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Early Blight of Potato

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Early Blight is one of the most common and widely distributed foliar diseases of potato (*Solanum tuberosum* L.) and is caused by the deuteromycetous fungus *Alternaria solani* (Ellis & Martin) Jones & Grout. The disease is prevalent wherever potatoes are grown throughout the world and is particularly destructive in warm, humid climates with alternating wet and dry periods. Under favourable conditions, Early Blight can cause defoliation of up to 70–80 per cent, leading to significant reduction in tuber yield and quality. Early Blight was first described by Ellis and Martin in 1882 in the United States, where they observed characteristic concentric-ringed lesions on potato foliage and named the fungus *Macrosporium solani*. Jones and Grout (1897) subsequently transferred it to the genus *Alternaria* based on its conidial characteristics, giving it the name *Alternaria solani*, which remains the accepted name. The disease was reported from Europe and Asia in the early twentieth century and is now recorded in all potato-growing regions of the world including India, USA, UK, Germany, the Netherlands, and Australia. In India, Early Blight is economically significant in the major potato-growing states of Uttar Pradesh, Punjab, West Bengal, Bihar, and Himachal Pradesh. It tends to appear earlier in the season than Late Blight (caused by *Phytophthora infestans*) and is often confused with it by farmers. The disease is especially severe on older, nutritionally stressed plants, and tends to intensify as the crop matures.

Causal Organism

Taxonomic Classification (Subdivision / Systematic Position)

The causal organism of Early Blight of Potato is Alternaria solani, classified as follows:

Kingdom: Fungi
Division (Subdivision): Deuteromycota (Fungi Imperfecti) — Mitosporic fungi; asexual stage
Class: Hyphomycetes
Order: Moniliales (Hyphomycetales)
Family: Dematiaceae (Pleosporaceae in teleomorph stage)
Genus: Alternaria
Species: *solani*
Full name: *Alternaria solani (Ellis & Martin) Jones & Grout, 1897*

Note: *Alternaria solani* is classified under Deuteromycota (the Fungi Imperfecti) because it was described and primarily identified based on its asexual (anamorphic) stage. When the sexual (teleomorphic) stage is taken into account, the organism is reclassified under the Ascomycota (see Section 3 on Perfect Stage).

In the modern phylogenetic classification system (based on molecular data), Alternaria species are placed under:

Phylum: Ascomycota
Subphylum: Pezizomycotina
Class: Dothideomycetes

Order: Pleosporales
Family: Pleosporaceae
Teleomorph genus: Lewia
Teleomorph species: *Lewia infectoria (Fries) Simmons & Crew*
Morphological Characters
Alternaria solani is a dematiaceous (dark, melanised) hyphomycete characterised by dark brown to olivaceous colonies on culture media. The mycelium is septate, branched, and brown to dark brown in colour. The fungus produces conidia exogenously on conidiophores.
Conidiophores: Conidiophores are simple or branched, erect, geniculate (knee-like), dark brown to olivaceous, septate, and measure 50–110 µm in length and 6–10 µm in width. They arise from the mycelium singly or in groups from stomata or directly through the cuticle.
Conidia: Conidia (asexual spores) are the primary means of reproduction and dispersal. They are the most important diagnostic feature of the fungus. Conidia of *Alternaria solani* are large (150–300 µm × 15–19 µm), solitary or in short chains, pyriform to obclavate (club-shaped) in form, with a long beak-like apical extension (rostrum) that is 2.5–5 times longer than the spore body. They are dark olive-brown, multicellular with both transverse and longitudinal septa (muriform septation), giving them a brick-like internal appearance. The beak is the most distinctive character separating *A. solani* from closely related species such as *A. alternata* (which has short or no beak).

Cultural characters: On Potato Dextrose Agar (PDA), colonies grow rapidly (25–30 mm/day at 28 °C), are initially white to grey and become dark olivaceous-brown to black with age. The reverse of the colony is also dark. The optimal temperature for mycelial growth is 28–30 °C; growth ceases below 6 °C and above 40 °C. Sporulation is stimulated by alternating light and dark cycles (near-UV light enhances conidiation).
Physiological Characters
Alternaria solani produces several pathogenicity and virulence factors. The most important is the host-selective toxin Alternaric Acid, a chlorinated aliphatic compound produced in culture and in planta, which contributes to tissue necrosis and symptom development. The fungus also produces cell-wall degrading enzymes including cellulases, pectinases, and proteases that macerate host tissue, and also produces the enzymes cutinase for penetration through the plant cuticle. The fungus is a hemi-biotroph — it has a short biotrophic phase at the beginning of infection followed by a necrotrophic phase where host cell death drives colonisation. Several races of A. solani have been reported, differentiated by their virulence on specific potato cultivars and by sensitivity to the fungicide mancozeb.

