would consist not only of the hepatitis B surface antigen but of a virus core protein. At the recent meeting in San Francisco of the First International Congress for Recombinant dna Research William J. Rutter of the University of California at San Francisco described such an approach. He and his colleagues put two viral genes in tandem into a plasmid. One gene coded for the hepatitis B surface antigen and the other for a molecule closely related to a protein found in the viral core. To turn on operation of these genes, Rutter included in the plasmid a regulatory stretch of DNA called the trp-promoter. It can direct the bacterial cell to devote most of its protein synthesis to the viral genes. Rutter reports bacterial production of "significant quantities" of both the hepatitis B surface antigen and the core protein, as opposed to low levels of hepatitis B surface antigen purified from virus particles taken from hepatitis B carriers or as previously made in bacteria with recombinant DNA techniques (SN: 5/26/79, p. 344). Because both the hepatitis B surface antigen and the virus core protein have been detected in hepatitis patients, Rutter hypothesizes that a two-protein vaccine might be more effective than one made only of hepatitis B surface antigen.

Insights into hepatitis B virus's role in causing liver cancer are emerging from the lab of William S. Robinson of Stanford University Medical School. At the recent meeting in Dallas of the American Society for Microbiology Robinson reported that liver cancer is 300 times more prevalent among persons persistently infected with hepatitis B virus than among control subjects. Other researchers have found that hepatitis B viral DNA is integrated into the DNA of cancerous liver cells but not into the DNA of noncancerous liver cells (SN: 8/6/80, p. 102). Although both lines of research suggest that hepatitis B virus can cause liver cancer, Robinson points out that a viral cancer-causing gene per se may not be responsible for turning a liver cell into a cancer cell. One reason to think this is not the case is that while a cancerous liver cell's DNA contains all of the hepatitis B viral DNA, and while all of the viral DNA is transcribed into RNA, only some of the RNA is translated into proteins, notably the hepatitis B surface antigen; yet this selective pattern of gene translation has also been observed in some noncancerous liver cells. Instead, the hepatitis B virus may turn a liver cell into a cancer cell by a nonspecific mechanism, Robinson speculates. For instance, because the viral DNA is integrated into the cellular DNA, it might interrupt cellular DNA functions, and this interruption in turn might be what makes the cell cancer-

"So I'm not at all sure," Robinson told SCIENCE NEWS, "that there is a gene in this virus that is directly responsible for changing cell function in a sense that we call [cancerous] transformation."

Coffee and cancer: A brewing concern

People who drink coffee may be at double or triple the risk of developing pancreatic cancer, a rare but lethal disease, according to epidemiologists at the Harvard School of Public Health. If their "unexpected" finding is confirmed, and "[i]f the distribution of coffee consumption in our control group reflects that in the general population," the Harvard group says, we estimate the proportion of pancreatic cancer that is potentially attributable to coffee consumption to be slightly more than 50 percent." But before banning the brew, remember that the jury is still out. Even Brian MacMahon and his Harvard colleagues point out that other data must be evaluated "before serious consideration is given to the possibility of a causal relation" between coffee and cancer.

MacMahon's project, described in the March 12 New England Journal of MEDICINE, was actually designed to reexamine the incidence of pancreatic cancer in relation to its victims' smoking habits and to explore whether alcohol consumption plays a confounding role. The survey involved 369 pancreatic-cancer patients and a "control" group of 644 hospitalized patients with other ailments. Each was asked about smoking habits. Questions also probed the frequency with which each consumed alcoholic beverages prior to onset of the disease, the age span over which they drank and the beverage they consumed most often. Data about tea and coffee habits were limited to the typical number of cups consumed before onset of their current disease became evident.

The researchers observed no link between pancreatic cancer and use of alcohol, tea, pipe tobacco or cigars. There appeared to be a "weak positive association" between cigarette smoking and the disease—also noted in at least two earlier studies—though "only the data for women showed a significant dose-response relation." Coffee consumption was another matter.

Coffee-drinking men — regardless of how much they drank — showed a "flat" but statistically significant excess risk of pancreatic cancer over men who avoided the brew. But among women coffee drinkers, pancreatic-cancer risk increased in proportion to how much coffee they consumed. Combining both sexes, the pancreatic-cancer risk (adjusted for age and sex) showed no elevation for non-coffee drinkers, a doubling (2.1) for those drinking a cup or two daily and more than a three-fold increase for those downing at least five cups a day. MacMahon plans a follow-up study hoping to confirm or rebut these findings.

