Synchytrium endobioticum:

Synchytrium endobioticum causes the black wart or wart disease of potato (Solanum tuberosum). According to Karling (1964), it has a broad spectrum of Solanaceous sp. in its host range. The potato wart disease is widely distributed in the potato growing regions of the world.

It is prevalent in areas with a cool moist climate. In India, it has been reported from the Darjeeling district (West Bengal) and areas of Nepal contiguous to the former. The fungal parasite cannot survive in hot places.

Black wart of Potato.

Symptoms of Synchytrium Endobioticum:

Usually the disease affects the underground parts of the host. Diseased potato tubers appear as dark brown or black cauliflower like outgrowths. Galls or tumours may be formed on aerial parts as well.

On shoots, the disease appears in the form of outgrowths on green twisted leafy structures. Due to the presence of the fungus, the host cells are stimulated to divide leading to increase in the number of host cells. Most of the host cells contain resting sporangia (Fig. 4.3).

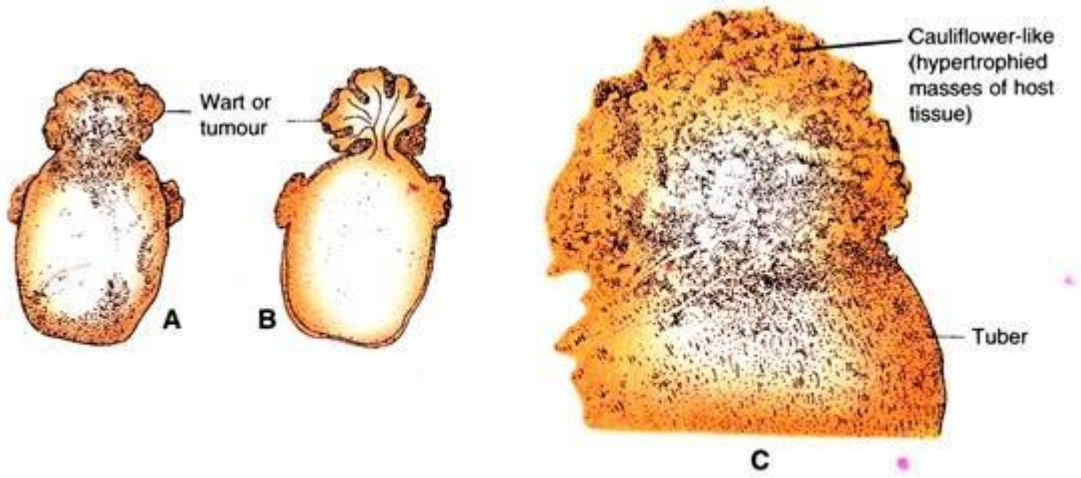

Fig.4.3 (A-C). A-B, Potato tubers infected by *S. endobioticum* showing black warts; C, portion of A slightly enlarged.

Thallus Structure of Synchytrium Endobioticum:

Synchytrium endobioticum is a holocarpic endoparasite. It is a holocarpic fungus because the thallus is naked and unicellular. The thallus forms a mass of naked, uninucleate amoeboid mass of protoplasm. Soon, a double layered chitinous wall develops around the thallus. The fungus is an endoparasite because it is endobiotic and occurs as a parasite in the epidermal cells of the host.

Life Cycle of Synchytrium Endobioticum:

The life cycle of S.endobioticum has been studied by Curtis (1921) and Kohler (1923, 1931).

