environmental problems than in producing solid international agreements. As Maurice Strong of Canada, United Nations under secretary for the environment, has said, the UN has generally failed in efforts to make nations change their ways internally; it has been successful only in dealing with problems that cannot be dealt with any other way but internationally.

So the conference is likely to have little impact, for instance, on Brazil's apparent resolve to secure industrial development of the polluting kind now common in Europe and North America. Nor could one imagine that any international police action would be possible against the apparent determination of U.S. oil companies and the Department of the Interior to ship Alaska's North Slope oil to the U.S. West Coast via a partly marine route, despite ecologists' objections.

However, the conference may require the United States to commit itself to participation in an international marine monitoring system and to eventual ratification of conventions against ocean dumping of harmful materials. In the case of the former, the United States might then be obliged by international agreement to do baseline studies of Alaska's Prince William Sound. Such studies are not now included in Interior's environmental impact statement for transfer of the Alaskan oil. And conventions against ocean dumping, if ratified by the United States, might require a revision in some future Alaska-type scheme.

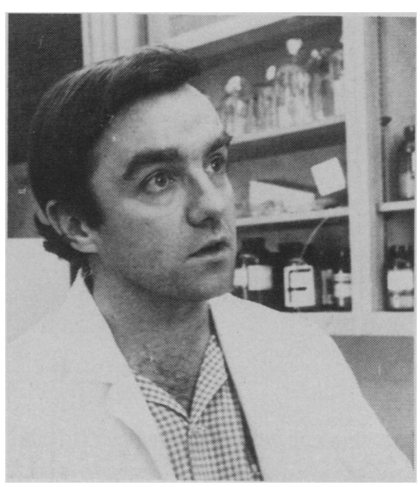
But areas where progress may consist mainly of only public education or public relations include those involving necessarily internal matters, or subjects not amenable to international control: migration from rural to urban areas with consequent impoverishment to both, the environmental aspects of natural resource management, and government support of economic practices which result in pollution or harmful resource depletion. The United Nations is not a world government, and Stockholm will not make it so.

There are some strong indications that the United States may be among the nations doing the most foot-dragging in securing effective movement in this direction. "I'm disturbed," Sen. Edmund S. Muskie said on the Senate floor last week, "that there will be little opportunity for the exchange of views and recommendations [at Stockholm] because U.S. positions have been predetermined in detail by the Administration." Muskie charges that there are no well-known nongovernment environmentalists in the U.S. delegation and that pollution control measures the United States is willing to support at Stockholm are far less stringent than ones already adopted domestically. Muskie contends that the U.S. delegation is committed to a weak UN environmental agency and that it goes to Stockholm with instructions to oppose adamantly any formula for additional foreign aid from the industrialized nations to developing nations for pollution control or nonpolluting economic development. □

Tryptophan repressor: Therapeutic potential?

If cells in different parts of an organism have the same genetic information, why do they differ so much in form and function? One hypothesis is that there is a class of regulatory genes that makes repressors, each of which functions to prevent a structural gene from making its product. In other words, when a protein repressor sits on a gene, the gene does not work at its usual capacity. It took a decade to bear out this theory, but in 1967, the first repressor was isolated by Mark Ptashne of Harvard University in material from bacterial cells. Since then, some dozen other protein repressors have been isolated, mostly by investigators at Harvard and Columbia Universities. Now a team of biologists from both schools, including Geoffrey Zubay, Daniel Morse, W. J. Schrenk and J. H. M. Miller, has isolated the repressor for the several sequential genes, or operon, in the bacterium *Escherichia coli* that shut off the production of tryptophan when the cell does not need it. Tryptophan is one of the 20 amino acids that form proteins in all living systems.

The work of Zubay and his colleagues is reported in the May PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES. Their paper shows that their method of repressor isolation is probably the method of choice for isolation of most repressors. But their work, seen within the larger context of biochemical research and genetic diseases, holds more significance. This is the first time that a repressor for enzymes that make products, such as an amino acid, has been isolated. The other repressors that have been isolated are for enzymes that break down, rather than build up, molecules in a cell. Of more interest, their accomplishment raises the question whether a gene repressor for tryptophan production might have use in the treatment of schizophrenia.



Columbia Univ.

Zubay: Isolating gene repressors.

In recent years the Lafayette Clinic in Detroit, the nation's largest center for schizophrenic research; the First Institute of Psychiatry of the Soviet Academy of Sciences in Moscow, the largest schizophrenia research center in the world; Lund University in Sweden, and the University of Oslo in Norway have been gathering evidence that schizophrenia may have a biochemical basis, specifically, an overproduction of tryptophan in brain cells. Might the tryptophan repressor isolated by the Columbia and Harvard researchers be injected into the brains of schizophrenics and improve their condition?

Zubay asserts that it would not work, because the tryptophan repressor as well as all others isolated so far have been taken from *E. coli* and other bacteria. "The bacteria repressor proteins will not cross-react with sites on human genes. They have evolved in a different way. In other words, human repressor proteins would have different amino acid sequences from bacteria repressors." Nonetheless Zubay acknowledges that if a tryptophan repressor is ever taken from human cells, it might have potential for schizophrenia treatment. However, he says, it would be virtually impossible to get a repressor into the cell of a schizophrenic, and even if one could get the repressor through the cell wall, the repressor would probably be destroyed by the normal breakdown processes in the cell. So, he concludes, probably the only way to get the repressor into the cell would be by sending the gene for the repressor in via a virus.

The problem, Zubay explains, is that biochemists and geneticists currently do not have the tools to pinpoint genes and repressors in higher organisms. "Not one gene in animals or in humans has had its mechanisms of control worked out yet," he declares. Yet since scientists have reached the point where they can map genes and their functions and can isolate gene repressors in bacteria, Zubay is hopeful they will soon be able to do the same in animals and humans. Thus, while the work of Zubay and his colleagues may not provide a direct practical use in genetic disease therapy, it is contributing to an incredibly detailed mosaic of information that will ultimately lead to treatment at the cellular level for schizophrenia and other metabolic diseases. □

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