It is also noticed that by cutting young cashew trees close to the ground (coppicing), production of a large number of sprouts can be encouraged which can be subsequently layered as described earlier. Theoretically such a stump after one set of layering should produce more number of shoots for further layering.

Detailed studies on the best time of stooling, effect on root formation with and without hormone application and optimum period for separation of layers after stooling have been undertaken at the station.

Central Plantation Crops Research Institute (Regional Station), Vittal-574 243, Karnataka, India,
March 9, 1979.

M. A. MENON,
S. NAGABHUSHANAM.

1. Argles, G. K., "*Anacardium occidentale*—Cashew" In : *The Propagation of Tropical Fruit Trees.* ed. R. T. Garner *et al.*, Commonwealth Bureau of Horticulture and Plantation Crops, East Malling, Maidstone, Kent, 1976, p. 184.
2. Coester, W. A. and Ohler, J. G., *Trop. Agric.*, 1976, **53**, 353.
3. Hartmann, H. T. and Kester, D. E., *Plant Propagation Principles and Practices.* Prentice Hall Inc., Englewood Cliffs, New Jersey, 1975.
4. Mukherjee, S. K. and Mazumdar, P. K., *Indian J. Hort.*, 1963, **20**, 204.
5. —, —, Bid, N. N. and Goswami, A. M., *J. Hort. Sci.*, 1967, **42**, 83.

A MODEL FOR THE GENERATION OF T CELL RECEPTORS

T LYMPHOCYTES show dual specificity, one for the foreign antigen (anti-X R_1 receptor), and the other for the self H-2 antigen (anti-H-2 R_0 receptor)[1–3]. Anti-X R_1 receptors are generated by somatic mutation of germ-line genes encoding specificity for self H-2 antigens (anti-H-2 R_1 receptors)[4,5]. On the other hand, the generation of anti-H-2 R_0 receptors is determined by the thymus epithelium and these R_0 specificities are directed against the H-2 antigens of the thymus[2,6]. I suggest here a model for the ontogeny of these receptors.

von Boehmer, Haas and Jerne[5] have recently suggested that the generation of R_0 receptors occurs by a process of positive thymic selection[5], while that of anti-X R_1 receptors occurs by a process of negative thymic selection[4]. Thus the hypothesis of von Boehmer *et al.* claims that the H-2 antigens present only on thymus epithelium can drive the generation of diversity. The present model proposes that the H-2 antigens which can bring about the generation of T cell diversity need not necessarily be present on the thymus epithelium. Although H-2 antigens are present both on thymus as well as on nonthymic tissues, the anti-H-2 R_0 receptors are determined only by thymus epithelium[2]. I suggest that the expression of genes encoding R_0 receptors is induced by thymus-specific molecules. It is proposed that: (*a*) the expression of V genes coding for R_1 receptors is independent of thymic environment, (*b*) R_1 mutates on interaction with the corresponding H-2 antigens present on thymus epithelium or on nonthymic cells, and the resulting anti-X R_1 specificity is directed against X alone or against the complex of X with the responder H-2 antigen, (*c*) the expression of genes encoding R_0 receptors is induced by thymus-specific effector molecules different from H-2 antigens, and (*d*) dual recognition is necessary for clonal selection and amplification. If R_1 specificity is directed against X alone, dual recognition is ensured by the thymus induced R_0 receptor. The notion of thymic induction is consistent with current ideas on eucaryotic gene expression[7]. Parenthetically it may be noted that the findings of Press and McDevitt[8] can be explained as a consequence of anti-X R_1 receptor being specific against the complex of the foreign antigen (X) with the responder H-2 antigen.

This model makes specific predictions which are amenable to experimental verification. For example, when nonresponder bone marrow stem cells are made to differentiate in a mixed environment of responder nonthymic tissues and nonresponder thymus, the present model predicts that the nonresponder cells would acquire responsiveness whereas that of von Boehmer *et al.* predicts that they would not. The experiment also permits one to distinguish between the following two possibilities: (*a*) anti-X R_1 specificity directed against X alone or (*b*) anti-X R_1 specificity directed against the complex of X and responder H-2 antigen. In case (*a*), the T lymphocyte shows specificities directed against the foreign antigen (X) and the thymic nonresponder H-2 antigen whereas in case (*b*) the T cell shows specificities directed against: (1) the antigen X, (2) the nonthymic H-2 antigen that drives the generation of anti-X R_1 receptor, and (3) the thymic nonresponder H-2 antigen. It should be noted that the specificities (2) and (3) would be directed against the same (responder) H-2 antigen when the T cells mature in a responder thymus.

The model also predicts that a thymic extract from which H-2 antigens are removed should induce anti-H-2 R_0 specificity in thymectomised and irradiated mice reconstituted with bone marrow stem cells. This R_0 specificity would be directed against the H-2 antigens of the thymus epithelium from which the extract is made. Further implications and details of the model will be discussed in a separate article.

