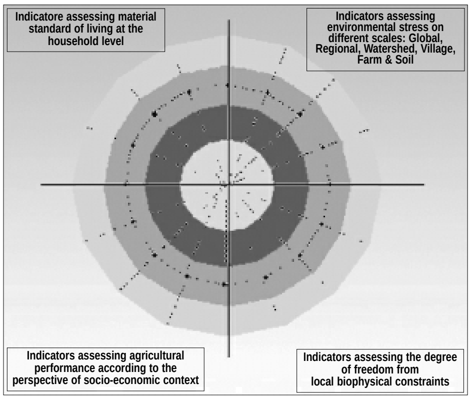
Farmer amoeba sows bacteria

Saving seeds for next year's crop is the prudent practice of some microbes, says s ananthanarayanan



THE cultivation of food is generally seen as the starting point of civilisation. When primitive humans learnt to grow food, they eliminated uncertainty, did not have to travel to forage or go in search of prey and could put down roots — which was the start of settlements, dwellings and communities. The act of cultivation was not just the technique of taking from the earth, it was an act of collecting and saving the seed, tilling the soil and investing in returns to come at the end of a season. It was a sea change from the "hunt for the next meal" way of existence and the basis of what today has become the civilisation of machines, transport, power, states and countries, conveniences and dangers.

Debra A Brock, Tracy E Douglas, David C Queller and Joan E Strassmann of the department of ecology and evolutionary biology, Rice University, Houston, report in the journal *Nature* their study of a strain of amoeba that was known to exhibit "social" behaviour and also actually cultivates and "farms" the bacteria on which it feeds!

Some form of cultivation, in the form of the dispersal of seed, providing nutrients and then harvesting is seen in some animals, notably varieties of ants and termites. The *attine* ants, a family of 200 or so species also famous as the *Leaf-Cutters*, can strip a grove of trees of every leaf in a few hours. But the ants cannot directly digest the material of the leaf; they process the leaves to become the food of a strain of fungus that fattens on the partly digested leaves and forms their actual food source. Although it is clearly a symbiotic relation, the role of the ant is one of cultivation of its food, by efforts to provide nutrients for the growth of the latter. Like the ants, there are fungus-farming termites that, unlike others of their kind, cannot digest lignin or cellulose, the main constituents of the plants on which they feed. Using roughly chewed or partly digested plant matter, the termites construct a framework, or fungus comb, on which the fungus *Termitomyces* can grow. This fungus, in turn, breaks down the vegetable matter into forms that the termites can use.

Further work on the specific fungi and termite species has revealed mechanisms by which the fungus organisms at one site can be retrieved and carried away by termites from another colony to set up their own "fungus farm". Understanding these mechanisms is seen as important in the drive to control the spread of this group of termites, which is the main cause of crop damage. More complex relations in the symbiotic termite-fungus pair have also been discovered, with ants carrying in their cuticles (the tough, outer covering of their skin) and transmitting from generation to generation a third participant, *Acyinmycete*, a bacterium of the family *Streptomyces*. *Acynomycete* produces antibiotics that target a specific parasitic fungus which would not allow *Termitomyces* to thrive.

Research carried out by Debra Brock and colleagues found that some amoebae sequester their food – particular strains of bacteria – for later use.

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The farmer amoeba

The work of the Houston group is a more direct example of farming, without the element of symbiosis. *Dictyostelium discoideum* is an *amoeba*, or single-celled animal, that has a way of communicating with others of its kind when food is scarce. In normal conditions, when food is plenty, the amoebae forage singly on bacteria, their staple food. But when food grows scarce, they secrete a signal molecule that other amoebae can sense. When an amoeba detects the signal, it begins to move towards the source and also begins to secrete the molecule, which strengthens the signal. The entire community of amoebae soon collects into a mass that displays its own structure and behaviour. The body, which is called the *slug*, has a defined front and back, responds to light and temperature and can move from place to place.

When conditions are right, the slug breaks up into groups of inactive cells, protected by a cell covering, to become new amoebae when food is available. The groups of cells are called *spores* and they are like bubbles that can disperse, and they have stalks of cellulose or dead cell material by which they are loosely connected to the main body.

The main thing in the formation of the slug, and then the spores, is that they form when the amoebae have nearly exhausted the supply of bacteria in their environment. The cells then depend on drifting to the presence of food before they can again feed and multiply.

But the remarkable thing about *D. discoideum* is that a few, about one-third, of the amoebae do not consume the bacteria to the end but preserve the last of the bacteria food in some of the spores, while the rest of the spores are bacteria-free. Thus, when conditions are right, the seed bacteria can be released from the first group of spores to grow as a fresh food source. These observations were confirmed by using light and shade variations to guide slug bodies from bacteria-rich to bacteria-free zones and then selecting spores that contained bacteria and also spores clear of bacteria and before culturing them in different conditions of food availability.

