

POTENTIALLY CATASTROPHIC POLICIES

by

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ABSTRACT

The rapid development of science and technology has created a new type of public policy problem: uncertain and complex risks which bring the possibility of large-scale catastrophe. I call this class: potentially catastrophic policies (PCP's). Whether the issue is biological research, immunization programs, ozone depletion, space travel, or nuclear power, the same generic questions of risk are involved.

Each of the participants in the decision making process -- the public, experts, and government -- have a difficult time with PCP's. Perceptual biases prevent individuals from accurately gauging the nature and extent of PCP risks -- they are forced to rely on trained experts for advice on the possible hazards. The public learning process about these risks takes time and is itself subject to distortions. Nor is the information passed on by the experts "objective": the various communities of scientific and technical experts must be seen as interest groups pursuing their own economic and professional objectives. The government in carrying out its role of protecting the public from societal dangers, further confounds the PCP problem by analyzing policies with decision techniques that are difficult to apply, easy to manipulate, and inaccessible to the uninitiated.

The central role of the experts in evaluating PCP's is illustrated by the recombinant DNA controversy, the swine flu immunization program of 1976, as well as many other examples.

Michael O'Hare, Associate Professor
Thesis Supervisor

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Chapter 1

INTRODUCTION

With the rapid development of science and technology in the latter part of the twentieth century, a new type of policy problem has emerged. As individuals and as a nation, Americans are more and more being called upon to make decisions on policies where there is a small chance of a major catastrophe. I call this type of policy a Potentially Catastrophic Policy, or PCP.

In the first part of this work, I shall give many examples of PCP's and I shall argue that, however different they may appear at first, all PCP's share the common property of pervasive uncertainty and complexity. Thus, whether the issue under study concerns nuclear power, immunization programs, recombinant DNA research, space travel, new drugs and chemicals, or irreversible ecological damage like ozone depletion, their similarities outweigh their differences. From a public policy point of view, they should be treated as a generic type of social decision problem.

As their name implies, PCP's bring the possibility of large scale catastrophe -- some may never occur or, indeed, may later turn out to be theoretically impossible. I am concerned here with the potential for catastrophe as perceived at the time of public discussion. When faced with PCP risks, the choices are clouded by what is not known as much as by what is. Originally I had intended to use the term low probability catastrophes to describe PCP's, but this would have begged the questions: "how probable?" and "according to whom?".

A discussion of the recombinant DNA controversy is central to the argument. During the last few years I have spent

a great deal of time reading and listening to the arguments on both sides of what has become a polarized debate. Though lacking training in microbiology (I studied chemistry), I have tried to learn the science upon which the estimates of risk are made. Nevertheless, I must admit that I do not know how hazardous these experiments are. Certainly, some are more dangerous than others; just as working with cancer viruses is riskier than working with influenza viruses. I am sure that the scientists themselves have insufficient cause for the confidence which they seem so eager to share with us.

In the pages that follow, one special kind of catastrophe, war, will be largely ignored. Together with pandemic disease, wars have caused widespread human suffering throughout human history. War represents a different kind of policy problem: it constitutes an adversary situation where one participant tries to outwit and overpower the other. There is one useful comparison to be drawn from the transition that has occurred in the strategy of conflict. With the development of thermonuclear weapons, the potential costs of war have changed. While war always brought widespread misery, there is now the possibility of destroying human life on earth. Similarly, many of the technological policies which I shall review have taken a step towards a large scale collective calamity.

What follows is divided into three parts: First, I shall show PCP's to be different from other policy questions

because of their uncertainties and complexities and catastrophic potential. In the second part, I shall review how PCP's are handled by individuals, the experts, and the government. I shall show that none of these, acting alone or with the others, is able to cope satisfactorily with PCP's. In the final part, I shall suggest some ways in which we can better deal with PCP's.

PART I

PCP'S: A NEW PUBLIC POLICY PROBLEM

Chapter 2

CATASTROPHES

Catastrophe has entered our common vocabulary to describe the effects of everything from a broken heart to a nuclear explosion. My first task, therefore, is to review some of the possible calamities that are of special concern to my argument. Before I begin, a word of caution is appropriate: many of the catastrophes I will describe in this chapter are believed to be likely by some but improbable by others. For the present, I will not detail arguments for or against their likelihood -- certainly, they are all possible.

A. What is a Catastrophe?

A safe beginning would be to identify a catastrophe with "the end of the world." The most obvious examples are astrophysical events, like those popularized in science fiction.

