Meher-Homji for providing an opportunity to not only clarify the points raised, but also to state our position concerning vegetation types.

1. Champion, H. G., *Indian For. Rec.*, 1936.
2. Puri, G. S., Meher-Homji, V. M., Gupta, R. K. and Puri, S., *Forest Ecology*, Oxford & IBH, New Delhi, 1983, vol. I.
3. Pascal, J. P., *Wet Evergreen Forests of Western Ghats of India: Ecology and Structure Floristic Composition and Succession*, French Institute, India, 1988.

T. GANESH
R. GANESAN
M. SOUBADRA DEVY
P. DAVIDAR

Salim Ali School of Ecology and Environmental Sciences,
Pondicherry University,
Pondicherry 605 014, India

K. S. BAWA

Department of Biology,
University of Massachusetts, Boston,
100 Morrissey Blvd.
Boston, MA 02125, USA

Electrostriction effect and solvent engineering

Anil Kumar on application of ion–solvent interaction and thermostability of DNA duplex in ionic solution (*Curr Sci.*, 1996, **71**, 289) highlights many important findings which bear relevance not only to DNA but are very much true for proteins and enzymes. The kinetic data presented clearly explain the thermostability acquired by DNA in ionic solution. Interestingly, similar role of electrostriction effect is indicated from several reports on enzyme stability by soluble additives, e.g. salts, sugars and sugar alcohols.

Nowadays the area of thermostability of enzymes is a challenging field for chemists, biochemists, biotechnologists and industrial microbiologists. Lot of work has been directed to achieve this by stabilization of native confirmation of protein in aqueous environment. T_m of lysozyme and ribonucleases and many other enzymes has been raised using sugars and polyols. Thermolabile enzyme preparations have been stabilized by high ionic strength solutions. The role of viscosity, intensification of the intramolecular hydrophobic interaction within the protein reduced volume and increased surface tension has been advocated in protein solvent and additive interactions[1].

The possible role of electrostriction (ES) effect leading to a hydrophobic environment in an aqueous system in enhancing the shelf life of enzyme, altering substrate specificity and rate of reaction, opens up new vistas in solvent engineering of enzyme mediated reactions.

1. Gupta, M. N. (ed.), *Thermostability of Enzymes*, Narosa Publishing House, New Delhi, 1993.

RANI GUPTA
R. K. SAXENA

Department of Microbiology,
University of Delhi, South Campus,
Benito Juarez Road,
New Delhi 110 021, India

Anil Kumar replies:

Gupta and Saxena have offered valuable information on the role of electrostriction in explaining several biological processes. We often stress, in the seminars and symposia about effective and fruitful collaboration among biochemists, microbiologists and biophysicists. The time has now come that we, in India define the areas of collaboration and intensify collective efforts to work on the solvent engineering of enzyme-mediated reactions.

ANIL KUMAR

Physical Chemistry Division,
National Chemical Laboratory,
Pune 411 008, India

Dengue haemorrhagic fever epidemic in Delhi

Dengue was endemic in India, but dengue haemorrhagic fever (DHF) is of recent origin, first reported[1] from Calcutta in 1963. Since then DHF and dengue shock syndrome (DSS) have been occurring in India sporadically and entering urbanized and industrialized settlements. DHF/DSS epidemics have also been reported from purely rural villages in Maharashtra, Karnataka, Tamil Nadu, Delhi, etc. Urbanization and rural water supply are the principal reasons for *Aedes* dispersal. So far there have been 70 reported outbreaks in the country and the 1996 epidemic was preceded by 6 outbreaks in Delhi. All four serotypes (Den-1, Den-2, Den-3, Den-4) occur in India. In the 1996 epidemic Den-2 virus has been isolated by Pradeep Seth at the All India Institute of Medical Sciences, New Delhi and Kalyan Banerjee at the National Institute of Virology, Pune. The epicenter of DHF was south Delhi affecting all communities, and later cases were reported from almost all localities. In this epidemic beginning in August, the government has reported 8900 cases and 375 deaths due to DHF. Reported cases may be the proverbial tip of the iceberg. Dengue fever and deaths are also being reported from neighbouring towns in Haryana and Uttar Pradesh. Dengue now occurs across the country with increasing reports of DHF/DSS. Army's help was sought, but ironically deaths and large number of admissions due to DHF were also reported from the New Delhi military hospital. The epidemic has receded with the onset of winter but DHF is known to become endemic, and might resurface periodically. In Delhi *Aedes aegypti* breeds profusely with larval index from 20 to 40 or more in many

