The Disease Cycle and Fungus-Host Relationships in Dallisgrass Ergot

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ABSTRACT

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Germ tubes from conidia of *Claviceps paspali* penetrate between cells of stigmas or styles of *Paspalum dilatatum* within 4 hr and invade the ovary through the style. In 2-3 days, intercellular hyphae permeate the ovary and break out between the epidermal cells. At 4 days, a thick plectenchymatous stroma envelops the disintegrating ovary. Conidia produced on the surface of the stroma ooze from infected florets in drops of honeydew by the 5th or 6th day. The extramatrical stroma develops into the mature, globose, white-to-brown ergot sclerotium in 2-3 wk. The ovary serves primarily as a portal of entry for the pathogen and is destroyed soon after infection. The fungus establishes a continuing compatible relationship with living host cells in a narrow interface between fungus stroma and noninvaded host tissue in the receptacle of the floret. Primary disease cycles are initiated by airborne ascospores forcibly discharged from perithecia produced on clavae arising from overwintered sclerotia. At least 6 wk of exposure to 5 C is required to break dormancy of sclerotia. Inoculations with conidia are most effective at anthesis. A 2-hr dew period is sufficient for infection after floret closure.

Additional key words: pathological histology, Ascomycetes, Clavicipitaceae.

Ergot of dallisgrass (*Paspalum dilatatum* Poir.) caused by *Claviceps paspali* F. L. Stevens & J. G. Hall results in losses in seed production but is more important as a source of alkaloids inducing toxicoses in grazing cattle. In the southeastern United States (2) the ergot sclerotia develop along with the seed in the succession of inflorescences produced by the perennial clumps of dallisgrass during the summer and early fall. The overwintered sclerotia germinate during the following season to form clavae with spherical heads containing perithecia. Ascospores from these perithecia furnish the primary inoculum. Repeated secondary cycles of infection occur throughout the growing season from conidia produced in drops of honeydew on infected florets and dispersed by insects or rain.

Although the disease cycle in the dallisgrass ergot is similar to that in the rye ergot caused by *C. purpurea,* morphological differences in the sclerotia of *C. paspali* and *C. purpurea* are obvious. Inconsistencies in reports on infection and on development of sclerotia in rye ergot (3, 4, 5, 6, 7, 11, 12) raise further questions in extrapolating from *C. purpurea* to *C. paspali.* Consequently, a study was made of the disease cycle in dallisgrass ergot with emphasis on host-parasite relationships. This study extends the report of Brown (2) on the life history of *C. paspali.*

MATERIALS AND METHODS

Germination of sclerotia.—Infected inflorescences collected in the field in September were placed in aluminum screen wire trays just above the ground and left outdoors over winter. Sclerotia picked from the trays in June were scattered over moist sand in culture dishes and covered with a 4-mm-thick layer of sand for germination. To determine effect of temperature on germination, sclerotia picked from inflorescences in September were sorted into lots of 50 each in wire packets, buried in moist sand, and stored at 5 C on October 1. Each week thereafter sclerotia from one packet were placed on layers of moist seed germination paper in 15-cm petri dishes and kept in the laboratory at temperatures around 20 C.

Inoculation with ascospores.—Germinated sclerotia with clavae discharging ascospores were distributed over wet filter paper in the bottom of a 2-liter beaker. The beaker was inverted above dallisgrass inflorescences taped over a disk of wet filter paper on a glass plate. After 1 hr the inflorescence was removed from the beaker, and the plant was placed in a dew chamber for 24 hr. Slides that had been exposed alongside the inflorescences at inoculation were transferred to the dew chamber with the plant.

Inoculations with conidia.—Dallisgrass transplants grown outdoors in 15-cm pots were inoculated with conidia in August and September. Pots with inflorescences on the verge of blooming were brought into the laboratory in the afternoon and placed in an east window. Florets that opened the following morning were inoculated at anthesis or at various times after anthesis. The positions of target florets were recorded at anthesis by serial numbers obtained by counting florets in each row on the individual racemes from base to apex. A suspension of conidia from plants naturally infected in the field was misted onto the inflorescences with a chromatography sprayer, and inoculated plants were placed in a dew chamber at 25 C for various periods. The plants were held overnight in the laboratory and placed outdoors the

following day. Appearance of droplets of honeydew at the tips of inoculated florets was used to confirm infection.

Histological techniques.—Material for microscopic study was taken from specific florets inoculated at anthesis and held in the dew chamber for 4 hr. Drawings (Fig. 1-6) were made with a camera lucida from squashes or free hand sections of living ovaries mounted in water.

