



Potato Tuber Lenticels: A Review of Their Development, Structure, Function, and Disease Susceptibility

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Abstract

Potato tuber lenticels are essential components of the potato skin. This review draws on more than a century of published literature to give a comprehensive overview of potato tuber lenticels. This review describes the development and structure of lenticels, as well as the number of lenticels per tuber. Lenticels facilitate gas exchange between the atmosphere and the interior of the tuber, and data on lenticel permeability to oxygen and carbon dioxide are summarized. Conditions that promote proliferation of filling cells and lenticel enlargement are described in the context of laboratory experiments and observations from the field. Lenticels are potential sites of infection by plant pathogens including common scab, powdery scab and late blight. Research demonstrating interactions between lenticels and various diseases is presented, with an emphasis on potato soft rot. Many aspects of lenticel biology remain poorly understood and a few compelling unanswered questions are highlighted.

Keywords Lenticel spot · Swollen and enlarged lenticels · Lenticel proliferation · Periderm

Introduction

Lenticels are multicellular structures that allow for exchange of oxygen (O₂) and carbon dioxide (CO₂) into and out of plant tissues. In this regard, they are analogous to stomata (Lendzian 2006). Lenticels are common in plant organs where the epidermis has been replaced by a phellem (Lendzian 2006). Stems, roots, and fruits that have undergone secondary growth often utilize lenticels for gas exchange. In potatoes, lenticels are present on stems above ground, stolons and tubers. This review focuses exclusively on potato tuber lenticels.

Lenticel Development and Structure

The development of potato tuber lenticels was described in detail by Fellows (1926) and Adams (1975a). Stomata are present on the youngest two internodes of a developing potato tuber and these stomata mark the sites where the first

lenticels form (Lindau et al. 1909; Artschwager 1918; Darling 1937; Adams 1975a; Peterson and Barker 1979). An internode is the region between two consecutive eyes. Periclinal divisions of subepidermal cells around the stomata cavity produce radial files of rounded, relatively undifferentiated filling cells that cause the stomata to be raised initially and shed eventually (Fig. 1a, b) (Lindau et al. 1909; Fellows 1926; Adams 1975a). Filling cells are also referred to as complementary cells. Intercellular spaces form between the filling cells as they enlarge. The volume occupied by the proliferating filling cells and accompanying airspaces increases until the overlying epidermis or newly formed periderm is ruptured (Fig. 1c). Once this occurs, the lenticel becomes fully functional as a conduit for gas exchange between the external environment and the underlying tissue. Lenticels become flatter and may become slightly concave as the tuber enlarges further (Adams 1975a).

A lenticel may form a cork barrier containing several layers of highly suberized cells (Jürgens 1872; Fellows 1926; Darling 1937; Adams 1975a; Tyner et al. 1997). The development of the cork layer has not been studied in detail. The illustration of a lenticel in Tyner et al. (1997) shows the cork layer immediately above the phellogen. In photographs of sectioned material, several layers of filling cells appear between the cork barrier and the underlying

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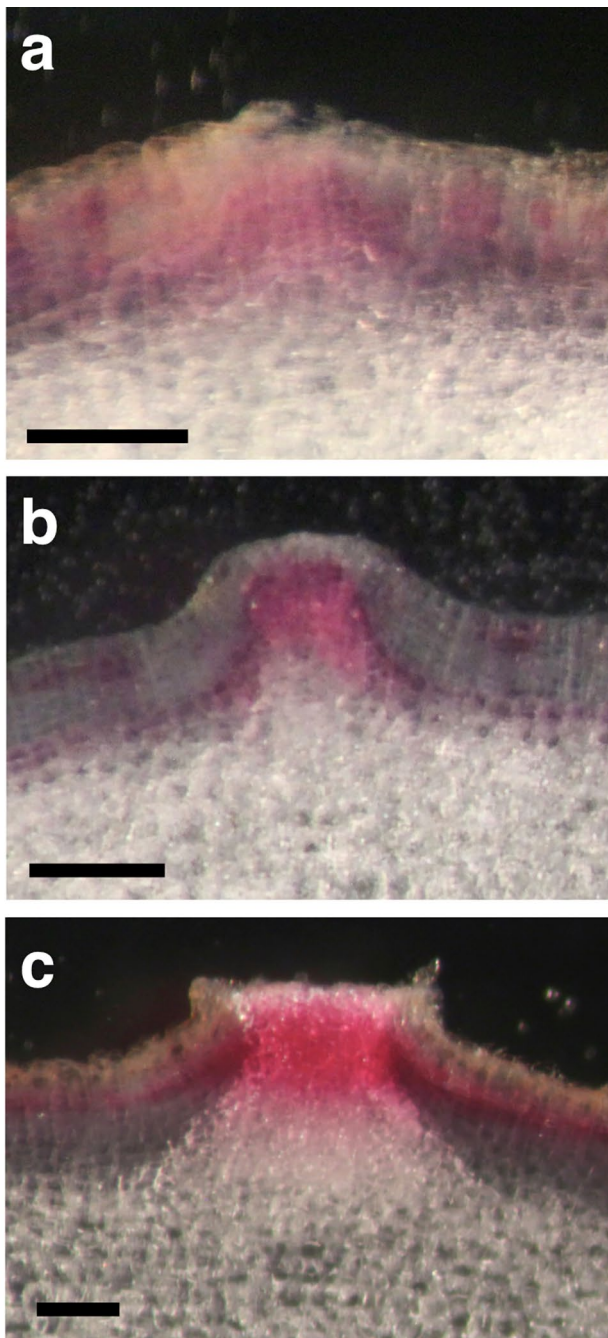


Fig. 1 Lenticel development in Red Norland. **(a)** Radial files of pale pink filling cells in a lenticel at an early stage of development. **(b)** A young lenticel that is raised above the tuber surface but has not broken through the skin. **(c)** A lenticel in which the filling cells have emerged through the potato skin. Free hand sections were prepared from developing potato tubers 10 mm or less in diameter and were photographed using a digital camera attached to a dissecting microscope. Scale bars indicate 250 μm

phellogen (Fellows 1926; Adams 1975a). It may be that the suberized layer is formed immediately above the phellogen and then displaced centrifugally as additional filling cells are produced, as is observed in other species (Rosner

and Kartusch 2003). Thickness of the suberized cork layer varied throughout the growing season and between varieties (Darling 1937; Tyner et al. 1997). For field grown potatoes, the suberized layer was usually 10–70 μm thick during the growing season (Tyner et al. 1997). For varieties Desirée and Cara stored at 4 °C for 5–6 months, the suberized cork barrier was 165–230 μm thick (Tyner et al. 1997).