localities, particularly during the transmission months following rains. Breeding opportunities for *Aedes aegypti* are enormous and evenly spread in most Delhi in the form of desert coolers, water storage tanks, leaking water supply, fountains, wells, tire dumps, and rain water collection on roof tops and a variety of receptacles. Vector control under the urban malaria scheme is erratic and limited to some localities by way of spraying of larvicides in surface drains, temephos treatment of water tanks and thermal malathion fogging. Field operations are wanting and mainly targeted to control *Culex* breeding. Malaria is endemic in Delhi and the vector *Anopheles stephensi* and *Aedes aegypti* largely share similar breeding habitats. DHF epidemic coincides with *P. falciparum* prevalence making diagnostics a problem. Dip-stick test[2] for *P. falciparum* would be desirable. While malaria is treatable, there is no specific treatment for viral infections. In dealing with DHF preventive vector control is the key to success. This is attainable by a well-organized *Aedes* control programme. The control of *Aedes aegypti* by chemical methods like spraying and fogging or ULV application is not productive and sustainable[3], except epidemic control[4]. *Aedes* control requires strong entomological support which is weak in the states and almost non-existent in the municipal corporations and smaller bodies[5]. The disease has come to stay and governments would do well by emphasizing community participation and taking help of legislative measures. The approach to DHF control should be community based directed towards species sanitation and learning from the DHF endemic countries.

1. Ramakrishnan, S. P. *et al.*, *Indian J. Med. Res.*, 1964, **52**, 633–650.
2. Peyron, F. G. *et al.*, *Lancet*, 1994, **343**, 1502–1503.
3. Gubler, D. J., *Am. J. Trop. Med. Hyg.*, 1989, **40**, 571–578.
4. Gratz, N. G., *J. Am. Mosq. Control Assoc.*, 1991, **7**, 353–365.
5. Sharma, V. P., *Nature*, 1994, **369**, 700.

V. P. SHARMA

Malaria Research Centre,
22, Sham Nath Marg,
New Delhi 110 054, India

Sex and the single X

In mammals, including human, it is believed that the Y chromosome by its presence or absence exerts a series of actions upon which develop the sexually dimorphic characters. Although a number of genes in this cascade of binary decision might be autosomal or X-chromosomal, there must be one or more gene(s) on the Y chromosome to govern the process of testis determination if the male development is a dominant pathway.

SRY is the only gene that is required on the Y chromosome for testis determination as the introduction of transgenic *SRY* alone into chromosomally female mice results in male development[1]. Recently a few autosomal genes have been identified that are probably involved in testis development. For example, mutation in *SOX9* gene is associated with XY sex reversal in human[2] and the null mutant for the gene *SF-1* shows severe abnormalities in the gonadal development of mice[3]. In contrast to our current understanding of the process of male development in mammals, the genetic cascade of the female pathway is almost completely unknown. It is possible that in some of the XY females, a 'loss-of-function' mutation in a gene that inhibits ovarian development (in males) could have been the cause for sex reversal. Although chromosomal deletion in 9p and 10q is associated with such XY females, interruption of testis development was viewed as the cause for sex reversal having led to the female development by default[4]. However, that the ovarian differentiation may not be a passive one is indicated by the fact that in wood lemmings, rearrangement in an X chromosome induces the development of reproductively active XY females even in the presence of a normal Y chromosome[5]. More interestingly, in two species of mole-vole, sex determination is in the absence of Y chromosome (XO male; XX female)[5,6]. The first indication for involvement of an X chromosomal gene in human sex determination was provided by the identification of a family with an X-linked mode of inheritance of 46,XY sex reversal[7]. Demonstration of duplication in short arm of the X chromosome (Xp21 region, called the *DSS* locus) in such 46,XY females raised the interesting possibility that the duplicated X chromosomes cause XY sex reversal by expressing double dose of a gene normally subject to X inactivation[8]. However *DSS*-negative 46,XY males develop testis and show normal external genitalia, indicating *DSS* is not involved in testicular development[8]. Therefore, it is puzzling to imagine how the inactivation of X chromosome and the dosage activity of this locus control gonadal development.

Recently Jimenez *et al.*[9] discussed an appealing model for the dosage effect of *DSS* speculating a female determining function for the *DSS*. It is proposed that in normal female the *DSS* down regulates autosomal genes involved in male development (e.g. *MIS*) but the autosomal factors are not inhibited in the 46,XY males owing to the suppressive action of *SRY* on *DSS*. Thus, testis development implies inhibition of female pathway genes and for the ovarian development, the male pathway genes must be suppressed. Therefore, deletion of *DSS* does not affect male development. However the duplication of *DSS* in XY individuals [46,XY *dup*(Xp)] renders the level of SRY protein insufficient to suppress the two active doses of *DSS*, thus leading to development of female phenotype. It is likely that such a dosage effect might have been the primitive mechanism of sex determination in mammals and that *SRY*'s role is a recent addition because some of the events of sexual differentiation in the primitive mammals, marsupials, are based on the ratio between X chromosome and autosomes[10]. In fact the effect of dosage