Chapter

Blister Blight Disease of Tea: An Enigma

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Abstract

Tea is one of the most popular beverages consumed across the world and is also considered a major cash crop in countries with a moderately hot and humid climate. Tea is produced from the leaves of woody, perennial, and monoculture crop tea plants. The tea leaves being the source of production the foliar diseases which may be caused by a variety of bacteria, fungi, and other pests have serious impacts on production. The blister blight disease is one such serious foliar tea disease caused by the obligate biotrophic fungus *Exobasidium vexans*. *E. vexans*, belonging to the phylum basidiomycete primarily infects the young succulent harvestable tea leaves and results in ~40% yield crop loss. It reportedly alters the critical biochemical characteristics of tea such as catechin, flavonoid, phenol, as well as the aroma in severely affected plants. The disease is managed, so far, by administering high doses of copper-based chemical fungicides. Although alternate approaches such as the use of biocontrol agents, biotic and abiotic elicitors for inducing systemic acquired resistance, and transgenic resistant varieties have been tested, they are far from being adopted worldwide. As the research on blister blight disease is chiefly focussed towards the evaluation of defense responses in tea plants, during infection very little is yet known about the pathogenesis and the factors contributing to the disease. The purpose of this chapter is to explore blister blight disease and to highlight the current challenges involved in understanding the pathogen and pathogenic mechanism that could significantly contribute to better disease management.

Keywords: tea, blister blight, *Exobasidium vexans*, Basidiospore, defense, control

1. Introduction

Tea is one of the most popular beverages worldwide, having gained popularity for its taste, stimulating effect, various medicinal properties, and related health benefits. Tea is processed from the leaves of evergreen, woody, and perennial tea plants (*Camellia sinensis*) belonging to the family Theaceae. Three indigenous varieties of tea plant are found *viz. C. sinensis* (L.) O. Kuntze (China type), *C. assamica* (Assam type), and (3) *C. assamica* sub *spp lasiocalyx* (Planchon ex Watt.) or Cambod type. These varieties are capable of cross-pollination and interbreeding, resulting in heterogenous hybrids. Under natural conditions, the tea plants can grow up to a maximum height of 15 m, while cultivated tea plantations are maintained as a bush with a height of 60–100 cm, which facilitates the plucking of tender leaves [1]. Tea was first used as a beverage in China in 2737 B.C and was introduced in India for commercial production by the erstwhile colonial British, through the East India Company in 1853 [2]. Tea plantations in India are found in three main geographic regions - the Northeast, (Assam, West Bengal, Tripura, Sikkim, Manipur, Nagaland, Meghalaya, Arunachal

Pradesh, and Mizoram), the South (Karnataka and Tamil Nadu), and the Northwest (Himachal Pradesh and Uttarakhand) [3]. The Darjeeling Tea from northeast India has been signified as the world's premium, and exotically flavored tea owing to its unique flavor and aroma, earning itself a GI tag.

Presently tea is cultivated worldwide across 61 countries of which China, India, Kenya, Sri Lanka, and Vietnam are the largest tea-producing countries, contributing 77% of world production and 80% of global exports (**Figures 1** and **2**). China is reported to produce 2700 million kg of tea of which 366.6 million Kg were exported for the year 2019 with one million hectares under tea cultivation. This was followed by India and Kenya with 1390.1 and 458.9 million kg of tea production. Recently, Kenya was listed with the highest exporter of tea for the year 2019 with 392.6 million kg of exports. India accounts for 23% of the total world tea production with an

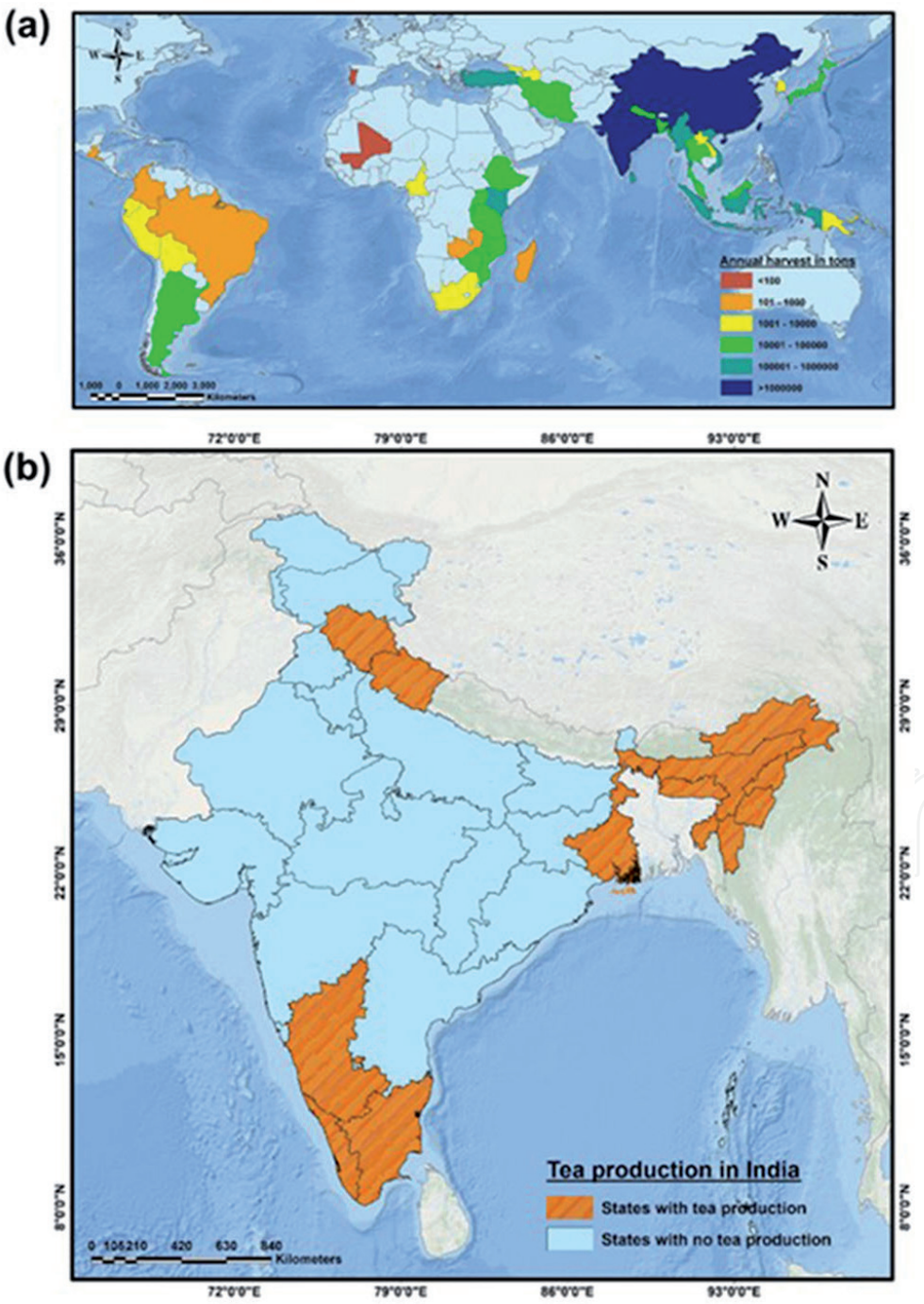

Figure 1.
Tea producing countries worldwide (a), area of tea cultivation in India (b).

