The matured basidiocarp lasts for 1–2 h and disintegrates dehiscing the spores in the substratum. The spores undergo development forming mycelium-utilizing nutrients, and later forming the basidiocarp[9]. Thus the life cycle continues.

1. Jamaluddin, Goswami, M. G. and Ojha, B. M., *Fungi of India 1989–2001*, Scientific Publishers (India), Jodhpur, 2004, pp. 1–158.
2. Brown, N., Bhagwat, S. and Watkinson, S., *J. Appl. Ecol.*, 2006, **43**, 11–17.
3. Ainsworth, G. C., James, P. W. and Hawksworth, D. L., *Ainsworth and Bisby's Dictionary of Fungi*, 1971, 6th edn, p. 166.
4. Alexopolous, C. J., Mims, C. W. and Blackwell, M., Phylum Basidiomycotina. In *Introductory Mycology*, John Wiley, New York, 1996, pp. 544–554.
5. Hemmes, D. E. and Desjardin, D. E., XVI International Botanical Congress, San Francisco, USA, 1999.
6. Lee, W. S., *Mycologia*, 1957, **49**, 156.
7. Tuno, N., *Ecol. Res.*, 1998, **13**, 7.
8. Martin, S., Fungi. In *Tropical Topics*, No. 72, March 2002.
9. Lockwood, T. F., *Gasteromycetes, Treasures from the Kingdom of Fungi*, 1999, pp. 59–60.

ACKNOWLEDGEMENT. We thank Mr R. Deepak for the photographs.

Received 7 March 2006; revised accepted 7 December 2006

SYED ABRAR
S. SWAPNA
M. KRISHNAPPA*

Department of Post Graduate Studies and Research in Applied Botany,
Kuvempu University,
Jnana Sahyadri,
Shankaraghatta 577 451, India
**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[1], the hormone increases the renal synthesis of 1,25 dihydroxy chole calciferol (1,25 DHCC), which in turn stimulates the active transport of calcium by the gut[2]. Removal of parathyroid glands in normal rats prevents this adaptive response[3]. Diabetic rats show a marked decrease in circulating 1,25 DHCC level due to poor synthesis of this active metabolite in the kidney[4]. In a bid to study the adaptive process to low-calcium diet in diabetic rat, 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 duodenal sacs were prepared as described earlier[5]. Active transport of calcium was measured as serosal/mucosal (S/M) ratio of ^{45}Ca, as described earlier[6].

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

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

	HNS	HDS	LNS	LDS
Active transport (S/M ratio)	2.8 ± 1.2[a]	2.6 ± 0.48[b]	6.3 ± 0.66[a]	6.3 ± 1.40[b]
Plasma calcium (mg%)	12.3 ± 0.69	10.4 ± 0.38	10.5 ± 0.38	10.2 ± 0.40
Plasma glucose (mg%)	111 ± 10[c]	463 ± 35[c]	115 ± 12[d]	486 ± 46[d]
Final BW (g)	185 ± 8.95	178 ± 9.5	182 ± 6.80	174 ± 8.8

Table 2. Effect of low-calcium diet on parathyroidectomized non-diabetic and diabetic rats

	HNP	HDP	LNP	LDP
Active transport (S/M ratio)	2.1 ± 0.36	1.7 ± 0.17[f]	2.8 ± 0.66	5.4 ± 0.99[f]
Plasma calcium (mg%)	12.68 ± 0.33[g]	12.0 ± 0.50	6.3 ± 1.02[g]	9.08 ± 0.99
Plasma glucose (mg%)	131 ± 17[h]	395 ± 19[h]	110 ± 8[i]	415 ± 38[i]
Final BW (g)	191 ± 4.9	138 ± 11.9	119 ± 14.2	111 ± 11.3

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.

low, confirming lack of an enhanced calcium absorption.

It is clear that the diabetic rat responds to low-calcium diet by a significant increase in active transport of calcium by the duodenum. Unlike normal rats, diabetic rats do not seem to require intact parathyroid glands for this adaptation. Diabetic rats do show a rise in plasma level of 1,25 DHCC in response to low-calcium diet[4]. However, such levels are still less than those encountered in normal rats on standard lab chow[4] and may still be insufficient to cause an increase in synthesis of calbindin9K, a key protein involved in the active transcellular transport of calcium[7]. In view of these observations, it is likely that some other mechanism is involved in the adaptation of diabetic rats subjected to low-calcium diet.

Recently, the existence of calcium sensor in the intestine with capabilities of modulating the transport in response to plasma calcium level has been proposed[8]. Perhaps such a sensor is upregulated in diabetic rats, which show an increase in the size of intestinal mucosa[9]. This in turn may promote calcium transport in the absence of parathyroids in diabetic animals. Our experiments provide an impetus to look into these possible avenues of research.

