NATURAL HISTORY MAG.
88:60-66 1979

Why Food Rots

When something spoils, microbes have won out in a contest with vertebrates for food both crave

by Daniel H. Janzen

The blissful microbe that has just found a cut in a ripe strawberry has three tasks in life: first, to convert the strawberry into more of itself, that is, to propagate and increase its population; second, to convert the strawberry into compounds that will be antibiotic to other microbes; and third, to convert the strawberry into an object of disgust to the passing bird or mammal, thereby defending its food supply against competitors. In other words, spoil the strawberry. Put in different terms, you are a youngster left alone in the kitchen for fifteen seconds before your mother returns, and there are two strawberries, one fresh and one moldy, on the counter. If you pop the fresh fruit in your mouth, the microbe has won.

Ecological tradition has it that the most intense competition for anything, including food, occurs among closely related species living in the same habitat. We assume that closely related species are likely to have similar needs. But there are cases of grossly dissimilar organisms eating the same kind of food. Perhaps the most dramatic example is the competition between microbes and animals over very concentrated food sources such as ripe fruit, caches of grain, and fresh carcasses.

According to the classic explanation, putrefaction, the so-called rotting of highly desirable foods by microbes, is the battlefield that remains after competition between different species of microbes or is a spinoff of microbial extracellular digestion, during which microbes release enzymes outside their cells and then take in the results of this chemical action. Indeed, putrefaction could be both these things; in either case I believe that it is also the result of microbes' explicit efforts to render food unattractive to higher animals.

This is not a simple area of study. One beast's poison is another beast's drink, and the ecology of rotting food consumption offers some of the very best examples: wine, penicillin, and the feeding habits of vultures. Humans call a fruit spoiled if it appears unattractive; fermented if it looks agreeable. Every human culture knows an intoxicant; drinking alcohol (ethanol) is the driving ingredient of more human drink, water and milk excepted, than any other single compound. Why do yeasts make and then discard that marvelously simple little molecule, literally excreted from yeast into fruit, that has such a strong disequilibrating effect on animals? My hypothesis is that yeasts discard the molecule as soldiers "discard" bullets. A primary adaptive reason why yeasts manufacture alcohol is to render ripe fruits distasteful or unacceptable to wild vertebrates, thereby maximizing the chance that a fruit will be ignored by the very animal for which natural selection produced the fruit in the first place.

The test of this hypothesis lies in observation of the choices wild animals make when presented with naturally rotting fruits. Unfortunately, no such observations have been published. We do know that long-term alcohol consumption, in the absence of malnutrition, can produce morphological damage to the central nervous system of mice. We also know that wild animals become stupefied, perhaps drunk, after feeding on partly spoiled fruit, as in this newspaper account from Redwood City, California (February 1978).

> Drunks are being slaughtered and maimed on the Central Expressway in Mountain View in record numbers, but city and Santa Clara County road officials say they have more important things to worry about. The official disinterest is because the drunks are birds, mainly robins. The birds are eating [fermented] pyracantha berries on bushes growing along the expressway. And after a morning or afternoon of imbibing, they fly into the path of trucks and cars or right into the sides of vehicles. There are times when dead and dying birds dot the landscape. Motorists say it is almost impossible to pass the pyracantha bushes without hitting at least one bird.

Cars are not the normal predators of drunk birds, but we can reasonably assume that a wild animal with a gut full of alcohol-rich fruits is more likely to make a mistake in the face of a predator, break a limb in a fall, or fail to find a cozy place in a storm than a sharp-eyed, if hungry, animal that has passed up a dinner of fermenting fruit.

But we don't know how often birds or other vertebrate frugivores reject spoiling or fermenting fruit in the wild or even whether animals reject rotten fruit because of the taste of ethanol or of the multitude of other nasty chemical compounds made by microbes. Pyracantha berries are very small fruits; when fermented, each one contains a very small amount of alcohol, which may be too dilute for a bird to detect. Only the cumulative effect of eating many berries intoxicates a bird. In other rotting foods, the animal may be insensitive to the taste, there may be no signal of rottenness, or there may be other cues still unfamiliar to us.