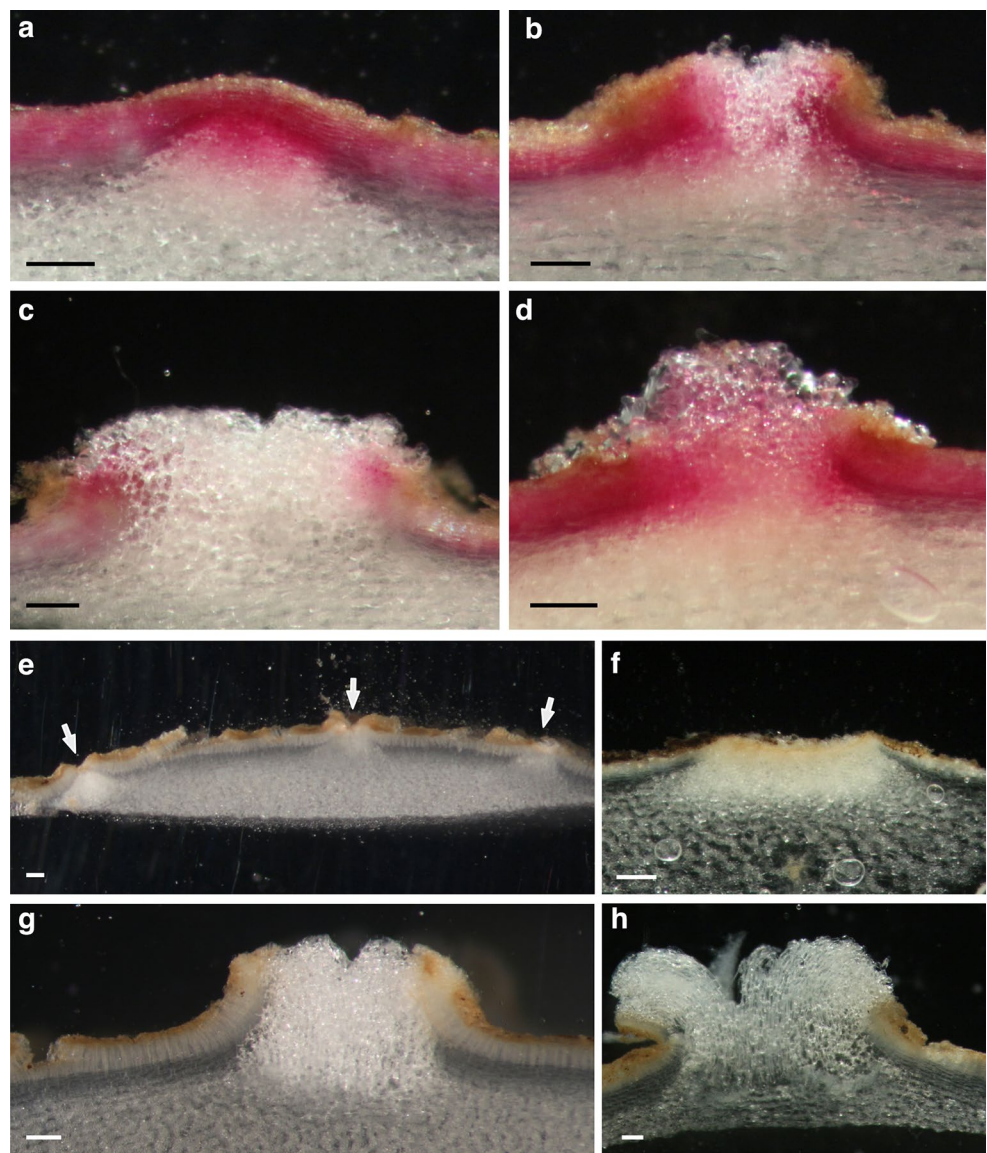
Lenticel Responses to the Environmental

Lenticels are dynamic organs that respond to the local environment. In dry soils, suberin is deposited on the outermost filling cells (Adams 1975a). The extent of suberization, as assessed by histological staining, increased during tuber development from internode 3 to internode 7 (Adams 1975a). Adams (1975a) estimated that stomatal guard cells persisted for about one week on growing potato tubers, and suberization of lenticel filling cells occurred over the subsequent week. Suberization of filling cells is not observed under moist conditions (Peterson and Barker 1979).

In wet soils, renewed proliferation and enlargement of underlying parenchyma cells may rupture and expand the lenticel aperture (Fig. 2). In the literature, this process is referred to as proliferation or lenticel enlargement. Proliferation ruptures the cork barrier in older suberized lenticels (Adams 1975a). Newly formed parenchyma cells may accumulate above the surface of the lenticel (Fig. 1b–d, g, h), producing mounds of loose tissue that are easily visible with the naked eye.

Cell division gives rise to additional filling cells during lenticel enlargement, but the location of the dividing cells giving rise to new filling cells has not been described precisely. A drawing in Lindau et al. (1909) (Fig. 3a) illustrates filling cells in the lenticel pore above densely packed rows of juvenile filling cells that extend radially to parenchyma cells in the cortex. Fellows (1926) presents a micrograph (Fig. 3c) of a recently opened lenticel that shows filling cells extending in columns from the lenticel pore to the cortex. Similarly, a micrograph in Adams (1975a) (Fig. 3b) presents a recently ruptured lenticel with loosely packed filling cells near the surface, and files of densely packed, smaller, flattened cells extending to the cortex. A variation on this arrangement is found in Pérombelon and Lowe (1975) (Fig. 3d) where the number of filling cells relative to the number of interior flattened cells is greater than that observed by Adams. One interpretation of these data, and the additional data presented in Fig. 2, is that the lenticular phellogen in mature lenticels is displaced substantially inward from the periderm phellogen and is located near the cortex below the lenticel pore (Fig. 3). This suggestion is consistent with a general view of lenticel formation in angiosperms and gymnosperms (Esau 1960).