Paraffin sections (Fig. 9-12) were made of entire florets fixed in formalin-propionic acid-ethanol, dehydrated in tertiary butyl alcohol, embedded in "Paraplast", and sectioned at 8 μm. Because of difficulty in cutting the indurated lemma and palea of the floret, sectioning was done at 5 C. Blocks were oriented to give longitudinal sections extending from the center of the flat palea through the ovary at the point of attachment of the ovule on the adaxial side to the center of the concave lemma on the abaxial side. Sections were mordanted in 4% iron alum 2 hr, washed in distilled water 30 min, stained in 0.5% hematoxylin 2 hr, and differentiated in a saturated aqueous solution of picric acid 15-20 min.

For electron microscopy (Fig. 7-8) ovaries were dissected in a drop of 3% gluteraldehyde on a sheet of dental wax, sliced, fixed in 3% gluteraldehyde in 0.025 M phosphate buffer 2 hr, post fixed in 2% OsO_4 2 hr, left overnight in 1.5% uranyl acetate in 30% ethanol, dehydrated in ethanol, and embedded in Spurr's medium. Sections were stained on the grids with lead citrate for 2 min.

RESULTS

Host morphology. — Potted plants grown outdoors were comparable to those growing normally in the field. Each of the succession of culms produced near its tip three to five horizontally diverging racemes. The florets were arranged in four imbricated rows on the lower surface of the flat rachis of each raceme. Blooming occurred progressively on successive days from the terminal to the lower racemes and progressively within racemes, beginning with the middle or upper florets in the two outer rows. Anthesis occurred between 800 and 1200 hours. Most florets opened about 900 hours and closed by 1030 hours.

The ovary developed in a relatively large cavity enclosed by a concave cartilaginous lemma and a flat cartilaginous palea that fitted into the incurved lip around the margins of the lemma. At anthesis lodicules on either side of the ovary forced the lemma and palea open. Two purple stigmas were extruded, followed by stamens with purple anthers. After the anthers dehisced, the lemma and palea closed, leaving the stigmas and withered stamens protruding from the tightly sealed margins. The grain remained enclosed by the lemma and palea at maturity. Its flat, adaxial face was appressed to the palea; its rounded abaxial surface followed the contour of the lemma.

Primary disease cycle. — *Overwintering and germination of sclerotia.* — When sclerotia overwintered outdoors were buried in moist sand, clavae from germinating sclerotia emerged in 1-2 wk. Some produced ascospores in 4 wk. At 4 wk, 90% of the sclerotia showed some signs of germination. Each sclerotium formed one to four yellow clavae with stalks 6-37 mm long and spherical heads 0.75-1.25 mm in diameter. The surface of the head was papillate with the protruding beaks of the immersed perithecia. Sclerotia left in the trays outdoors produced the first mature clavae in mid-July, which was soon after dallisgrass began to head in the field.

No germination occurred in sclerotia stored indoors at 5 C for less than 6 wk. Germination in lots exposed to 5 C for 6-12 wk and examined at weekly intervals was 8, 12, 36, 26, 84, 50, and 76%, respectively. Germination among sclerotia in the same lot was erratic. Some produced mature clavae even before others began to germinate.

Ascospore discharge. — Heads of mature clavae placed on moist filter paper squares in lids of petri dishes discharged ascospores that were dispersed singly on the agar below. Germ tubes arose from any cell of the filiform ascospore. In 2-3 days conidia developed at the tips of erect phialides scattered over the thin mycelium. Within 1 wk fluffy white colonies developed, and no conidia were produced thereafter.

Ascospore discharge was observed directly by placing opened dishes of sand containing sclerotia with mature clavae under a dissecting microscope and focusing on the beaks of the perithecia. Ascus tips emerged through the ostiole and, in a quick burst, successively discharged the eight ascospores through the apical pore. As the heads dried, ascospore discharge slowed until the spores oozed from the ascus, each succeeding spore overlapping the tail of the one that preceded it through the pore. Ascospores extruded under drying conditions collected around the perithecial beaks on the surface of the head.

Infection by ascospores. — In three inflorescences exposed to airborne ascospores, percentages of infection after 7 days were 4, 3, and 0. No record was made of the proportions of florets that were in a susceptible stage at the time of inoculation. Slides exposed to the ascospore shower with the inflorescences showed ascospores with germ tubes 4-33 μm long when they were removed from the dew chamber 24 hr after inoculation.

Secondary disease cycles. — *Sequence of events.* — Evidence of conidial infection was noted 3 days after inoculation as white mats of mycelium on the surface of the ovary, but this could be found only by dissecting the florets. Droplets of honeydew oozed from the tips of infected florets 4-7 days after inoculation. Most florets showed this external sign of infection on the 5th or 6th day. Production of conidia continued at a maximum rate for about 5 days, declining with the emergence of the sclerotia 9-11 days after inoculation. Production of conidia was extended in florets that did not form sclerotia. Of 193 infected florets whose development was followed, only 64% produced sclerotia. The globose, white-to-brownish sclerotia appeared mature in about 2 wk, whereas normal grain required about 3 wk to reach maturity. The largest sclerotia (4 mm diameter) remained seated in the floret; small sclerotia (1 mm diameter) were largely enclosed within the lemma and palea. Dehiscence occurred, as in normal grain, by detachment of the entire floret.