(1) The earth might leave its present orbit and begin travelling either towards, or away from, the sun. The possible climatic results were graphically depicted in the British thriller, The Day the Earth Caught Fire, in which the earth had been jolted out of orbit by nuclear tests. Temperate regions became deserts, and vice versa. Alternatively our planet might move without direct interference from man: a team of American and English scientists has claimed that the ice ages were caused by periodic

(1) Many of these threats to the biosphere are adapted from a paper delivered by Walter Orr Roberts at the 1976 meeting of the American Association for the Advancement of Science.

changes in the earth's orbit. (2) Also, another planet or a comet might collide with us. While such events seem incredibly unlikely, they could happen -- in 1976, an asteroid had what some astronomers would call a "near miss" with the earth; it passed within three quarters of a million miles of us. (3) If anything happened to the sun, we would quickly be aware of the change. The sun is not expected to "go nova" for millions of years, but when it does, the explosion will vaporize our oceans and sterilize the atmosphere. Much sooner, the sun could suddenly flare up, dramatically increasing the concentration of ultraviolet radiation bombarding the earth, and thereby disrupting life on the planet. We know very little about how the sun shines: if, as some have hypothesized, the sun's core is at a lower temperature than its surface, the earth may one day suffer a new ice age when the "cooler" nuclear reactions reach the sun's exterior. (4)

While it is interesting to speculate about such calamities, they occur at intervals measured on an astronomical time-scale -- on the order of the age of the earth and the solar system. They are much rarer than the unlikely events which concern me here. (5) More important, we could not protect

(2) John Noble Wilford, "Ice Age Held Caused by Earth Orbit Shift," New York Times, November 30, 1976.

(3) "Asteroid Has 'Near Miss' with Earth 750,000 Miles," New York Times, November 3, 1976. See also footnote (5).

(4) Walter Sullivan, "Doubts Are Raised on Why Sun Shines," New York Times, January 23, 1976.

(5) It has been estimated that the probability of a meteorite

ourselves from these catastrophes. Nevertheless, they do have two characteristics which are useful for thinking about PCP's: they all entail collective and involuntary risks. In Tom Lehrer's words, "we will all go together when we go," ready or not, we will all "drop our agendas and adjourn."

Apart from such cataclysmic threats, life on earth may be quite resistant to total annihilation. A committee of the National Academy of Sciences (NAS) explored the consequences of a war in which half of all nuclear weapons in current arsenals was detonated: this is the equivalent of ten billion tons of TNT. Philip Handler, the president of NAS, summarized the study's findings with the perhaps surprising conclusion that the biosphere and the species Homo sapiens would survive. (6) (The study group offered no thoughts about the quality of life for the survivors.)

The consensus of scientists, supported by the empirical evidence all around us is that man is extraordinarily

crashing onto the earth's surface with a lethal area of over 100 square miles is five chances in ten million per year. K. A. Solomon, et al., Estimates of the Hazards to a Nuclear Reactor from the Random Impact of Meteorites, Report No. ENG-7426, (UCLA: School of Engineering and Applied Science, March 1974), p. 11. This estimate must be reduced further to predict loss of life since the meteorite is much more likely to fall on an isolated region than on a populated metropolitan area. The reliability of this probability is another matter: see Chapter 10.

(6) Letter from Philip Handler to Fred Ilke in Long Term Effects of Multiple Nuclear Weapons Detonations (Washington, D.C.: National Academy of Sciences, 1975). The findings caused an immediate stir. Lest anyone saw advantage in the committee's conclusion, Dr. Handler was quick to add: "[N]o nation can deliver what is intended as a massive pre-emptive strike without automatic catastrophic natural consequences to itself." P. Handler, "No Escape," New York Times, November 26, 1975.

adaptive; at least some of us will live through most calamities. Given this natural limit, I can distinguish four different classes of catastrophes:

1. Global Hazards. Some of the things we are doing --partly because there are more of us doing them and partly because what we are doing is new with unknown consequences-- are jeopardizing the stability of the earth's energy budget and the essential elemental cycles. Incoming and outgoing radiation and the myriad gases and particles in the atmosphere must all be in balance if we are to maintain the present weather and climate. Nor should we upset the oxygen, carbon, and nitrogen cycles, for these basic elements produce our diverse and complex environment. Such global impacts are primarily outgrowths of decentralized decisions: no single policy can undo the potential for disaster --indeed usually there is no existing institution which has the authority to regulate these hazards.