The amoeba behaviour then is one of desisting from consuming all the food bacteria at the time the supply becomes scarce to preserve this remnant till conditions are suitable and then to infect the new environment with seed bacteria, for the new crop, which thrives while nutrient for the bacteria lasts. The whole cycle is one of directed and planned prudence of holding back when conditions are bad and then planting the seeds when fertile ground is found.

In the case of the leaf-eating termite, the arrangement is one of symbiosis, where the fungus also depends on the termite; but in the case of amoebae, they are *pure predators* and perform no service for the bacteria. Specific, earlier studies have looked for and eliminated any symbiotic relationship.

A difficulty that has been raised is that if the new location is already rich in bacteria, saving bacteria and carrying the load was avoidable, and there is the question of non-farming amoebae exploiting the farmers by enjoying food security without bearing the costs. But this is not seen as a problem because the population of amoebae is structured and the benefits of farming pass on to many generations within a feeding cycle – a case of "long lived groups of kin". All members of the population, further, are seen to be the same species.

Reasoning has also been advanced for why all spores do not carry bacteria – that this would be a way of ensuring that useless or harmful bacteria are not transported in mass to the new pasture. If such were the case, it would be the farmers who were benefiting from the transport provided by the whole community, despite the negative stowaways admitted by the farmers.

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Eating right

tapan kumar maitra **prescribes a diet chart for people using toxicants and explains what they must avoid and why**

WORK with chemical means of protecting plants must be performed with great care, special attention and precision. Persons using toxicants must be able to select and properly use the means for individual protection.

The prevention of poisoning by pesticides is determined to a major extent by the strict observance of the instructions and of the rules of personal hygiene.

The toxic action of pesticides on a human depends on the state of his or her organism, therefore rational conditions of work, nutrition and recreation must be observed in the period of working with them.

Smoking is prohibited when using toxicants because it facilitates the entry of the poisonous substances into the organism. The action of toxicants on persons drinking alcoholic beverages before or during work is amplified scores of times and this is why the use of such beverages is strictly prohibited.

An important role in the prevention of poisoning is played by rational nutrition, which increases the resistance of an organism to the action of poisonous substances. The food must be rich in proteins, vitamins and should preferably contain products having enveloping properties — starch, gelatin — that diminish the irritating action of chemical compounds and prevent their being sucked in.

Food must be taken before beginning work with toxicants. The absence of food in the gastro-intestinal tract creates conditions promoting the more rapid suction of chemical substances into the blood and increasing the harm to the organism.

The toxic action of pesticides on a human depends on the state of the worker's organism. Therefore, rational conditions of work, nutrition and recreation must be observed.

The breakfast and lunch of persons working with pesticides must contain an adequate amount of liquid, not very salty food — soup, milk, stewed fruit, tea. Such food facilitates the rapid excretion of poisonous substances from the organism. It is not good to use food products retaining water in the organism — salty fish, vegetables, etc.

Persons working with organochlorine pesticides must take food rich in animal proteins — meat, cottage cheese, fish — and calcium salts and vitamin B_2 — riboflavin. Fats must be avoided because they facilitate the suction of poisonous substances into the organism.

The food ration of persons handling organophosphorus compounds must include cottage cheese, cheese, sour milk, sugar, vegetables, fruit, greens, buckwheat porridge and a large amount of vitamin C. Spicy dishes and fats must be avoided.

Persons working with copper-containing pesticides must use food products rich in proteins and vitamins — beef, porridge, vegetables, fruit, sugar and honey. Workers having to do with copper-containing pesticides must never take fats and milk and those handling zinc phosphide must not eat egg, fatty food and milk.

People working with toxicants must wash their hands and face with soap and rinse their mouth before eating. After finishing work, they must take a shower.

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The smelling test

Why are some people more sensitive to odours than others? And why do no two people experience a scent in the same way? The answer lies in our genes, writes laura spinney

IN 2004, American neuroscientists Linda Buck and Richard Axel shared a Nobel Prize for their identification of the genes that control smell, findings that they first published in the early 1990s. Their work revived interest in the mysterious workings of our noses, interest that is now generating some surprising insights – not least that each of us inhabits our own, personal olfactory world.

"When I give talks, I always say that everybody in this room smells the world with a different set of receptors, and therefore it smells different to everybody," says Andreas Keller, a geneticist working at the Rockefeller University in New York City. He also suspects that every individual has at least one odorant he/she cannot detect at all – one specific anosmia, or olfactory "blind spot", that is inherited along with his/her olfactory apparatus.

The human nose contains roughly 400 olfactory receptors, each of which responds to several odorants, and each of which is encoded by a different gene. But, says Boris Schilling, a biochemist working for Givaudan, the world's largest flavour and fragrance company based in Geneva, Switzerland, "unless you are dealing with identical twins, no two persons will have the same genetic make-up for those receptors."

The reason, according to Doron Lancet, a geneticist at the Weizmann Institute of Science in Israel, is that those genes have been accumulating mutations over evolution. This has happened in all the great apes, and one possible explanation is that smell has gradually become less important to survival, having been replaced to some extent by colour vision – as an indicator of rotten fruit, for example, or of a potentially venomous predator.

