Jesson admitted that DuPont appears to be hedging its bets on the chlorofluorocarbon controversy.

"DuPont is taking what I regard as the prudent course, that is, to investigate intensively the kinds of alternative products that one might put on the market should the chlorofluorocarbons be found environmentally unacceptable."

At the same time, he said that two chlorofluorocarbon alternatives that looked like they might do the job without interfering with the ozone layer were later rejected because of their extremely high toxicity in animal tests.

For his part, Molina argued that the two years of additional research proposed by Jesson is unnecessary in the face of the large volume of supportive data that has already been accumulated. Even if the incremental effects of the chlorofluorocarbons in the stratosphere are as low as Jesson predicts, Molina believes that it is possible that the same kind of argument will be used to block or delay decisions about continuing manufacture any time in the future, while the overall threat to health continues to grow.

Whether or not the upcoming NAS reports will resolve the ozone controversy and trigger government action against the manufacture of the chlorofluorocarbons is not clear. A hint that the situation still remains unresolved comes from panel member Fred Kaufman of the University of Pittsburgh, who told the ACS press conference that the report will give "uncertainty a generous range of uncertainties." In fact, it was uncertainties about the chlorine nitrate controversy and other disagreements in data from computers, laboratories and various levels of the atmosphere that have already delayed release of the NAS report for more than six months. □

Guadeloupe volcano: Watch and wait

Its reincarnation was heralded last November by swarms of small earth tremors which in July gave way to ominous clouds of steam and ash. Most recently, it belched out a glowing avalanche of rock and gas, called a *nuée ardente*. The still-threatening object is La Soufrière (the sulfur mine), a volcano on the pair of connected French Carribean islands called Guadeloupe. Some 72,000 of Guadeloupe's residents living nearest to the volcano were evacuated about three weeks ago.

Although the Aug. 30 explosion was the volcano's most violent in this current episode of activity, volcanologists disagree on whether or not it was the main eruption they have predicted from the beginning. Four French scientists working at the fissure's rim were injured and others had to be lifted to safety by helicopter when the explosion launched clouds of ash and debris thousands of feet into the air. Richard Fiske and W.T. Kinoshita, two U.S. Geological Survey scientists assisting French volcanologists to monitor La Soufrière, have now returned from Guadeloupe. The pair were halfway up the volcano's slope when it exploded and they escaped injury. It was a "dramatic event," Fiske says, and "kind of scary."

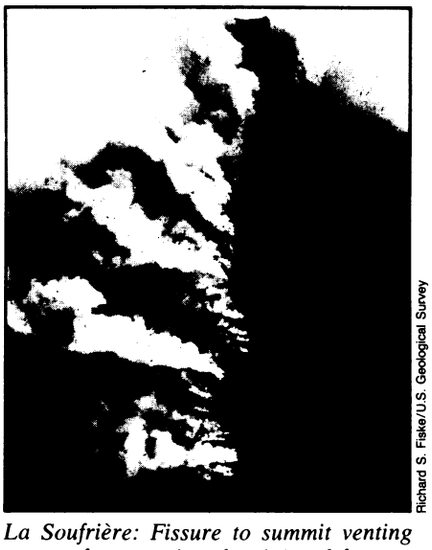
While on the island, they installed a number of tiltmeters to monitor the ground deformations around the volcano. The devices, which can detect even the slight movements caused by human footfalls, are implanted in an array extending halfway up from the base of the 4,815-foot-high volcano. The instruments will measure ground swelling, an indication that the volcano is storing energy in probable anticipation of a major eruption. The recent explosion was a "large one," says Fiske, and there had been "significant inflation" in La Soufrière's slopes to forecast its occurrence.

Since the tiltmeters have just been installed, Fiske says, "we don't know the results" of the recent explosion. "The quakes are continuing," he explains, "about 50 to 150 [of them] per day."

Recently, a statement made by Haroun Tazieff, one of France's leading volcanologists, highlighted the disagreements that have attended this situation from the beginning. In a rather emotional confession he belatedly criticized the decision to evacuate the people, calling it a reaction to "panic." Explaining that "moral pressure" from French authorities had kept him from speaking out sooner, Tazieff asserted that the scientists who made the initial predictions about La Soufrière's impending eruption are "incompetent" and "have never seen an eruption."

