Smoke toxicity debate rekindled

The vast majority of the 8,000 fire fatalities in the United States each year are due not to burns, but rather to the inhalation of smoke. Carbon monoxide (CO), a combustion product of wood and most other materials, long has been considered the primary toxic threat in such smoke. In recent years, however, fire safety officials and industry representatives have intensely debated whether the increased use of plastic and other synthetic building materials — which emit hydrogen cyanide and many other toxic gases in addition to CO when burning — heighten the danger of fires.

Now, a recently released report on combustion toxicity — prepared for New York state by Arthur D. Little, Inc., a consulting firm in Cambridge, Mass. — is expected to spur the next round in this heated debate. The report states that technology exists to identify toxic gases emitted when building and furnishing materials burn and recommends that the state adopt regulations requiring manufacturers to submit such combustion toxicity data to a designated agency. The report is particularly noteworthy in that it follows on the heels of a National Institute of Building Sciences report that reached the opposite conclusion — specifically, that available tests for combustion product toxicity are inadequate and therefore should not yet be incorporated into codes or regulations. Fire safety officials charged that the Washington, D.C.-based NIBS — which released its report in preliminary form last summer and in final form last month — was pressured by plastics industry representatives into reaching that conclusion (SN: 8/7/82, p. 86).

It was a string of highly publicized hotel fires — in the last few years — that brought burning plastics under close scrutiny. In the wake of those fires, NIBS, which was created by Congress in 1974 to organize and improve the heterogeneous network of U.S. building codes, was asked to examine whether combustion toxicity tests could be incorporated into building codes. And, at about the same time, the New York legislature contracted Arthur D. Little to evaluate the various published methods for testing combustion product toxicity and to determine whether they could be incorporated into any type of regulation.

The Arthur D. Little study, conducted by Rosalind C. Anderson and colleagues, concluded that the most useful test is one developed by Yves Alarie and Anderson when she was at the University of Pittsburgh. (Anderson told SCIENCE NEWS that she saw no conflict of interest in recommending a test that she helped design. Anderson said she also helped develop another combustion toxicity test that was rejected in the Arthur D. Little study.) In the Pittsburgh test, mice are placed in special chambers where they are exposed to smoke from burning test materials. The test is designed to indicate an LC_{50} — the weight of a test sample that is lethal to 50 percent of exposed animals. Using the Pittsburgh test, the LC_{50} of Douglas fir, for example, is 31 grams, while that of polytetrafluoroethylene (Teflon)-coated wire is 3 grams.

Anderson and colleagues recommend that manufacturers submit such LC_{50} data to a state agency, where they would be accessible to architects, engineers and the general public.

Not surprisingly, the Arthur D. Little study has met with mixed reviews. Gordon Vickery of the Foundation for Fire Safety in Seattle, Wash., says Anderson and associates "are to be complimented." G. R. Munger, president of the Society of Plastics Industry, on the other hand, says that because the Pittsburgh test does not take into account several fire safety factors such as resistance to ignition, flame spreadability and extinguishability, it could give negative marks to materials that have good overall fire safety qualities.

The New York state legislature now must decide whether to adopt laws to implement the recommendations of the Arthur D. Little study. The study also could play a role in four other state legislatures considering similar laws and in congressional fire safety hearings scheduled for July. —*L. Garmon*

Scanning the receptors in human brains

Keener observation of living brain chemistry from outside the skull is becoming possible. Scientists now report a method to visualize in the human brain, by a non-invasive means, receptors implicated in a variety of neuropsychiatric disorders, including Parkinson's disease.

For several years positron emission tomography (PET) has been used to measure energy consumption, and therefore general activity, of nerve cells (SN: 1/31/81, p. 76). In the technique, a scanning device detects within the body specially prepared chemicals, injected or inhaled, containing radioactive atoms that emit positrons.

The new work, tested on one human volunteer, employs a radioactive derivative of an antipsychotic drug. This chemical binds to receptors for a nerve signal chemical, dopamine, in the brain. Michael Kuhar of Johns Hopkins Medical Institutions in Baltimore says the newfound ability to label receptors is "an important turning point,"enabling scientists to determine more specific characteristics of intact cells.

At a conference this week at the National Institutes of Health in Bethesda, Md., Kuhar and Henry Wagner reported that the drug derivative called ^{11}C-*N*-methyl spiperone concentrated in those areas of the human subject's brain known to contain nerve endings that release dopamine. These areas are the caudate and putamen of the basal ganglia.