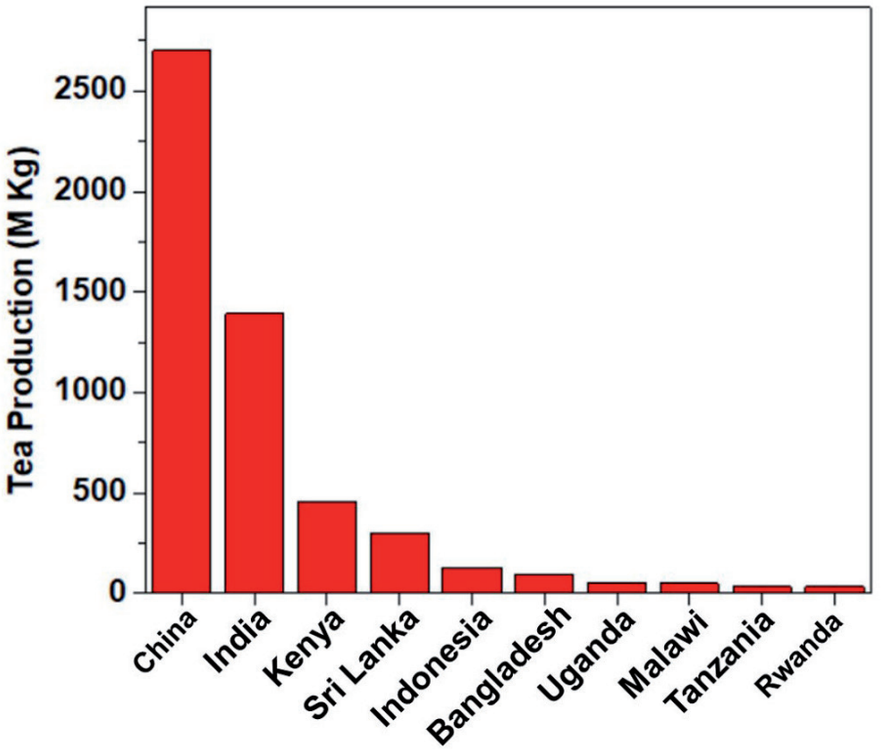

Figure 2.
Tea production of top 10 countries for the year 2019–2020 (source: Tea board of India).

area of 400,000 hectares under tea cultivation. This contributes to about US $803 million to the Indian economy **(Tea board of India)**. India is followed by Sri Lanka with an export worth US $721.3 million. In addition to contributing majorly to the economies of the tea growing countries, the tea industry also provides livelihood to a significant part of the population in these countries.

Different forms of tea have been produced from the same tea plantations depending on the method of processing and plucking of leaves. Some of them are black tea, green tea, oolong tea, white tea, pure tea, and dark tea. Black tea is the most popular form of tea produced in all the major tea producing countries including India, Kenya, and Sri Lanka. Production of black tea in India accounts for 85% of total worldwide production and green tea is being produced by a few tea gardens. Green tea is the most popular form of tea in China followed by black tea.

Tea being a perennial and monoculture crop, the microclimate of tea plantations makes it prone to various pests and pathogens [4]. Chen and Chen recorded around 400 pathogens 507 species of fungi infecting tea plants [5, 6]. Although all the parts of the tea plant including leaf, stem, and root are prone to infection, the pathogens invading leaf parts are of great concern as the main source of commercial production of tea is the young and fresh leaves. The incidence of diseases in the leaves significantly affects the crop yield and quality of made tea. This also directly affects the economy of agronomic countries where tea is considered an important cash crop. The various diseases of tea can be categorized into primary and secondary diseases. In case of the primary diseases, the pathogens directly invade healthy tea bushes while secondary diseases are caused by weaker parasites infecting already diseased/infected tea bushes. In this context, some of the most important diseases infecting the leaf, stem, and root of tea plantations are listed in **Table 1**.

Blister blight disease is one of the most serious primary foliar tea diseases that significantly affects the crop yield and quality throughout various regions of tea-producing countries across the world. The causal organism of Blister blight disease is the biotrophic fungus *Exobasidium vexans* Massee. Peal was the first to recognize

Infection Site	Disease	Causal organism	References
Leaf	Blister blight	*Exobasidium Vexans*	[7, 8]
	Black rot	*Corticium invisum* and *Corticium theae*	
	Leaf red rust	*Cephaleorus mycoidae*	
	Brown blight	*Colletotrichum camelliae*	
	Gray blight	*Pestalozzia Theae*	
Stem	Poria Disease	*Poria hypobrunnea*	
	Nectria	*Nectria cinnabarina*	
	Black root rot	*Rosellinia arcuata*	
	Brown root rot	*Fomes lamaoensis*	
	Jew's ear fungus	*Auricularia auricula*	
	Thorny blight	*Aglaospora aculeata*	
	Ganoderma	*Ganoderma applanatum* and *Ganoderma lucidum*	
Root	Red root rot	*Poria hypolateritia*	
	Tarry root rot	*Hypoxylon asarcodes*	
	Purple root rot	*Helicobasidium compactum*	
	Charcoal stump rot	*Ustulina zonata*	
	Violet root rot	*Sphaerostilbe repens*	
	Thorny blight	*Aglaospora* sp.	

Table 1.
Commonly prevalent diseases of tea plantation.

the occurrence of blister blight in the year 1868 in North East India [9]. The disease mainly attacks young harvestable tender leaves which are used for the commercial production of tea. Blister blight causes enormous crop loss throughout the major tea-growing countries of Asia including India, Sri Lanka, Indonesia, China, and Japan causing a yield loss of 40% globally [10]. The incidence and severity of blister blight depend on the nature of the tea cultivar, geographical, and environmental conditions of the tea growing areas. The percent yield loss of made tea due to blister blight incidence across the major tea producing countries is represented in **Figure 3b**. Some of the most susceptible tea cultivar prone to blister blight infections are UPASI-1 and UPASI-3 (Assam), UPASI-9 and UPASI-15 (China), and UPASI-17 and TRI-2025 (Cambod), BSS-1, etc. [11]. Here is a comprehensive discussion on the incidence of blister blight disease in different countries, the causal organism, disease cycle, epidemiology, severity, and different approaches employed for the control of blister blight.