A bird's hangover from eating rotten fruit may be due to other compounds besides ethanol and could be as dangerous as the drunken state—but do other animals suffer hangovers? We do know that crops of rotting fruits often remain untouched on the ground beneath fruiting trees, apparently ignored by the animals that normally eat them, and become dispersal agents of the trees' seeds. Despite our own fondness for alcoholic beverages, we usually drink them under protected circumstances—they lower our physical fitness in many ways, and we are fussy about which rotten fruits we eat.

Humans appear to be the vultures of the world of spoiling fruits; I know of

The sight of mold on a strawberry is usually enough to turn away a hungry human, and probably many birds and animals, leaving the microbe to contend with insects and other microbes for the fruit.

Thomas Page

Bob Clay

Partly decomposed fruit does not necessarily repel birds. Every year in California, scores of birds, mainly robins, feed on fermented pyracantha berries. But. . .

no other vertebrate animal that regularly specializes in eating fermenting fruits. However, a number of insects feed on spoiling or fermenting fruits before they fall or as they lie on the ground beneath the tree. Some types of beetles and fruit flies (*Drosophila*) are the best-known examples. Resistant to alcohol, they burrow through fermenting fruits, eating the yeast. A microbe is rarely so toxic that it keeps all insects out, and besides, these insects present much less of a threat to the microbe than does a vertebrate. First, insects often just take some juices, rather than consuming the entire fruit. Second, they may be primary dispersal agents, enabling microbes to travel from rotting to uncolonized fruits. Third, their feeding activity, which makes holes and burrows in the fruit, may break down its physical structure and thereby make it a more attractive food for a microbe. The work of these insects is directly analogous to the importance of carrion-feeding insects, which break down a carcass for invading microbes. For these reasons I suspect there is great heterogeneity in the degree to which the contents of rotting fruit are toxic or repugnant to the different species of insects that feed on ripe fruit. Some microbes will repulse all; others will not discourage some. Some insects specialize in consuming microbial products; others turn away.

The function of a ripe, juicy fruit is to propagate its parent plant by enticing vertebrates that will eat it and eventually defecate, spit, or regurgitate the seeds it contains in places where new adult plants will grow. Evolution has engineered fruit color, consistency, taste, and nutrient content to be attractive, in certain amounts, to certain animals and repulsive to others. In the light of microbial consumers of ripe fruits, we have to add another dimension to the adaptive significance of a fruit's chemical traits. Theoretically, a fruit should evolve in a manner that will reduce the chances of its being so modified by microbes that it becomes unacceptable to those vertebrates that raise the parent plant species' chances of survival by dispersing seeds to appropriate places. Why do many juicy fruits have a tough skin? Why do grapes have all that tannin in their skins? Many ripe wild fruits are astringent or rich in other antimicrobial compounds. Until the 1960s, a major source of vegetable tannin for India's leather trade was the ripe fruit of *Terminalia chebula*, an Indian tree whose fallen fruits are eaten by deer, porcupines, squirrels, and other animals. High citric acid content, which renders many ripe wild fruits sour, is probably of similar biological significance. In short, odd flavors may discourage microbes but not all members of a plant's disperser coterie.

. . . once the berries are digested, the microbes strike back. This robin is one of many inebriated birds that are killed when they fly into the path of, or collide with, oncoming vehicles.

Bob Clay

The natural food of black bread mold is not old slices of bread. *Aspergillus*, *Penicillium*, *Alternaria*, *Fusarium*, and the other genera of fungi that grow so readily on stored grain and grain products are likely professional colonists of rodents' grain caches and of ungerminated seeds and fruits in the wild. In harvesting both kinds of resource, these fungi are in intense competition with granivorous birds, rodents, and insects; not surprisingly, they are the only known species of fungi whose hyphae, a kind of rootlike structure, produce compounds that are extremely toxic to higher animals.

