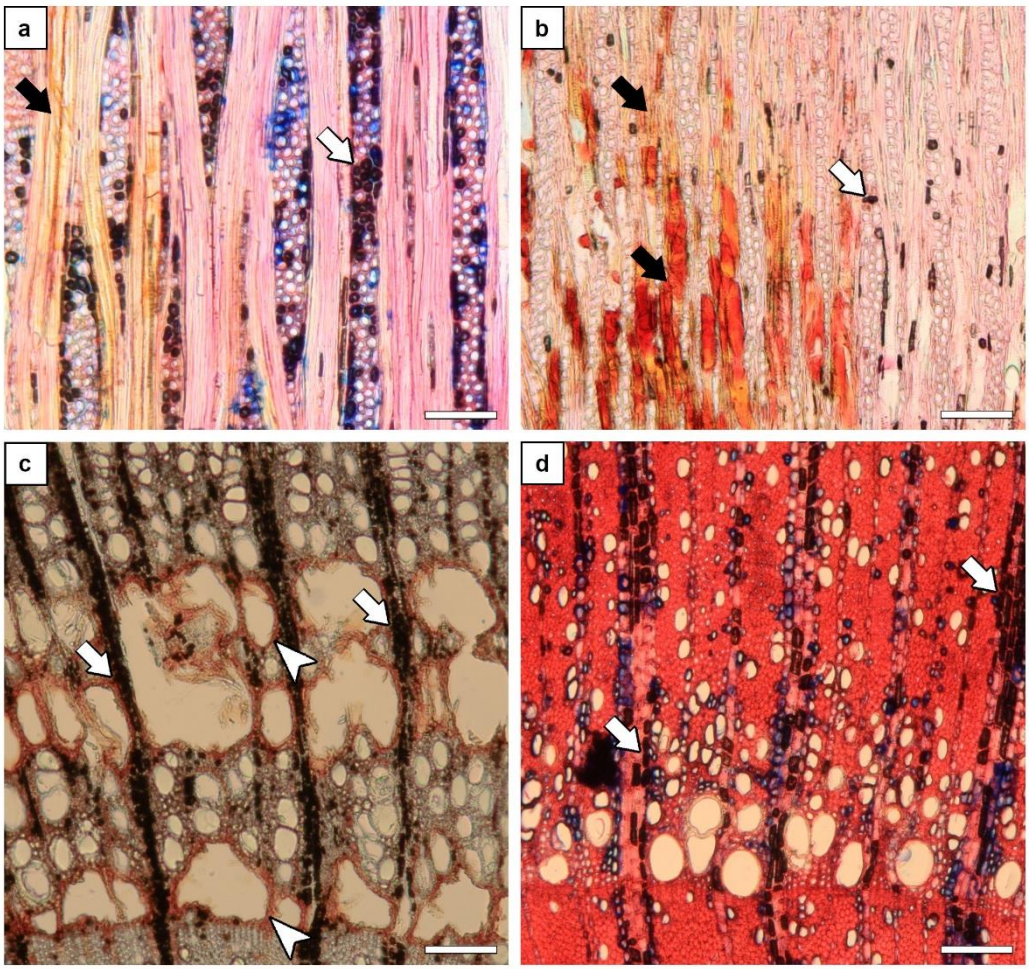

Figure 4: Images (a) and (b) present tangential cross sections of the infected wood. Image (c) shows a cross section with traumatic gum ducts and (d) shows a cross section with vessels of a normal size. White arrow with a black outline point at oxidized extractives, black arrows point at areas where the gum is filling out cell element lumina, white arrows head with a black outline point at traumatic gum ducts. The scale in all images is 100 µm.

Figure 4c shows a section with traumatic gum ducts (white arrowheads with black outline) and Figure 4d shows vessels of a normal size. Compartmentalization of the infection can be seen in both figures, as oxidization in ray parenchyma cells (white arrows with black outlines).

Measurement of traumatic gum ducts and vessels

According to Boothby (1983), Carolina and Kusumoto (2020), Setia (1983) gum ducts normally occur in healthy tissues of some broad-leaved tree species. However, most of broad-leaved species form gum ducts following microbial infection, insect attacks, mechanical injury, water stress, and chemical stimuli. Microbial infection was the cause of traumatic gum duct development in the studied apricot tree.