1.1 History

Balidon in his book 'Tea in Assam' has indicated the prevalence of blister blight disease on wild indigenous tea in Assam shortly after the beginning of tea cultivation during 1863 [12]. Shortly afterwards Peal in 1868 recognized the existence of blister blight disease of tea and Sir George Watt was the first to report the disease symptoms in Assam in the year 1895. [9, 13]. Later the confirmation of causative pathogen of blister blight as *Exobasidium vexans* was reported by Massee, the Mycologist of Kew Botanical garden in 1898 based on samples sent by Dr. Watt from Upper Assam, India [14]. In 1908, a sudden outbreak of the disease occurred in Darjeeling, West Bengal, India, and

(a)

Climatic condition	Range
Temperature	20-25
Relative humidity	>80%
Sunshine	3.5 h
Rainfall	Moderate
Leaf wetness	15 h

(b)

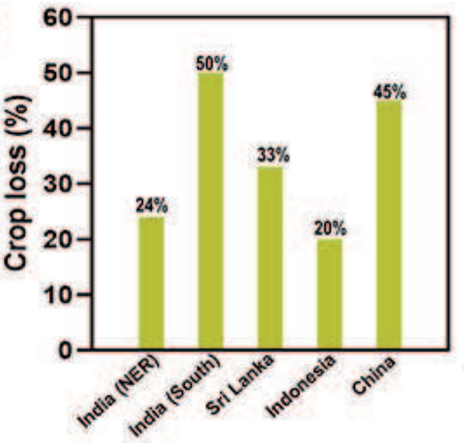

Figure 3.
(a) Climatic condition influencing blister blight disease, (b) blister blight mediated crop loss (%) across major Asian countries [NER: North east region].

was then subsequently reported from Formosa (Taiwan) in 1912, Japan in 1922, and Taiwan in 1938 [15–17]. This was followed by the emergence of the disease in southern India in August 1946, wherein blister blight incidence was first reported in tea estates of Mundakayam and Peermade valley in Kerala. The disease soon spread in the West-Northwest and Southwards direction to Anamallais, other tea estates of Kerala, Nilgiris, Wynaad, and Chikmagalur in Karnataka, due to the effect of South West and North East monsoon winds, thereby affecting the entire tea growing regions of southern India [18]. Later on, the disease was reported from Sri Lanka in 1947 [19], from Sumatra and Java in Indonesia in 1949 [20], Nepal in 1948, East Pakistan in 1951, Thailand in 1953, and South Vietnam and Cambodia in 1959 (CMI, 1970). Hence, blister blight has eventually become a devastating tea disease throughout all the major tea plantations of Asian countries including India, Sri Lanka, Bangladesh, Cambodia, China, Indonesia, Japan, Malaysia, Nepal, Taiwan, Thailand, and Vietnam.

1.2 Blister blight disease symptoms, pathogen, and life cycle

The causal organism of blister blight disease *Exobasidium vexans* is known to be an obligate biotrophic fungus with no alternate host completing its entire life cycle in tea (**Table 2**). The pathogen mainly attacks young, succulent, and tender harvestable leaf and shoot thereby inflicting an enormous effect on the quality and quantity of consumable tea production. The pathogen reproduces through basidiospores which are commonly known to get dispersed by wind. The basidiospores germinated upon lodging on the surface of susceptible tea leaf surfaces under a humid atmosphere with a minimum relative humidity of 80%. The infection is facilitated by the formation of infection peg from appressoria either directly penetrating the cuticle of host tissue or penetration through stomata [21]. The first apparent sign of infection appears in young leaves in the form of pink translucent spots which are considered as the first stage of blister blight infection and are visible after three days of fungal penetration. The spots enlarge along with the leaves and approximately reach a diameter of 3–12.5 mm. In the second stage, the enlarged spots develop into white and velvety convex blister lesions on the lower surface of tea leaf, and on the upper surface, the area with blister lesions becomes sunken resulting in concave depression [22]. The disease progress to its third stage characterized by the curling of the infected tea leaf, browning of blister lesions, and consequently necrosis of the infected leaf tissue. The life cycle of blister blight disease is represented in **Figure 4**. During the off-season, the pathogen is reported to survive on these necrotic leaf parts which facilitate the infection to occur in the

subsequent season under favorable climatic conditions. In a study was carried out to detect the survival of *E. vexans* during the off-season the presence of basidiospores in the atmosphere was reported indicating the active state of the pathogen throughout the year. However, the spore concentration being very low (10 spores/m3) the basidiospores failed to sporulate [23]. Under favorable climatic conditions, the pathogen completes its life cycle within 11 days, although it is reported at times extend to 28 days

Kingdom	**Fungi**
Phylum	Basidiomycota
Class	Exobasidiomycetes
Subclass	Exobasidiomycetidae
Order	Exobasidiales
Family	Exobasidiaceae
Genus	*Exobasidium*
Species	*vexans*

Table 2.
The taxonomic position of E. vexans as described by Massee [14].

