Ancient biochemical fossil detected

The earliest forms of life, in all likelihood, were single-celled, prokaryotic (lacking a true nucleus) organisms. Their delicate remains, unable to withstand biologic and geologic destruction, often are not preserved as fossils. But they have left other traces. The ancient organic molecules that once comprised an organism can be detected in sedimentary rock. Called biochemical fossils, they allow researchers to affix a date to those first organisms.

Now, two researchers report in the January PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES potential evidence for a 2.7-billion-year-old biochemical fossil, making it the oldest known chemical evidence of life. Earlier work showed evidence for a 2.3-billion-year-old fossil (SN: 7/5/75, p. 7). University of Arizona's Bartholomew Nagy and Deborah S. Sklarew, now at Battelle Pacific Northwest Laboratories, have found traces of a compound, 2,5-dimethylfuran, which they believe to be a remnant of an ancient sugar, in rock samples from outcroppings of the well-preserved Rupemba-Belingwe stromatolites in Rhodesia. (Stromatolites are laminated sedimentary rocks formed with the help of blue-green algae and bacteria.)

The uncertainties of identifying a biochemical fossil are many, but Nagy and Sklarew are fairly certain they have the real thing. For instance, organic molecules have been detected in 3.4- and 3.0-billion-year-old rocks (SN: 11/8/75, p. 302). But the compounds were found in soluble portions of the rock, indicating they might be modern contaminants picked up from groundwater or introduced by laboratory procedure. The 2,5-dimethylfuran, however, was detected in an insoluble fraction of the rock, a coal-like substance called kerogen. Further experiments on the permeability of the kerogen convinced the researchers that the furan compound could not be a contaminant and must have been deposited as the rock formed.

Another problem is ascertaining that the compound does have a biological source. Furan compounds can arise from organic as well as inorganic reactions. Were inorganic reactions responsible, a variety of dimethylated furans in addition to the 2,5-dimethyl form would have been produced. Organic reactions involving 5- and 6-carbon sugars, on the other hand, yield only the 2,5-dimethyl form. Because that was the only form present, the researchers conclude the compound must have an organic origin. □

The case of the mother who was twins

If it had been the father whose genes did not show up in the children, there might have been grounds for divorce. But as it was the mother who did not seem to fit into the family tree, Austrian scientists were perplexed.

The puzzle arose when the fourth child of a woman of blood type A_2B was found to have blood type O. By basic genetic rules all offspring of an AB parent must be type A, B or AB. The woman's physician consulted specialists who examined more than 20 genetic markers in blood and skin samples from the family. Each of the four children had markers that they had not inherited from either their mother or father and that would logically exclude the woman as their parent.

A hospital mix-up of each of the four babies seemed unlikely, and the obstetric staff where the children were born convinced investigators that there had been no such exchanges. So someone else's genes clearly had infiltrated the family, and it appeared to be an inside job.

The researchers called in the woman's parents. In samples of their blood were all those genetic markers that must have been transmitted by the woman (but which were not detectable in her blood). Thus, a sister of the woman could be imagined who would have the appropriate genes. But the mother had no twin sister with whom she might have swapped some cells.

The solution the scientists propose is that the mother is herself non-identical female twins, or rather a genetic mosaic. Her parents had produced two fertilized eggs, as in the case of fraternal twins, but early in development the embryos merged to develop into just one body. Cells derived from one fertilized egg ended up making the woman's blood and skin cells, while cells derived from the other eventually produced her ova.

In laboratory experiments, biologists deliberately create animals comprised of two genetic types (SN: 2/12/77, p. 107; 11/18/78, p. 344; 1/27/79, p. 60); such "chimeras" also occur occasionally in nature. Among people, chimeras arise from the exchange of blood-forming cells between non-identical twins in the uterus. Human chimeras resulting from fusion of early embryos have been identified by sex abnormalities and mixed blood cell populations. The woman in Vienna is the first case where the cell mixture is detectable only by analyzing offspring.

In their report in the Jan. 18 NATURE, W.R. Mayr, V. Pausch and W. Schnedl of the University of Vienna conclude, "Such individuals [chimeras whose blood and skin differ genetically from their eggs or sperm] must be extremely rare, but the practical implications of this observation for problems of parentage are clear." Blood and tissue typing are powerful techniques for establishing — or denying — parentage. □

Alcohol: Stalking the real villain

The loose tongues and rubbery knees that accompany overimbibing are well-known. But the exact mechanisms that cause alcohol intoxication have yet to be pinpointed. Scientific theory recently has leaned toward the alcohol metabolite, acetaldehyde, as the primary instrument of intoxication (SN: 4/19/75, p. 257).

A new set of results at Texas A & M University, however, suggests that alcohol itself, rather than its by-product, is the chemical culprit. This means that "any future research on intoxication — such as attempts to develop a [preventive or] 'sober up' remedy, need to be directed at alcohol, not acetaldehyde," says biologist William R. Klemm.

Klemm and Joseph A. Mikeska measured the brain waves of 29 rats, including a subgroup that received acetaldehyde at a steady, continuous infusion rate comparable to acetaldehyde buildup during drinking. The few EEG-acetaldehyde studies done previously indicated intoxication effects in the form of a characteristic "deactivated" EEG. But those studies involved large, single doses of the metabolite that did not reflect the true ingestion rates found in drinking, says Klemm.

In their own work with lower level, continuous infusion, Klemm and Mikeska report in the Jan. 19 SCIENCE that EEG was unaffected, even when "concentrations of acetaldehyde in the blood were equal to or greater than those that occur during intoxication. It appears that acetaldehydalone, in concentrations that are similar to those found in an ethanol-intoxicated rat, is not adequate " to produce intoxication as reflected by brain wave changes, they say.

Acetaldehyde, however, may not be totally benign in the drinking process, Klemm notes. The metabolite is believed to interact with brain neurotransmitters (primarily catecholamines) and may be involved in the long-term processes of addiction and alcoholism. But in intoxication, Klemm says, alcohol seems to be the key.

Since it takes time for the human body to convert alcohol to acetaldehyde, both substances are present simultaneously in a drinking person, Klemm says. If his animal results are correct, this means alcohol, before its conversion, is responsible for intoxication.

Because alcohol is known to dissolve through cell membranes, Klemm suggested in an interview that intoxication may result from this dissolving process, primarily in nerve cells. Therefore, the key to any hypothetical anti-intoxicant might lie in its ability to prevent or reverse this dissolving process, Klemm said. "Not much research has been done on stopping intoxication," he says. "If I get any clever ideas, I'll pursue it." □