Effect of moisture on infection. — In early inoculations in which percentages of infection were obtained by dividing number of florets infected by total number of florets in the raceme, incidence of infection was 3-31% with a mean of 12%. Varying periods in the dew chamber after inoculation from 4-24 hr had little effect on infection.

When florets reaching anthesis on the day of inoculation were used as a base for determining percentages of infection, higher percentages were obtained. Mean percentages of infection obtained from three to six replicates of inflorescences inoculated when florets were at anthesis and kept under dry conditions in the laboratory or placed in the dew chamber for 1 or 4 hr are in Table 1. Although a short dew period following inoculation doubled the percentage of infection, a good proportion of the florets inoculated at anthesis became infected in the absence of dew. Plants with florets closed for 2-6 hr after anthesis were inoculated and placed in the dew chamber for 2-20 hr. Mean percentages of infection for four to five replications at each dew period (Table 1) showed that no increase in infection resulted from extending the dew period beyond 2 hr.

Effect of age of florets on infection. — Period of susceptibility of florets was determined by inoculations followed by a minimum dew period of 2 hr on plants at varying periods after the florets closed following anthesis. Percentages of infection obtained from four to eight replications of inoculations made 2-24 hr after floret closure are in Table 2. The highest percentage of infection obtained in inoculations following floret closure (57%) was less than two-thirds of that obtained in inoculations made at anthesis (95%). Percentages of infection gradually declined with increasing intervals after anthesis, but some infection occurred when florets were inoculated 24 hr after anthesis.

Repetitive germination of conidia. — Drops of honeydew in the field often were covered with a sparse, white layer of phialides projecting above the surface of the drop and bearing conidia singly at their tips. Conidia were likewise produced on honeydew that dripped over other parts of the inflorescence. Slides touched to drops of honeydew were placed face up and face down on glass rods over moist filter paper in petri dishes at 25 C in continuous light and in continuous darkness for 24 hr. Conidia germinated to form phialides producing a single conidium (Fig. 3). Where drops of water condensed on slides incubated face down, the phialides extended through the surface of the drop and produced conidia on the free tips. When conidia were streaked or sprayed onto plates of V-8 juice and potato-dextrose agar, germ tubes developed within 6 hr. Many of the germ tubes were erect phialidic structures. After 20 hr the phialides were forming conidia abundantly, and a second conidium was developing on some phialide tips. Other germ tubes, often from the same conidia producing phialides, developed into a mycelium. Transfers freed from contaminants produced fluffy white colonies in which no conidia developed.

TABLE 1. Effect of length of dew period on infection by *Claviceps paspali* following inoculations of dallisgrass florets at anthesis and 2 to 6 hr after anthesis

Dew period (hr)	Florets (no.)	Florets infected (%)
At anthesis:		
0	111	40
1	45	76
4	142	95
After anthesis:		
2	163	56
6	134	53
10	134	51
20	209	57

TABLE 2. Infection obtained with *Claviceps paspali* following inoculations of dallisgrass florets at various periods after anthesis. The dew period after inoculation was at least 2 hr

Time after floret closure (hr)	Florets (no.)	Florets infected (%)
2	283	57
4	226	48
6	212	47
9	256	39
12	249	17
24	63	14

Penetration and infection. — Conidial germ tubes penetrated between the cells of the stigma (Fig. 1) and style (Fig. 2) and grew downward inside the style to the ovary. Stages in penetration shown in Fig. 1-2 were reached in the dew chamber 4 hr after inoculation. Germ tubes from conidia on the tips of stigmatic cells grew over the surface until they reached a suitable portal of entry (Fig. 1). Most germ tubes from conidia on styles penetrated immediately (Fig. 2). Some grew extentively over the surface with no evidence of penetration (Fig. 2, upper left). Some that penetrated the style grew upward away from the ovary (Fig. 2, upper right).

Invading hyphae entering the apex of the ovary through the style penetrated immediately to the inner layers of the ovary wall and spread around the ovary in the inner layers of the wall and between the ovary wall and the ovule. From the inner layers, hyphae spread outward between the cells of the ovary wall and, to a lesser extent, inward between the large cells of the nucellus of the ovary (Fig. 4). Two days after inoculation an intercellular mycelium occupied the ovary wall (Fig. 4, 8) and the ovule (Fig. 4).