Perfect Stage (Sexual / Teleomorphic Stage)

The perfect stage (teleomorph) of Alternaria solani is Lewia infectoria (Fries) Simmons & Crew. The teleomorphic stage is rarely observed in nature and has been reported only infrequently from field conditions. It belongs to the class Dothideomycetes under the phylum Ascomycota.
Sexual Fruiting Bodies: The perfect stage produces pseudothecia (flask-shaped, dark brown ascomata embedded in host tissue). Pseudothecia are globose to sub-globose, measuring 200–400 µm in diameter, with a distinct papillate ostiole (opening). They are immersed in host tissue and later become erumpent.
Asci and Ascospores: Asci are bitunicate (double-walled), cylindrical to clavate, and contain 8 ascospores each. Ascospores are brown, muriform (with both transverse and longitudinal septa), ellipsoidal, and measure 30–45 µm × 13–18 µm — similar in septation pattern to the conidia but smaller and without the long beak.
The teleomorphic stage (Lewia infectoria) is classified as follows:
Kingdom: Fungi
Phylum: Ascomycota
Class: Dothideomycetes
Order: Pleosporales

Family: Pleosporaceae
Genus: Lewia
Species: *infectoria (Fries) Simmons & Crew*
The rarity of the perfect stage under field conditions means that genetic recombination through sexual reproduction is uncommon and the pathogen population is predominantly clonal. However, the sexual stage, when it does occur, can generate new virulent genotypes, making it of evolutionary significance. Most disease epidemics in the field are driven entirely by asexual conidia produced on diseased leaf tissue.

Symptoms

Early Blight primarily affects leaves, stems, and tubers of potato. Symptoms appear first on older, lower leaves and progress upward — a pattern characteristic of diseases caused by necrotrophic pathogens that prefer nutritionally stressed tissue.

Leaf Symptoms

The most diagnostic and characteristic symptom is the appearance of circular to angular, dark brown to black necrotic lesions on the leaves, each surrounded by a conspicuous yellow chlorotic halo. The most distinguishing feature of Early Blight lesions is the presence of concentric rings within the lesion, giving it a target-board or bull's-eye appearance — a feature that is pathognomonic for this disease and is caused by alternating zones of fungal growth and sporulation corresponding to periods of high and low humidity during lesion development. Lesions begin as small (2–3 mm), irregular, dark brown spots that enlarge to 5–15 mm in diameter. Multiple lesions coalesce under heavy infection, leading to blighting and premature defoliation of leaves. A dark, velvety sporulation of the fungus (conidia and conidiophores) is visible in the concentric zones of the lesion under humid conditions. Severely affected leaves turn yellow, wither, and abscise, causing progressive defoliation from the base of the plant upward.

Stem Symptoms

On stems, the disease causes dark brown, irregular, slightly sunken lesions that may girdle the stem in severe infections, leading to stem girdling and collapse of the foliage above. Stem lesions also show the characteristic concentric ring pattern under magnification.

Tuber Symptoms

Tuber infection occurs through lenticels, wounds, or through the stolons. Affected tubers show dark brown to black, circular to irregular, slightly sunken lesions on the surface that are dry and leathery in texture. A section through the lesion reveals dark brown, corky, dry rot extending a few millimetres into the tuber flesh, with a sharp boundary between healthy and diseased tissue. Tuber Early Blight makes tubers susceptible to secondary infection by soft-rot bacteria during storage.

Disease Cycle

Primary Inoculum Sources and Survival

Alternaria solani overwinters (or survives the dry/off-season) as dormant mycelium and conidia in infected plant debris (dead leaves, stems, and haulms) left on the soil surface or incorporated into the soil, as well as on and in infected seed tubers, on volunteer potato plants and solanaceous weed hosts (Solanum nigrum, S. americanum, Datura stramonium, Lycopersicon esculentum — tomato), and occasionally in the soil itself (though survival in soil without host material is limited).

Primary Infection

At the beginning of the growing season, conidia produced on infected debris are disseminated by wind, splashing rain, and irrigation water to the lower leaves of emerging potato plants. The conidia land on leaf surfaces and germinate in the presence of free moisture (dew, rain) within 1–2 hours at temperatures of 24–29 °C. The germ tube penetrates the leaf either directly through the cuticle (using cutinase enzymes) or through stomata and wounds.

Infection and Colonisation

After penetration, the fungus establishes an initial biotrophic relationship, colonising inter- and intra-cellular spaces in the mesophyll. Within 2–3 days, the necrotic lesion becomes visible (incubation period: 2–7 days depending on temperature and humidity). As the lesion matures, the fungus produces conidiophores and conidia on the lesion surface, particularly in the concentric rings — this is the secondary sporulation that drives epidemic development.

