argues against research and development of other energy sources—including fission. What we do argue against is a national policy that would require that fission power—or anything else—*must* precede and thereby delay or exclude the inception of fusion.

In our democratic society, decisions are ideally arrived at by integrating the input from all sources. We therefore agree with Parsegian on the need for the broadest input possible to what we all feel is perhaps the most critical technical-political issue that we in this century have been asked to resolve—from whence will come the energy to build a better future world?

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Thioctic Acid and Mushroom Poisoning

In Barbara J. Culliton's article "The destroying angel: A story of a search for an antidote" (News and Comment, 16 Aug. 1974, p. 600), Frederic C. Bartter, clinical director of the National Heart and Lung Institute at the National Institutes of Health, refers to experiments performed at the Food and Drug Administration (FDA) in which thioctic acid and glucose cured mushroom poisoning in dogs. As the unnamed researcher who conducted the experiments and analyzed the data, I wish to point out that my findings are reversed in Culliton's report. Bartter is quoted as saying, "So, in the later experiments we carefully maintained glucose levels in the dogs, just as you routinely would in a patient." In fact, I found that the low blood glucose levels in mushroom-poisoned dogs were *not* restored to normal by six hourly injections of dextrose and thioctic acid. Culliton continues, "It appears that this was the problem. When dogs receiving toxin and antidote were also given glucose, they survived. The researchers decided then that it is reasonable to use thioctic acid experimentally to try to save the lives of victims of the destroying angel." In fact, I found that four of five mushroom-poisoned dogs given thioctic acid and glucose and five of five mushroom-poisoned control dogs *died*. I reported this finding to my FDA colleagues, including Alan K. Done, who, as Culliton indicates, communicated with Bartter. Culliton's article suggests that there is renewed hope for victims of mushroom poisoning and appears to encourage doctors to request emergency shipments of thioctic acid antidote. Unfortunately, the negative results I obtained with thioctic acid in both mice and dogs do not justify this hope or course of action.

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As Culliton reported, I was asked to assume the IND (investigational new drug) application for thioctic acid on the basis of numerous more or less favorable reports of its clinical use from Europe, and a few from the United States.

I did indeed discuss with Alan Done experiments designed to test its efficacy in animals. He showed me reports of mouse studies which did not show a therapeutic effect for thioctic acid given in distilled water. We agreed that no test of the effectiveness of thioctic acid in which blood sugar was not maintained could be of value. This is because both amanitin poisoning and thioctic acid may produce hypoglycemia.

I wish to emphasize that I had no part in the design of the experiments which were then done on dogs. I was simply informed later (August 1974) by the Food and Drug Administration (FDA) that it was reasonable to proceed with the IND.

Recently (December 1974), I received the details of the dog studies. From them one may infer the reasons the FDA decided to proceed from the clinical evidence not to withhold thioctic acid from human subjects poisoned with amanitin. It was clear that dog studies designed as these were had done nothing to clarify the issue. Glucose had been given in tiny doses of 500 milligrams by six hourly injections (3 grams a day). Of course the dogs that died (four out of five) showed hypoglycemia. In human subjects, thioctic acid should be given with a sustained intravenous drip of glucose, and the total dose of glucose should be at least 100 grams a day. With this regimen, no human subject has been reported to show hypoglycemia.

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