Development of the sphacelial stage. — Hyphae emerged between the cells of the outer epidermis of the ovary and spread laterally beneath the cuticle (Fig. 7), which was lifted and ruptured (Fig. 4). By the 3rd day hyphae formed a compact, plectenchymatous, extramatrical stroma surrounding the ovary (Fig. 5). Free tips of elongating hyphae covered the surface of the stroma. The intramatrical hyphae were less extensively developed, and the cells of the ovary were intact.

By the 4th day a massive stroma surrounded the ovary (Fig. 9). The stroma was relatively thin and appressed to the palea on the adaxial side. On the abaxial side it expanded into the space enclosed by the concave inner surface of the lemma. Although the outlines of the ovary were still visible, the ovarian cells were largely disintegrated and replaced by a loose fungus plectenchyma. Cells at the apex of the ovary and base of the style remained intact, and some contained nuclei. Dissections in water revealed the ovary as a small, ellipsoidal, brownish, translucent sack embedded in the base of the white stromatic mass. The extramatrical stroma spread upward,

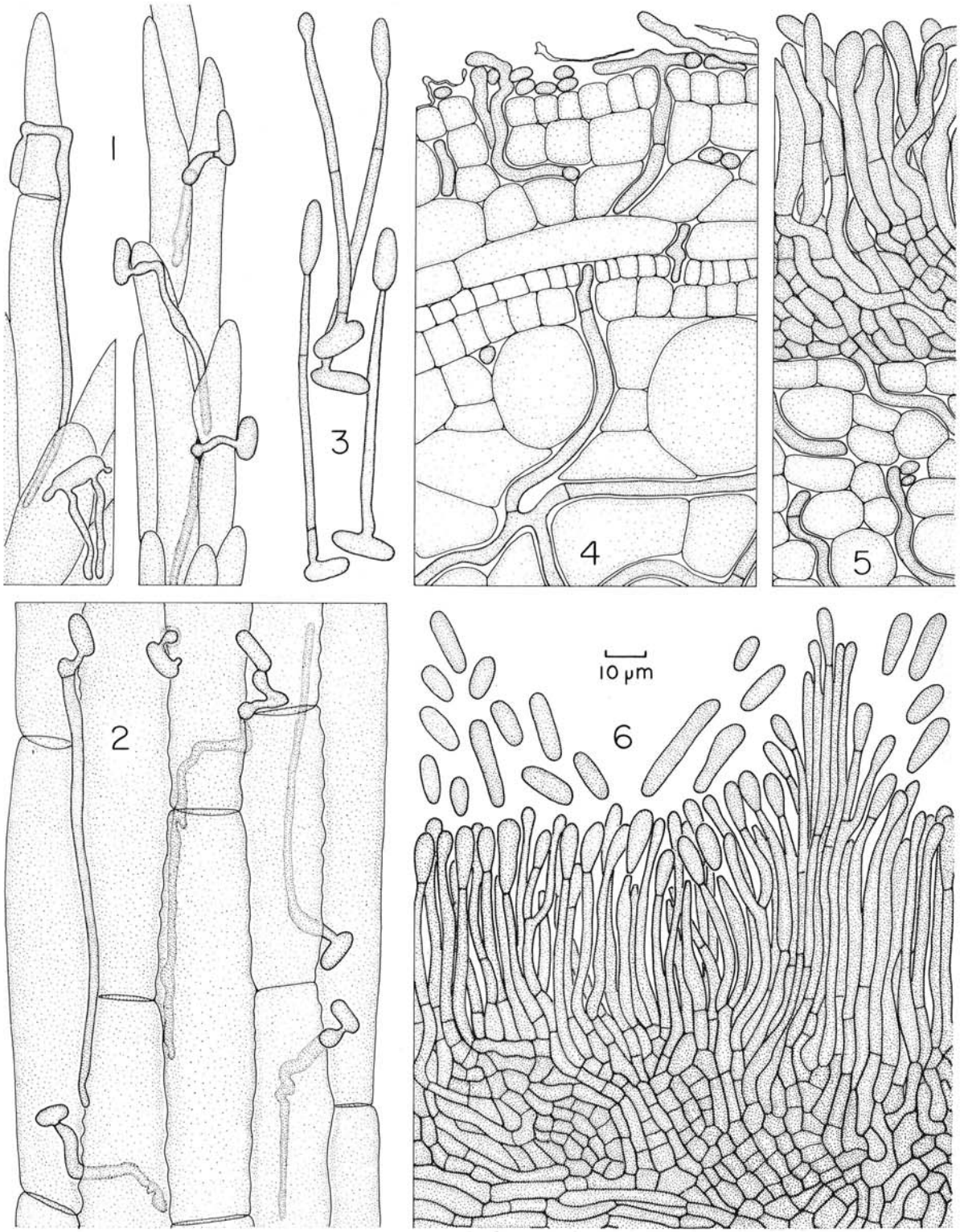

Fig. 1-6. *Claviceps paspali.* Camera lucida drawings of fresh material mounted in water; all conform to scale bar in Fig. 6. **1)** Germ tubes from conidia penetrating between cells of stigma. **2)** Conidia germinating on surface of style; some germ tubes penetrating between cells and growing downward inside of style toward ovary. **3)** Conidia in honeydew germinating by phialides that produce secondary conidia on their free tips. **4)** Cross section of infected ovary 2 days after inoculation; intercellular mycelium occupying large celled nucellus of ovule (lower) and wall of ovary (upper) with hyphal tips emerging between cells of ovary epidermis and rupturing cuticle. **5)** Cross section of infected ovary 3 days after inoculation; hyphae emerging between cells of epidermis (across middle) and forming plectenchymatous extramatrical stroma (above). **6)** Cross section 4 days after inoculation showing production of conidia from palisade of phialides over surface of extramatrical stroma.