The comparison of traumatic gum ducts to normal vessels is depicted in Figure 4c and Figure 4d. Traumatic gum ducts look like they were 2 or more separate vessels, but the cell walls either collapsed or were damaged by the pathogen. In comparison to the oval or round shape or normal vessels, the lumen of traumatic gum ducts has a very irregular shape The Figure 4c microsection was not stained intentionally, in order to observe small colour changes in the cell elements. The traumatic gum ducts have a reddish-brown cell wall, which is not usual for vessels. In unstained microsections, extractives in ray parenchyma cells are usually of a reddish-brown colour.

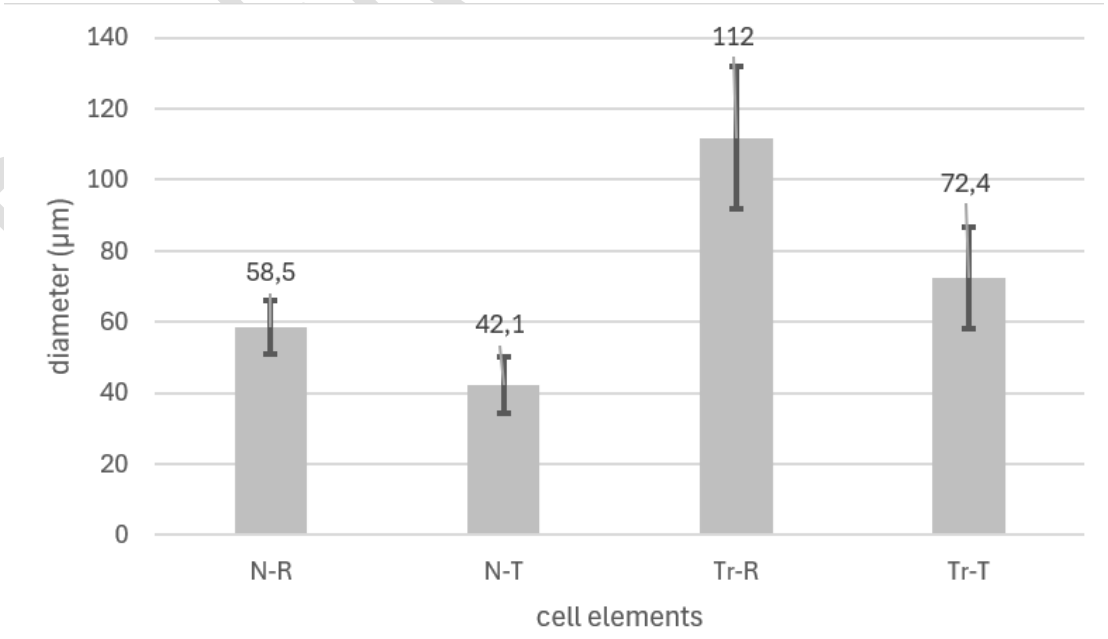

Figure 5: Radial (R) and tangential (T) sizes of normal vessels (N) and traumatic gum ducts (Tr) with standard deviations. 25 vessels were measured per each N-R, N-T, Tr-R and Tr-T.

Measurement of the normal and traumatic gum ducts is presented in Figure 5. Since some of the traumatic gum ducts looked like they were two or more separate vessels, but their cell walls collapsed, only unusually large vessels without the signs of collapsed cell walls were measured. The radial size of traumatic gum ducts is almost double the radial size of normal vessels. Tangential size of traumatic gum ducts is 1,72 times wider than tangential size of normal vessels. As the measurement shows, the traumatic gum ducts were prolonged radially; the ratio of radial to tangential sizes of traumatic gum ducts was 1,55 whereas this ratio in normal vessels was 1,39.

Using the CODIT model to describe compartmentalization of the infected wood

The CODIT (Compartmentalization of Decay in Trees) model is made up of four 'so-called' walls in two- parts, which are conceptual and explain the anatomical divisions or compartments that exist in woody plants at the time of wounding (part I), and divisions built after wounding (part II) by the plant to limit the spread of air or decay progressing into new wood (Morris *et al.* 2016). When trees are wounded, the cambium responds by laying down a thin layer of unique cells called a barrier zone. This zone separates the normal tissue formed prior to wounding from the normal tissue formed after wounding (Shigo 1984).