Fig. 2 Proliferation of filling cells before (**a, e, f**) and after (**b, c, d, g h**) they expand the lenticel pore and emerge above the tuber surface. Red Norland (**a–d**) and Snowden (**e–h**) potatoes were wrapped in wet paper towels and incubated at 21 °C to stimulate lenticel enlargement. Free hand sections of fresh tuber tissue were photographed using a digital camera attached to a dissecting microscope. Scale bars indicate 250 μ m. Arrows in (**e**) point toward lenticels



The rate of proliferation increased with temperature in the range of 10–20 °C for potato tubers taken out of storage and wrapped in wet paper toweling (Adams 1975a). In field experiments, the extent of proliferation and the intensity of cell wall staining with safranin, used as an indirect measure of suberin deposition in cork barriers, varied throughout the growing season, corresponding somewhat to soil moisture content (Adams 1975a). Rain in July and August increased proliferation, decreased safranin staining intensity, and decreased the percentage of lenticels with cork barriers. Proliferation stopped when drier weather returned. In plots that were watered to field capacity for three weeks prior to sampling, tubers dug in July and August showed proliferation but those collected in late August and September did not (Adams 1975a), suggesting that older tubers have a

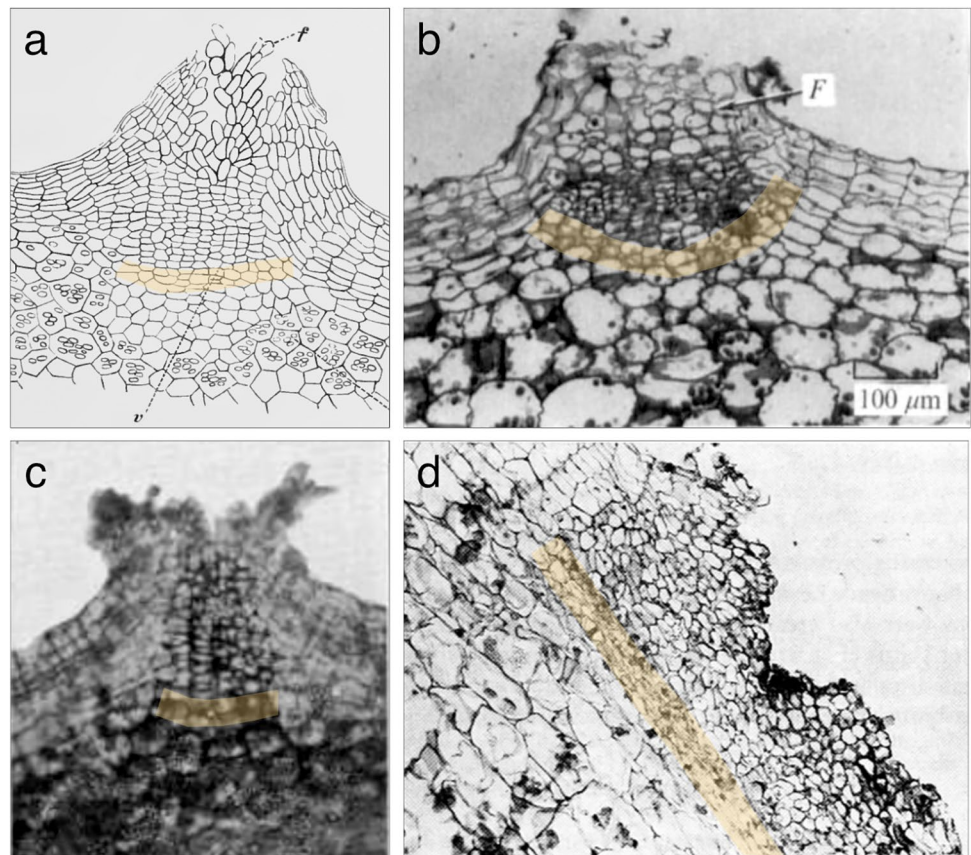
reduced predisposition to proliferate under wet conditions (Weber 1990).

When the surface of potatoes with enlarged lenticels and exposed filling cells is dried, the filling cells die and a suberized closing layers form (Fig. 4). Such lenticels are easily seen on potatoes with a smooth skin. This physiological defect is referred to as lenticel spot.

Lenticel Number and Size

The number and size of lenticels varies between potato varieties, among potatoes of a particular variety, and with growing conditions. Meinel (1966) found that lenticel number varied between growing seasons and with soil type for some varieties. Table 1 summarizes data on lenticel number per potato and number per unit surface area for several potato

Fig. 3 Arrangement of cells in mature (a) and proliferating (b–d) lenticels. Images reproduced from (a), Lindou 1909, (b) Adams 1975, (c) Fellows 1926 and (d) Pérombelon and Lowe 1975. Suggested locations for the lenticular phellogen are indicated by beige shading



varieties. For Early Ohio, Irish Cobbler and Green Mountain, the number of lenticels per kilogram and the number per surface area increased as tuber size decreased. As an example, small, medium and large Irish Cobbler tubers had 3.19, 2.80 and 2.08 lenticels per square centimeter, respectively (Michaels 1932). The number of lenticels per surface area also decreased with increasing tuber weight for varieties Schwalbe and Ora (Meinl 1966).

For a given variety, the number of lenticels per potato varies, suggesting that lenticel initiation does not follow a rigid developmental program. Although the first lenticels are formed below stomata, a few reports suggest that additional lenticels are produced from the cambial layer in the periderm as potatoes enlarge (Burton 1965; Khatri et al. 2013). For example, King Edward and Majestic tubers weighing less than 5 g had about 100 lenticels per tuber, whereas tubers greater than 5 g had 150–180 lenticels per tuber (Burton 1965). These data for the number of lenticels in King Edward tubers are similar to those reported in (Choudhury 1939) and (Wigginton 1973). Lenticel density values reported by (Choudhury 1939), however, were unusually high at approximately 20 lenticels per cm^2 .