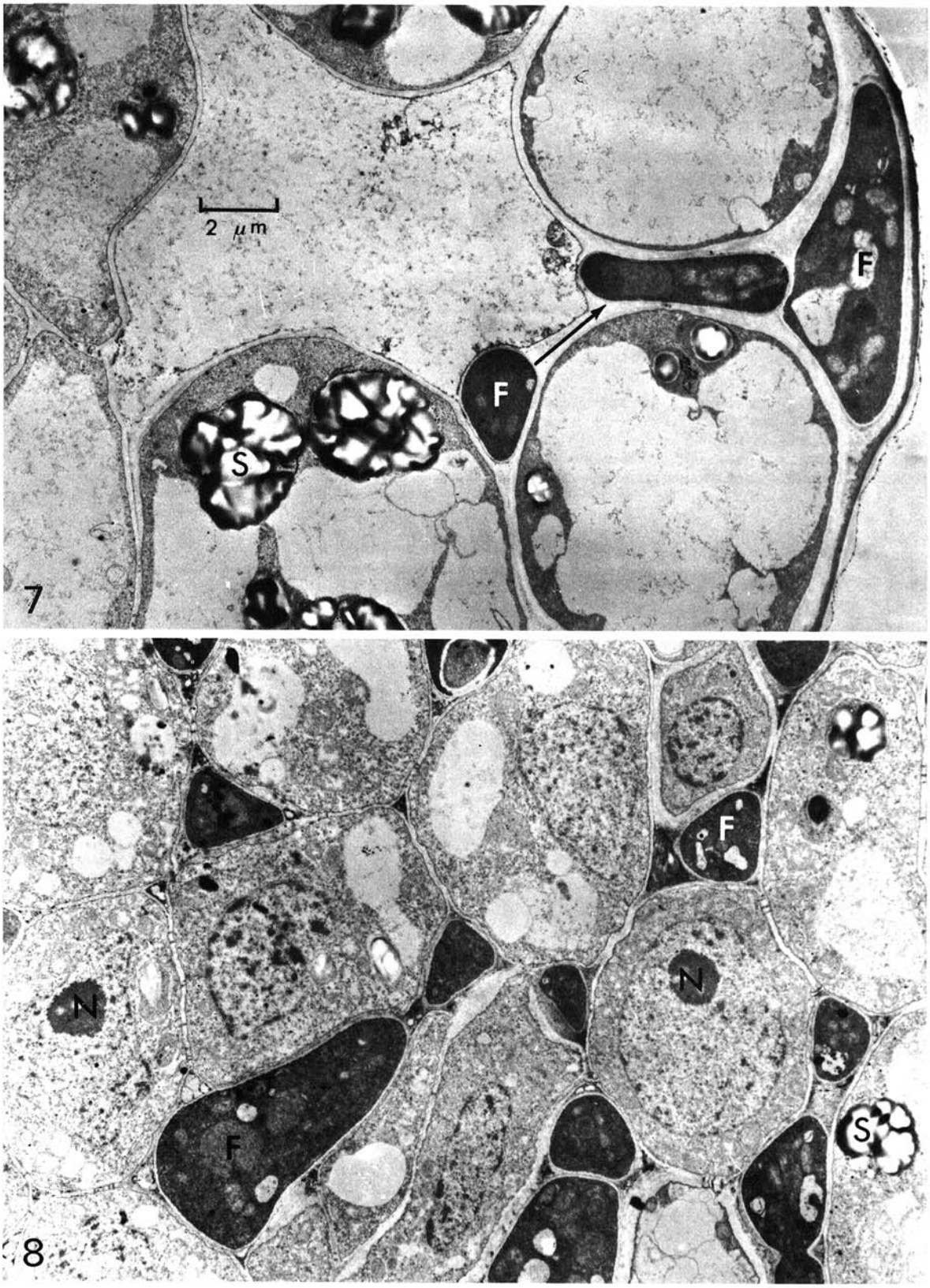

Fig. 7-8. *Claviceps paspali.* Electron micrographs of cross sections of infected ovary wall 2 days after inoculation; all conform to scale bar in Fig. 7. Symbols: F = fungus hyphae, N = nucleoli of host nuclei, and S = starch grains in host cells. **7)** Hypha (arrow) emerging between two epidermal cells and spreading laterally beneath cuticle. **8)** Deeply stained intercellular hyphae among host cells in ovary wall.