Secondary Spread

Secondary spread is entirely through asexual conidia produced on diseased leaf lesions throughout the growing season. Conidia are dispersed by wind and rain splash to upper leaves and adjacent plants, initiating new infection cycles. Multiple secondary infection cycles occur during a single growing season (polycyclic disease), and each cycle takes 5–7 days under optimal conditions, resulting in rapid and progressive disease buildup. The number of secondary cycles, combined with the rate of lesion expansion, determines the ultimate severity of the epidemic.

End-of-Season Survival

At the end of the growing season, the fungus survives on infected haulms, leaves, and tubers left in the field or on storage tubers. In the soil, conidia and mycelium can remain viable for several months in the presence of infected plant debris. The infected debris thus forms the primary inoculum source for the next crop season, completing the disease cycle.

Favourable Conditions for Disease Development

- **Temperature:** The optimum temperature range for infection and disease development is 24–29 °C. The fungus can infect between 6 and 34 °C, but infection is most rapid at 28–30 °C. Alternating warm days and cool nights are particularly conducive to heavy sporulation.
- **Humidity and Leaf Wetness:** Relative humidity above 80% and prolonged leaf wetness (dew, mist, light rain) of at least 1–2 hours are essential for conidial germination and infection. Even brief periods of leaf wetness are sufficient for infection at optimal temperatures.
- **Alternating Wet and Dry Periods:** The characteristic concentric ring pattern is produced because the fungus sporulates during humid periods and growth pauses during dry periods. Crops in regions with alternating dry and wet weather experience the most severe epidemics.
- **Plant Age and Nutritional Status:** Older, senescing plants are far more susceptible than young, actively growing plants. The disease is strongly associated with nitrogen and potassium deficiency, which weakens host resistance. Symptoms typically appear first after flowering when the plant begins to senesce naturally.
- **Dense Canopy and Poor Air Circulation:** Dense planting and lush canopy growth retain moisture around leaves for longer periods, increasing the duration of leaf wetness and the number of infection events per day.
- **Crop Residue:** High levels of infected crop debris from the previous season in the field provide abundant primary inoculum at the beginning of the new season, leading to early disease onset.
- **Susceptible Cultivars:** Varieties with thin, waxy cuticles and without the R-gene or quantitative resistance loci (QTLs) for Alternaria resistance are highly vulnerable under the above conditions.

Management / Control Measures

Cultural Control

- **Resistant Varieties:** Planting moderately resistant potato varieties wherever available is the primary and most economical defence. In India, Kufri Bahar, Kufri Sindhuri, Kufri Jyoti, and Kufri Surya show moderate field resistance to Early Blight compared to highly susceptible varieties like Kufri Chandramukhi. Varieties with thicker, waxy cuticles and higher chlorogenic acid content tend to be more resistant.

- **Certified Disease-Free Seed Tubers:** Using certified, pathogen-free seed tubers eliminates the seed-borne primary inoculum source and reduces the risk of early-season outbreaks.
- **Crop Rotation:** Avoid planting potato or other solanaceous crops (tomato, brinjal, chilli) in the same field for at least 2–3 years. Rotation with non-host crops (cereals, pulses) allows infected debris to decompose and the inoculum to decline.
- **Field Sanitation:** Complete removal and destruction (burning or deep burial) of infected haulms, leaves, and plant debris immediately after harvest eliminates the principal source of primary inoculum for the next season.
- **Balanced Fertilisation:** Ensuring adequate nitrogen, phosphorus, and potassium nutrition strengthens plant vigour and extends the green canopy life, delaying the natural senescence that predisposes the crop to Early Blight. Split nitrogen application is recommended to avoid luxury nitrogen that promotes succulent tissue.
- **Optimum Plant Spacing:** Avoid overcrowding; maintain recommended plant spacing (30 × 60 cm) to ensure adequate air circulation within the canopy, reducing leaf wetness duration.
- **Irrigation Management:** Prefer furrow or drip irrigation over overhead sprinklers to reduce leaf wetness duration. Irrigate in the morning so that leaves dry out before evening, minimising the period of nocturnal leaf wetness.

Mechanical Control

- **Removal of Affected Leaves:** In small holdings and kitchen gardens, manually removing the first-affected lower leaves at the onset of the disease can slow the build-up of secondary inoculum in the canopy.
- **Haulm Destruction:** Mechanical killing or removal of haulms (above-ground parts) 2–3 weeks before harvest — by flailing, chemical desiccation, or manual cutting — reduces the inoculum available to infect developing tubers during harvest operations.
- **Wound Prevention at Harvest:** Careful mechanical harvesting to minimise tuber wounding reduces the entry of the pathogen into tubers during and after harvest.