Aflatoxins are the compounds that receive the most attention in contemporary nutritional literature. Made by many species of *Aspergillus* and other grain-inhabiting fungi, they are among the most potent carcinogens known. Only 15 parts per billion of aflatoxin B_1 in rat diets produces a high incidence of liver tumors. A major problem for turkey farmers is contamination of feed by aflatoxin-producing fungi. Peanut farmers find that unharvested crops left in the ground for more than a month run a high risk of aflatoxin contamination. However, fungi that inhabit grain make many other objectionable compounds. Ergotism, also known as Saint Vitus' dance, is caused by alkaloids from *Claviceps purpurea* on field grains. During World War II, grains left standing in fields in central Europe over a moist winter were harvested with disastrous results for livestock and human beings; the grains had been poisoned by cladosporin and fusarigenin produced by seven species of grain-inhabiting *Fusarium* fungi. The toxins were still in the grain seven years later. From a 1976 Costa Rican newspaper comes the following report:

> More than 29,000 tons of contaminated corn were finally incinerated this week, after a long and hopeless battle to use it productively. The corn was first purchased in 1974 by the National Production Council (CNP), and upon discovery that it contained dangerous toxins, alternate uses other than sale to the public were sought. After last attempts to resell it to another country, the decision to burn the corn was finally reached several months ago.

Pharmacological literature abounds with grim tales of spoilage fungi:

> Moldy [with *Penicillium rubrum*] field corn was shelled and fed to five pigs, each weighing approximately forty pounds. Two died within four days, showing typical manifestations of the field toxicosis. The remaining three pigs refused to eat the moldy corn and subsequently were sacrificed; necropsy indicated starvation. . . . Two additional pigs were force-fed, by stomach tube, milled moldy field corn. One perished in three days, and the other in eight days; necropsy findings were indicative of the acute field toxicosis. . . . A 280-pound calf was force-fed daily six pounds of milled moldy field corn. The animal developed depression, weakness, excessive salivation, and became bloated. In addition, the animal ran short distances, would twist and turn, and work its mouth vigorously during the exertion. The animal perished on the third day during one of the seizures.

Small wonder that many farm animals will starve to death before eating moldy feed or grain. Of course, not all molds on grains produce poisonous compounds. In a test of 247 cultures of 63 *Aspergillus* species on chicks and mice, 74 cultures killed the chicks or mice and 52 more stunted the growth of one or both animals when 50 percent of the diet was moldy wheat or soybeans.

Although grain-consuming fungi make complex compounds that are very toxic to vertebrates, most bacteria, fungi, and algae are insensitive to these poisons. Therefore, these compounds probably did not evolve as a weapon in the warfare that fungi and microbes wage over high-quality foods

such as grain. Nevertheless, even potent antimicrobial chemicals may be valuable to their parent fungus partly because of their effects on vertebrates. To digest food, most vertebrates require aid from microflora dwelling in their guts. Hence, a potent antibiotic may be just as dangerous to a vertebrate as a compound that attacks the animal's more personal, cellular physiology. Not only compounds such as citreoviridin but also penicillin made by *Penicillium* may protect the fungus's food. A guinea pig will die if fed too much tetracycline.

In nature, a rodent cannot store a grain cache in the ground or in a rotting log without risking its contamination by spores of toxin-producing fungi. To protect its investment, the animal depends on cold or dry weather. Desert rodents probably make well-drained burrows, as much to keep their cached seeds dry and, therefore, mold free, as to keep the seeds from germinating. In the wet tropics, very few rodents make seed caches. When they do, they lose many to fungi; they often cache only large single seeds that have coats impenetrable or otherwise resistant to fungi during the normal length of time when seeds are cached. Another way in which rodents could protect caches would be to develop their own resistance to the metabolites that grain-inhabiting fungi manufacture. Compared with other animals, mice are extremely resistant to aflatoxins, although they are by no means immune. Their sensitivity to corn moldy with *Penicillium* varies widely, and natural selection could change this variation in habitats where mice have regular access to grain stores. Of the few birds that have been tested, the highly granivorous quail are the least susceptible to aflatoxins. They certainly must consume some moldy seeds in nature.