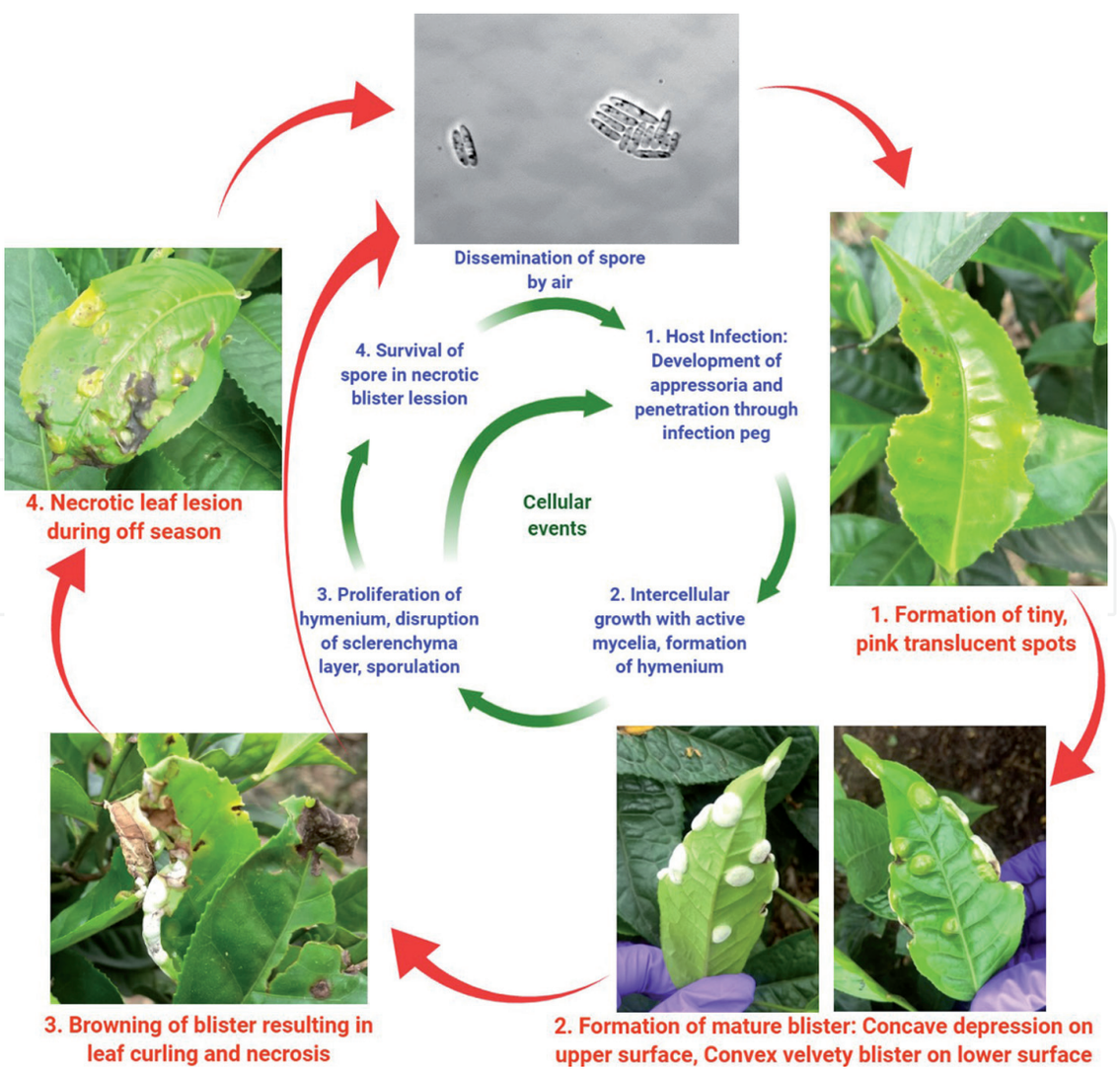

Figure 4.
Disease cycle of blister blight along with life cycle of causal organism Exobasidium vexans.

depending on the prevailing climatic conditions. Owing to the short span of the life cycle, multiple generations of the pathogen are completed within a single cropping season. For the development of blister blight infection sporulation to germination takes place in 4 h to 5 days, germination to penetration takes 4–9 days, penetration to the appearance of visible symptoms takes 3–10 days, development of mature blister and subsequent sporulation takes 11–28 days [11].

A histological study of blister blight disease on tea leaves provided insights into the cellular alteration of host tissue during infection. The study revealed that during the first stage of infection, the enlargement of the translucent spots is a result of hypertrophy as the size of the cells in the mesophyll layer of the infected leaf was substantially higher as compared to the healthy leaf. In the lower epidermis of infected tea leaves with mature blisters, the development of hymenium was prominent in the second stage. This disrupts the lower epidermis completely and gets filled with networks of intercellular hyphae which subsequently develop into basidia that bear basidiospores. However, in tea leaves with blister infections localized in veins, the proliferation of hymenium was apparent in both the lower and upper epidermis. This results in the disruption of the sclerenchyma layer in the vein thereby rupturing xylem and phloem resulting in leaf curling and necrosis of infected leaf in the third stage. The hymenium consisting of bundles of hyphae on maturity forms the clavate to cylindrical basidia (46.98–86.42 μm × 4–5 μm) with normally two and rarely three to four sterigmata [24]. The basidiospores of *E. vexans* are formed at the apex of these sterigmata and two nuclei from the basidium pass into spore via fission. The basidiospores are hyaline and elliptical and measure 7–15.5 X 2.3–4.5 μm when observed under a microscope. It has been reported that 10,000 basidiospores are produced per mm^2 of the blister lesion while the mature blister lesion can produce up to two million basidiospores in 24 hrs [25, 26]. Although the basidiospores are single-celled when immature, three to four septa are reported to form during germination [27]. In a recent study, stages of basidiospore germination were reported under *in vitro* conditions (**Figure 5**). The basidiospores were found to germinate on agar surface 4 h post-incubation followed by germ tube growth from either one or both ends. The spores were initially observed to be aseptate and at a later stage, as many as four transverse septa were formed. At 8 h post-incubation, the formation of hyphae was observed that differentiated into branches to form a complex network of hyphae [28]. So far, the identification of *E. vexans* is being carried out by studying the basidiospore morphology as discussed above, and blister blight disease is identified symptomatically. Molecular based identification remains a major challenge as very few sequences for the ITS region of *E. vexans* have been deposited in NCBI to date. This urges the development of a specific molecular barcode for the identification of *E. vexans*. *E. vexans* being an obligate biotrophic fungus, establishing *in vitro* culture to study the pathogen is another major challenge. In this context, Sundstrom reported that thiamine is a significant supplement in culture media [29]. This was followed by the use of media based on natural substrates for the growth of *E. vexans* under laboratory conditions [30, 31]. Also, vitamin B_5 and calcium pantothenate has been indicated as necessary supplements to maintain *in vitro* culture of *E. vexans* for a period of up to 48 h [32]. Viable *in vitro* culture of *E. vexans* was achieved up to four-five weeks from an optimization study on three different basal media. The carbon source was found as the most significant parameter for czapek dox and v8 juice while tea leaf extract for potato dextrose along with optimal temperature and pH range to be 25–27°C and 7–8 respectively [28].

