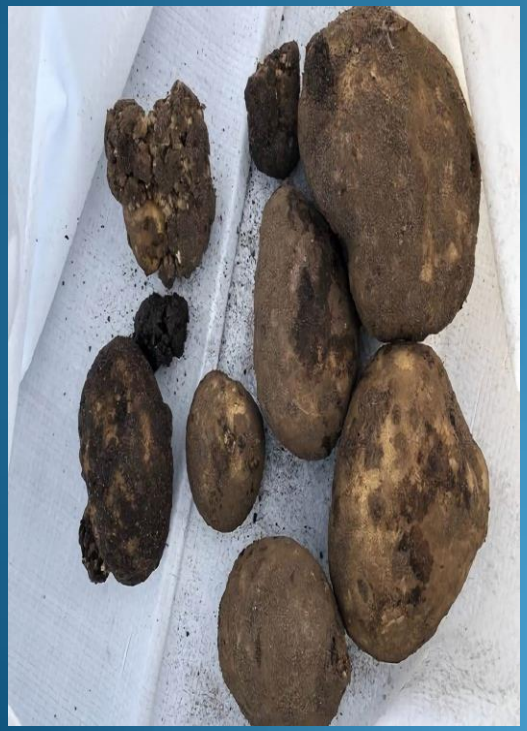
Wart Disease Of Potato... (Black wart)



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PG DEPT. OF BOTANY

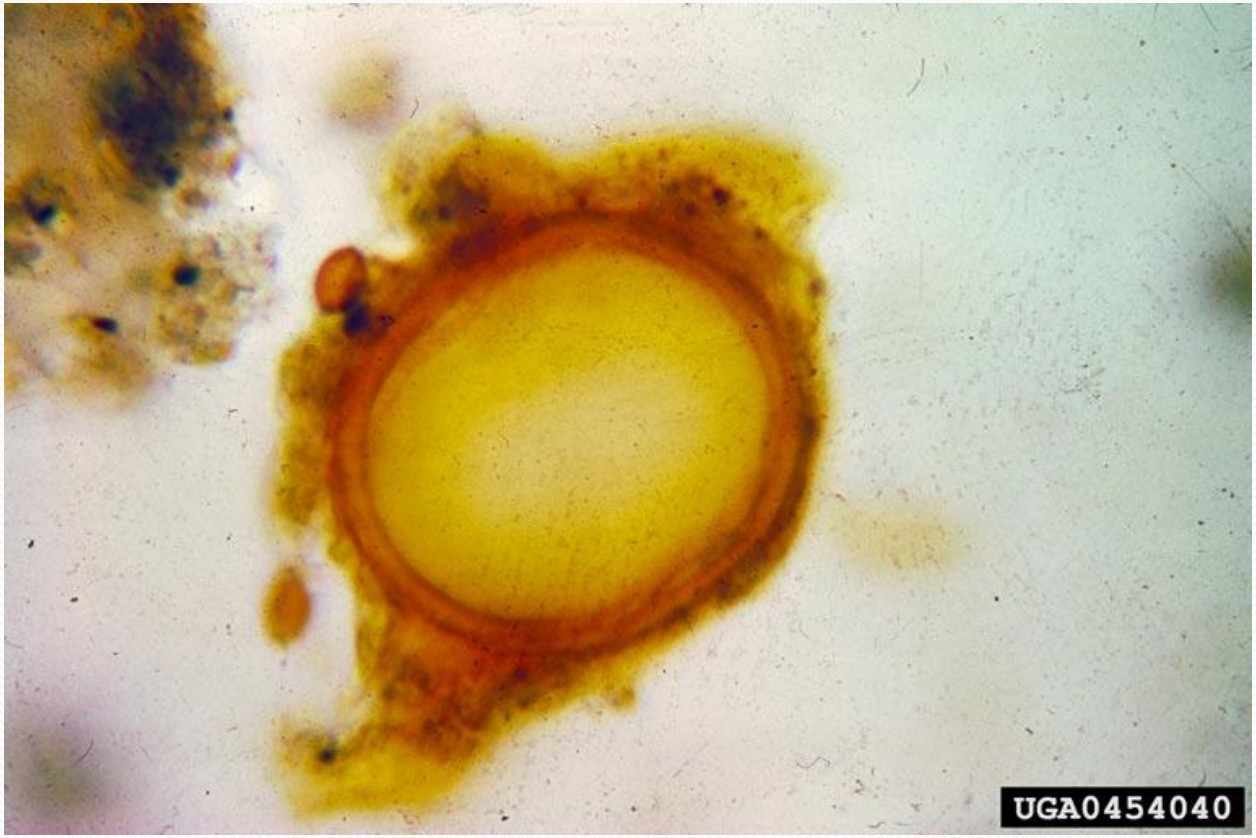
GOVT. COLLEGE FOR GIRLS, LUDHIANA, PUNJAB

What is wart disease of potato?

