tional polymorphism (SSCP) and minisequencing. Studies carried out on *Bt* resistance have shown the possibility of involvement of single gene or limited alleles. Characterizing the gene responsible for *Bt* resistance in *H. virescens* has enabled efficient DNA-based screening for resistant heterozygotes by directly detecting the recessive allele. Similar studies on identification of DNA markers to monitor *Bt* resistance in the American bollworm, *Helicoverpa armigera* are being carried out, to provide further impetus on efforts to develop *Bt* resistance management tactics.

Mariamma Jacob of the University of Kerala, Thiruvananthapuram highlighted the insect cell, tissue and organ culture as a tool in molecular endocrine research. Studies on the hormonal regulating mechanisms of spermatogenesis of *Oryctus rhinoceros* showed that testis fragments underwent meiosis in the presence of 20-hydroxyecdysone. Electrophoresis (SDSPAGE) of culture media, before and after spermatogonia culture showed new proteins in response to ecdysone added in the culture. Spermatocytes differentiate to spermatozoa when brain, corpus cardiacum (cc) and corpus allatum (ca) from adults are added in culture, thus giving evidence that other hormones/neuronal factors are essential for spermatogenesis. Most of the lepidopteran studies show that ecdysone is the only hormone promoting spermatogenesis.

Aspects relating to genetic and molecular implications in the improvement of *B. thuringiensis* were discussed by J. S. Kennedy of the Tamil Nadu Agricultural University, Coimbatore. He illustrated five different techniques for using genetically engineered *Bt* endotoxin gene to combat insect pests. These include spores and crystals, bioencapsulation, epiphytes or microbes that colonize the roots or leaves of plants, endophytes or microbes that live inside plant tissue and transgenic plants.

Delivering the valedictory address, T. M. Manjunath, Consultant, Monsanto Research Centre, Bangalore discussed opportunities and challenges afforded by transgenic crops. Transgenic technology offers exciting opportunities to develop plants with new traits, which include resistance to biotic stresses, pests, diseases, herbicides as well as abiotic stresses, drought, heat, cold, salinity. Transgenic technology also helps in increasing crop yield and enhancing quality traits such as nutrition and shelf-life of fruits, vegetables and flowers. Transgenic crops bestowed with any of these traits will significantly contribute towards improving sustainable agriculture. Similar economic and environmental benefits from transgenic crops had been reported from other countries also. Transgenic crops with various other traits are at various stages of experimentation in several countries. 'Golden Rice' fortified with vitamin A and iron, mustard with increased beta-carotene, potato with enhanced protein content, fat free oil, corn with enhanced animal feed products like lysine, edible vaccines are good examples.

S. Chelliah, Tamil Nadu Agricultural University, Coimbatore, while moderating the open session concluded that younger scientists should take up shores on molecular entomology, since there is need for a paradigm shift from traditional study of insects to address specific problems posed by biotypes and races of insect species for which molecular tools may be used in the diagnostics of biotypes. He emphasized once again that any success in agricultural revolution through innovative approaches is possible only if it is accepted and adopted by farmers.

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RESEARCH NEWS

Obesity and gut flora: Link revealed?

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The spreading epidemic of obesity is engulfing not just industrialized countries but developing nations as well[1]. It is also associated with other disorders like type II diabetes, cardiovascular pathology, hypertension and non-alcoholic fatty liver diseases. India is no exception to this and weight gain and obesity are beginning to pose a growing threat to the health of citizens[2]. The reasons attributed to this are industrialization, urbanization, increased living standards, change of diet and life style. But in their recent publication, Backhed *et al.*[3] make a startling revelation that our gut microbes are responsible for fat deposition in our body.

Influence of gut microbes on human health is not new, it was realized long back[4]. Their sheer number in the gut milieu speaks of their value in human health. In a normal healthy adult, there are as many as 10^{23} microbes in the gut, this is about 10 times the total number of cells present in the human body. There is increasing belief that this microbiota should be viewed equivalent to an 'organ', exquisitely tuned to carry out metabolic functions that we are unable to perform ourselves. Like any other branch of microbiology, the components of this microbiota remained poorly defined due to the limitations of being able to culture them in the laboratory. Recently developed approaches based on direct amplification and sequencing of genes like 16S rRNA estimate that total number of different species present in the gut could be 500–1000, this again is equivalent to 100 times more genes than the human genome[5].