Published data on lenticel size are uncommon, but large differences in lenticel size have been observed (Darling 1937; Burton 1950; Zhang et al. 2016). For example, Burton

(1950) used 0.2 mm^2 as an estimate for lenticel surface area whereas mean surface area for lenticels of variety Atlantic was 1.5 mm^2 (Zhang et al. 2016).

Gas Exchange Through Lenticels

Lenticels are the pores through which O_2 enters and CO_2 exits a potato tuber. Oxygen diffusion through the periderm was below the level of detection in areas of the periderm that did not contain lenticels (Wigginton 1973). Permeability of the periderm to O_2 was measured for potatoes at various stages of development. Oxygen permeability of Majestic and King Edward tubers decreased from a maximum of 2.4×10^{-4} to less than $0.7 \times 10^{-4} \text{ ml O}_2 \text{ s}^{-1} \text{ cm}^{-2} \text{ atm O}_2 \text{ tension}^{-1}$ as the potatoes matured (Burton 1965). Variation in O_2 permeability between individual lenticels has been observed (Wigginton 1973). For individual lenticels, O_2 diffusion rates varied widely, from 0.024 to $0.296 \text{ cm}^3 \text{ h}^{-1} \text{ atm}^{-1}$. Values for O_2 permeability of lenticels were also measured by Banks and Kays (1988). In general, they found permeability values lower than those observed by Wigginton. Banks and Kays suggested that variation in lenticel permeability might be caused by changes in lenticel structure, such as the presence and extent of a suberized cork layer.

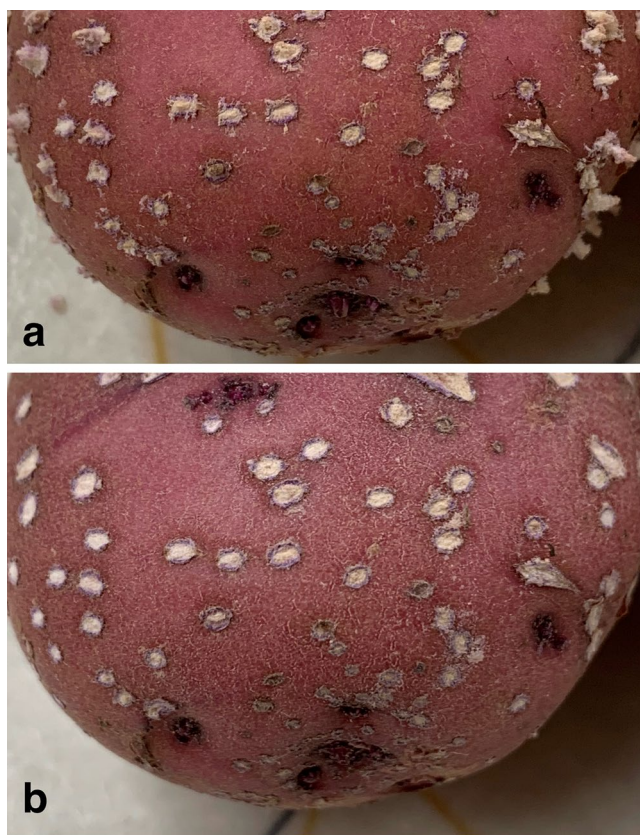


Fig. 4 Lenticel spot on Red Norland potatoes. Exposed filling cells dry quickly in air (a). Gentle abrasion of the tuber surface removes dead filling cells and leaves behind blemishes where a closing layer has formed (b)

Table 1 Lenticel number per tuber and number per square centimeter for several potato varieties

Variety	Lenticels per tuber	Lenticels per cm ²	Source
Early Ohio	–	2.35–3.09	Michaels
Irish Cobbler	–	2.08–2.69	1932
Green Mountain	–	2.86–3.24	
King Edward	150–200	19–20	Choudhury 1939
King Edward and Majestic	100 (tubers < 5 g) and 150–180 (tubers > 5 g)	–	Burton 1965
King Edward, mature tubers	74–141	–	Wigginton 1973
Desirée	–	1.66	Zhang et al. 2016
Atlantic	–	3.69	
19 varieties in two years	–	~1.5-8	Meinl 1966

Lenticel surface area in mature potato was 4–5% of the phellem area for varieties Combi and Erna (Lendzian 2006) and less than 1% for other varieties (Burton 1950). Lenticel surface area, however, is not a useful predictor of gas permeability. This is not surprising given that the

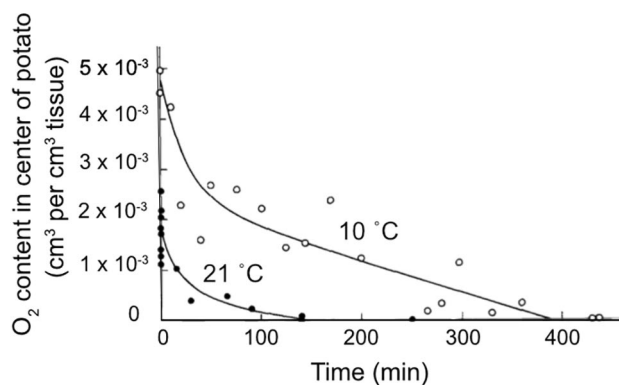


Fig. 5 Oxygen content in the center of a potato when covered with a film of water for various lengths of time. Figure modified from Burton and Wigginton (1970)

cross-sectional area and length of air passages between the filling cells determine the resistance to gas exchange, not the surface area of the lenticel. Burton estimated that the cross sectional area of the intracellular space in filling tissue was on the order of 10^{-6} of the tuber surface area based on measured rates of O₂ diffusion across the periderm (Burton 1965). Once oxygen has passed through the lenticel, it is distributed by diffusion and convection through air spaces within the potato. The air space within a potato is approximately 1–2% (Burton 1950; Davis 1962) or 2–4% (Weber 1990) of total potato volume.