1.3 Epidemiology, occurrence, and disease severity

The weather condition plays a very important role in the epidemiology and severity of blister blight disease. Low temperature, high humidity, cloudy condition with

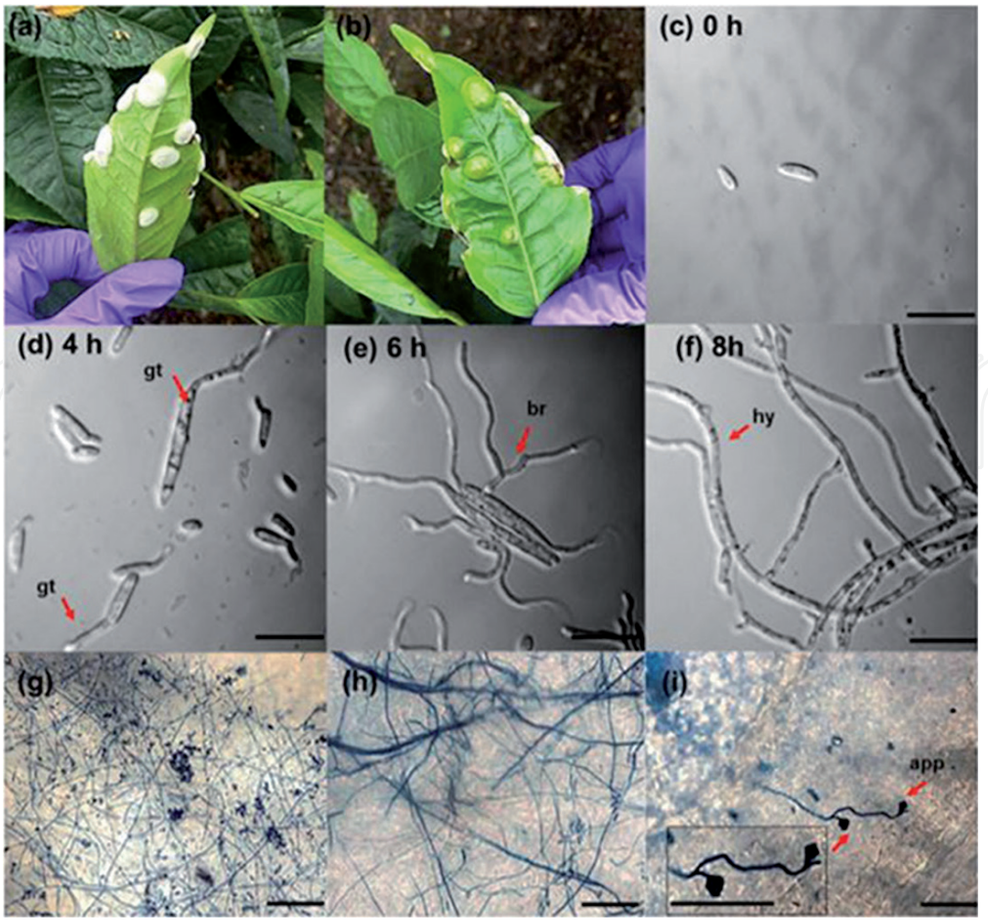

Figure 5.
Representative image of dorsal (a) and ventral (b) surfaces of blister blight infected tea leaf (Ananda tea estate, Lakhimpur, India). Different phases of germination of E. vexans basidiospores on agar: Basidiospores (c); germ tube growth from single end of basidiospores (d); germ tube growth from both ends of basidiospores (e); and hyphal growth of E. vexans with branching (f). Infected leaf part with extensive hyphal growth (g–h) and formation of appressorium (i) (scale: 10 μm, gt-germ tube, br-branching of hyphae, hy-hyphae, app-appresorium) [adapted with permission from Chaliha et al. 2020].

moderate rainfall has been found to play a profound impact on the development of pathogen and disease incidence. As such the incidence of the disease is most favored in monsoon season and facilitated with relative humidity (RH) of more than 80% and availability of water on the leaf surface. In a study carried out by Huysmans (1952), blister blight incidence was recorded with a 5-day average of RH of greater than 83%. On the other hand, Homburg (1953) studied that RH below 80% over 5 days was unfavorable to blister infection. Venkata Ram has reported that the optimum period of leaf wetness to facilitate infection was 11 h and the maximum infection occurred at 13 h [33]. The requirement of moisture content for the germination of basidiospore is reported to be provided by approximately 0.1-inch rain per day while the optimal growth temperature was recorded to be 20–25°C with a maximum tolerance limit of 34°C [29, 34]. Sporulation was found to be inhibited at a temperature greater than 35°C and a temperature of 32°C was reported to be lethal for the basidiospores of *E. vexans* [35]. The incidence of blister blight disease is inversely related to the period of sunshine. Visser et al. in 1961 found a reduction of blister blight disease with an average of 3.5 h of sunshine per day for 5 days at a stretch [34]. Following this report, an exercise of cutting shade trees to medium height for allowing penetration of sunlight to the tea canopy was practiced in Sri Lanka [36]. In a different study, the UV-B (290–320 nm) component of sunlight was found to reduce the sporulation in blister thereby decreasing the number of spores at the end of the disease cycle [37]. Variation in the nature of spores and sporulation behavior was also observed in basidiospores

developed under adverse climatic conditions. The basidiospores produced during unfavorable months were found to be thick-walled that failed to germinate. Also, the atmospheric spore count was less during these months. However, in tea plantations close to the ravine that recorded low temperature and high humidity, blister blight disease was noticed during the unfavorable months. Also, tea plants in these areas experienced surface wetness of 15 h which is ideal for blister blight disease incidence [23]. The optimal climatic condition influencing blister blight disease (**Figure 3a**) and percentage crop loss in major Asian countries (**Figure 3b**) is depicted in **Figure 3**.

The spore liberation in the air over blister blight infested tea plantations follows a diurnal rhythm and resembles a nocturnal pattern of spore discharge of other basidiomycete pathogens. The maximum liberation of basidiospores was found to occur between midnight and 4.00 am [38]. The spore deposition on tea plants was found to be directly proportional to the number of spores in the atmosphere. However, the difference in spore deposition in different bushes was observed with higher spore deposition in susceptible hosts [39].

Blister blight disease, being a foliar disease, directly affect the quality and quantity of consumed tea. Severe disease incidence has been recorded after pruning of tea plantations owing to the abundance of young and tender leaf and stem. Also, during infection of tender stem the entire shoot withers and falls along with the curled infected leaf making it unusable for plucking [33]. As such along with enormous yield loss a quality deterioration below 35% disease threshold level is imposed due to blister blight infection [10, 40]. The percentage of crop loss varies with the geographical condition of different countries. In Sri Lanka, Loos reported 50% crop loss in tea plantation without protection, and 33% in plantations protected with copper fungicide [41]. Indonesia reported a loss of ~10 million kg of tea which is 20–25% between 1951 and 1952 [16]. In southern India, during the initial years of blister blight infection, enormous crop losses were observed with an annual loss of about 18 million kg of tea between 1948 and 1952 [42]. North-east India reported, crop loss up to 24% due to blister blight infection, the infection occurring mostly in the hilly region. Darjeeling has been reported with the worst effected tea plantations with blister blight, owing to the favorable climatic conditions. The onset of the disease has been recorded in June with the starting of monsoon and reaches its severity in August till October. In Assam, India blister blight incidence was associated with early rain in February and reaches its severity in the month of March–April [43].