Scientists have found that many human activities perturb these processes. Carbon dioxide is building up in the atmosphere, heating the planet; aerosol propellants, refrigerants, organic solvents, and SST emissions are all predicted to deplete the ozone layer; fertilizers may not only harm the ozone layer but also modify the nitrogen cycle. We are far from an understanding of the long-term consequences of upsetting the balance nature has established over the past millenia. (7) Disruptions could be catastrophic. During another

(7) Reid A. Bryson, "A Perspective on Climatic Change," Science, 184 (May 17, 1974), pp. 753-760; and Inadvertent Climate

ice age, mountains of ice could devastate regions now producing our food. Conversely, a warming trend could melt the polar ice caps flooding the earth's coastal cities.

The combustion of all carbon-bearing fuels (oil, coal, wood, and natural gas) produces carbon dioxide which absorbs infrared radiation emitted by the earth's surface and re-radiates it back to the earth. Carbon dioxide keeps us warm, but if the recent upward trend in its atmospheric concentration continues, we may suffer serious climatic changes. One expert has hypothesized: "by the first decade of the next century we may experience global temperatures warmer than any in the last 1000 years." (8) A panel of the National Academy concluded that while society could adapt to a build-up of carbon dioxide over the long run, the short term outlook was bleaker: "the effects might be adverse, perhaps even catastrophic." (9) George Woodwell describes the magnitude of the potential problem in no uncertain terms: "There is almost no aspect of national and international policy that can remain unaffected by the prospect of global climatic change. Carbon dioxide, until now an apparently innocuous trace gas in the atmosphere, may be moving rapidly

Modification, Report of the Study of Man's Impact on Climate
(Cambridge, Mass.: MIT Press, 1971).

(8) Wallace C. Broecker, "Climatic Change: Are We on the Brink of a Pronounced Global Warming?" Science, 189 (August 8, 1975), p. 461.

(9) National Research Council, Energy and Climate Studies in Geophysics (Washington, D.C.: National Academy of Sciences, released 1977, publication pending). See "Carbon Dioxide from Fossil Fuels: Questions of Climate Change," NAS News Report, 27 (October 1977), p. 5.

toward a central role as a major threat to the present world order." (10)

2. Pervasive Hazards. In contrast to the global hazards which may wreck the planet's ecology, there is a second set of PCP's which can induce changes just as pronounced but whose members are sensitive to identifiable policy decisions. Here again scale is a crucial determinant of hazard.

For example, a catastrophe can result from the planned or inadvertent spread of a toxic agent. International attention has focussed on the perils of a number of chemicals such as DDT, PCB's, asbestos, methyl mercury, and vinyl chloride. Each individual substance has had a tragic history, but the far more dangerous outcome is that the many known and other still unknown toxic chemicals are together having a cumulative, irreversible impact. Chemical carcinogens may have already sown the seed for a future disaster. Dr. Bruce Ames, developer of the most popular "quick" test for detecting carcinogens, told the National Cancer Institute's Cancer Advisory Board that "the modern chemical world hasn't hit us yet;" when it does by 1990, there will be a "cancer epidemic." (11)

The list of known chemical carcinogens to which we are,

(10) George M. Woodwell, "The Carbon Dioxide Question," Scientific American, 238 (January 1978), p. 43.

(11) "Bruce Ames Tells NCI Advisory Group that a Cancer 'Epidemic' is Coming," Pesticide and Chemical News, September 21, 1977, pp. 15-16. Ames is not alone in suspecting cancer incidence is increasing. The Blue Cross and Blue Shield of Greater New York sponsored a symposium on carcinogens, "Cancer Epidemic?" Bulletin of the New York Academy of Medicine, Second Series, 54 (April 1978).

or have been, exposed grows almost daily: red dye No. 2, in everything from maraschino cherries to chocolate pudding, cyclamates and saccharin in soft drinks and mouthwashes, chlorinated hydrocarbons as simple as carbon tetrachloride and as complex as PCB's in our water, coal ash in our air, asbestos in both our air and water, pesticides and chemical additives in our food, benzo-pyrene in cigarettes, estrogens in drugs, and tris on our children's sleepwear, as well as scores of other chemicals used in hair dyes, solvents, plastic packaging, and myriad industrial processes. In fact, one method of detecting cancer, mammography, causes the breast tumor it is designed to diagnose!