In earlier experiments with baboons, Kuhar and colleagues also observed preferential accumulation of spiperone in the basal ganglia and demonstrated that the accumulation resulted from specific binding to receptors. This binding, as measured, reflects both the number of receptors and their affinity for the radioactive material. Kuhar and colleagues plan soon to examine with PET the receptors of patients with Parkinson's disease, who are thought to have a deficit in dopamine receptors.

At the NIH meeting, Arnold M. Friedman of the University of Chicago and Michael Welch of Washington University School of Medicine in St. Louis also reported animal experiments showing specific binding of spiperone derivatives to brain areas containing dopamine receptors. "We are just about to put it into a human," Welch says.

Dopamine receptors are not the only brain sites being targeted with the PET technique. J. James Frost of Johns Hopkins is attempting to use the method to study the sites that bind opiate-like substances. He and colleagues have made positron-emitting analogs of two substances, called diprenorphine and lofentanil, that bind to opiate receptors. Frost says, "We will soon carry out *in vivo* experiments in baboons and humans." —*J. A. Miller*

Study links coffee to high cholesterol

A new study reports that heavy coffee drinking increases serum cholesterol and may provide a link between coffee consumption and increased risk of heart disease. Researchers Dag S. Thelle, Egil Arnesen and Olav H. Førde of the University of Tromsø in Norway examined the relationship between coffee consumption and levels of serum cholesterol, high density lipoprotein cholesterol and triglycerides in more than 14,500 men and women. After subtracting for the effects of other factors that might cause high cholesterol, such as age, weight, amount of exercise, smoking and alcohol consumption, the researchers revealed an "unexpected finding" — the more coffee the subjects drank, the higher their serum cholesterol levels, the researchers report in the June 16 NEW ENGLAND JOURNAL OF MEDICINE.

For those who drank one to four cups per day, serum cholesterol was more than 5 percent higher than for non-coffee drinkers. And "heavy" drinkers, subjects drinking nine or more cups per day, had serum cholesterol levels 11 percent or more higher than non-coffee drinkers. The

other two variables—HDL cholesterol and triglycerides — did not show any strong trends after adjustment.

Although the researchers indicate that this study suggests "at least a two-fold increase [among heavy coffee drinkers] in the risk of coronary heart disease," a spokesman for the National Coffee Association said "there is nothing in the current report that would substantiate [this]. The researchers focused on cholesterol, not heart disease," he added, "and failed to consider other factors known to affect cholesterol levels, such as dietary intake of saturated fat."

Basil M. Rifkind, chief of the lipid metabolism and atherogenesis branch of the National Heart, Lung and Blood Institute in Bethesda, Md., agrees that dietary fat intake should not be overlooked. He adds that "relationships of the magnitude that this group was reporting came as somewhat of a surprise" to him. "If coffee is raising cholesterol by that amount," he said, "one would expect it to show through as a definite risk factor for heart disease," and this has not been the case in previous studies. Still, he says, "this is a good group of investigators," and their findings of an association between coffee and cholesterol should not be taken lightly. Rifkind's group conducted a study of heart disease risk factors in the early 1970s, in which dietary information, such as coffee consumption, was recorded but not evaluated. Now he plans to re-evaluate these data to see if he can verify the coffee-cholesterol connection. *—P. Taulbee*

Schizophrenia clues in angel dust

The street drug called "angel dust," or PCP, is known to cause bizarre behavioral changes strikingly like those seen in schizophrenic psychosis—hallucinations, aggression coupled with extraordinary physical strength, catatonic rigidity. As a result PCP "psychosis" has for some time been used as an animal model in the study of schizophrenia. This week two Baltimore scientists announced that they have identified the pathway through which PCP affects the brain — a finding that could shed light on the brain abnormality underlying the severest of psychiatric disorders.

According to physiologist Mordecai Blaustein and pharmacologist Edson Albuquerque of the University of Maryland School of Medicine, PCP (short for phencyclidine) achieves its effects by altering the chemical activity in the membrane of neurons — a change that in turn prolongs chemical communication among cells and presumably distorts normal information processing. Specifically, they found that PCP alters brain activity by binding to a poorly understood molecule in the membrane called the potassium ion channel.