Blister blight infection results in significant degradation of quality in made tea owing to changes in biochemical characteristics [44, 45]. Gulati in 1999 carried out the analysis of biochemical parameters in diseased leaf. In the infected leaves catechin content, total phenols, nitrogen, chlorophyll, amino acids, and polyphenol oxidase activity was recorded in decreasing concentration in comparison to healthy leaf. In orthodox tea processed from infected tea leaves theaflavins, caffeine, catechin polymer thearubgins, and aroma components were significantly found in reduced concentration [46]. Tea shoots with blister infection were also reported with a decrease in catechin content, flavor component 2-phenyl ethanol, and enzyme activity of prephenate dehydrase [47].

1.4 Blister blight disease control

Considering the severity of blister blight disease and its related agronomic and economic losses control of the disease is of utmost necessity. Various control measures have been adopted for the protection of tea plantations against the disease of which the use of therapeutic approaches at the field scale started around 40 years ago [48]. Over the last few years, the various control measures adopted against blister blight disease can be categorized into cultural, chemical, biological,

and host tolerance approaches. Different studies have been carried out concerning control measures against *E. vexans* infection. An overall representation of various approaches in controlling blister blight disease is shown in **Table 3**. Details of these approaches and their application at field scale are discussed below.

1.4.1 Cultural practice

E. vexans mainly infect young succulent harvestable leaves which are normally plucked for commercial tea production. The cultural practice of hard plucking also known as fish leaf plucking and early pruning has been employed in tea plantations to reduce the severity of blister blight infection. Eden (1947) reported that hard plucking practice after every two to three months in a year causes no major damage to the tea bushes. However, the long-term hard plucking can result in decreasing crop yield as the bushes get weakened and become susceptible to the attack of mite [57]. Also, tea plantations after hard plucking get delayed and irregular with the growth of new foliage, and as such the tea plantations look worn out [41]. Severely infected tea plants are pruned immediately to control blister blight diseases. Pruning was carried out during hot dry weather to ensure the growth of foliage in the period when the disease presented no danger. However, this resulted in the sunscald damage of the stems that developed into cankers [58]. In line with cultural practices, pruning of shade trees is also rehearsed to allow sunlight to fall on tea bushes as long-term sunlight is reported to inhibit the germination of basidiospores of *E. vexans* [59]. UV-B solar radiation component has been reported to play a significant role in the natural regulation of blister blight disease. The study has shown that removal of UV-B component from sunlight falling on tea bushes resulted in an increasing number of blisters formation while on the other hand, complete sunlight reduced the number of sporulating blisters post 62 h of inoculation. This proves that prolong durations of sunlight can have a negative impact on completing several generations within the same cropping season [37].

Treatment		Disease inhibition	References
Chemical control	Copper oxychloride	85.43%	[49]
	Hexaconazole	78.10%	[49]
	Tridemorph	67.8	[49]
	Propiconazole	78.50%	[49]
	Bitertanol	72.50%	[49]
	Nickel Chloride	84%	[50]
Biological control	*Trichoderma harzianum, Gliocladium virens, Serratia marcescens, Pseudomonas fluorescens, Bacillus subtilis*	40%	[51]
	PGPR	83.94%	[48]
	Ochrobactrum anthropi	73.40%	[52]
	Azoto II-1, *Acinetobacter* sp., Endo-5, Endo-65, and Endo-76	33%	[53]
Systemic acquired resistance	Acibenzolar-S-methyl and salicylic acid	40.80%	[54]
	Calcium chloride ($CaCl_2$)	80%	[55]
	Chitosan	67.70%	[56]

Table 3.
Disease inhibition (%) with various control measures.

1.4.2 Chemical control

The importance of chemical control of blister blight disease and the use of economically feasible chemical therapeutics dates back to 1960 when the disease incidence was recorded in southern India. Protectant fungicides, eradicant fungicides, and systemic fungicides are used as foliar sprays against blister blight disease. Bordeaux mixture and copper oxychloride are the two most commonly used protective fungicide formulations. The acceptance level of the use of copper in tea leaves to control blister blight was set at 150 ppm (Lamb, 1950) as copper-based fungicides also possess collateral damage of phytotoxicity and release of copper residues to environment causes human health hazard, effect soil microflora, and marine population. The formulation of copper oxychloride was able to control blister blight disease at a usage rate of 0.21 Kg metallic copper per hectare. The concentration of copper at 50% wettable powder was used in copper oxychloride formulations [59–61]. Eradicant fungicide nickel chloride hexahydrate was found effective in controlling blister blight disease by antisporulant activity. The reduction in infection was achieved from 84–24% post 3 weeks of treatment and up to 13% after 5 weeks. However, the treatment with nickel chloride was found severely phytotoxic which rejected its use as a potent fungicide [50]. Owing to the phytotoxicity and collateral health hazard from chemical fungicides, organic fungicides were introduced in Sri Lanka, Indonesia, and southern India. However, the disease resistance efficacy was lower in comparison to copper-based fungicides. Also, the high cost related to the processing of organic fungicides discarded its use for blister blight control [25, 60, 62, 63]. Two common brand names for organic fungicides used for blister blight control are Daconil and Difolatan [64].

Conventionally, around 26 rounds of spraying of these fungicides are carried out at 7-days intervals during the disease season to control blister blight incidence. However, since climatic conditions play a significant influence on the severity of blister blight incidence the spraying interval of fungicides differs from region to region. In Indonesia and Sri Lanka an extended period of spraying based on sunshine hours at a specific period of the disease season mediated the control of disease at the economic threshold. On the other hand, control of blister blight disease was not achieved even after a 7-day spraying interval in southern India [65]. Systemic fungicides are often used against plant pathogens owing to its sustained control of plant for example, blister blight could be controlled in southern India by administering pyracarbolid (Sicarol) over 3 weeks. This treatment exhibited strong antisporulant activity reducing the sporulation in mature blisters, while eradicating the latent blister lesions. Also, plant growth was found to be stimulated with the use of pyacarbolid [33]. In a different study with systemic fungicides ergosterol biosynthesis inhibiting (EBI) fungicides tridemorph, bitertanol, hexaconazole, and propiconazole were studied for its effect on physiological parameters of the tea plant and controlling blister blight disease in southern India. EBIs were found with antisporulant activity with a significant reduction in spore size, viability, and inhibited spore germination except for tridemorph treatment. As such, inhibition in spore germination reduced the viability of spore which mediated the reduction in spore load thereby controlling blister blight incidence. The effectiveness of treatment lasted for 7 days, with a reduction of the occurrence of the disease by half relative to untreated plants. Additionally, the EBIs were found with a positive effect on the physiological parameters of the tea plant. The stomatal conductance, total chlorophyll, carotenoids, and photosynthetic rates were found to be induced with EBIs treatment along with an increase in biometric parameters like dry weight and shoot length [49]. In North-East India, 2–3 rounds of systemic fungicides like propiconazole or hexaconazole has been used at 5% EC @ 1:1000 as a foliar spray at 14 days interval to control blister blight infection **(Tea board of India)**.