If a substance is sufficiently toxic and persistent, relatively small quantities could, depending on nature's own forces of distribution, pollute huge areas of the planet. One such chemical is dioxin. The British medical journal, Lancet, has called it, "one of the most toxic compounds known." (12) One correspondent made the following extrapolation: "If there were an exact analogy between guinea pig and man (which there isn't) the lethal dose for an adult would be 70 millionths of a gram." (13) On July 10, 1976, an explosion at a chemical plant released some two kilograms of dioxin over the town of Seveso, near Milan, Italy, contaminating an unsuspecting population. (14) Hundreds

(12) "Seveso," Lancet, 2 (August 7, 1976), p. 297.

(13) Lawrence McGinty, "The Graveyard on Milan's Doorstep," New Scientist, 71 (August 19, 1976), p. 383.

(14) For a description of the Seveso incident, see Thomas Whiteside, "A Reporter at Large: The Pendulum and The Toxic Cloud," New Yorker, July 25, 1977, pp. 30-55, and John G. Fuller,

of people were evacuated from their homes (they have not yet returned), and many who were exposed soon developed chloracne, a skin irritation, sometimes requiring hospitalization. But the most serious hazards are still unknown: dioxin may be carcinogenic and mutagenic. Many pregnant women unwilling to take the risks had abortions. (15) One pessimistic view was expressed by a dermatologist who had treated the victims of a similar explosion in Britain: "I would evacuate everybody in the contaminated areas at Seveso, and not allow them to take anything with them -- not even their clothes. Then I would seal off the area, and leave it barren, if necessary for ever." (16)

No one sets out to make dioxin, rather it is a by-product of other reactions, as in the manufacture of trichlorophenol, the chemical being produced in the Seveso plant at the time of the explosion. (17) Since trichlorophenol is a precursor of the herbicide 2,4,5-T, both contain traces of dioxin. Though less dramatic than a sudden explosion, the continual spraying of Agent Orange (a mixture of 2,4,5-T and 2,4-D, another herbicide), over Vietnam during the war may have

The Poison that Fell from the Sky, (New York: Random House, 1977).

(15) Alastair Hay, "Seveso Solitude," Nature, 267, (June 2, 1977), p. 385. In January 1978, the International Agency for Research in Cancer began an epidemiological survey of dioxin-exposed persons. A. Hay, "Dioxin Meeting Recommends Cancer Study," Nature, 271 (January 19, 1978), p. 202.

(16) "Doomwatch, Italian Style," Times (London), August 1, 1976.

(17) Janice Crossland and Kevin P. Shea, "The Hazards of Impurities," Environment, 15 (June 1973), pp. 35-38.

an even more serious aftermath. (In Chapter 6, I shall compare the perceived risks of "bang"-type to "fizzle"-type catastrophes.) While only two kilograms were released in Seveso, some 100 kilograms were scattered over the Vietnamese countryside. (18)

Toxicologist Samuel Epstein believes that cancer is "now a killing and disabling disease of epidemic proportions." (19) Will the trend continue or worsen? Is the latency period between exposure to carcinogens and the onset of symptoms obscuring the coming pandemic? Or are we merely dying of cancer because we have learned how to prevent many of the other diseases which used to kill us? If we solved the mysteries of cancer, would we live any longer or would we succumb to some other disease? We do not know the answers to these questions, and until science catches up, we can only do as Epstein, among others, has counseled: eliminate human exposure to known carcinogens. (20)

Few drugs have a greater potential for catastrophe than the birth control pill. With the introduction of the pill, a group of doctors and drug companies offered fertile women -- many

(18) Alastair Hay, "Vietnam's Dioxin Problem," Nature, 271 (February 16, 1978), p. 597.

(19) Samuel S. Epstein, "The Political and Economic Basis of Cancer," Technology Review, 78 (July/August 1976), p. 35.

(20) John Cairns has also urged that exposure to carcinogens be reduced; he has pointed out that such an approach "should not be taken as counsel of despair. After all, it was largely preventive medicine that eradicated the infectious diseases." "The Cancer Problem," Scientific American, 233 (November 1975), p. 78.

of whom were childless, but still planning to have a family -- a method of contraception based on altering their hormone cycle. Over the years, the medical profession has grown wary of the pill; with the emerging evidence of harm, recommended dosages have shrunk, and the risks have been found unacceptable to more and more age groups. By 1974, some 150 million women around the world had used the birth control pill and, despite apprehension about its effects, some 50 million women (10 million American) were still taking it. (21) Given how little doctors knew about the human reproductive system, I am amazed that they believed themselves able to safely regulate it with a estrogen-progestogen based drug. Indeed, they were so confident that they were willing to risk the health of just those members of society who could assure its continuation.

