Dictyophora cinnabarina

The mushroom fungus *Dictyophora cinnabarina* commonly called dancing lady or skirted stinkhorn, is a macrofungus that belongs to the order Phallales in the fungal phylum Basidiomycota. The members are commonly known as stinkhorns. The fungus grows at a temperature of about 21°C, altitude of 690 m asl and relative humidity of 45–85%. *D. cinnabarina* is commonly found at Kuvempu University, Shankaraghatta during October/November.1,2

*D. cinnabarina* is a partial saprobe growing on dead tree trunks. The macrofungus is attractive because of its orange-coloured ‘inducium’ (skirt). Mycelium is white in colour, spreading all over the tree trunk and beneath the soil intermittently with the formation of reproductive structures called ‘eggs’ (young stage of basidiocarp) with a conspicuous rhizomorph at the base existing in the substrate. The eggs are hypogeous when young and become epigeous at maturity.3

The growth stages of *D. cinnabarina* have been studied by us. The matured basidiocarp is about 9–12 cm tall, receptacle white 1.5–2.5 cm thick, cylindrical, spongy, perforated with bulbous base. Gleba (cap-fertile portion of basidiocarp) 1.5–2.5 cm, dark metallic green and celled with an apical pore, sticky, gelatinous, odoriferous and decreasing with age. Inducium 5–9 cm in length, 6–29 cm in diameter, orange, porous, margin wavy, semi-elastic and increasing with age; volva 2–2.5 cm, white and thick (herb no. KUABSAK-192, Department of Applied Botany, Kuvempu University, Shankaraghatta).

The life cycle of *D. cinnabarina* is for 18–21 days, including vegetative (formation of mycelium) and reproductive (formation of basidiocarp) stages. The reproductive stage passes through four phases: egg phase, growth phase-I (receptacle phase), growth phase-II (inducium phase) and adult phase.

From the egg phase we have made detailed study on *D. cinnabarina* during October/November 2005.

Egg phase: Eggs are white in colour, surrounded by three layers of semi-elastic and rubbery peridium (exoperidium, mesoperidium and endoperidium). The development and differentiation of different parts of *D. cinnabarina* take place within the eggs in 6–14 days. The global areas within the eggs are surrounded by yellowish white mucilage (Figure 1a).

Growth phase-I (receptacle phase): The receptacle is differentiated and characterized within the eggs. It is protected by the surrounding peridial layers, breaks up giving rise to receptacle with the gleba at its tip. Breakage of peridium occurs due to the pressure exerted by the internal tissue or by the external force of any medium. Remnants of the peridium form the volva. Complete development of the receptacle outside the layer takes place within 2–5 min. In some cases, the basidiocarps are formed beneath the rocks under pressurized state and when the rocks are removed the peridium of the basidiocarp breaks up and the receptacle with the gleba at its tip stands up within 5 s (Figure 1b).

Growth phase-II (inducium phase): In this stage a porous orange-coloured inducium emerges out from the base of the gleba in 2–3 h. The green gleba undergoes disintegration or autodigestion and as a result the length decreases (Figure 1c).

Adult phase: After 2–3 h white receptacle stands out with colourful orange inducium and reduced dark metallic green, sticky, gelatinous, odoriferous, gleba at its tip. The insects that are attracted feed on the internal tissue of the gleba and help in the dispersal of spores (Figure 1d).

Figure 1. Growth stages of *Dictyophora cinnabarina*. 
The matured basidioecarp lasts for 1–2 h and disintegrates debishing the spores in the substratum. The spores undergo development forming mycelium-utilizing nutrients, and later forming the basidioecarp. Thus the life cycle continues.


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SYED ABRAR
S. SWAPNA
M. KRISHNAPPA

*For correspondence.

e-mail: krishnappam4281@yahoo.com

Non-involvement of parathyroid glands in adaptation to low-calcium diet in diabetic rats

Active transport of calcium in the intestine is significantly increased when experimental animals are subjected to low-calcium diet. In rats, fall in calcium level leading to an increased secretion of parathyroid hormone (PTH) seems to be the primary event in this process. Although there is evidence for a direct action of PTH on the intestine, the hormone increases the renal synthesis of 1,25-dihydroxy cholecalciferol (1,25 DHCC), which in turn stimulates the active transport of calcium by the gut. Removal of parathyroid glands in normal rats prevents this adaptive response. Diabetic rats show a marked decrease in circulating 1,25 DHCC level due to poor synthesis of this active metabolite in the kidney. In a bid to study the adaptive process to low-calcium diet in diabetic rats, we performed the following experiments.