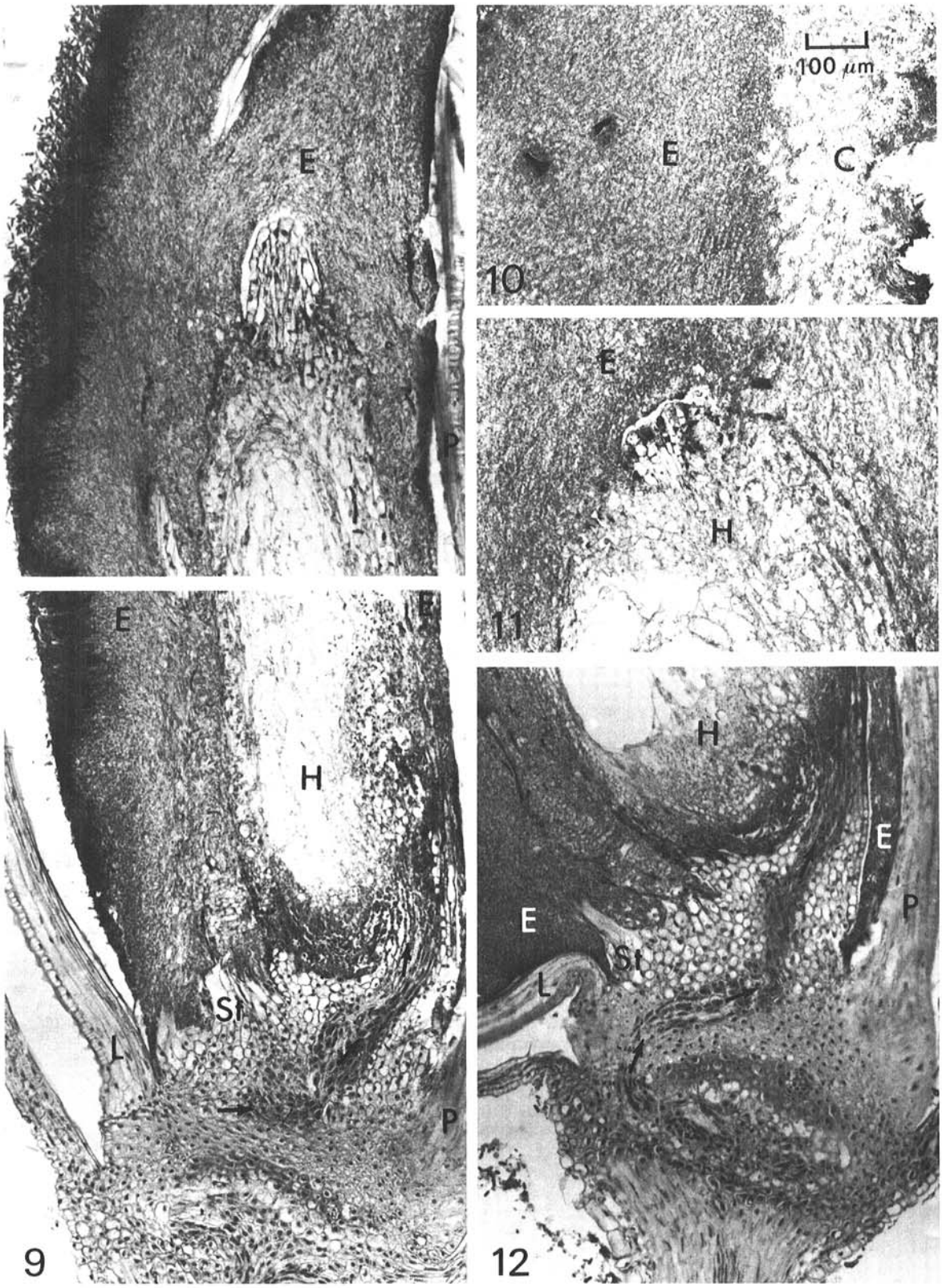

Fig. 9-12. *Claviceps paspali.* Light micrographs of longitudinal sections of entire infected florets; all conform to scale bar in Fig. 10. Symbols: L = host lemma, P = host palea, St = stamen, arrows = vascular bundle winding upward through host receptacle into stalk of ovary, H = disintegrated ovary, E = extramatrical fungus stroma or sclerotium, and C = cortex of sclerotium. **9)** Floret 4 days after inoculation; extramatrical stroma surrounding ovary (disintegrated except for cells in apex and basal stalk), extending downward over surface of receptacle between stalk of ovary and lemma and palea, engulfing fragments of stamen (top and left middle), and over surface at left covered by deeply staining layer of phialides producing conidia in cavity enclosed by concave lemma. **(10-12)** Various levels of mature sclerotium in floret 15 days after inoculation. **10)** Apex of sclerotium with cortex at right and dark fragments of stigma embedded in sclerotial plectenchyma at middle and upper left. **11)** Tissue near base of sclerotium with outline of ovary apex still visible. **12)** Base of floret with massive sclerotium extending downward around base of disintegrated ovary into space between ovary stalk and widely spread lemma and palea; host cells in ovary stalk and receptacle still alive and nucleated.

engulfing stamens and styles. Over its surface, especially the free surface enclosed by the lemma, it produced a palisade of conidiophores (Fig. 6, 9). Ridges of longer hyphae, appearing as synnema-like tufts in section (Fig. 6), anastomosed over the surface of the stroma.

Host cells in the receptacle and in the stalk of the ovary remained alive. The fungus stroma and the noninvaded host tissue of the receptacle were separated by a narrow interface of living host tissue into which intercellular hyphae penetrated. The vascular bundle extending up the adaxial side of the ovary stalk terminated in the fungus tissue of the stroma. There was no tendency for hyphae to follow the vascular bundle downward. In fact, host tissue persisted to a higher level on the adaxial side of the ovary. The stroma extended irregularly downward around the outer surface of the stalk of the ovary and filled the spaces between lemma and palea (Fig. 9).

Development of the sclerotium.—Further development consisted of expansion of the stroma to form the mature ergot sclerotium. By the 9th day the stroma filled the space inside of the lemma and protruded from the tip of the floret. At 15 days after inoculation the sclerotium appeared mature (Fig. 10-12). The entire exposed surface of the stroma beyond the margins of the palea and lemma was covered by an irregular brownish cortex made up of layers of empty cells (Fig. 10). Remnants of the conidial layers persisted at points on the surface. Inside of the cortex the sclerotium was composed of a rather uniform, compact plectenchyma. Fragments of host stigmatic cells were embedded in the apex. In the base of the sclerotium the outlines of the ovary were apparent in its original position (Fig. 11, 12). A loose plectenchyma filled the space originally occupied by the ovary.