Mass immunization programs using vaccines which have undergone only limited testing entail possibly grave risks. In the course of a vaccination drive, entire populations may voluntarily, or through coercion, be inoculated. The consequences could be catastrophic if the vaccine is contaminated. It is hard to imagine a more direct way of exposing an entire society to a toxin than by inadvertently injecting it into everyone's body. Vaccines have sometimes caused the illness which they were intended to fight; in such cases a few individuals suffer due to negligence or to a hidden error; death and disease soon bring the campaign to a

(21) Elizabeth B. Connell, "The Pill Revisited," Family Planning Perspectives, 7 (March/April 1974), p. 62.

halt until the source is identified. But the much greater danger is that ill-effects remain hidden long enough for the vaccination campaign to be completed. Undoubtedly, there have been fabulous rewards from vaccines over the years; my quarrel is not with immunization per se but with the comprehensive degree to which it is sometimes attempted. The objective of the 1976 swine flu immunization drive was the vaccination of every American. By trying to prevent a tragic viral pandemic, health officials were risking a remote, though ever-present, man-induced tragedy.

A number of new technologies present the possibility of large-scale catastrophe. Foremost among them is nuclear power which brings a laundry list of potential disasters: (1) nuclear reactor accident: the worst accident could cause anywhere from 3,300 to 33,000 immediate deaths and up to an additional 304,000 latent fatalities, depending on who is doing the counting (though both sets of estimates are admittedly very uncertain). (22) (11) Proliferation of nuclear weapons: as nuclear technology is transferred from one country to another, some of which have not signed the 1968 Non Proliferation Treaty, the odds are that a number of them will develop atomic weapons (as India has done), with the almost inescapable result of a nuclear war. (23) Also,

(22) The low estimate is from: U.S. Nuclear Regulatory Commission, Reactor Safety Study. (Springfield, Va.: National Technical Information Service, October 1975), Main Report, p. 83; the high estimate is from: Union of Concerned Scientists, The Risks of Nuclear Power Reactors, (Cambridge, Mass.: Union of Concerned Scientists, August 1977), p. 125. In addition to the loss of life, the Reactor Safety Study estimates the total property damage to be about \$14 billion.

(23) For example see the discussion of the multi-billion dollar

with the growing trade in nuclear materials, the risks of their diversion and use for terrorism will increase. (24) (111)

Environmental contamination from radioactive wastes: the by-products of military and commercial nuclear program must be contained for what can be considered forever. Unless some solution to the disposal problem is found, the consequences could overshadow what we now consider to be serious pollution problems: "Should the military and commercial waste inventories grow as [government energy agencies] foresee, by sometime after the turn of the century it would take a thorough mixing of the hazardous radionuclides in the waters of all the world's oceans to lower the concentrations to within the presently acceptable limits. And, perhaps by the middle of the 21st century, the radioactivity could be quite beyond all possibility of dilution to safe levels. The hypothesis is of course farfetched, for no such mixing could conceivably occur. But, by the same token, the potential contamination of local and regional environments is significant."

nuclear deal between Brazil and West Germany: Norman Gall, "Atoms for Brazil, Dangers for All," Bulletin of Atomic Scientists, 32 (June 1976), pp. 4-48. David Lilienthal, the first chairman of the Atomic Energy Commission, perceives the danger to be so great that he advised a Congressional committee to unilaterally stop all exports of nuclear hardware and materials. David Burnham, "U.S. Export Ban on Nuclear Equipment Urged by Former Atomic Energy Chief," New York Times, January 20, 1976. See also: Warren H. Donnelly and Barbara Rather, International Proliferation of Nuclear Nuclear Technology, House Committee on Interior and Insular Affairs, Committee Print No. 15, 94th Congress, 2nd Session, April 15, 1976.

(24) Mason Willrich and Theodore B. Taylor, Nuclear Theft: Risks and Safeguards, (Cambridge, Mass.: Ballinger, 1974).

(25) (iv) Low level radiation: with each new source of radioactivity, there is a greater chance of increasing the genetic mutation rate with uncertain effects to human and animal populations. (26)

The transportation of liquified natural gas (LNG) presents some dramatic risks. A government report concluded that a major rupture of a LNG storage installation could cause up to tens of thousands of deaths. (27) LNG tankers coming in and out of busy ports entail similar hazards. On another front, Paul Brodeur has charged that exposure to microwave radiation is a serious and neglected danger. (28) Sources of microwaves are ubiquitous: radio and television broadcast antennas, radar stations, point-to-point communication systems, ovens, CB radios, industrial sealing equipment, etc. Foreign scientists believe

(25) Luther J. Carter, "The Radioactive Waste Inventory," Science, 195 (February 18, 1977), p. 662. See also Chapter 3.