- Potato wart is a serious disease,caused by a soil borne fungus ***Synchytrium endobioticum***.
- It is an obligate parasite which does not produce mycelium but an abundance of dissemination sporangia which are responsible for tumour formation on the underground potato organs.

Causative agent-

- ***Synchytrium endobioticum***



Host-potato

- **Distribution-**
- This disease was first introduced to europe and was then spread nearly to all potato growing countries in europe
- After the statutory measures were taken its spread was restricted only to the local countries of europe including UK.

SYMPTOMS-

- The diagnostic symptoms of potato wart are galls poduced on several plant parts.
- Galls are primarily parenchymatous and may form on stem tissues including stem base, stolon ,buds and tuber eyes.
- They are formed on underground portions of the plant.
- Vary in size from small protuberancesto large lumps of galls.
- These are greenish white in colour when exposed to light and then turn black when exposed to air and release a black spore powder.

- The wart tumours are never formed on the roots.
- These are produced due to the hypertrophy and hyperlapsia of infected cells.

Transmission of a disease-

- When the warts/galls rot or disintegerate , they release a thick wall resting spores of fungus into the soil.
- These spores can survive for very long period of time and remain viable for atleast 30 years.
- The main means of spread is in infected seed potatoes.
- Disease is favoured by cool and wet soil.
- When the crop is lifted specially after the dry season many of the warts/galls may be extremely small and can be overlooked.

Causal organism-

- It is caused by non-myceial,unicellular holocarpic chytrid fungus ***Synchytrium endobioticum.***
- It is a biotrophic parasite.
- Warts contain host cell that is stimulated to divide by presence of fungus.
- Host cell contain resting sporangia.
- These are spherical cells of thick dark brown walls with folded plate like extensions and are released by decay of warts and remain viable for long periods(40 years).
- Thallus is holocarpic and spherical sac surrounded by a wall.

Disease cycle-

- Infected tissue release the zoospores that are naked and uniflagellated.
- They swim in film of water for 2 to 3 hours and then comes to rest and withdraw their flagella.
- Then it peneterates in the epidermal cell and contents of zoospore cyst are tranferred to host cell through minute pore.
- Fungal thallus inside the host enlarges.
- Host cell is also stimulated to enlarge.
- This leads to the formation of rosette of hypertrophied cells surrounding the central infected cell.

- Their walls get thickened and assume a dark brown colour.
- Infected cell dies and parasite passes to bottom of the host cell and enlarge and become spherical and fills the lower portion of the host cell.
- This leads to formation of double layered chitinous wall of golden brown colour around the thallus (PROSORUS).
- Till this stage the thallus is UNINUCLEATE.
- Inner wall of prosorus protrudes out through pore in outer wall.
- Then it expands as a vesicle and enlarge upward filling the upper half of the host cell.

- Then the contents and nucleus of prosorus are transferred to the vesicle.
- Nucleus divides mitotically
- Thus the vesicle now contains 32 free nuclei.
- Cleavage of cytoplasmic content into number of sporangia takes place.
- Sorus of 4 to 9 sporangia develops.
- Then the nuclear divisions in sporangia occurs and number of nuclei increases.
- Then each nucleus with its surrounding cytoplasm becomes differentiated to form a zoospore.
- Sporangia gets mature,absorb water and it gets swell
- Host cell gets burst and is opened.

- Zoospores are released in sporangial wall(500 to 600).
- Swim actively in water film.
- Comes in contact with host tissue.
- Encyst on epidermis and peneterate it.
- Enlarge within host thallus and forms prosorus again and surrounds host cell enlarges forming the rosette.
- Therefore several generations of prosorus are produced in this way throughout the growing season of the host.
- At end of growing season resting sporangia are formed as a result of sexual reproduction
- Zoospores released from soral sporangia fuse in pairs and forms the zygote.