Once a deleterious mutation occurs in a certain gene, that gene ceases to produce a working receptor and becomes a "pseudogene". But Lancet says that although all people may have the same proportion of olfactory pseudogenes, each may have different ones. The result is that every individual has a different genetic "bar code" and a different combination of olfactory sensitivities.

Genetic variability is reflected in behavioural variability, as Keller, with colleague Leslie Vosshall and others, recently demonstrated when they asked 500 people to rate 66 odours for intensity and pleasantness. The responses covered the full range from intense to weak, and from pleasant to unpleasant, with most falling in the moderate range – a classic bell curve in each case. The researchers also tested people's subconscious responses to odorants by presenting them at much weaker doses. At these doses, the volunteers were not conscious of smelling anything, but they did show physiological responses to the odorants, such as an increased skin conductance due to minute increases in perspiration. "There's a surprisingly large variability in all those measures, and maybe more so in the subconscious than in the conscious measures," Keller says.

One compound that people famously perceive differently is androstenone, a substance that is produced in boars' testes and is also present in some people's perspiration. "For about 50 per cent of people androstenone is nothing," says Chuck Wysocki of the Monell Chemical Senses Center in Philadelphia. "For 35 per cent it's a very powerful stale urine smell, and for 15 per cent it's a floral, musky, woody note."

While most people enjoy the scent of roses, an unlucky few will be unable to smell it.

Androstenone is a special case, however. Of the specific anosmias that have been identified to date, most affect between one and three per cent of the population, an example being the inability to smell vanilla. In 2007, Keller, Vosshall and colleagues linked a specific anosmia for androstenone to the combination of alleles or variants a person inherits of a gene called OR7D4. It was the first such gene-behaviour link to be made in the domain of smell, but it convinced Keller that a person's olfactory blind spots can be directly linked to their genetic make-up.

At the University of Dresden in Germany, Thomas Hummel and colleagues are trying to identify other, similar links by carrying out genetic analyses in people who share a certain anosmia to find out what receptors they lack. The study, which involves 3,000 volunteers, has already revealed that, when it comes to anosmias, not all odorants are equal. "Specific anosmias are significantly related to the molecular weight of the odour," Hummel says, with anosmias becoming more common as the molecular weight of the odorant increases. He suspects that the reason is that smaller, simpler molecules are more likely to fit the binding pockets of several receptors, making them detectable even if one of those receptors isn't working. A heavier, more complicated molecule, on the other hand, might only bind to one specific receptor, and so become undetectable if that receptor's gene becomes a pseudogene.

Most of the odorant "partners" of the 400 or so olfactory receptors – the so-called "primary scents" – remain unknown, but perfumers dream of the creative possibilities, if only they knew what these were. That's because, although each receptor may bind to only one or a very few odorants, triggering an electrical signal to the brain, what the brain perceives is a result of the combination of incoming signals from all receptors. It's that combinatorial power that creates the richness of our olfactory worlds. Think what a painter can do with three primary colours and a chef with five categories of taste, and imagine what a perfumer could do with a palette of 400 primary scents.

Lancet says that the genetic tools that are now available could help researchers to solve another olfactory puzzle, too: why some people are more sensitive overall to smells than others. One in 5,000 people is born without any sense of smell at all, while at the other end of the spectrum are those individuals who have a higher than average general sensitivity, some of whom may gravitate to the perfume industry.

He suspects that the biological culprits in this case are not the olfactory receptors themselves, which are responsible for specific anosmias, but the proteins that ensure the efficient transmission of the signals elicited by those receptors to higher processing areas in the brain – transmission pathways that are shared by all receptors. "What is fascinating to me is the idea that we could discover a gene or genes that underlie this general sensitivity to odorants, so that we might be able to 'type' those professional noses and say, 'Aha, we now understand why you are in your profession,'" says Lancet.

The implications of the new research go wider than smell, however. Most of our sensation of taste comes from the odorants in food stimulating our olfactory receptors. "The wonderful enjoyment of a fresh tomato is practically only in the nose," Lancet says. Awareness of individual variation in smell has already filtered through to the wine world, launching a debate about how valuable experts' advice really is, when they may be having different smell – and hence taste – experiences from other people.

The science of smell could even throw light on patterns of human disease. It's now known that many diseases are polygenic – that is, they are the products of the cumulative, small effects of many genes, just as a person's range of olfactory sensitivities is the product of a certain combination of genes and pseudogenes. In both cases, the effect of a single mutation is minimal, and mutations spread fairly easily through a population. With monogenic diseases such as haemophilia, on the other hand, a single mutation is so disruptive that natural selection generally acts to eliminate it from the gene pool, usually by killing an affected individual before he or she reaches reproductive age.

Thanks to Buck and Axel, scientists know a lot more about the genetics of olfaction than they do about most polygenic diseases, and they are now studying the former in order to understand how the latter arise and spread through a population – an inspired piece of lateral thinking which the Nobel Prize committee may or may not have foreseen when they bestowed their honour in 2004.

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