(26) National Research Council, The Effects on Populations of Exposures to Low Levels of Radiation, Washington, D.C.: National Academy of Sciences, 1972); John Gofman and Arthur Tamplin, Poisoned Power, (Emmaus, Penn.: Rodale, 1971); see also Chapter 10.

(27) David Burnham, "G.A.O. Warns of a Disaster Peril Posed by Liquified Gas Storage," New York Times, January 26, 1978; this article was based on the summary and conclusions of the report; at the time this thesis is being completed for submission, the GAO has just released its study, Liquified Energy Gases Safety, July 31, 1978, in three volumes. See also: Elisabeth Drake and Robert Reid, "The Importation of Liquified Natural Gas," Scientific American, 236 (April 1977), pp. 22-29.

(28) Paul Brodeur, The Zapping of America, (New York: Norton, 1977): see also: U.S. General Accounting Office, Efforts by the Environmental Protection Agency to Protect the Public from Environmental Nonionizing Radiation Exposures, Washington, D.C., March 29, 1978.

that low levels of microwaves are implicated in neurological, genetic, and behavioral effects, as well as being potentially carcinogenic. The U.S. occupational guideline for microwave exposure is 1000 times greater than the Russian occupational standard, and 10,000 greater than its ambient standard. According to government statistics more than 2 million Americans are exposed to microwave power densities above the Russian standard. (29) Like the possible disaster caused by chemical carcinogens, microwave radiation may be responsible for serious long-term health effects.

3. Self-Propagating Hazards. An organism with the ability to self-replicate could bring catastrophe if it had a combination of pathogenic and persistent properties that allowed it to survive and spread itself around the world. Such microorganisms could inadvertently be returned to earth on a spaceship (backcontamination), (30) created in a biology laboratory, or bred naturally by abusing certain drugs. This class of PCP's has the unique property that it takes only one "event" to bring tragedy --though the more pressure that is applied to the introduction or creation of a new organism (more space exploration, more laboratory research, and more drug abuse), the greater the risks.

(29) Statement of William J. Thaler, Office of Telecommunications Policy, Radiation Health and Safety, Hearings before the Senate Committee on Commerce, Science, and Transportation, 95th Congress, 1st Session, June 1977, p. 696.

(30) This catastrophe is better known as the "Andromeda Strain" scenario, after Michael Crichton's best selling book.

Scientists hope to travel to new worlds to unravel the mysteries of the birth and development of our solar system, the galaxy, and the universe. The unknown generates both excitement and dangers; the search might lead to the return to earth of a dormant spore, possibly one trapped inside a moon or a Martian rock. Would the organism awaken on contact with the earth's atmosphere? Would it be antagonistic to life on earth? Could we protect ourselves from its pathogenic properties? If the foreign organism did not poison us directly, might we be unable to compete with it for resources? Even if we could adapt to it, we might not do so in time. As the National Academy's Space Science Board speculated: "exotic soil organisms with unfamiliar metabolic capabilities conceivably could sequester a nutrient, such as fixed nitrogen, in a stable form which could not be attacked or utilized by terrestrial organisms. In time, the terrestrial flora would experience nitrogen starvation." (31) Without nitrogen, protein production would stop and life would soon end. The chairman of the NAS Board later stressed the need to consider the potential hazards, however remote they may be: "While the risks cannot be calculated, concern is based not on irrational fear of the unknown, but on appreciation of how very serious the consequences may be. It is this awareness that prevents us from dismissing the danger as trivial, just because the risk of disaster from back contamination -- even though we

(31) Space Science Board, Conference on Potential Hazards of Backcontamination from the Planets (Washington, D.C.: National Academy of Sciences, Revised, February 19, 1965), p. 5.

cannot compute it -- must surely be very small." (32)

By joining the genes from multiple sources, microbiologists can create hybrid DNA molecules and, on inserting them into bacteria, new genetic instructions can be passed on to the bacteria's offspring. The DNA of these "recombinant" forms of life may have pathogenic properties. Thus, scientists can now "create" bacteria which might be the carriers of disease. As the Berg Committee, the group which in 1974 alerted the world to the potential dangers of recombinant DNA research, warned: Some experiments would "result in the creation of novel types of infectious DNA elements whose biological properties cannot be predicted in advance." (33) Erwin Chargaff, a pioneer of molecular biology, whose work led to the successful prediction of the DNA structure, was more outspoken about the "awesome irreversibility" of the risks: "You can stop splitting the atom; you can stop visiting the moon; you can stop using aerosols; you may even decide not to kill entire populations by the use of a few bombs. But you cannot recall a new form of life." (34)