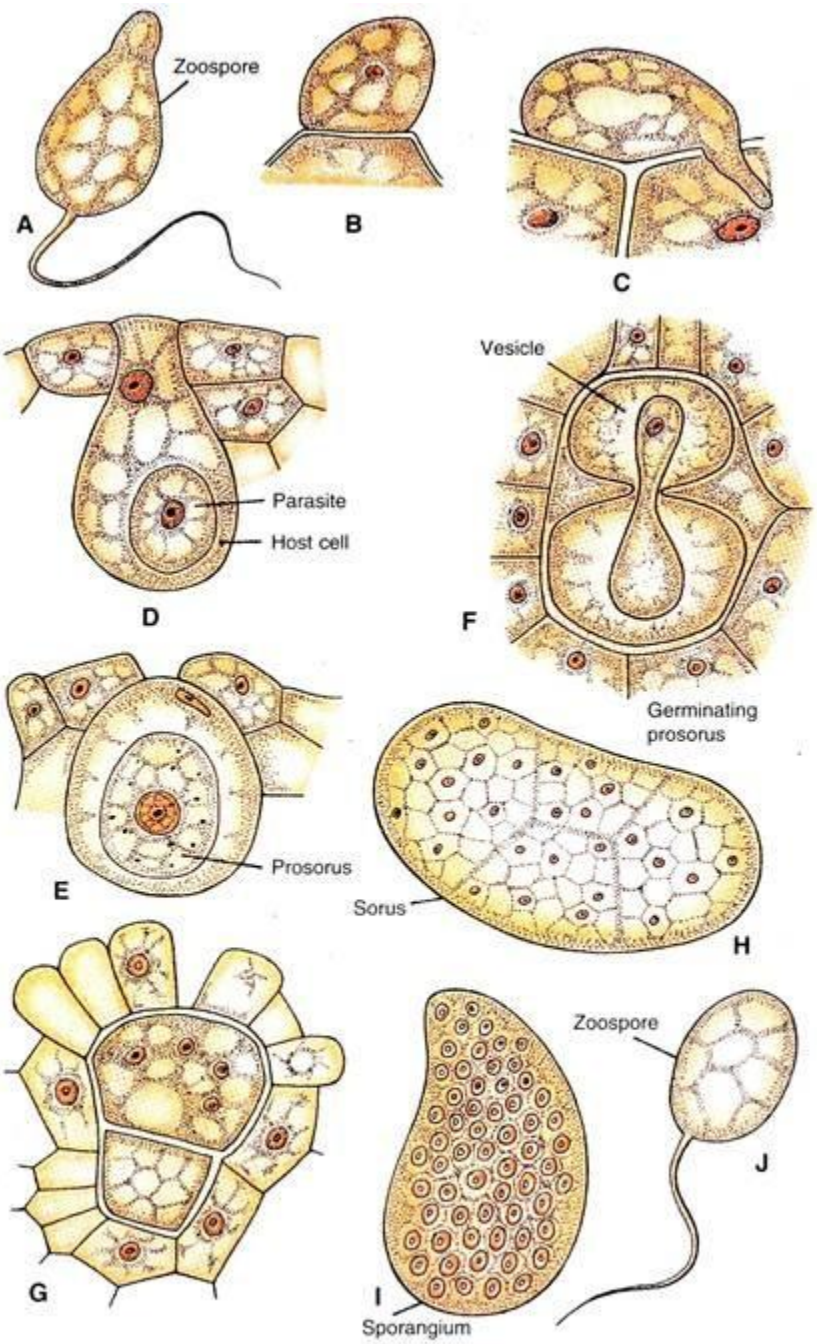

Fig. 4.4 (A-J). *Synchytrium endobioticum*. Asexual phase in the life cycle. A, Zoospore from a resting sporangium; B-C, Host penetration; D, Parasite in the host epidermal cell; E, Prosorus; F, Prosorus migrating into the vesicle in the upper part of the host cell; G, Later stage of F; H, Sorus with four multinucleate young sporangia; I, sporangium (summer); J, Released zoospore. (After Curtis)

Reproduction in Synchytrium Endobioticum:

Asexual Phase:
The asexual phase of the life cycle of this parasitic fungus starts with the infection of the host by the parasite.

(i) Infection (Fig. 4.4):
The causal organism (S. endobioticum) is present in the wart tissue of potato tuber or in soil in the form of resting sporangia. Some mycologists call them resting or winter spores.