Another recent epidemiological study (SN: 1/31/81, p. 71), this one by Irving Kessler and Ruey Lin at the University of

Maryland, also suggests that "one or more constituents (or contaminants) of coffee may contribute to the risk of pancreatic cancer." Because so many pancreatic-cancer patients in their study drank decaffeinated coffee, solvents such as the animal carcinogen trichloroethylene — used in decaffeinating coffee — "come immediately to mind," the researchers said. But many other factors were also linked with the cancer.

What does all this mean to the hard-core coffee addict? "Prognosis of [pancreatic] cancer is very unfavorable and therefore even a preliminary finding such as that of Dr. MacMahon's study arouses great concern," says a National Cancer Institute announcement. In fact, pancreatic cancer accounts for about 20,000 deaths in the United States annually, more than any other except colorectal, lung and breast cancers. However, NCI adds, "caution should be exercised regarding overreacting to a preliminary finding until results of further studies are reported."

But Kessler offers a further note of caution in interpreting the Harvard group's findings. After looking at a "whole constellation" of risk factors, he concludes that "there is substantial evidence" for believing that most human pancreatic cancers result from no one single factor—such as coffee drinking—but rather from a synergistic host of interacting factors.

Metal drops that dig into graphite

Chemical reactions go faster with catalysis. That arouses the interest of physical chemists, who want to know how the catalyst speeds things up. It also attracts the interest of industrialists who see more efficient industrial processes in prospect. So as Reese Terrence Keith Baker of the Exxon Research and Engineering Co. in Linden, N.J., pointed out at the meeting this week in Phoenix of the American Physical Society, a study of the effects of metals in increasing the reaction rate of gasification of graphite can have a number of purposes. Such catalytic reaction rates may determine how long graphite structures will last, particularly in an oxygen-aiding atmosphere. Knowledge of such rates could aid development of better ways of removing graphite from places where it collects during coking and other such processes. Finally, the work may help in finding the best catalyst for gasification of coal. Gasification is often said to be the best way to make a nonpolluting fuel from coal.

In the course of their work, Baker and his co-workers have developed a tentative model for the mechanism of graphite gasification catalysis, which is always a most fascinating physical chemical point, and they have found that under the conditions of the experiment the solid metals

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act somewhat like liquids. That is, they "wet" the graphite surface — they spread themselves in a thin film over it. Wetting could promote highly efficient catalysis, and if it does, it could be important in devising industrial processes that demand swift completion of wholesale lots.

The experiments are done in a special cell placed inside an electron microscope so that a continuous video record of the process can be made. In the cell metal pieces of about 100 angstroms in size are placed on the surface of the graphite. The atmosphere in the cell is the gas appropriate to either an oxidizing or a reducing reaction, that is, either oxygen or hydrogen. The temperature is near 1,000° C. This is called controlled atmosphere electron microscopy (CAEM).

There are two kinds of catalytic processes: pitting and channeling. Pitting goes perpendicular to the basal plane of the graphite crystal. A metal particle happens to lie on the surface at some defect in the crystal. The metal chews its way down, excavating a hexagonal pit that gradually turns circular. Channeling goes in from the side, making a tunnel through the crystal parallel to the basal plane. Channeling is the faster, and one aim of the research is to see how to encourage it. Here, too, the wetting phenomenon can be observed especially strikingly. Baker showed films in which the metal particles wet their tunnels as they went.

Baker stresses that this liquid-like behavior occurs although the temperature in the chamber is well below the bulk melting point of the metal in question. It is not a well understood kind of physical behavior. Baker suggests that a search for good catalysts for graphite or coal gasification might use their behavior on the graphite or coal surface — whether they wet might be a criterion.

There are two kinds of gasification reaction under study here: one, oxygen plus graphite yields oxides of carbon; two, hydrogen plus graphite yields methane. Comparison showed that the two reactions go at different rates with the same catalyst. Reducing reaction, the one with hydrogen, particularly seems to go at a rate that depends on the size of the catalyst particles.

From all of this comes the suggestion that the reducing reaction depends on the surface area of the metal particle, that the metal surface traps hydrogen and introduces it to the carbon. The oxygen-aiding reaction, on the other hand, would go by diffusion of carbon atoms through the bulk of the metal - again this liquid-like behavior of metal. This model is very tentative, but Baker concludes, "Although we are not yet in a position to make any definite conclusions with respect to the mechanisms of catalyzed gasification of graphite, the CAEM data is certainly providing some interesting leads into novel methods of both inhibiting and promoting the reaction rates."