Host relations were essentially the same on the 15th day (Fig. 12) as on the 4th day (Fig. 9). The stromatic tissue of the fungus remained sharply marked off from the living cells in the ovarian stalk.

DISCUSSION

My observations on the disease cycle in dallisgrass ergot agree with those of Brown (2) except on ascospore dispersal, which he suggested was accomplished by insect transport from globules extruded from the beaks of perithecia. Similar disagreement on ascospore dispersal has arisen from studies of rye ergot although Engelke (5) gave a clear account of forcible ascospore discharge in *C. purpurea* in 1902. Ascospores of *C. paspali* may collect in globules on the perithecial beaks under conditions suboptimum for completion of the normal process of forcible discharge. Under suitable moisture conditions ascospores are forcibly discharged, and the primary inoculum is airborne.

Brown's (2) assumption that conidia of *C. paspali* are dispersed from the drops of honeydew by insects is probably valid, although insect transmission has not been confirmed by experiment. Demonstration of the production of secondary conidia on phialides projecting singly into the air as a consequence of repetitive germination of conidia suggests the possibility of infection by airborne conidia; but this must be only a subsidiary means of dissemination in the secondary disease cycles.

Exposure to low temperatures is required to break dormancy in sclerotia of *C. paspali*, and adequate moisture for germination of sclerotia and production of ascospores is required for initiation of the primary disease cycle. Otherwise, the fungus seems to be largely sequestered from the external environment. Conidia introduced into the florets during anthesis produce a high percentage of infection in the absence of external moisture, presumably as a result of germination in the microenvironment within the closed lemma and palea. Conidia deposited on the stigmas after closure of the florets require only a 2-hr dew period for infection. Development of the extramatrical stroma and production of conidia also occur in the protected microenvironment within the floret. High humidities are necessary for the formation of conspicuous globules of extruded conidia at the floret tips, and production of large drops of honeydew may increase the probabilities of dissemination by insects. Splashing or wind driven rain also may diseminate the conidia.