They remain viable for a long period. Under suitable conditions (presence of potato seedlings in the field, moist soil and suitable temperature), the resting sporangium or spore germinates releasing posteriorly uniflagellate, naked haloid zoospores which are liberated by the rupture of the enclosing membrane.

The liberated zoospore (A) swims in a film of water in the contaminated soil. It may finally reach a potato plant and come to rest on the host surface (tuber or stolon). The quiescent zoospore (B) then retracts its flagellum and germinates by putting out a small, thin, naked, peg-like germ tube called the infection peg or penetration tube (C).

The latter pierces the cuticle and the wall of the epidermal cell of the tuber or bud tissue in the stolons. Subsequently the uninucleate protoplast of the zoospore enters the host as a naked mass. The old view that the zoospore dissolves a minute pore in the epidermal wall through which it penetrates is now discarded.

(ii) Prosorus Stage:
Once within the epidermal cell of the host it promptly sinks to its lower part (D) where it develops into a uninucleate intracellular thallus with a comparatively large nucleus (D) at the expense of the food material absorbed from the host cell. The unicellular parasite rounds off and secretes a thin wall around it. It increases in size as the infected host cell is induced to enlarge and become pear-shaped.

Reaching a certain size, the unicellular pathogen thallus secretes a wall around it which is differentiated into an outer, thick, golden yellow exospore and an inner, thin, hyaline endospore (E). The nucleus also increases in size. The mature thallus with a heavy golden wall around it is called the prosorus (pi. prosori).

Meanwhile the adjacent epidermal cells and the surrounding cortical cells become stimulated to activity. They divide rapidly and repeatedly to form a minute gall or tumor or a wart-like tissue. The infected hypertrophied cell containing the prosorus in its lower part by now is dead. It is in the centre of a rosette of more or less hardened epidermal cells.

(iii) Germination of Prosorus:
The mature prosorus germinates within the dead host cell. A pore is formed in the thick exospore layer. The thin, hyaline endospore layer extrudes throuth the pore in the form of vesicle (F). The contents, of the prosorus migrate into the vesicle which extends into the upper half of the host cell. During migration, prosorus nucleus divides (G) repeatedly to form about 32 daughter nuclei.

The multinucleate protoplast of the vesicle is now partitioned by newly formed thin, hyaline walls into four to nine multinucleate polygonal compartments (H) or segments. Each segment has a wall of its own and functions as a zoosporangium. The whole mass of 4 to 9 sporangia compose a sorus.

(iv) Sporangia:
The nuclei in each sporangium undergo further division to form 200 to 300 daughter nuclei. The multinucleate protoplast is finally organised into uninucleate daughter protoplasts by aggregation and rounding off of the cytoplasm round the nuclei (H).

Each daughter protoplast becomes metamorphosed into a posteriorly uniflagellate zoospore. In this way about 1500 zoospores are produced from the single, original zoospore which initiated the infection of the host.

The mature sporangia in the sorus absorb water and swell. The pressure thus set up from within ruptures the host cell wall and vesicle membrane. The zoosporangia are pushed out of the sorus on to the surface of the host (potato tuber).

The zoospores escape through an opening (a slit) in the sporangial wall, or upon the rupture of hyaline projections in the sporangial wall which are termed papillae. The released zoospores (J) swim in a film of water in the soil.

Some of these may reinfect the host and repeat the sequence of events outlined above. This completes the asexual phase in the life cycle of Synchytrium. From the account of

asexual phase of the life cycle of the parasite given above it becomes evident that the parasite multiplies during the growing crop season releasing several successive generations of zoospores.

The latter cause repeated infections to the standing crop thus spreading the disease in an epidemic form.