The bacterium that has found a dead mouse has much the same problem as does the yeast with its plum or a fungus with its grain cache. Putrefaction is no simple biochemical accident. The more rapidly and intensively a bacterial clone can fill a carcass with noxious compounds, the less chance that a vertebrate carnivore will take it away. The bacterial game is certainly played with compounds called amines, perhaps with other substances as well. Bacteria inside a carcass are presented with a great deal of protein. Proteins are long chains of amino acids; an amino acid with its carboxyl (–COOH) group (which makes it an acid) stripped off is an amine. Bacteria produce amines in great quantity but apparently do not use them for anything in their own metabolism. I would interpret amines as possible preemptive toxicants to vertebrates and as warning signals that there are other, less volatile toxicants in the carcass. Putrescine and cadaverine are well-known examples of the former; isoamyline and p-hydroxyphenylethylamine are examples of the latter.

There is another reason why the advertised presence of bacteria in a carcass may keep most vetebrates away. Diseases such as salmonellosis, botulism, and avian cholera that may have killed the animal could still infect carnivores that devour the carcass.

The system I have postulated above calls for the evolution of vertebrates able to effectively recognize either microbial toxins or signs of dangerous bacteria in carcasses. For example, human revulsion to microbial presence, either natural or learned, is strong. When single cell protein (bacterial cell protein) is added to human food in amounts greater than about 15–25 grams per day, it generates gastrointestinal upsets. On the other hand, I would expect certain animals to become adept at recognizing physiologically acceptable bacterial levels even when the bacteria are generating some pretty fierce signals. Some of the more fragrant cheeses are a familiar example; the animal registers the bacteria's presence, but realizes that they are not producing too much poison.

Given that bacteria frequently win their contests with ordinary carnivores for food supplies, a few exceptional carnivores have specialized in capitalizing on microbe-infected carcasses, making them their major source of food. Vultures, crows, marabou storks, hyenas, and carrion-feeding insects come immediately to mind. To feed on carrion, these animals must have an incredible gut chemistry, but it remains unstudied. Olfactory preferences are not the physiological mystery of prime interest when a hyena pulls a putrefied eland carcass out of the water and makes a meal. On the other hand, why don't the putrefaction bacteria come up with some really potent aflatoxinlike compounds that will bequeath the carcass completely to the microbial world? I suspect the answer lies in the importance of scavengers in moving putrefaction bacteria around in the world and in the importance of carrion-feeding insects in mechanically breaking down carcasses, thereby helping microbes to enter. If carcasses are protected from all multicellular consumers by fine screen cages, they decompose slowly and form mummies even in July-August temperatures in midwestern latitudes.

There are numerous times when humans—and, I suspect, other animals—hold their noses or put on spices and take a bite, either because the particular spoiled food is known to contain harmless mimic microbes, because humans are immune to that particular toxin, because the concentration of toxins is low, or because intoxication is desired. Ignoring signals of the remains of the microbes' presence can backfire at New Year's Eve parties or other occasions where too much booze or cheese is consumed or when unexpected microbes get into the brew; for example, *Clostridium botulinum* is a lethal contaminant of Japanese izushi, a fermented fish and vegetable dish. A lot of drunks and antibiotics users are happy that microbes fight back and produce useful materials, but the battleground is littered with chemical booby traps.

When an animal eats putrefied food of any kind, it usually risks poor nutrition, microbial infection, or injury from toxins or microbe-produced antibiotics. Of course, all these consequences may simply be the byproduct of microbes interacting with each other. But I suspect that microbes putrefy food to avoid the generally maladaptive event of having themselves and their resources eaten by larger animals. Fruits, seeds, and meat spoil because that is the way microbes compete with bigger organisms for food. □

Hans D. Dossenbach

There are a few exceptional animals whose intestinal chemistry is mysteriously resistant to putrefaction microbes, so that they can feed on rotting flesh. At right, hyenas and vultures divide spoils.

By eating microbe-infested carcasses and excreting the remains, vultures and other scavengers play an important role in distributing putrefaction microbes around their habitat.

Fran Allan, Animals Animals