Both direct observations and the results of inoculations made after dallisgrass florets were closed and only the stigmas were exposed indicate that infection by *C. paspali* occurs through the stigma and style. Although Kirchhoff (6) and Campbell (3) stated that *C. purpurea* enters through the base of the ovary, Engelke (5) reported growth of the fungus downward through the pistil to the base of the ovary. This seems the more probable course of normal infection. Infection through the stigma followed by preferential growth of invading hyphae in the inner layers of the ovarian wall to the base of the ovary is characteristic of ovarian infections caused by such diverse fungi as smuts (1) and deuteromycetes (8, 13), although penetration also may occur through the ovary (1, 8).

As Brown (2) noted, the dallisgrass ovary is completely surrounded by an extramatrical stroma and is destroyed within 4 days after infection by *C. paspali*. When rye ovaries are infected by *C. purpurea*, development begins at the base of the ovary with the appearance of the sphacelial stage (12). A convoluted white stroma spreads rapidly upward and almost obliterates the ovary. The hairy, cap-like apex of the ovary, however, is carried upward on the apex of the sphacelial stroma, where it persists as an identifiable mass of ovarian tissue. Campbell [Fig. 22 of (3)] illustrated the eruption, as in *C. paspali* (Fig. 5), of the intercellular hyphae between the epidermal cells of the ovary to form the extramatrical sphacelial stroma, but early stages in invasion of the ovary by *C. purpurea* have not been adequately documented.

Structure of the ergot in *C. purpurea* is more complex than in *C. paspali*. The ergot sclerotium of *C. purpurea* is clearly differentiated from the sphacelial stroma. It develops in the base of the sphacelial stroma and carries the sphacelial stroma upward on its tips (4, 12). Immediately above the foot-like base of the sclerotium, which maintains contact with the vascular system of the host receptacle (4, 9), is a layer of vertically oriented generative hyphae (4). Above the generative zone the sclerotium is covered by a dark cortex and is further differentiated internally into regions of fine hyphal cells and regions of isodiametric cells (3).

Despite the differences in development and morphological complexity of the sclerotia, host relationships in *C. paspali* and *C. purpurea* are essentially the same. The host ovary serves as little more than a portal of entry. The imperative in infection is establishment of a compatible

relationship with living tissue in the tip of the host receptacle. Development of the sphacelial stage and of the sclerotium depends on nutrients translocated from the host through the narrow region in which this compatible relationship is maintained (10).

LITERATURE CITED

1. BHATT, R. S. 1946. Studies in the Ustilaginales. I. The mode of infection of the bajra plant (Pennisetum typhoides Stapf.) by the smut Tolyposporium penicillariae Bref. J. Indian Bot. Soc., Madras 25:163-186.
2. BROWN, H. B. 1916. Life history and poisonous properties of Claviceps paspali. J. Agric. Res. 7:401-406.
3. CAMPBELL, W. P. 1958. Infection of barley by Claviceps purpurea. Can. J. Bot. 36:615-619.
4. DICKSON, J. G. 1947. Diseases of field crops. McGraw Hill, New York. 429 p.
5. ENGELKE, C. 1902. Neue Beobachtungen über die Vegetations-Formen des Mutterkornpilzes Claviceps purpurea Tulasne. Hedwigia 41:221-222.
6. KIRCHHOFF, H. 1929. Beiträge zur Biologie und Physiologie des Mutterkornpilzes. Zentralbl. Bakteriol. Parasitenk. Abt. 2, 77:310-369.
7. KYBAL, J. 1964. Neue Beobachtungen über die Bildung der Sklerotien von Claviceps purpurea (Fr.) Tul. Planta Med. 12:166-168.
8. LUTTRELL, E. S. 1976. Ovarian infection of Sporobolus poretii by Bipolaris ravenelii. Phytopathology 66:260-268.
9. MILOVIDOV, P. 1954. Příspěvek k mikroskopiko-morfologickčmu studiu vývoje námele (Claviceps purpurea (Fr.) Tul.) Preslia 26:415-425. [Biol. Abstr. 30:11517. 1956].
10. MOWER, R. L., and J. G. HANCOCK. 1975. Mechanism of honeydew formation by Claviceps species. Can. J. Bot. 53:2826-2834.
11. RAMSTAD, E., and G. GJERSTAD. 1955. The parasitic growth of Claviceps purpurea (Fries) Tulasne on rye and its relation to alkaloid formation. J. Am. Pharm. Assoc. (Scientific Edition) 44:741-743.
12. TULASNE, L. R. 1853. Mémoire sur l'ergot des glumacées. Ann. Sci. Nat. 20:5-56.
13. WEIHING, J. L. 1956. False smut of buffalograss. Nebr. Agric. Exp. Stn. Res. Bull. 180:1-26.