Sexual Phase (Fig. 4.5):

Gametangia:
Under conditions of scarcity of water (dry weather), which means the end of growing season, the segments of the prosorus function as gametangia (A) which are in no way different from sporangia. The gametangia produce planogametes (B) similar to zoospores in every respect except size. The gametes are smaller than the zoospores and fuse in pairs (C).

Fusion occurs after liberation in a film of water on the surface of the host or in the soil, between planogametes from different gametangia in the same sorus. Plasmogamy (fusion of the gametes) is followed by karyogamy which probably takes place at the time of host penetration.

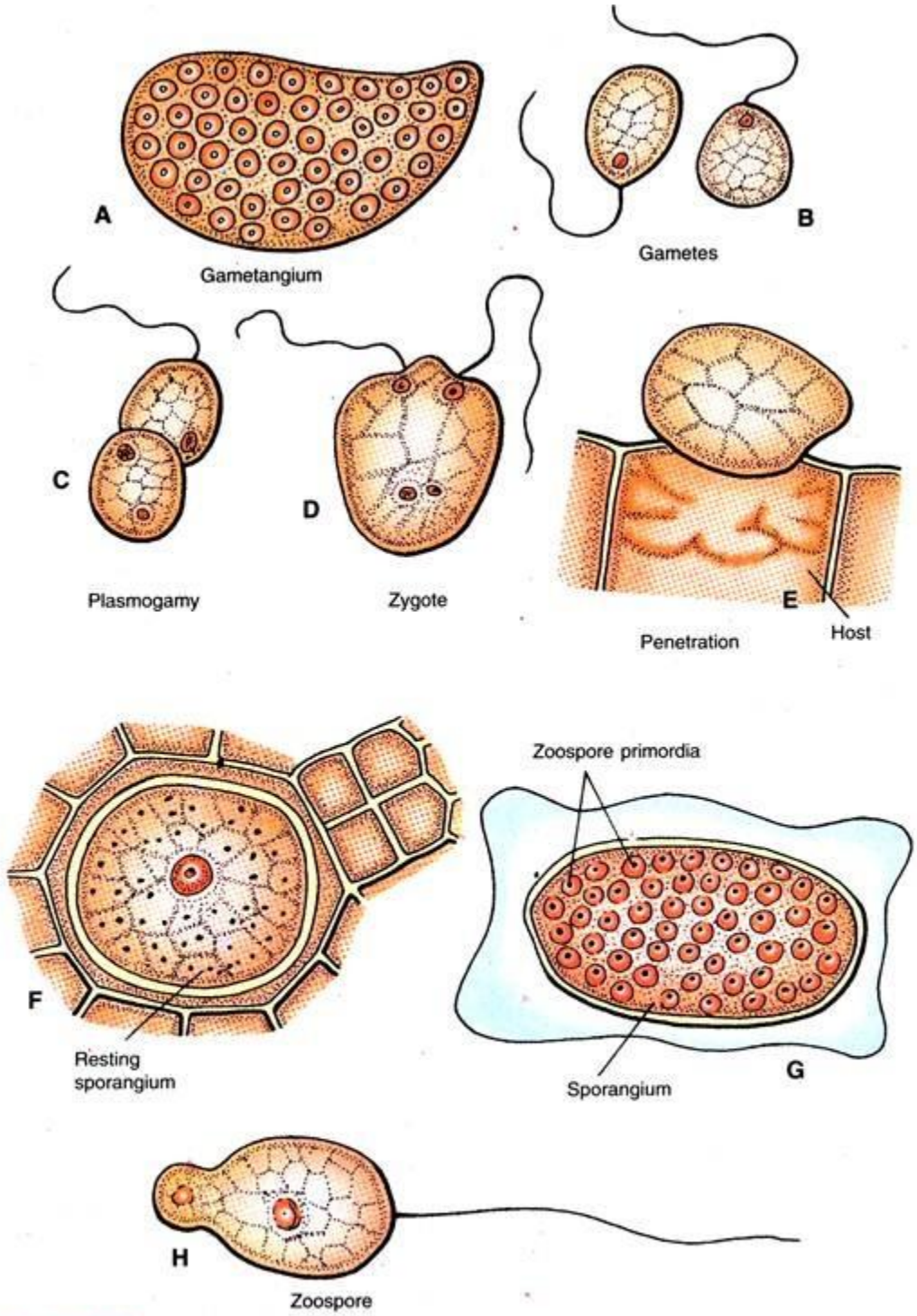

Fig. 4.5 (A-H). *Synchytrium endobioticum*. Sexual phase of the life cycle. A, Gametangium; B, two gametes released from the gametangium; C, Fusing gametes; D, Zygote; E, Zygote penetrating the host epidermal cell; F, Resting sporangium; G, Germinating resting sporangium with zoospore primordia; H, Released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

Zygote (Fig. 4.5 D):
The diploid zygote formed by the fusion of the planogametes is biflagellate. It swims about in a film of water in the soil. Finally it comes to rest on the surface of the host (tubers) and penetrates the epidermal cell (E) in much the same way as the haploid zoospore.

The flagella are retracted and absorbed before entry into the host. Within the host the zygote sinks to the bottom of the infected hypertrophying epidermal cell of the host and grows in size.

Resting Sporangium (Fig. 4.5 F):
The presence of the parasite (zygote) in the host causes hypertrophy and hyperplasia of the surrounding cells. They are stimulated to divide repeatedly. Consequently the infected cell is soon buried deep within the host tissue. The diploid zygote (parasite) enlarges and is enclosed in a thick, reticulately ornamented, two-layered wall to become a resting sporangium. Some authors call it a resting or winter spore.

The resting spores are released into the soil by the decay of the infected tubers. In the soil they remain dormant through winter. It has, however, been reported that resting spores can remain viable in the warty tubers in soil for five years and air dry soil for fifteen months.