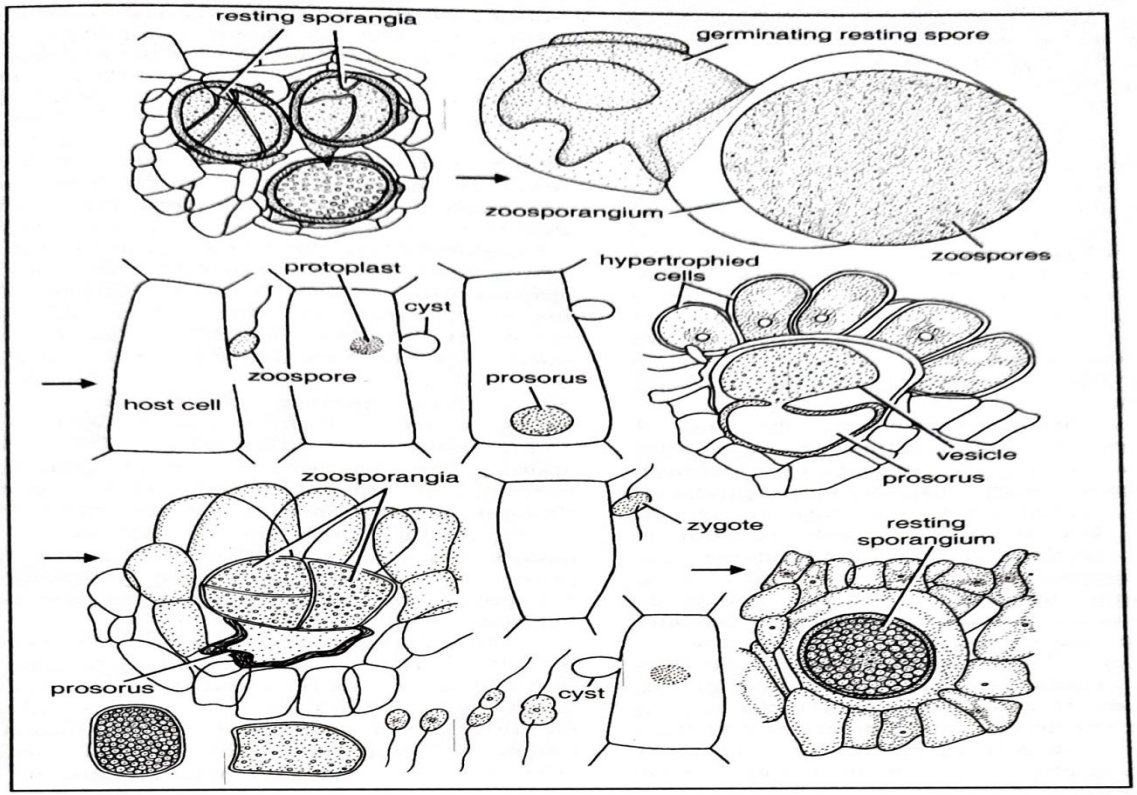


Fig. 13.2. Successive stages in the disease cycle of wart of potato.

During further development, the inner wall of the prosorus protrudes out through a pore in the outer wall and then expands as a vesicle which enlarges upwards and fills the upper half of the host cell. The contents of the prosorus including the nucleus are transferred to the vesicle. During its passage into the vesicle the nucleus generally divides mitotically and the vesicle contains about 32 nuclei. Cytoplasmic contents of the vesicle become cleaved into a number of sporangia. Thus a sorus of 4-9 sporangia develops. Further nuclear divisions in each sporangium occur and number

Fertilization-

- Zoospore that functions as a gamete are similar in size and shape(copulation is isogamous).
- Zoospores from single sporangium may not fuse.
- Male zoospores swim actively and female ones secreate hormone(sirenin).
- It chemotatically attracts male gametes towards itself.
- After swimming for sometime with its flagella zygote encyst on surface of host epidermis.
- Then it peneterates into the host cell.
- Karyogamy occurs before penetration and this leads to an infection.

Infection-

- It is of two types-
- HYPERTROPHIED-

Occurs due to the zoospore.

In this there is increase in the cell volume.

In this the host also enlarges to form a rosette.

HYPERLAPSIA-

Occurs due to the zygote.

In this there occurs a repeated cell division.

Due to this the fungal protoplast is seen burried several cell layers deep beneath the epidermis.

- Zygote enlarges in size.
- Secreates two layered wall around itself
- Outer and inner
- Outer wall is thick and is dark brown and is thrown into the ridges or folds.
- Inner wall is thin and hyaline and it surrounds the granular cytoplasm.
- Host cell dies and its content get deposited on outer layer of the parasite.
- This thick walled structure is uninucleate and is called as the resting sporangium.
- Then the host tissue decays and the resting sporangia is released in the soil.

- Before germination-
- The nucleus divides repeatedly
- Forms many daughter nuclei
- Outer wall epispore gets burst and opens by an irregular aperture.
- The inner wall endospore balloons out and forms avesicle within which a single sporangium differentiates.