Oxygen permeability of the periderm as a whole was sufficient to support fully aerobic tuber metabolism in arable soil (Burton 1965). Devaux (1890) measured internal gas concentrations of potatoes and found 11.2–14.9% O₂ and 4.4–9.1% CO₂. Banks and Kays (1988) found that internal oxygen concentration ranged from 16 to 18% for potatoes exposed to ambient air at 20 °C. Internal CO₂ concentrations were 4–8%. Burton (1950) measured internal oxygen concentrations between 5 and 20 °C and found O₂ saturation to be 65–95%. Magness (1920) reported that the concentrations of O₂ and CO₂ in the intracellular space of potatoes at 22 °C were 5.7% and 34.4%, respectively. These values are substantially different from those found by subsequent researchers. Whether this difference reflects a methodological error, or a different physiological state of the potato cores used for the study compared with other studies is unknown.

The diffusion coefficients for O₂ and CO₂ in air are approximately 10,000 times greater than they are for O₂ and CO₂ in water. Because of this, oxygen limiting conditions are imposed on potato tubers in water-saturated soil and when tubers are covered by a film of water. The center of mature Bintje variety potatoes became anoxic in 2.5 h at 21 °C and 6.5 h at 10 °C when covered with a film of water with an average thickness of 3×10^{-2} mm (Fig. 5) (Burton and Wigginton 1970). Interestingly, Smith (1920) noted

that covering lenticels with Squibb's petrolatum to restrict gas exchange caused cells under the lenticels to begin to divide in some species. Squibb's petrolatum was a paraffin oil likely enriched in cyclic hydrocarbons (Langmuir 1933).

Under oxygen limiting conditions, glycolytic respiration declines and anaerobic respiration is the primary energy source for continued metabolism. In one study, CO₂ production under anerobic conditions was approximately 40–50% of that under aerobic conditions (Choudhury 1939).

Tuber Lenticels are Infection Sites for Pathogens

Lenticels are openings in the protective periderm of the potato tuber. As such, they are potential infection sites for pathogenic bacteria, fungi and oomycetes. Lenticels that are newly formed, and those that have enlarged following proliferation of filling cells, tend to be the most susceptible to infection since a protective, suberized cork layer has not been produced or has been ruptured. Diseases that can initiate an infection through lenticels and which have significant commercial importance are listed in Table 2.

Bacterial Soft Rot Caused by *Pectobacteria Carotovorum*

One of the most serious diseases affecting potatoes is soft rot caused predominantly by *Pectobacterium carotovorum* var. *carotovorum* and *Pectobacterium carotovorum* var. *atro-septicum*. Tuber soft rot results in wet breakdown of the tuber flesh. Early-stage lenticel infections are characterized by raised, water-soaked areas around the lenticels. If the infected area around the lenticels is dried, the periderm often becomes slightly sunken but remains intact (M. A. Smith and Ramsey 1947). Such tubers are said to have bacterial lenticel spot (Fig. 6) (Inglis et al. 2011; Robinson and Secor 2016). Infections that continue to progress can result in tubers that are reduced to a gluey, gassy mass enclosed in the periderm (Pérombelon 1972).

An association between lenticels, wet conditions and bacterial soft rot has been described on numerous occasions. Bacterial infection of enlarged lenticels resulting from wet conditions was observed by Sorauer and described in his Manual of Plant Diseases (Sorauer 1886). Smith (1920) proposed that lenticels are routes for bacterial infection of potatoes, based on observations he made as early as the late 19th century. He used a mixture of *Bacillus phytophthorus* and *Bacillus solanisaprus* to demonstrate lenticel infections in the laboratory (Smith 1920). It is likely that *Bacillus phytophthorus* was *Pectobacterium carotovorum* var. *atro-septicum*, and that *Bacillus solanisaprus* was *Pectobacterium carotovorum* var. *carotovorum* (Hellmers 1959). Regarding soft rot, Smith noted that “all potato varieties are said to be subject to this disease”. “Are those varieties whose lenticels

Table 2 Diseases and pathogens associated with potato tuber lenticels

Disease	Pathogen	Selected references
Soft rot	<i>Pectobacterium</i> species	M. A. Smith and Ramsey 1947; Pérombelon 1972; Pérombelon and Lowe 1975
Common scab	<i>Streptomyces scabies</i>	Fellows 1926; Adams and Lapwood 1978
Powdery scab	<i>Spongospora subterranea</i>	de Boer et al. 1982; Harrison et al. 1997
Late blight	<i>Phytophthora infestans</i>	Adams 1975b; Walmsley-Woodward et al. 1975; Walmsley-Woodward and Lewis 1977
Black scurf	<i>Rhizoctonia solani</i>	Hughes 1980
Skin spot	<i>Polyscytalum pustulans</i>	Allen 1957; Nagdy and Boyd 1965
Pink rot	<i>Phytophthora erythroseptica</i>	Goss 1949; Lonsdale et al. 1980
Silver scurf	<i>Helminthosporium solani</i>	Heiny and McIntyre 1983; Errampalli et al. 2001
Charcoal rot	<i>Macrophomina phaseolina</i>	Stevenson et al. 2001
Gangreen	<i>Phoma exigua</i>	Stevenson et al. 2001



Fig. 6 Bacterial lenticel spot of potato. Arrows point to sunken lenticels surrounded by discolored haloes

open freely in wet soils specially subject to this rot?” he asks; and answers “I believe they are.”

Nielsen (1974), working in North Carolina, observed that when mature plants of varieties Superior and Pungo were flooded in early June, lenticel infection developed in 38% of the tubers within 72 h in one year and in 9% of the potatoes within 52 h in another year. Pathogenic bacteria isolated from the infected lenticels were identified as *Erwinia carotovora* var. *atroseptica* and *Erwinia carotovora* var. *carotovora* (*Erwinia carotovora* has been renamed *Pectobacterium carotovorum*).