I am grateful to Drs. H. Sharat Chandra and V. Nanjundiah for constant advice and encouragement. I thank the Department of Science and Technology, Government of India, for the award of a fellowship.

Department of Biochemistry, B. SESHU.
Indian Institute of Science,
Bangalore 560 012, India,
September 14, 1979.

1. Zinkernagel, R. M. and Doherty, P. C., *Nature*, 1974, 251, 547.
2. —, Callahan, G. N., Klein, J. and Dennert, G., *Ibid*, 1978, 271, 251.
3. Waldmann, H., Pope, H., Brent, L. and Bighouse, K., *Ibid.*, 1978, 274, 166.
4. Jerne, N. K., *Eur. J. Immunol*, 1971, 1, 1.
5. von Boehmer, H., Haas, W. and Jerne, N. K., *Proc. Natl. Acad. Sci. U.S.A.*, 1978, 75, 2439.
6. Waldmann, H., Pope, H., Bettles, C. and Davies, A. J. S., *Nature*, 1979, 277, 137.
7. Britten, R. J. and Davidson, E. H., *Science*, 1969, 165, 349.
8. Press, J. L. and McDevitt, H. O., *J. Exp. Med.*, 1977, 146, 1815.

INFLUENCE OF AN ORAL CONTRACEPTIVE ON STEROIDOGENESIS

LYNDIOL 1 mg (Organon Laboratories) is a well-reputed oral contraceptive which is widely used in India. Relationship between oral contraceptive and serum cholesterol has been already established[1] and the role of gonadal ascorbic acid in steroidogenesis has been explained by Dennert who has shown an increased concentration of the same in hypofunctioning ovaries. Cholesterol serves as the precursor of steriod hormones and the seat of the sex hormone synthesis lies in the gonads. Ovary synthesizes sex steroids and cholesterol serves as the precursor of steriod hormones[11, 12]. Among the different steps of steroid biogenesis, oxidoreduction and hydroxylating process occupy the most important. Oxidoreduction process is catalysed by DPN linked dehydrogenase as $\Delta^5 3\beta$ hydroxy steroid dehydrogenase. Oral administration of cholesterol increases 11 hydroxysteroid excretion.[2] Ascorbic acid has been reported to induce the synthesis of ovarian steroids in rats[8]. The enzyme, $\Delta^5 3\beta$ hydroxysteroid dehydrogenase plays an important role in steroid biosynthesis, and the increase of its activity stimultates steroidogenesis.

The oral contraceptive taken for the present study is a combination of Lynesterol and Etlenylestradiol. High dose (0·02 mg/100 g) of this drug was used daily for three months and the cholesterol, ascorbic acid levels were estimated from ovary and adrenal to study its influence on them and the activity of the enzyme $\Delta^5 3\beta$ hydroxy steroid dehydrogenase was detected histochemically *paripassu* for studying the effect on steroidogenesis of the oral contraceptive.

Methods and Materials

Mature female albino rats (20 in number) weighing between 100 to 115 g were kept on laboratory stock diet for 7 days. They were then individually caged and divided into 2 groups of 10 animals each. The first group was considered as control. The experimental group was administered Lyndiol orally at a dose of 0·02 mg/100 g body weight daily, mixed with 15 g of food. All the animals took their daily food completely. The animals were sacrificed after 90 days and the adrenals and ovary removed quickly and weighed. The cholesterol and ascorbic acid concentrations were measured both in control and experimental groups. Cholesterol was determined by the method of Sperry and Webb[11] and ascorbic acid by the method of Roe and Kuether[9].

A second group of 10 animals were subdivided into two equal groups and caged. They were administered Lyndiol in a similar manner. A control group of 5 animals was also kept. After 90 days of drug administration, these animals were sacrificed and the ovarian and adrenal enzyme activity determined. The activity of the steroidogenic enzyme, and the $\Delta^5 3\beta$ HSD were determined histochemically[6].

Results

The results are summarised in Tables I and II. The weights of the ovaries and adrenals showed dissimilar changes after drug treatment. The ovary showed a 22% decrease in weight, whereas a 65% increase was observed in the weight of the adrenals after 90 days of

TABLE I

Changes in ovarian cholesterol and ascorbic acid in rat after Lyndiol treatment for 90 days

Daily Dose : 0 02 mg/100 g of body wt./15 g of food.

Parameters	Control	Treated	% change
Wt. of the ovary (mg) (10) Mean ± SEM	35·62 ± 0 065	27·79 ± 0·163	21·98 decrease
Cholesterol in mg/g of wet tissue (10) Mean ± SEM	17·80 ± 0·41	30·04 ± 0·45	68·76 increase
Ascorbic acid in mg/g of wet tissue (10) Mean ± SEM	0·3318 ± ·0061	0 4392 ± ·0093	32·36 increase

The figures within parenthesis indicate the number of animals sacrificed.