Nevertheless, the volcano, which has erupted on 14 occasions since Columbian times, is of the dangerously explosive "strato" variety. By contrast, shield-type volcanos, like most Hawaiian ones, are characterized by "oozing" eruptions of massive lava flows. The various disagreements are easily understood because of the little experience volcanologists have in making predictions of this sort. "The first formal prediction [affecting a volcano] in Hawaii," Fiske notes, was only recently made (SN: 3/27/76, p. 199).

Seismic studies, volcanic gas measurements, monitoring the amount of ash and noting the relative amounts of exuded fresh magma and old rock are techniques employed to predict the likelihood and severity of a volcanic eruption. Observations of this kind indicate that La Soufrière remains in an "unstable state," according to Fiske. The situation now is one of just watch and wait. □



Richard S. Fiske/U.S. Geological Survey

La Soufrière: Fissure to summit venting steam after eruption that injured four.

Arsenic in wine: A bubbling brouhaha

A tempest in a wine bottle is probably a fair assessment of the furor that developed over a paper that was scheduled for presentation, then withdrawn at the last minute, from the 172nd national meeting of the American Chemical Society in San Francisco. And even though the paper was never released it sent shivers down the collective spine of many California wine producers and stimulated a flurry of claims and counterclaims among the principals involved.

It all began with a paper that indicated that some California wines and several other foodstuffs contained potentially toxic levels of arsenic, a known poison and a suspected carcinogen. The authors included Richard K. Vitek of Bio-Metals Analysis, Inc., New Berlin, Wis., William C. Houser of Milwaukee County Hospital, Stanton Deeley, formerly of West Allis Memorial Hospital, in West Allis, Wis., and James J. Bors of Wauwatosa, Wis. The snowball started to roll when a Milwaukee newspaper reporter—inadvertently or otherwise—published news of the findings several days before the paper would have been delivered at the ACS meeting.

The report was triggered by the discovery that a "wino" who consumed about two quarts of wine a day showed signs of arsenic poisoning when examined at the West Allis Memorial Hospital. That suspicion was strengthened after it was determined that the alcoholic's urine contained 439 micrograms per liter (μg/l) arsenic when he entered the hospital. After six days off the wine, the arsenic level dropped to 329 μg/l and to 19 μg/l after 15 days. Further investigation showed that the wine imbibed by the patient contained abnormally high levels of arsenic. The levels found ranged from 66

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to 78 μg/l.

That finding prompted a more extensive study of other foodstuffs and the discovery that some California wines contained far more arsenic than other foodstuffs did. For example, two bottles of a California pink Chablis, purchased in Milwaukee, contained 240 μg/l and 287 μg/l of arsenic respectively, while some California Burgundies, rosés, ports and vermouths contained lesser, but still high concentrations of arsenic.

According to the authors of the still unreleased report, present U.S. Public Health Service directives assert that the maximum allowed level for arsenic in drinking water is 50 μg/l and that a safe level is 10 μg/l.

Spokesmen for the California wine growers argued that arsenic levels in a few bottles of wine do not constitute enough evidence to damn the entire output of an industry. Nevertheless, based on the unreleased ACS meeting paper, the public health department of the state of California will begin immediately to measure arsenic levels in wine produced in the state. As for where all that arsenic may be coming from, agricultural chemists speculate that it is probably the residue of the arsenical pesticides that were used extensively in California grape fields until about two years ago.

Following the premature newspaper story of the data that was never presented formally, public information personnel at the ACS were bombarded with statements which gave a few clues as to why the paper was withdrawn. A dispatch from the lawyer of Vitek, Bors and Houser stated that "the sensationalism created from portions of the data being printed in the newspapers in advance of the agreed upon release date has detracted from the purely scientific nature of the paper."

From the West Allis Memorial Hospital public relations director, Bob Betts, came the explanation that "this material was being utilized without the knowledge or consent of the director of the hospital's laboratory, Dr. Harold J. Conlon."

To counter that statement, Vitek, Houser and Bors said they had a perfect right to use information from their own laboratory records and that they had followed normal procedure for obtaining authorization to use such data.