Based on his inspections of potato shipments coming into Chicago, Ramsey (1937) observed that “The increasing practice of washing southern-grown potatoes has also

led to serious consequences in some regions. In many lots of potatoes not properly dried before loading, the surface moisture has greatly favored the development of bacterial soft rot during transit” (Ramsey 1937). Smith and Ramsey (1947) would later report that in 132 carlots of potatoes, losses caused by bacterial soft rot ranged from 2 to 98%. “Losses of 1,000 to 10,000 lb. were common, and in many lots 15,000 to 24,000 lb. out of a 30,000-lb. load were worthless. Most of these shipments originated in States having excessively wet weather before and during harvesting, and most of the decay that followed originated at lenticels” (Smith and Ramsey 1947). They proposed a scenario that links lenticel proliferation to soft rot development. In this scenario, tubers grown in heavy or wet soils develop greatly proliferated lenticels. When tubers are washed before shipment, the proliferated tissue is rubbed off and the injured areas resulting are readily invaded by bacteria present in the wash water. In some cases, infected lenticels remain as mere blemishes. Under “favorable” conditions, however, bacterial soft rot develops at such centers. They emphasized that “warm potatoes, bagged wet, and loaded into non-refrigerated cars are under conditions most favorable for the development of extensive bacterial soft rot.” (Smith and Ramsey 1947).

The prevalence of soft rot bacteria on apparently healthy tubers was assessed by placing seed potatoes with water in sealed polyethylene bags and incubating them at 18 °C for 10–15 d. The relatively high temperature and high relative humidity inside the bags caused extensive soft rot. Most of the lesions appeared to have started at lenticels; only rarely was it evident that rotting originated at the stolon end. Of 790 rotted tubers sampled, isolates resembling *P. carotovorum* were obtained from 693 tubers (Pérombelon 1972).

Scottish seed potatoes were evaluated post-harvest for the presence of soft rot bacteria. *P. carotovorum* var. *carotovorora* and *P. carotovorum* var. *atroseptica* were both present, the latter being detected four times more frequently. Most contamination was located on the tuber surface and in the lenticels rather than in the vascular ring or tuber cortex. The level of contamination of lenticels was only slightly affected after winter storage. On the other hand, winter storage substantially reduced surface contamination (Pérombelon 1973). These data suggest that the presence of soft rot bacteria in lenticels is widespread and that lenticels might shelter soft rot bacteria through winter storage. Nielsen (1978) came to a similar conclusion based on experiments conducted with seed potatoes grown in four U.S. states and one Canadian province.

Conditions that promote soft rot were explored by Smith and Ramsey (1947) using *P. carotovorum* isolated from water-soaked areas around previously infected lenticels. When freshly harvested potatoes were immersed in water

suspensions of these bacteria for more than 1 min, infections of lenticels were observed in tubers subsequently held at 72 °F and close to 100% RH for 48 h (Smith and Ramsey 1947). A similar finding was observed when potatoes were immersed in the water used for washing the potatoes used in this experiment. The authors concluded that “commercially washed potatoes may carry on their surfaces sufficient numbers of virulent soft-rot bacteria to later bring about lenticel infections if favorable environmental conditions are provided”.

To better define environmental conditions favorable for soft rot development, experiments were conducted with varieties White Rose and Bliss Triumph. Potatoes were surface sterilized with 80% ethanol, dried, immersed in water containing soft-rot bacteria for 5 min, and stored at 16 °C or 22 °C at various levels of relative humidity (RH). Lenticel infection did not occur at either temperature within 72 h when RH was 90% or less. At 95% RH, lenticel infection was apparent after 48 h at 22 °C but did not occur at 16 °C. At 98% RH, both varieties showed some infection at 16 °C after 48 h. Finally, at 100% RH, all Bliss Triumph and half of the White Rose tubers were infected after 24 h of incubation. None of the non-inoculated control tubers showed infection at 72 h. The researchers concluded that few or no infections through lenticels will occur in the field or in transit within 3 days at a temperature below 72 °F and a relative humidity less than 94.8 per cent. Under moist or wet soil conditions, however, these temperature and humidity conditions are frequently exceeded, especially in some regions growing early potatoes (Smith and Ramsey 1947).

In one study where the extent of soft rot was observed after potatoes were packaged in polyethylene bags, the extent of rotting was found to be much greater when tubers were packaged immediately after harvest compared to bagging after they had ‘cured’. Washing tubers with clean water before packaging them in polyethylene bags reduced the number of tubers that rotted compared with unwashed tubers, but washing with water containing *P. carotovorum* increased the extent of rotting compared to other washing treatments (Scholey et al. 1968).

The link between infection by soft rot, lenticel enlargement and anoxia was explored by Pérombelon and Lowe (1975). Lenticels were individually inoculated with a suspension of *P. carotovorum* var. *atroseptica*. Soft rot lesions were not observed after a 7-day incubation at 20 °C with 100% RH air or N₂ when the tuber surface was dry and lenticels were “closed” i.e., lenticels had not recently proliferated and a suberized layer was present. If the suberized cork layer of the lenticel was punctured, however, all lenticels became infected in the N₂ atmosphere, and 20% or less were infected in air. When tubers were wrapped in wet tissue paper, 40–60% of closed lenticels became infected when the

tuber was incubated in N₂. Infection was not observed when similarly wrapped tubers were incubated in air. Finally, 80–100% of enlarged lenticels inoculated with soft rot bacteria developed lesions when incubated in N₂, although rotting was not observed when tubers were incubated in air. It is important to note that rotting was defined as the presence of an active lesion and not by the presence of small, arrested lesions, which were frequently visible at enlarged lenticels under aerobic conditions. The authors concluded that anaerobic conditions are a prerequisite for the initiation of soft rot but are not sufficient to cause extensive tuber rot. Rather, soft rot development occurred when anaerobic conditions were combined with free water on the tuber surface (Pérombelon and Lowe 1975). Nielsen (1978), likewise, found that anaerobic conditions and moisture stimulate infection of naturally inoculated lenticels. Tuber rot developed faster for tubers immersed in water compared with those in a desiccator flushed with humidified CO₂-N₂ gas (Nielsen 1978).