Biological Control

Biological control of Early Blight has been investigated extensively, and several bioagents have shown promising results both in vitro and under field conditions:

- **Trichoderma viride / T. harzianum:** Trichoderma species are the most widely used bioagents in potato cultivation in India. Soil application of Trichoderma-enriched FYM (2.5 kg Trichoderma formulation per 100 kg FYM per acre) at planting suppresses soilborne inoculum and induces systemic resistance. Foliar sprays of T. viride (0.5% WP formulation) at 7–10 day intervals from the appearance of first symptoms have shown 30–40% reduction in disease severity in field trials.
- **Bacillus subtilis:** Bacillus subtilis strains (particularly PGPR strains) produce iturin A, bacillomycin, and surfactin — lipopeptide antibiotics that disrupt the fungal cell membrane and inhibit germination and growth of Alternaria solani. Foliar sprays of B. subtilis-based formulations (e.g., Serenade, available in some markets) at 2–5 mL/L at 7-day intervals have shown 40–60% reduction in foliar Early Blight under field conditions.
- **Pseudomonas fluorescens:** Pf-based formulations applied as seed tuber treatment (10 g/kg) and foliar spray (2 g/L at tillering equivalent stage) induce ISR and reduce lesion development, with efficacy reports of 25–40% disease suppression.
- **Neem-based products:** Neem leaf extracts (5%) and neem oil-based formulations (3 mL/L) have demonstrated antifungal activity against A. solani in vitro and modest suppression in field conditions when applied preventively.

Chemical Control

Fungicide application remains the most reliable and widely used method for Early Blight control in commercial potato cultivation. Timely and correct fungicide application, combined with cultural measures, is the basis of integrated disease management for this disease.

Spray scheduling: Fungicide sprays should begin at the first appearance of symptoms (or prophylactically 2–3 weeks after canopy closure, around flowering) and repeated at 7–14 day intervals depending on disease pressure and weather conditions.

- **Mancozeb 75 WP (Dithane M-45):** The most widely used and economical protectant fungicide for Early Blight. Apply at 2.5 g per litre of water as foliar spray. It has multi-site action (no resistance risk), broad-spectrum activity, and also provides secondary control of Late Blight.
- **Chlorothalonil 75 WP (Kavach):** Broad-spectrum, multi-site protectant. Apply at 2 g per litre. Highly effective against Early Blight and provides additional control of other foliar diseases. Residue concerns limit its use close to harvest.
- **Iprodione 50 WP (Rovral):** A systemic fungicide (SDHI group) with both protectant and curative action. Apply at 2 g per litre. Particularly effective in wet conditions where systemic activity is needed.
- **Azoxystrobin 23 SC (Amistar):** A strobilurin (QoI) fungicide with both protectant and systemic curative action. Apply at 1 mL per litre. Highly effective against Early Blight when used preventively or at early infection stage. Resistance risk: avoid more than 2 sprays per season with strobilurins; always alternate with multi-site fungicides.
- **Difenoconazole 25 EC (Score):** A DMI (triazole) fungicide with strong systemic curative and protectant action. Apply at 0.5 mL per litre. Effective against Alternaria diseases of potato and tomato.
- **Cymoxanil + Mancozeb 8:64 WP (Curzate M8):** A combination of systemic (cymoxanil) and contact (mancozeb) components providing both curative and protectant action. Apply at 2.5 g per litre. Also controls Late Blight, making it a dual-action product under mixed disease conditions.
- **Propiconazole 25 EC (Tilt):** Systemic triazole. Apply at 1 mL per litre. Useful mid-season when curative action is needed after the onset of infection.

Resistance Management: Fungicides should be rotated between chemical groups (FRAC codes) to prevent resistance development. Strobilurins (QoI), DMIs (triazoles), and SDHIs should always be alternated with multi-site protectants (mancozeb, chlorothalonil). A recommended sequence is: Mancozeb → Azoxystrobin + Mancozeb → Difenoconazole → Mancozeb, cycling through the season.

Conclusion

Early Blight of Potato, caused by Alternaria solani, is a polycyclic foliar disease with significant economic impact on potato production globally. The fungus belongs to the subdivision Deuteromycota (anamorph) and to the Ascomycota-Dothideomycetes in its teleomorphic (perfect) stage as Lewia infectoria. The disease thrives under warm temperatures (24–30 °C), high humidity, and alternating wet and dry periods, and is most severe on older, senescing plants under nutritional stress. Effective management of Early Blight requires an integrated approach: selection of moderately resistant varieties, use of certified seed, crop rotation, field sanitation, balanced nutrition, biological agents (Trichoderma, Bacillus subtilis, Pseudomonas fluorescens), and timely application of appropriate fungicides with proper resistance management strategies. Understanding the complete disease cycle, from primary inoculum survival in debris and seed tubers through asexual conidial dispersal and multiple secondary infection cycles, is critical for correctly timing both cultural and chemical interventions.