Even the indiscriminate use of antibiotics can lead to the creation of new pathogenic organisms: antibiotic-resistant strains of bacteria. All organisms undergo random mutations,

(32) A. H. Brown, "'Back Contamination' and Quarantine Problems and Perspectives," in Biology and the Exploration of Mars, ed. by C. S. Pittendrigh, W. Vishniac, and J. P. T. Pearman, (Washington, D. C.: National Academy of Sciences, 1966), p. 444. [emphasis Brown's]

(33) Paul Berg, et al., letter, Science, 185 (July 26, 1974), p. 303.

(34) Erwin Chargaff, letter, ibid., 195 (June 4, 1976); p. 938.

with those able to compete with the predominant types surviving -- new dominant strains emerge rarely since the changes in the DNA are more likely to hinder than improve the odds of survival. With a rapid reproductive rate bacteria can test many new variations in a short time. Antibiotics will wipe out the majority strains, allowing new resistant genetic types to grow and multiply. For many years, immune bacteria have been isolated in hospitals but now such strains are emerging in environments with less direct antibiotic pressure. Growing abuse of antibiotics is contributing to a global problem. (35) In some countries, antibiotics are sold over the counter; in the United States, a prescription is required -- though evidence is mounting that doctors are ignorant about many aspects of their use. (36) Most controversial is the widespread addition of antibiotics to cattle feeds to promote growth: in 1975, nearly half of all antibiotics produced in the United States were fed to animals. (37)

The fear of PCP's arises from the ability of the bacteria to transfer their antibiotic resistance (coded within their DNA on the so-called "R" plasmid) from one strain to another. In 1977 alone, doctors in the United States have

(35) Janice Crossland, "Power to Resist," Environment, 17 (March 1975), pp. 6-11.

(36) H. C. Neu and S.P. Howrey, "Testing the Physician's Knowledge of Antibiotic Use," New England Journal of Medicine, 293 (December 18, 1975), pp. 1291-1295.

(37) Colin Norman, "Action on Antibiotics," Nature 269 (September 8, 1977), p. 99.

documented the resistance to one or more antibiotics in gonorrhea, (38) salmonella, (39) pneumonia, (40) and tuberculosis. (41) Up to now, the drug companies have always had an effective antibiotic in reserve to control resistant bacteria. But the costs have already been large. Substitute drugs are often expensive -- in some cases ten times greater -- and in order to fight off a disease, doctors may be forced to prescribe toxic substitutes. Some strains of Haemophilus influenzae, the bacterium which causes meningitis in children, are now resistant to penicillin. As a result children diagnosed with this type of infection are being treated with chloramphenicol, a strong antibiotic which can damage bone marrow. (42) And during 1972-1973, chloramphenicol resistant bacteria in Mexico caused many thousands of cases of typhoid with a death rate, according to the World Health Organization, "comparable to the pre-antibiotic era." (43) Similarly bacteria

(38) Morbidity and Mortality Weekly Report, 27 (January 13, 1978), pp. 10, 15.

(39) Ibid., 26 (April 22, 1977), pp. 129-130

(40) Ibid., 26 (September 2, 1977), pp. 285-286, and ibid., 26 (October 21, 1977), pp. 345-346.

(41) Ibid., 26 (December 23, 1977), pp. 417-418, 423.

(42) Barbara J. Culliton, "Drug Resistance Growing Worse," Science, 194 (December 24, 1976), p. 1396. A few isolated cases of chloramphenicol resistant Haemophilus influenzae have already been reported. Morbidity and Mortality Weekly Report, 25 (December 10, 1976), p. 385.

(43) "WHO Committee Reports Increasing Antibiotic Resistance in Intestinal Bacteria Recommends Improved Education on Risks of Antibiotic Abuse," United Nations Press Release, H/2462, November 3, 1977.

resistant to many different antibiotics caused epidemics of dysentery in Central America and Bangladesh during the late 1960's and early 1970's. (44) One day the costs may be much greater: there may not be a potent substitute antibiotic available, and a pandemic will result.