Under normal circumstances, Blaustein told SCIENCE NEWS, a resting neuron contains relatively more potassium and less sodium than there is in the surrounding blood or cerebrospinal fluid. When the nerve is excited, the positively charged sodium leaks in through pores, called sodium ion channels, creating an "action potential" in the cell; this charge travels to the terminal, where it causes calcium to leak in and, in effect, order the release of chemical messengers. Finally, the positively charged potassium ions exit through their own channels, restoring normal charge to the cell and ending neurotransmission.

PCP binds to the potassium channel and blocks the normal flow of potassium, Blaustein says, and as a result it prevents the normal cessation of chemical firing into the synapse. Blaustein's results come from test tube experiments on rat brain tissue, but importantly, he notes, only PCP and chemical analogs that have demonstrable behavioral effects in living animals were found to block the potassium channels. Compounds with chemical structures very similar to PCP that do not affect behavior also failed to alter membrane activity or synaptic transmission.

Albuquerque had previously demonstrated that PCP binds to potassium channels in muscle-firing cells (a finding that could explain the great physical strength shown by those under the influence of angel dust), but this is the first time the same physiological effect has been seen in brain tissue. In addition, Blaustein says, they have found a way to radioactively label the potassium channels, which could make it possible to characterize the nature of the molecule.

The connection to schizophrenia is entirely speculative right now, Blaustein emphasizes, but the link between identifiable psychotic behaviors and a specific physiological abnormality could provide a useful lead. Schizophrenia is presumed to be related to abnormal neurotransmission, but it remains unclear which neurochemicals are involved. Blocked potassium channels, he says, could account for several synaptic abnormalities. *—W. Herbert*

Deciphering the history of ocean crust

Along the undersea ridge of the East Pacific Rise, which slashes like a gigantic wound along the seafloor, molten rock pushes up from the mantle to create ocean crust. The ridge is peppered with vents streaming heated waters laden with minerals leached from crustal rocks. That this hydrothermal process occurs today is clear; details of its history are not. Thus, when the drillship *Glomar Challenger* pulled out of Tahiti for its 92nd cruise in February as part of the Deep Sea Drilling Project, the scientists on board had several questions in mind.

One question is, over time, have there been changes in the rate of accumulation and composition of sediments altered by hydrothermal fluids? If they have changed, "that has very profound implications for the chemical history of the oceans," says Margaret Leinen of the University of Rhode Island in Kingston. She and David K. Rea of the University of Michigan in Ann Arbor were co-chief scientists of the cruise.

It is believed that every speck of ocean water circulates through the world-wide system of spreading centers every 10 million years or less, and that the rate of circulation and the accumulation of hydrothermal sediments varies directly with the spreading rate. The faster the crust spreads apart, the more cracking and fracturing occurs, allowing the seawater to percolate more rapidly through the crust. As more water circulates close to the buried cauldron of magma and back to the seafloor surface, more hydrothermal sediment is generated. Faster spreading would affect ocean chemistry because the reactions that take place when hydrothermal fluids react with basalt — molten material from the upper mantle — affect the ratios of many constituents of ocean water, including major ones such as magnesium and calcium.

During the cruise the *Challenger* drilled 19 holes into six sites in a line so that the ages of crust drilled increased with distance from the ridge. This path enabled the scientists to study how crust generated at the same location on the EPR varied with time. The drillholes spanned crust from 4.7 to 28.6 million years old. The researchers found that the sedimentation rates were higher than today's between five and eight million years ago, and again between 20 and 24 million years ago, but there are not yet enough data to allow conclusive answers about the past changes in ocean chemistry.

The researchers hope that the variety of experiments performed will reveal information about the spectrum of reactions at the vents. What controls the degree of alteration that takes place? What sort of solutions come out, and does this circulation continue even after the fractured crust has moved away from the spreading center?

Some answers may be provided by an experiment conducted at a hole drilled on a previous DSDP cruise. During the recent trip the *Challenger* returned to hole 504B near the Galapagos Islands, and a seismometer was emplaced in the hole. Seismic charges were set off nearby. The paths of the seismic waves may reveal the pattern and depth of cracking in the crust, indicating whether the hydrothermal activity goes on continually. Preliminary studies suggest that in the sea floor around the drillhole, the amount of fracturing varies greatly from one location to another.

—C. Simon