1.4.3 Biological control

Besides showing appreciable control of blister blight disease with chemical therapeutics, the related phytotoxicity and health hazard have initiated the approach of biological control of blister blight disease. The use of biological control agents like *Trichoderma harzianum*, *Serratia marcescens*, *Gliocladium virens*, *Bacillus subtilis,* and *Pseudomonas fluorescens* have been studied against blister blight disease [66–69]. However, the use of these bioformulations was not found efficient in controlling blister blight disease. Plant growth-promoting rhizobacteria *Pseudomonas* and *Bacillus* were tested for the control of blister blight disease. Foliar application of *Pseudomonas fluorescens* Pf1 bioformulation at 0.5% concentration weekly showed appreciable efficiency against blister blight disease and the lowest mean disease index of 16.06% was achieved which was at par with the chemical fungicide (14.57%). Reduction in disease incidence was achieved for two seasons and the treated tea plants were found with an induced accumulation of defense enzymes peroxidase, polyphenol oxidase, b-1,3-glucanase, chitinase, phenylalanine ammonia-lyase, and phenolics as compared to control. As such, PGPR mediated induced systemic resistance of tea plants against blister blight infestation [48]. Tea phylloplane bacteria have been isolated and screened for their inhibitory action against blister blight disease, of which isolate identified as *Ochrobactrum anthropi* and designated as BMO-111 was found efficient for the biocontrol of *E. vexans*. Foliar application with BMO-111 at 15 days intervals up to 120 days recorded a reduction in disease incidence. Treatment with BMO-111 resulted in 73.4% protection, compared to 64.7% protection with chemical treatments. An inhibitory effect of basidiospore germination and antifungal effectivity was achieved against *E. vexans*. However, the mechanism of action is still unknown and urges further investigation [52]. Although the majority of studies in this context have been carried out in India, a recent study in Indonesia reported the potential use of soil bacteria (Azoto II-1) and three endophytic (Acinetobacter sp., Endo-5, Endo-65, and Endo-76) bacteria against blister blight incidence. The bacterial suspension was to the soil of infected tea plantations at a dose of 2 l ha^{-1}, applied six times at a 1 week interval. The bacterial formulations were observed to control blister blight intensity only up to 2 weeks after treatment. However, the reduction of disease was not significant as compared to control. Also, the treatment was not accompanied by an increase in yield of fresh shoots except for *Acinetobacter* sp. that showed a 17.26% increase in fresh shoot yield [53]. Thus, in the context of biological control of blister blight disease, bioformulation of microbes and its commercialization at the field scale urges further studies for the screening of beneficial microbes that may be applied for efficient management of blister blight disease.

1.4.4 Host resistance against blister blight disease

In the context of blister blight disease, a variety of tea cultivars have been reported that are found to be resistant to *E. vexans* infection. Various studies have been carried out on the morphological characters of these cultivars and the various blister blight defense-related enzymes and pathways have been analyzed to study the nature and basis of resistance. A study was carried out with blister blight resistant clone SA-6 and a susceptible clone TES-34. Both the clones were studied for anatomical differences such as cuticle and epidermal thickness, stomatal length and breadth, palisade tissue, and quantification of epicuticular wax. They have reported that the resistant clone SA-6 showed a higher amount of epicuticular wax, the high thickness of cuticle with greater stomatal frequency when compared to TES-34. Upon pathogen infection, PR proteins are reported to induce systemic acquired resistance in plants. As such, the study also analyzed the difference in the production of PR protein chitinase and have found higher constitutive expression of chitinase in

resistant clone [70]. In a recent study with these cultivars, SA6, and TES34, the difference in gene expression against blister blight infection was accessed for chitinase, glucanase, phenylalanine ammonia-lyase, and genes in the flavonoid pathway. The relative intensity of the expression of these genes was found to be higher in the resistant cultivar SA6 in comparison to susceptible cultivar TES34. Also, the expression of these pathogenesis-related genes was found to increase along with each successive stage of blister blight infection [71]. A similar study was conducted recently on the biochemical characterization of resistant tea cultivar AV-2 and susceptible cultivar B-157. Secondary metabolites phenol and proanthocyanidin content were reportedly higher in the resistant clone AV-2. As such, the inferred resistance may be attributed to the antifungal properties of phenol and free radical scavenging activity with the chelation of transition metals by proanthocyanidins. Also, a higher concentration of hydrolyzing enzyme acid phosphatase, peroxidase, catechol oxidase, and superoxide dismutase occur in AV-2 cultivar as compared to B-157 [72].