The effect of submerging seed tubers of varieties Katahdin and Sebago in water at different temperatures was examined by Hardenburg (1950). Tubers submerged at 4 or 10 °C for up to 10 days remained sound and sprout growth was like that of control tubers. In contrast, none of the tubers submerged at 21 °C for more than 5 days was free of soft rot and suitable for use as seed. Sunken and decayed lenticels were usually present on tubers submerged for more than 3 or 4 days at 21 °C. These findings were extended by Nielsen (1974) who immersed tubers in water at about 34, 24, 15, and 8 °C and found that they developed lenticel infections in approximately 20, 44, 96, and 216 h, respectively. Nielsen attributed the strong temperature effect to increased bacterial action at higher temperatures (Nielsen 1974). Compared to cooler temperatures, warmer temperatures also increase tuber respiration rates, more rapidly decrease tuber oxygen content, and decrease the time needed for lenticel enlargement. Thus, the conditions favoring bacterial growth also favor entry of the bacteria through lenticels.

Common Scab Caused by *Streptomyces Scabiei*

Common scab, caused by the bacterium *Streptomyces scabiei*, produces unsightly lesions and pits on the surface of potatoes. The idea that lenticels are avenues for infection of potatoes by common scab was proposed over a century ago (Lindau et al. 1909) and this suggestion has been confirmed many times. Relationships between tuber development and tuber growth rate and common scab infection have long been recognized. Fellows (1926) found that infections by *S. scabiei* were more common on the younger, apical end of growing tubers than on the basal end. Furthermore, the percentage of tuber surface area covered with scab lesions increased with tuber size at harvest. Detailed investigations

showed that the initial point of infection was in a recently developed lenticel or in stomata that would develop into a lenticel as the tuber matured. In explaining these observations, Fellows wrote “The condition of the lenticels in the apical end of a tuber at the time of infection may predispose the tuber to infection in that region. When the lenticels first break open, the parenchyma cells below are freely exposed. This enables the organism or its products to come into direct contact with these cells. The parenchyma cells thus exposed have not been found to be suberized” (Fellows 1926). Jones examined lenticels infected recently by *S. scabiei* and found that the lenticels were not suberized at this early stage and the filling cells were tightly packed without air spaces between them (Jones 1931). Others have confirmed these findings and proposed that internodes that form successively at the apex of developing potatoes each pass through a period of scab susceptibility as stomata develop into lenticels (Hooker and Page 1960; Adams and Lapwood 1978). The youngest 1–2 internodes have stomata but not lenticels, and these appeared resistant to infection, perhaps because they had not yet been colonized by the disease (Adams and Lapwood 1978). Young lenticels in internodes 3–5 that do not have suberized filling cells were susceptible to infection, whereas lenticels on older internodes were often suberized and were resistant to infection. These early findings were explored further by inoculating tubers developing on plants growing in a hydroponic system at specific growth stages. Infection percentages were highest when tubers were inoculated early. Two weeks after initiation, 68% of tubers became infected whereas only 4% were infected when inoculated 8 weeks after tuber initiation (Khatri et al. 2011).

Powdery Scab Caused by *Spongospora Subterranea*

Powdery scab is a blemish disease caused by the soilborne pathogen *Spongospora subterranea*, which is also the vector for potato mop-top virus (PMTV). PMTV causes growth defects and spraing in tubers. PMTV is thought to persist in soils in resting spores of *S. subterranea* (Harrison et al. 1997). A survey of apparently healthy Foundation seed potatoes in Victoria, Australia, found *S. subterranea* spore balls in lenticels of six out of seven varieties examined six months after harvest (de Boer et al. 1982). The authors noted that they could not determine if the spore balls developed in the lenticels or were superficial contaminants. When *S. subterranea* inoculant was applied to potato plants at various times after tuber initiation, the amount of disease was highest when plants were inoculated at an early stage of tuber development, when tubers were 1–2 cm in diameter (Hughes 1980). That potatoes are most susceptible to powdery scab shortly after initiation was also observed by Diriwächter and Parbery (1991). They inoculated individual growing tubers

with suspensions of *S. Subterranea* spore balls at the first appearance of stolon tip swelling and 20–25 days later, when tubers were 1 cm in diameter. None of the later inoculated tubers were infected. Of the early inoculated tubers, 6 of 14 had scab lesions over 10% or less of their surface. Scabs covered 10–50% of the surface of the other eight. Infections occurred in the filling cells of lenticels, with a higher percentage of infection in lenticels where proliferation of filling cells had ruptured the periderm. Other data suggest that apparently healthy potatoes can carry a low level of *S. subterranean* inoculum in their lenticels (de Boer et al. 1982; Diriwächter and Parbery 1991). In a pot trial with varieties Desirée and Russet Burbank, inoculations with *S. Subterranea* 0, 10 and 20 days after emergence resulted in tuber disease incidence of 30% or more while later inoculations caused less than 5% disease. Data on relative rates of lenticel infection were not collected in this experiment (Than-gavel et al. 2015).

Late Blight Caused by *Phytophthora Infestans*

It has long been known that potato tuber lenticels are susceptible to infection by *Phytophthora infestans* (see (Jones et al. 1912) and references therein). Lenticels of infected tubers are brown and surrounding tissue may appear sunken during the early stage of an infection. Susceptibility of tubers (Boyd and Henderson 1953; Lacey 1967) and lenticels to late blight infection is greatest in young tubers and declines with tuber age (Walmsley-Woodward and Lewis 1977). After lifting, lenticels become very resistant to infection (Zan 1962; Adams 1975b). Zan (1962) suggested that increased resistance may be caused by suberization of the lenticels, based on a thesis published by Löhnis in 1922. An English summary of this work, however, suggested that infection of tubers in the field most likely occurs through slight wounds in the skin and not through eyes, lenticels and unbroken epidermis (Löhnis 1922). Adams thought that increasing resistance likely reflected increasing suberization of the lenticels and the formation of a cork barrier. Proliferation of lenticel filling cells led to an increase in the number and size of late blight lesions found at lenticels (Adams 1975b). There is evidence for chemical signaling between lenticels and late blight zoospores. For example, zoospores accumulated on lenticels even when they were covered with a layer of agar. This observation led the authors to propose that zoospores are drawn to lenticel by chemotaxis (Walmsley-Woodward et al. 1975). The proportion of late blight zoospores germinating on lenticels of King Edward tubers was close to 100% on July 16 and decreased to less than 20% eight weeks later (Walmsley-Woodward et al. 1975). Differences between potato varieties to lenticel infection have been observed, with King Edward being more susceptible

than Record, Maris Peer and Majestic (Walmsley-Woodward and Lewis 1977).