DSDP explores the Gulf of Mexico

In figuring out the puzzle of global tectonics, fitting in the small pieces is often the hardest part. It's obvious, for example, how South America and Africa fit together, how the Mid-Atlantic Ridge is the geologic zipper along which the ocean opened. But it's not so obvious, as the crew of the Deep Sea Drilling Project's Leg 77 recently found, how the small ocean basins, such as the Gulf of Mexico, opened up.

There are a lot of different explanations for how the Gulf of Mexico formed, but little physical evidence has actually been gathered, explains Wolfgang Schlager of the University of Miami, who was co-chief scientist on the cruise. Leg 77 aimed to fill in that data gap.

According to Schlager, some geologists believe that the Gulf of Mexico is not an ocean basin at all, but part of a continent that was gradually melted and transformed into basalt, the volcanic rock that characterizes ocean basins. Others, he says, suggest that the Gulf is what is called a back arc basin, an ocean that forms behind a line of volcanic islands, such as the Sea of Japan. Some believe that the Gulf is the oldest ocean on earth, possibly 230 million years old.

Schlager favors a model that sees the Gulf as about 150 million to 170 million years old. In this theory, the central North Atlantic and the Gulf of Mexico were part of the same spreading system and were "like twin brothers, doing everything at the same time," until about 140 million years ago when they parted ways. After that, according to the theory, the North Atlantic kept on spreading while the Gulf cooled and subsided. Because of this early halt in its growth, says Schlager, there is no active spreading ridge in the Gulf; it is "just

a big open spot with margins that record that it subsided."

It was with all these possibilities that the DSDP's drilling vessel, the Glomar Challenger, dug into the sediments beneath the Gulf of Mexico. And while the data are not conclusive evidence for any of the theories, says Schlager, the results give geologists something substantial to consider for future models.

Among other results, the researchers found that the edges, or margins, of the Gulf consist of what is known as "transitional crust" - a combination of continental and oceanic material. Transitional crust is believed to have formed as the continental rock gave way to the spreading ridge that was erupting beneath it; the volcanic rock that welled up from the ridge intruded into the cracks and fractures of the weakening continental rock. While geologists have suspected that this sort of rock would form in the early stages of ocean development, such rocks have not been recovered before, says Schlager, because the margins of most oceans lie too deeply buried beneath sediments. This means, he continues, that similar rock is likely to be found at great depths along the east coast of North America. This find and other evidence, such as rocks that document the transition from terrestrial to marine sediments, tend to support the view of the Gulf as an ocean basin, he says. Most of the competing models, however, depend on different timescales of events, he adds, and only further analysis of the microfossils used to date the rocks will determine when the transitional crust formed, when the ocean stopped spreading and when marine sediments began to be deposited. "We have just taken the first tentative steps toward exploring the Gulf," Schlager says, " ... we have to carefully work into the jungle and make sure we don't throw out any hypotheses before we're sure.'

Federal rules change for autistics

A recent change in the definition of autistic children under the federal handicapped education law appears to be more than a semantic flip-flop. The Department of Education announced in the Jan. 16 FEDERAL REGISTER that these children are no longer categorized as "seriously emotionally disturbed." They now make up a subgroup of the "other health impaired" category because, says the department, "not all autistic children are seriously emotionally disturbed."

Infantile autism is a condition marked by severe communication and other developmental and educational problems (SN: 3/7/81, p. 154). The National Society for Autistic Children in Washington has worked for the definitional change since 1975. Its view is that the shift will increase the chances of providing instruction aimed at the special needs of autistic children in public schools. Not all children with autism will automatically be placed in special classes, but state and local school systems must begin to adapt their educational programs to the needs of these children.

Change will not come quickly, though. School systems were slow to adopt the original handicapped education law in 1977, and the NSAC does not expect them to be any faster this time around. But the rules change will serve more immediately as an aid in informing the public that autism is primarily a neurological, not an emotional, disorder. It also will boost efforts to train and certify more teachers with appropriate skills to educate autistic youngsters. Such instructors are in short supply. The NSAC plans to monitor progress in teacher certification and state compliance to the new regulations.