In line with understanding the basis for resistance of tea cultivar against blister blight disease transcriptome profiling of two cultivars P-1258 (resistant) and T-78 (susceptible) have been carried out to identify defense-related transcripts particularly in a resistant cultivar. cDNA-AFLP mediated the screening of differentially expressed candidate transcripts which mainly showed homology with an acyl-CoA binding protein, zinc finger family protein, ubiquitin, and proline-rich protein that were upregulated after infection. Suppression subtractive hybridization-based transcriptome analysis resulted in a comprehensive study of transcripts induced in resistant cultivar P-1258 after infection. The induced contigs showed similarity to proteins such as ubiquitin family protein, an iron–sulfur cluster scaffold protein, short-chain dehydrogenase, ribulose-1,5-bisphosphate carboxylase/oxygenase small subunit, thioredoxin, pathogenesis-related proteins (chitinase, endo-glucanase, beta-glucosidase, wound-induced protein, protease inhibitor, thaumatin-like protein, cystatin, blight associated protein p12, aspartic proteinase) and proteins with a function in defense signal transduction pathway (serine/threonine-protein kinase, oxo-phytodienoic acid reductase, mitogen-activated protein kinase, leucine-rich repeat transmembrane protein kinase, salicylic acid-binding protein, calcium ion binding or calmodulin-related protein, hydrogen peroxide-induced protein, chitin-inducible gibberellin responsive protein, calreticulin). qRT-PCR based expression analysis of the genes showed greater than two-fold upregulation in P-1258 when compared to T-78 post-infection. Hence, the expression profiling mediated the molecular characterization of resistant tea cultivar involved in developing possible systemic acquired resistance against *E. vexans* infection [73]. To provide further insights into the molecular mechanism of host resistance against blister blight disease, a similar study of transcriptome profiling was carried out with tea cultivars SA6 (resistant) and Kangra-Asha (susceptible) at different stages of blister blight disease. In SA6 cultivar at stage 1 (post 24 h infection), salicylic acid metabolism and secondary metabolite biosynthetic processes were recorded to be enhanced. At stage 2 (7 dpi) hydrogen peroxide metabolic processes, cellular metabolism, and ion transport metabolism-related genes were found to be enriched in resistant variety. As such, hydrogen peroxidase activity suggests efficient scavenging of free radicals enabling restricted penetration/germination of spores inside the host tissue thereby conferring resistance. At stage 3 of infection (14 dpi) increased expression of phenylpropanoid (PAL) and aromatic compound category of gene synthesis probably lead to the synthesis of antimicrobial compounds that might have protected intercellular hymenium development in SA6 cultivar. It was interesting to note that at stage 4 (20 dpi) during necrosis of blister infected part Jasmonic acid-mediated signaling pathway genes are enriched in resistant variety SA6 along with induced monooxygenase and ACC oxidase activity and ethylene production [10]. Here, the induction of both SA and JA signaling pathways again urges to examine the hemibiotrophic existence of the pathogen which is otherwise reported to

be biotrophic. In a recent study presence of hypothetical proteins (HPs) was identified and assigned with novel putative defense-related functions in a resistant cultivar of tea SA6 against blister blight disease. The HPs proteins were functionally categorized into LRR, WRKY, NAC, chitinases, and peroxidases. Additionally, different pathways playing a significant role in SA6 resistance against blister blight were annotated based on the KEGG database including plant-pathogen interaction, biosynthesis of secondary metabolites, metabolic pathways, amino sugar, and nucleotide sugar metabolism, and phenylpropanoid biosynthesis which have probably [74].

Plants use various defense mechanisms to shield themselves from infection by pathogens. The cell wall itself acts as an insulation against invading pathogens. Pathogens invading plants breach the cell wall by releasing enzymes and the products get accumulated in the apoplastic region. These are termed elicitors and are capable of activating a complex array of defense signaling called pathogen triggered immunity. These elicitors also mediate the induction of systemic acquired resistance (SAR) in plants. In the last few years, various biotic and abiotic elicitors have been tested and found to mediate SAR in plants against the pathogen. Two chemical elicitors acibenzolar-S-methyl and salicylic acid were tested for their efficiency to induce SAR against blister blight in tea. Plants treated with 0.1% ASM provided 40.8% protection against blister blight. Salicylic acid was used at 250 ppm to achieve significant induction of resistance. Tea plants treated with elicitors were recorded with an induced level of β-1,3-glucanase, phenylalanine ammonia-lyase, and peroxidase activity thereby conferring resistance against blister blight disease [54]. In a similar study treatment of tea plant with abiotic elicitor calcium chloride ($CaCl_2$), found to induce activities of defense enzymes like phenylalanine ammonia lyase (PAL), polyphenol oxidase, peroxidase, and b-1,3-glucanase along with a higher accumulation of total phenolics, thaumatin, cinnamate 4-hydroxylase, flavonoid 30-hydroxylase when compared to control plants [55]. In this context, the use of chitosan as elicitors in tea plants to provide resistance against blister blight disease has been tested and the possible mechanism of resistance has been analyzed. Chitosan solution applied as a foliar spray at 0.01% concentration and 15 days interval reduced blister blight incidence for two seasons. The induced resistance was found to be facilitated by nitric oxide (NO) signaling and the level of total polyphenol content and expression of defense-related enzymes (peroxidase, polyphenol oxidase, phenylalanine ammonia-lyase, and b-1,3 glucanase) was induced [56].

Genetic improvement of tea has been made possible with transgenic technology started from the year 2000. Gene technology and the development in agrobacterium-mediated transformation mediated the incorporation of a foreign gene into crop plants for the development of cultivar with resistance against various diseases. Agrobacterium-mediated transformation was used to develop a disease-resistant variety of tea against blister blight with the introduction of *Solanum tuberosum* class I chitinase gene into tea genome. Plant selectable marker hygromycin phosphotransferase (hpt) gene was used to confer hygromycin resistance. 12 tea plantlets were confirmed with stable integration of transgene that showed resistance against blister blight disease tested in a detached leaf infection assay [75]. In a study carried out by the same group transgenic tea cloned with *Solanum tuberosum* endo-1,3-beta-D-glucanase using Agrobacterium-mediated transformation technique showed significant resistance against blister blight disease. Upregulation of pathogenesis-related (PR) genes like PR3 (chitinase I) gene and PR5 (thaumatin-like protein) gene was recorded in the transgenic tea plantlets which can be attributed to the resistance against blister blight disease [76]. A similar study was carried out for co-transformation of tea genome with LBA4404 pCAMBIA 1301-Chi (carrying potato class I chitinase gene expression cassette) as reported in Singh et al. (2015) and LBA4404 pBI121-Def (carrying mung bean defensin gene expression cassette) together. Although resistance against

blister blight was achieved with the co-transformation the resistance was better with *Solanum tuberosum* class I chitinase gene introduced transgene.

2. Conclusion

Widespread research has been carried out so far on understanding the incidence of blister blight infection and on its control measures for the survival of the tea industry. However, to date identification of the pathogen is being carried out symptomatically and morphologically. In the context of molecular identification of *E. vexans* a few sequences for the ITS region are found in the NCBI database which urges to carry out a more detailed study on the development of molecular barcode-based identification. Also, very little is known so far about the genome and transcriptome profile of the pathogen which could be a basis for understanding the molecular mechanism behind the pathogenesis of *E. vexans*. Understanding the molecular basis of pathogenesis of *E. vexans* would likely mediate control measures to be applied more specifically and efficiently. In line with control measures against blister blight, most of the studies reported so far have worked on defense responses and resistance mediated by tea cultivars and hence urges further studies to elucidate the molecular pathogenesis cycle of the pathogen. As such, from the pathogen point of view, there is a significant gap in understanding blister blight disease and a thorough analysis of the pathogen is likely to address the various associated challenges.

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Conflict of interest

The authors declare that they have no known potential conflict of interest.

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