Sharma and Commack (1976) reported that the resting spores of S. endobioticum may remain dormant in the soil for 25-30 years. Several investigators such as Curtis (1921), Glynne (1926), Karling (1967) and Sharma and Commack (1976) reported that the resting spores of S. endobioticum function as sporangia giving rise directly to zoospores.

Germination of resting Sporangium (Fig. 4.5 G):
At the onset of conditions favourable for growth (following spring when the host is available), the resting sporangium germinates. A number of granule-like structures make their appearance in the cytoplasm.

These are zoospore primordia. Further, each zoospore primordium extrudes chromatin. This is suggestive of meiosis. The multinucleate protoplast undergoes cleavage to form numerous uninucleate, dauthter protoplasts.

Each daughter protoplast gets metamorphosed into a posteriorly uniflagellate zoospore. The outer layer of the sporangial wall ruptures. The zoospores (H) escape throuth the exit tube. They are larger in size than the zoospores released from the sporangium of the asexual cycle. They, however, function in the same way and may infect the host to repeat the asexual phase.

The effect of zygote infection on tuber is more serious and destructive as compared with zoospore infection. The zygote infection induces excessive rate of division (hyperplasia) not only of the adjacent host cells but also of the cells some distance away from the site of infection.

Hyperplasia is followed by excessive enlargement of the resultant cells (hypertrophy). Hypertrophy and hyperplasia of the surface cell layers of the infected potato tuber and the resultant abnormal growth activity leads to the formation of large, unsightly and useless masses of crinkled black warty tissue (4.3) known as the wart or tumour.

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

Control:
To reduce spread of the pathogen, quarantine regulations are practised. In India, the entry of potatoes from Darjeeling Hills, where wart disease is known to occur, has been prohibited.

A number of varieties which are immune to the disease have been produced in recent years. Their use has considerably prevented spread of disease. The application of lime and use of fungicides in the soil before planting have given some useful results.