1. Nemere, I. and Larsson, D., *J. Cell. Biochem.*, 2002, **86**, 29–34.
2. Kenny, A. D. (ed.), In *Intestinal Calcium Absorption and its Regulation*, CRC Press, Boca Raton, Florida, 1981, pp. 79–85.
3. Pento, J. T., Waite, L. C., Tracy, P. J. and Kenny, A. D., *Am. J. Physiol.*, 1977, **232**, E336–E342.
4. Wilson, H. D., Horst, R. L. and Schedl, H. P., *Diabetes*, 1982, **31**, 401–405.
5. Wilson, T. H. and Wiseman, G., *J. Physiol.*, 1954, **123**, 116–120.
6. Martin, D. L. and De Luca, H. F., *Am. J. Physiol.*, 1969, **216**, 1351–1356.
7. Stone, L. A., Weaver, V. M., Burns, M. E., and Welsh, J., *Diabetes Res.*, 1990, **15**, 165–172.
8. Chattopadhyay, N., *Int. J. Biochem. Cell Biol.*, 2000, **32**, 789–804.
9. Noda, T. *et al.*, *Metabolism*, 2001, **50**, 259–264.

ACKNOWLEDGEMENTS. This scientific correspondence is dedicated to the memory of our mentor Alexander D. Kenny. This work was made possible through a grant from the NIH, Maryland to J.P.R.

Received 28 February 2005; revised accepted 26 December 2006

J. PRAKASA RAO[1,*]
INGRID GREENE[2]

[1]*Department of Physiology, Kasturba Medical College, Manipal 576 104, India*
[2]*Department of Pharmacology, Texas Tech University Health Sciences Centre, Lubbock, Texas 79430, USA*
**For correspondence.*
e-mail: jprao2001@hotmail.com

Reclamation and status of tsunami damaged soil in Nagappattinam District, Tamil Nadu

The 26 December 2004 earthquake (9.4 on the Richter scale) in the Indian Ocean led to generation of tsunami which caused devastative damages to human life, natural resources, livelihood assets and infrastructure. In the context of agriculture, the tsunami caused severe damage to standing crops as well as agricultural fields in Kancheepuram, Villupuram, Cuddalore, Nagapattinam, Thirunelveli and Kanyakumari Districts in Tamil Nadu, of which Nagapattinam was the worst affected district. In this district, the basic resources such as soil and water in the farm were severely affected; while common and grazing lands got salinized. Nearly, 4657 ha of cultivated area were affected due to salinization, of which 1367 ha were subjected to sand and silt/clay deposition.

The soil in Nagapattinam District is predominantly sandy in texture and clayey in certain pockets with slight salinity/alkalinity. The soil of the region belongs to the Valudalakudi series and possesses characteristics of dark brown to brown, deep, sandy and mild to moderate alkalinity levels[1]. During the pre-tsunami period the soil pH ranged between 6.1 and 8.55 and electrical conductivity (EC) between 0.2 and 1.1 dsm^{-1}, whereas the post-tsunami analysis (January 2005) showed a steep increase in pH to more than 8.5 and seldom exceeded 9 in selected locations and EC up to 23.7 especially in the clay deposited fields[1].

The type and intensity of damage to soil resources due to intrusion of tsunami varied across the affected areas in the district. Thus it was necessary to understand the area-specific issues as well as the multidimensional nature of the problem. The explorative soil analysis and discussion during July 2005 with the local farmers through a travelling workshop across the affected areas was organized by the M.S. Swaminathan Research Foundation (MSSRF, Chennai) in collaboration with 11 technical and academic institutions. The outcomes revealed three different kinds of major damages to soil such as: (i) Deposition of slushy greyish brown clay deposit; (ii) Sandy soils, and (iii) Sea water intrusion, which receded from the field (within 3 h to one week) leaving behind salts in the field. Among the devastating damages, the clay deposit was a cause for concern among scientists, NGOs and farmers for its mineral constituents and hence heavy metal analysis was carried out. The results showed that heavy metals like, nickel (12 mg/kg), zinc (32.5 mg/kg), chromium (35.5 mg/kg), copper (99.8 mg/kg) and lead (56 mg/kg) were comparatively less than the permissible limits of Indian compost standards with the exception of cadmium (31 mg/kg). (Heavy metal analysis of tsunami clay deposited material collected from Pushphavanam (a village located in Vedaranyam block, south of Nagapattinam) during July 2005 and April 2006 and analysed at the Indian Institute of Technology, Chennai.) At the same time, the clay deposit was rich in organic matter (0.6–1.2%), having good water-holding capacity and cation exchange capacity with an exchangeable sodium percentage