The most prominent member of this community is a Gram-negative anaerobic organism called *Bacteroides thetaiotami-*

cron. It exists as commensal in the distal intestine of both humans and mice. Earlier analysis of global intestinal transcriptional response has shown that it modulates expression of genes involved in several important intestinal functions including nutrient absorption, mucosal barrier fortification, xenobiotic metabolism, angiogenesis and post-natal intestinal maturation[6]. Complete genome sequence of this organism is available and the proteome contains 172 glycosylhydrolases that are predicted to cleave most glycosidic linkages encountered in human diets, thus explaining the extraordinary capacity of this organism to acquire and degrade plant polysaccharides[7].

Backhed *et al.*[3] take this study further and analyse the role played by these capacities in body fat accumulation. They have done comparative studies on Germ Free (GF) and the mice colonized with conventional microflora (Conventionalized). Interestingly, they found that the mice with microbiota have as much as 42% more total body fat though they consumed 29% less diet. Moreover, if mice that were borne Germ Free were introduced, 8–10 weeks after the birth with unfractionated microbiota obtained from distal intestines of conventionally raised adults, they showed 57% increase in their total body fat.

This was associated with increase in fasting blood sugar levels and insulin-resistant state. Increase in blood sugar levels was understandable with known ability of gut microbes to degrade plant polysaccharides, but how does this result in increased body fat? They further went on to dissect the biochemical pathways and molecular mechanisms behind this intriguing observation. Since glucose and insulin induce synthesis of lipogenic enzymes in liver, they looked at activities of two key enzymes of *de novo* fatty acid synthesis, acetyl-CoA carboxylase and fatty acid synthetase. Both had increased level in mice containing gut microbes. Likewise levels of transcription factors Steroid Response Element Binding Protein I (SREBP I) and Carbohydrate Response Element Binding Protein (ChREBP) too were higher in these animals. These helix–loop–helix/leucine zipper transcription factors mediate hepatocyte lipogenic responses to glucose and insulin, and are known to act synergistically. Colonized mice had higher level of xylulose-5-phosphate, an upstream regulator of ChREBP. Direct biochemical evidence for increased monosaccharide uptake by 'Conventionalized' mice was obtained by measuring 2-deoxyglucose levels. These mice not only have an increased uptake but also higher density of blood capillaries in the intestine, thus further facilitating the transfer of absorbed monosaccharide to portal circulation.

Thus it is clear that gut microbes metabolize plant polysaccharides and induce lipogenic activity in liver. The numbers of fat cells in the fat pads of Germ Free and Conventionalized mice were same, as evidenced by DNA content, which means that increased fat content is due to growth in the size of these cells rather than number. Lipoprotein lipase (LPL) is key regulator of fatty acid release from triglycerides in liver and other organs. Its activity was 122% more in epididymal fat pads and 99% higher in heart of Conventionalized mice. It is regulated by Fasting-induced adipocyte factor (Fiaf), a member of the angiopoietin-like family of proteins and is an inhibitor of LPL. It is induced in GF animals during suckling–weaning transition and this transition does not occur in Conventionalized mice, leading to continued expression of LPL. During suckling–weaning transition, the diet changes from lactose/lipid rich milk to low fat polysaccharide rich chow. This period is also concomitant with expansion of the microbiota and shift from facultative to anaerobic flora. The authors conclude that gut microbiota regulate LPL activity by mediating expression of Fiaf, this was further confirmed in mice where Fiaf is inactivated by gene disruption (knock out). The Conventionalized knock out mice have the same body fat as their GF controls. This establishes role of Fiaf as key mediator of body fat regulation by microbiota. The microbiota stimulate hepatic triglyceride production through transcription factors such as ChREBP and promote incorporation of these in adipocytes through transcriptional suppression of LPL inhibitor, i.e. Fiaf.

These studies indicate that human and gut microbes have co-evolved a symbiotic relation and one manifestation of this is ability of microbes to process dietary components and deposit extracted energy in the form of fat. This ability of storage could be of paramount importance to ancient humans who had variable access to food. Microbiota enabled them to draw maximum energy from available diet, store extra energy in the form of fat which could be utilized when in need. But in the modern day life, where food is not scarce, this benefit becomes detrimental. The authors further speculate that changes in microbial ecology promoted by western diets and difference in the microbial ecology of individuals in these societies could be the 'environmental factors' that affect predisposition towards obesity.

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