Young male rats of Sprague–Dawley strain weighing about 150 g were placed on high-calcium diet (H, 1.5% Ca) on day-0. On day-6, the rats were subjected to sham operation (S) or parathyroidectomy (P) (Tables 1 and 2). Rats which did not show any significant fall in plasma calcium after 24 h of surgery, were included in the sham group. On day-7, rats were randomly either continued on H or placed on a low-calcium diet (L, 0.02% Ca) supplied by Teklad Test Diets. On day-14, they were randomly injected with streptozotocin (70 mg/kg, ip) dissolved in pH 4.5 citrate buffer (D) or buffer alone (N). On day-19, the rats were bled for determination of plasma calcium and glucose using auto analyzer and urine was tested for the presence of glucose using the enzyme strip. Animals which did not show glucosuria were eliminated from the study. Everted duo-denal sacs were prepared as described earlier. Active transport of calcium was measured as serosal/mucosal (S/M) ratio of 45Ca, as described earlier.

Both diabetic and non-diabetic rats with intact parathyroid glands showed increase in active transport of calcium by the duodenum, when subjected to low-calcium diet. While the diabetic animals retained this ability to adapt after parathyroidectomy, the non-diabetic rats failed to respond in a similar manner. Their plasma calcium levels remained

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**Table 1.** Effect of low-calcium diet on sham-operated non-diabetic and diabetic rats

<table>
<thead>
<tr>
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<th>HNS</th>
<th>HDS</th>
<th>LNS</th>
<th>LDS</th>
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<tr>
<td>Active transport (S/M ratio)</td>
<td>2.8 ± 1.2*</td>
<td>2.6 ± 0.48*</td>
<td>6.3 ± 0.66*</td>
<td>6.3 ± 1.40*</td>
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<tr>
<td>Plasma calcium (mg%)</td>
<td>12.3 ± 0.69</td>
<td>10.4 ± 0.38</td>
<td>10.5 ± 0.38</td>
<td>10.2 ± 0.40</td>
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<tr>
<td>Plasma glucose (mg%)</td>
<td>111 ± 10*</td>
<td>463 ± 35*</td>
<td>115 ± 12*</td>
<td>486 ± 46*</td>
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<tr>
<td>Final BW (g)</td>
<td>185 ± 8.95</td>
<td>178 ± 9.5</td>
<td>182 ± 6.80</td>
<td>174 ± 8.8</td>
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**Table 2.** Effect of low-calcium diet on parathyroidectomized non-diabetic and diabetic rats

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<thead>
<tr>
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<th>HNP</th>
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<tbody>
<tr>
<td>Active transport (S/M ratio)</td>
<td>2.1 ± 0.36</td>
<td>1.7 ± 0.17*</td>
<td>2.8 ± 0.66</td>
<td>5.4 ± 0.99*</td>
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<tr>
<td>Plasma calcium (mg%)</td>
<td>12.68 ± 3.33*</td>
<td>12.0 ± 0.50</td>
<td>6.3 ± 1.02*</td>
<td>9.08 ± 0.99</td>
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<tr>
<td>Plasma glucose (mg%)</td>
<td>131 ± 17*</td>
<td>395 ± 19*</td>
<td>110 ± 8*</td>
<td>415 ± 38*</td>
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<tr>
<td>Final BW (g)</td>
<td>191 ± 4.9</td>
<td>138 ± 11.9</td>
<td>119 ± 14.2</td>
<td>111 ± 11.3</td>
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All values are expressed as mean ± SEM of 4 to 8 observations. Values bearing identical superscripts are significantly (P < 0.05) different from each other. Statistical analysis using Student’s ‘t’ test was performed. P value was set after applying correction for multiple comparisons. H, High-calcium diet; L, Low-calcium diet; N, Non-diabetic; D, Diabetic; S, Sham-operated; P, Parathyroidectomized, and BW, Body weight.