Part I is represented by three walls. Wall 1 is the weakest, since the growing tree must maintain vertical transport (Shigo 1984). It is is formed of tracheary elements (conductive cells of either angiosperms or conifers), and the adjacent living axial parenchyma contact cells (Morris *et al.* 2018). After injury and infection, plugs form in the vertical elements (Shigo 1984) to impede bi-directional fungal spread along the stem above and below the wound (Morris *et al.* 2018). There is a trade-off here, where to limit decay spread up or down the stem, the conduits must be sealed off with tyloses or gels, originating from contact parenchyma at the expense of continued water and solute movement (Schmitt and Liese 1990, Clérivet *et al.* 2000, Morris *et al.* 2016). According to this description, Wall 1 in the examined tree was presented in Figure 3b (cross section) and Figure 4b (tangential section). The vessel and libriform lumina are clogged with gum, which may limit the flow of nutrients and water.

Wall 2 is moderately strong (Shigo 1984). It comprises the latewood of annual rings of temperate tree species, and ray parenchyma present intermittently along a growth ring (Morris *et al.* 2020) and acts by halting the spread of decay inward toward the pith. It usually comes into effect after the wounding of the cambium (Morris *et al.* 2016). In the researched apricot tree, Wall 2 is presented in Figure 2d (dark arch in red rectangle) and Figure 3a and Figure 3b. In the latter mentioned figures, a distinct almost black zone is visible on the dark brown infected wedge and along the callus. This black zone is running along the growth ring.

Wall 3, the strongest wall in part 1. It restricts pathogens from spreading in a lateral direction (Shigo 1984). It is composed completely of ray parenchyma. These walls extend outward from the heartwood-sapwood boundary, forming a physical and dynamic barrier and thereby compartmentalizing much of the stem into discreet units in species where the walls also extend for significant axial distances (Morris *et al.* 2016). The presence of Wall 3 is visible in Figure 4a, Figure 4c and Figure 4d, it is denoted with white arrows outlined black. The ray parenchyma

in these figures is oxidized, they therefore appear dark, almost black. The flow of nutrients through the oxidized ray parenchyma is restricted and this structural response may help limit the adjacent tissue from spreading of the pathogen.

Part 2 may not begin to form for nearly a year, depending upon the time of wounding in relation to the next surge of cambial activity (Shigo 1984). Wall 4 or the barrier zone is a region composed of usually multiple layers of enlarged ray and axial parenchyma cells (up to 30 cell layers wide; Pearce 1996) that prevents fungal decay spread into new xylem laid down to the barrier zone's exterior (Morris *et al.* 2020). Included under the umbrella of wall 4 (any new tissue formed after injury), is the generation of new parenchyma cells after a wound has been created, forming a callus tissue (not wound wood in this case) that seals the damaged region and restricts the ingress of both air and pathogens (Morris *et al.* 2016). Callus tissue is presented in Figure 2d, Figure 3a and Figure 3b. In Figure 2d, the callus is growing around the whole branch, almost as new growth rings. This callus is denoted with a white arrow outlined black. Figures 3a and Figure 3b show a macroscopic close-up on the callus and a microscopic cross-section; Figure 3b, shows some oxidized cell elements at the border between the callus and infected tissue.

Conclusions

The focus of this article was the macroscopical and microscopical observation of apricot (*Prunus armeniaca*) wood samples with signs of branch dieback and gummosis and observation

of the anatomical response and signs of the disease in the wood. The fungus was determined as *Valsaria insitiva* (Tode) Ces. & De Not. As releasing large amounts of gum is a host response to irritation, large traumatic gum ducts formed in the infected tissue. The gum was found flooding the lumina of vessels and libriform fibers, which may disrupt the flow of water and nutrients and contribute to branch dieback in the studied tree. This can be understood as a defense mechanism of the tree, however it is also causing a slow decline of the infected tree as the infection seems to have spread at a much faster rate than the tree's ability to compartmentalize the infected sites.

Authorship contributions

B. S.: Conceptualization, data curation, formal analysis, investigation, methodology, resources, supervision, validation, visualization, writing – original draft, writing – review&editing. O. M.: Formal analysis, investigation, methodology, resources, supervision, validation, visualization, writing – original draft, writing – review&editing. P.H.: Investigation, methodology, writing – original draft.

Conflicts of interest

The authors declare there are no conflicts of interest for each author.

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