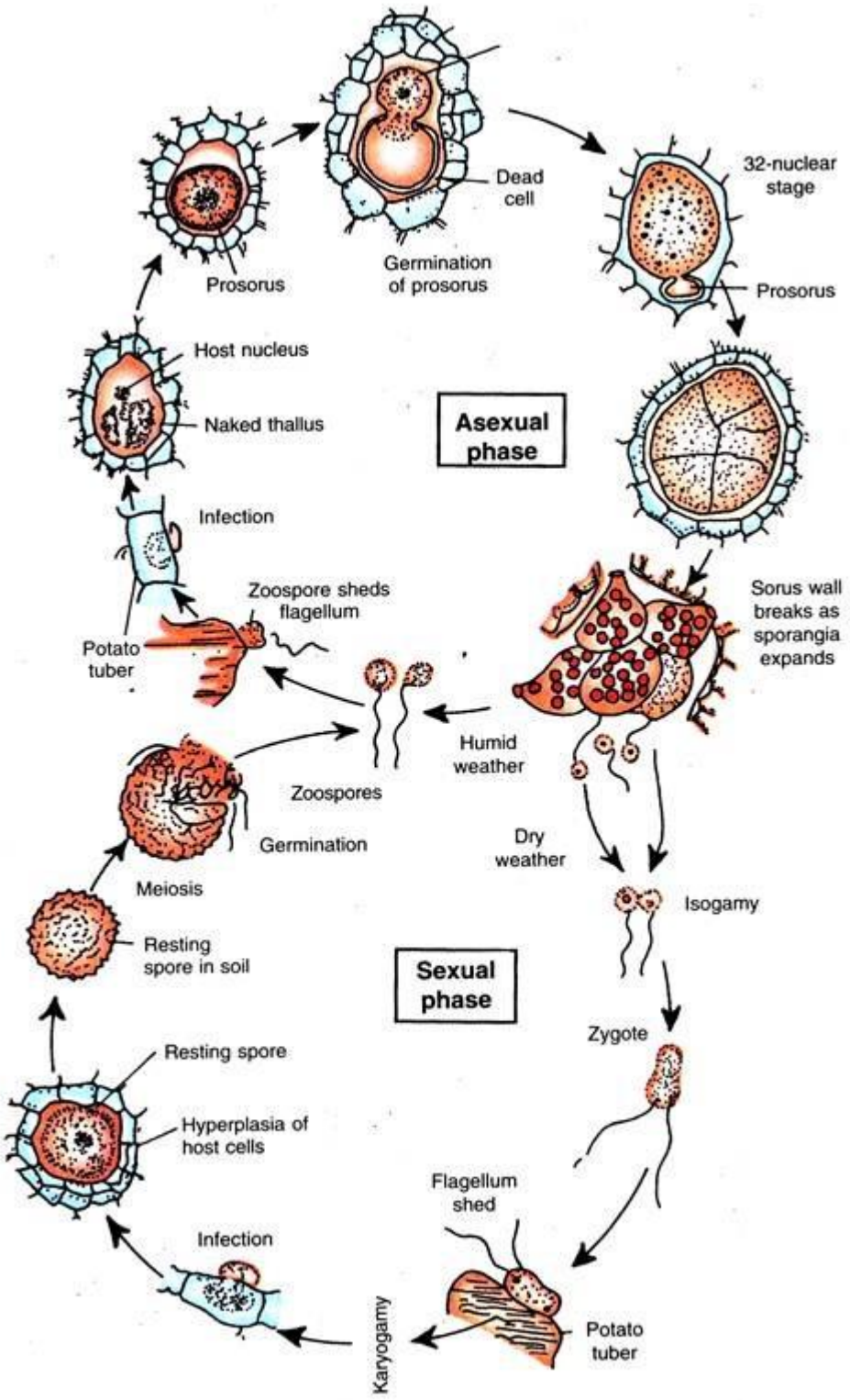

Fig. 4.7. Pictorial life-cycle of *Synchytrium endobioticum*.