"However, due to a misunderstanding, some hospital officials were unaware this data was to be published and have sought to retract the permission previously granted," the authors' lawyer said, adding: "In withdrawing their paper from the ACS program, the authors voluntarily accede to this request, even though substantial portions of the paper are the result of the authors' independent research not involving the facilities of the hospital's laboratories." In the lawyer's statement, it was stressed that the authors' method for analyzing arsenic levels in foodstuffs and other substances (the chief reason the paper was scheduled) "is valid and that the data generated by their study are confirmed and substantiated and can be readily duplicated."

Further, he noted that their paper calls for further research into arsenic and foodstuffs "and is not intended to imply that all wines or fruit juices pose a substantial hazard or that scientific investigation was complete."

Controversy over the paper and its findings will undoubtedly continue, but it is distinctly possible that if the results had not appeared prematurely in the Wisconsin newspaper, where they provoked the flurry of statements, the paper would have appeared quietly on the ACS program with only a fraction of the attention it ultimately received.

At the same time, the ruckus it caused was primarily responsible for getting the California public health officials to start checking arsenic levels in the state's wine output. □

Localizing missing cholesterol receptors

Lipoproteins (dots) bind to cholesterol receptor region of healthy membranes.

There are several kinds of inherited diseases whereby persons make too much cholesterol and suffer heart attacks. One is familial hypercholesterolemia. One out of every million Americans is a homozygote (carries a gene from both parents) for the disease and usually suffers a heart attack by age 20. One out of every 500 Americans is a heterozygote (carries a gene from one parent) for the disease and usually suffers a heart attack around age 40. First genetic and biochemical evidence, and now electron microscopic evidence, are unmasking the genetic defect underlying the disease. It is a paucity of cell membrane receptors for cholesterol.

During the early 1970s, Joseph L. Goldstein and Michael S. Brown of the University of Texas Health Sciences Center in Dallas learned that cells called fibroblasts have receptors on them that bind with cholesterol-lipoprotein complexes in the blood. When the complexes bind to the receptors, an enzyme that regulates the rate of cholesterol synthesis reads that as a signal that no further cholesterol is needed. The cholesterol production is arrested until the complexes have dwindled enough in the blood to turn cholesterol synthesis back on.

Goldstein and Brown had reason to believe that there is nothing wrong with this enzyme in persons with familial hypercholesterolemia. Instead it seemed that the enzyme does not receive the proper cues for turning off cholesterol synthesis because cholesterol-lipoprotein complexes do not bind to fibroblasts. They tested their hypothesis by radioactively tagging lipoproteins and putting them in the presence of cells from healthy subjects, from heterozygotes and from homozygotes for familial hypercholesterolemia. The lipoproteins bound efficiently to the cells from the healthy subjects, but with only 40 percent efficiency to the cells from the heterozygotes and with only 3.6 percent efficiency to the cells of the homozygotes. Clearly then, the genetic defect underlying the disease consists of missing cholesterol receptors. Because heterozygotes have one faulty gene, they make some receptors, but an insufficient number. Because homozygotes have two faulty genes, they make virtually no receptors (SN: 7/13/74, p. 22).

Now Goldstein and Brown, with help of Richard G. W. Anderson, also at the University of Texas Health Sciences Center, have pinpointed those areas of the cell membrane where the cholesterol receptors are normally present but lacking in persons with familial cholesterolemia. They radioactively tagged lipoproteins and put them in the presence of healthy fibroblasts and of fibroblasts from a patient homozygous for familial hypercholesterolemia, then examined the materials under an electron microscope. The lipoproteins could be seen to bind preferentially to specific receptor sites on the cell surface membrane of healthy fibroblasts—specifically, at indented regions. Although the defective fibroblasts had the same number of indented membrane regions per millimeter of cell surface as did the normal cells, no lipoproteins could be seen bound to these regions.

"The present ultrastructural data," the investigators conclude in the July PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES, "are consistent with previous biochemical and genetic evidence indicating that the lipoprotein exerts its regulatory action on cellular cholesterol metabolism in fibroblasts through an interaction with a specific cell surface receptor and that this receptor is defective in homozygous familial hypercholesterolemia fibroblasts." Although they did not examine the cell membranes of heterozygotes, presumably they would show some lipoprotein binding, but not nearly as much as healthy cells.

This basic research has therapeutic implications, the researchers believe. As Goldstein told SCIENCE NEWS, "The most important, I think, is that as we get more molecular information, we will be able to apply rational therapy at that level rather than just lower serum cholesterol with drugs or diet." □