The wart is soft and pulpy and thus invaded by bacteria which cause rot. The warty tubers become useless and a source of infection.

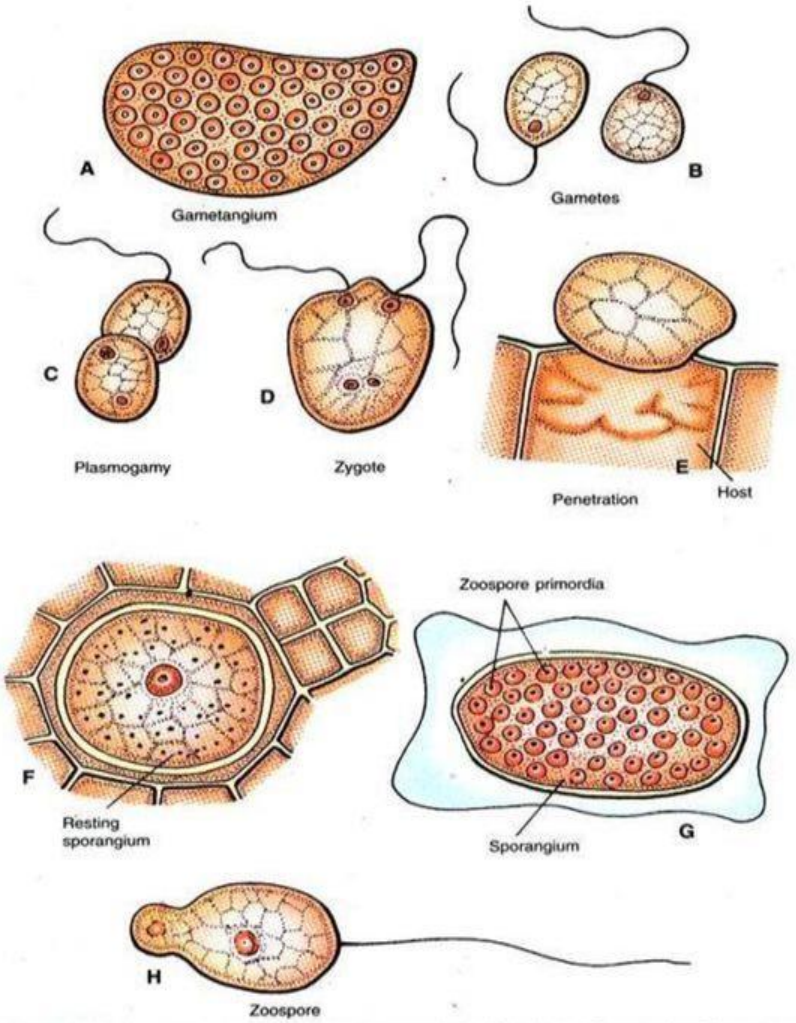


Fig. 4.5 (A-H). *Synchytrium endobioticum*. Sexual phase of the life cycle. A, Gametangium; B, two gametes released from the gametangium; C, Fusing gametes; D, Zygote; E, Zygote penetrating the host epidermal cell; F, Resting sporangium; G, Germinating resting sporangium with zoospore primordia; H, Released Zoospore. (After Curtis)

Germination-

- Resting sporangium function as PROSPORANGIUM.
- Zygote and young resting sporangia are diploid and meiosis occurs prior to the zoospore formation.
- Thus zoospores , prosori and soral zoospores are haploid.
- Thus the pathogen survives as resting spores freely in the soil.

Dispersal-

- Occurs through the movement of the diseased tubers and infested soil through the agency of the man.(feet and shoes , tools , vehicles moving through the infested areas.
- The pathogen may also attack tomato and the other solanaceous plants(datura and physalis.)

Disease control-

- Field sanitation by eradicating pathogen bearing plant debris from the fields and eradication of other solanaceous host helps in reducing the disease incidence.
- Soil treatment with steam and chemicals like mercuric chloride , ammonium sulphocyanate,copper sulphate,formalin etc.eradicate the resting spores.
- These methods are very expensive.
- The most effective and practical method to eradicate the disease is to cultivate immune or high resistant varieties.

- For example-in Darjeeling areas a resistant cultivar of potato ,Kufri jyoti was introduced in 1975.
- Later on many wart immune cultivars were developed and released for cultivation.
- For example-kufri muthu

  kufri sheetman

  kufri sherpa

  kufri khasi garo

  kufri bahar

  kufri kumar

  kufri kanchan

  kufri swarna

These varieties were found resistant to the disease.

THANK YOU