Skin Spot Caused by *Polyscytalum pustulans*

Skin spot disease, which is caused by the fungus *Polyscytalum pustulans* (formerly *Oospora pustulans*), produces circular, raised lesions on tubers after 3–4 months in storage, especially under humid conditions (Allen 1957). Skin spot disease is prevalent in cold and damp regions of potato cultivation. Varieties differ in their susceptibility to skin spot (Nagdy and Boyd 1965). Skin spot disease enters tubers through lenticels and eyes. Infections under lenticels may be contained for weeks by the formation of a cork cambium under the infected tissue (Allen 1957). At later times, however, the periderm outside of the of the infected tissue begins to degenerate, producing the visible ‘spot’ (Allen 1957). Inoculation experiments indicated that lenticels were the primary path of infection (Allen 1957). *P. pustulans* persists in soil for at least 5 years and can be transmitted from one generation to the next by infected seed (Carnegie and Cameron 1990).

Several other diseases are frequently associated with lenticels (Table 2). Pink rot and silver scurf are widespread and cause significant economic losses. It is unclear, however, if lenticels are primary infection points for the pathogens that cause these diseases. Charcoal rot (*Macrophthonina phaseoli*) and gangrene (*Phoma exigua* var. *foveata*, *Phoma exigua* var. *exigua*) are not discussed further, but these diseases have been reported to begin with lenticel infections.

Pink Rot Caused by *Phytophthora Erythroseptica*

Pink rot produced by the soil-borne oomycete *Phytophthora erythroseptica* was spread widely in the early part of the 20th century and causes tuber rot under wet or very humid conditions (Cairns and Muskett 1933; Goss 1949). *P. erythroseptica* often develops under conditions of high soil moisture and is associated with poor drainage and heavy, late season rainfall. Infected tubers often show symptoms first at the stolon attachment point, but occasionally infected tubers do not show symptoms at the stem end. This observation led Goss to suggest that infection may also occur through eyes, lenticels or wounds under favorable conditions (Goss 1949). Adams, however, suggested that lenticels are not a site for infection by *P. erythroseptica* (Adams 1973). Hooker (1981) in *The Compendium of Potato Diseases* states that lenticels are a potential infection site for pink rot. However, the second edition of that text does not make that claim (Stevenson et al. 2001).

Silver Scurf Caused by *Helminthosporium Solani*

Silver scurf is a serious disease of the tuber surface that results in extensive regions of discoloration and increased rates of water loss during storage ((Errampalli et al. 2001), and references therein). Schultz examined fixed sections of badly infected tuber tissue and stated “it was evident that the mycelium may enter the tuber through the lenticels or between the lenticels through the epidermis” (Schultz 1916). Likewise, Burke concluded that “ingress may be directly through a cell of the outer layers, or may be through a lenticel” (Burke 1938). These early studies have given rise to the widely disseminated idea that *H. solani* infects tubers through lenticels. However, reports from extensive investigations using light and electron microscopy did not present evidence of lenticel infection by *H. solani* (Heiny and McIntyre 1983; Martinez 2004). Rather, Martinez et al., suggested that penetration of tuber periderm cells by the pathogen likely involves a mechanical and enzymatic process (Martinez 2004). This suggestion is consistent with an analysis of the *H. solani* genome that showed an abundance of genes for cell wall degrading enzymes (Mattupalli et al. 2014).

Unanswered Questions

Research on potato tuber lenticels has been carried out for over a century. These efforts have revealed much about lenticel development, physiological responses to the soil and postharvest environment, and interactions with pathogens. However, many aspects of potato tuber lenticel biology remain poorly understood or are unexplored. Some of these areas are highlighted here, in the hope that doing so may encourage research in these areas.

Lenticels form beneath stomata during tuber development. How is the position of stomatal pores relayed to underlying cells and what are the molecular changes that result in a highly localized initiation of cell division? It seems plausible that apoplastic signals produced by guard cells could be used as positional indicators. One might also wonder about the similarity between cells in the phellem and adjacent filling cells in a developing lenticel. Differentiation of phellem cells requires thickening, lignification, and suberin deposition in the cell walls. Do filling cells differ from phellem cells primarily in that they lack extensive cell wall development with the result that the cells are larger, rounder and less tightly joined?

Oxygen deprivation causes lenticel proliferation. One might suppose that changes in cellular redox status or metabolite concentrations promote proliferation, but data relating to this question are lacking. Also lacking is an understanding of why filling cells are produced under low

oxygen conditions, but cell division is not promoted in adjacent periderm cells. There are data from lenticels in other species suggesting that ethylene promotes proliferation. Is the same true for potato tuber lenticels?

Potato lenticels often form a suberized barrier beneath the filling cells, but the properties of this barrier are largely unknown. To various degrees, suberized cell walls hinder pathogen spread and limit the exchange of O₂, CO₂ and water vapor. The permeability of the suberin barrier to gases is largely unknown but the scant data that exist indicate that permeability to gases is likely to vary with the extent and composition of waxes in the barrier. The suberin barrier is ruptured as lenticels enlarge. What is the signal that leads to the production of a new suberized layer once the environmental conditions promoting proliferation are removed?

As lenticels mature, they become less susceptible to infection by common scab, powdery scab, late blight and perhaps other diseases. How is this accomplished? Does increased disease resistance result primarily from the mechanical properties of the suberized barrier, or do chemical defenses also contribute to observed changes in disease susceptibility? A related question relates to the observation that soft rot bacteria and powdery scab spore balls persist in lenticels throughout the storage period. What other microbes are harbored in lenticels, how is infection avoided, and what are the consequences of their presence on the next generation of potatoes grown from tuber seed?

Clearly, there are numerous opportunities to explore lenticel cell biology, physiology, and interactions between lenticels and other species. Such investigations will shed new light on an essential piece of tuber anatomy and will likely produce frameworks for understanding lenticel biology in a wide range of species.

Declarations

Conflict of Interest The author declares no conflict of interest.

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