4. Natural Hazards. This last type of PCP is different from the others in that the occurrence of natural disasters cannot be controlled by human decisions. Though we may not be able to stop a natural upheaval from occurring, we can reduce its severity through the regulation of settlement patterns and building codes in high-risk areas and encouraging investments in hazard prediction and warning systems.

Natural disasters routinely cause large-scale misery. In 1976, some 20,000 people were killed in a Guatemalan earthquake, and the following year an equal number died when a cyclone hit the eastern coast of India. But there is also the potential for much greater catastrophes: a series of earthquakes and tidal waves in or around highly urbanized areas could kill millions. The 1976 earthquake in Tangshan, a city in Northern China, caused more than 650,000 deaths and an equal number of injuries! (45) While no American earthquake has ever claimed more than a thousand lives, the potential for catastrophe is everpresent. An estimated 31 million Americans in 21 states live

(44) Ibid.

(45) "Quake Toll in China Put at 665,000 in Report Said to Come from Area," New York Times, January 6, 1977; and Andrew H. Malcolm, "Chinese Disclose that 1975 Quake was Deadliest in Four Centuries," New York Times, June 2, 1977.

in "high" seismic risk zones. (46)

The perceived seriousness of the earthquake threat has prompted a crash effort in earthquake prediction so that people can be evacuated before a disaster strikes. Of course, the policy issues associated with imperfect early warning systems are themselves thorny: the displacement of huge populations may in itself have serious economic and social impacts. (47) This lesson was learned by the French government after it ordered the evacuation of the entire population of Guadeloupe (73,600 people) on the basis of what now seems to be an unsupportable prediction that the island's volcano, La Soufriere was about to erupt. (48)

B. What Would It Be Like?

No one can predict what living through and surviving a catastrophe would be like. Studies of the Holocaust, the Irish potato famine, the atomic blasts over Hiroshima and Nagasaki, the black plague, and the countless wars and epidemics paint a horrifying picture. Beyond the grim statistics of death and disease and the obvious economic ruin, there are the psychological repercussions.

Some 63,000 to 240,000 Japanese died in the first atomic blast in Hiroshima. As terrifying as the thought of being

(46) Senate Committee on Commerce, Earthquake Hazard Reduction Act, 94th Congress, 2nd Session, Report No. 94-862, 1976, p. 7.

(47) National Research Council, Earthquake Prediction and Public Policy, (Washington, D.C.: National Academy of Sciences, 1975).

(48) Haroun Tazieff, "La Soufriere, Volcanology, and Forecasting," Nature, 269 (September 8, 1977), pp. 96-97.

caught in the mushroom cloud is that of being a survivor. As Robert Jay Lifton documented, the catastrophe did not end so quickly for them -- life and death became inextricably intertwined. (49) Among survivors, there was a "frequent sense of being 'as-if-dead,' or what [Lifton] called an 'identity of the dead,' which took the following inner sequence: I almost died; I should have died; I did die or at least am not really alive; or if I am alive, it is impure of me to be so, and anything I do which affirms life is also impure and an insult to the dead, who alone are pure." (50)

The late historian, William Langer wrote about the black plague, which he thought was "in all probability, the greatest single disaster that has ever befallen European mankind." (51) During the first ravages of 1348-1349, most localities lost a third or even a half of their population within a few months. The plague would continue for the next three centuries. As might be expected, Europe entered a period of economic decline, but less well known, is the mood of those times, one described by Langer as marked by "misery, depression, and anxiety, and a general sense of impending doom." (52)

(49) Robert Jay Lifton, Death in Life, (New York: Vintage, 1969).

(50) Robert Jay Lifton, Boundaries, (New York: Random House, 1969?), p. 13.

(51) William L. Langer, "The Next Assignment," American Historical Review, 63 (January 1958), p. 292. See also: Langer's "The Black Death," Scientific American, 210 (February 1964), pp. 114-121.

• (52) Ibid., p. 297.

Langer warns of the long-term psychic impact of any major catastrophe:

[I]t is perfectly clear that disaster and death threatening the entire community will bring on a mass emotional disturbance, based on a feeling of helpless exposure, disorientation, and common guilt. Furthermore, it seems altogether plausible to suppose that children, having experienced the terror of their parents and the panic of the community, will react to succeeding crises in a similar but even more intense manner. In other words, the anxiety and fear are transmitted from one generation to another, constantly aggravated. (53)

Conclusion

All these catastrophes present non-adaptive social risks. We learn about most hazards through experience, adjusting to a given risk by a reiterative process of public outcry and corrective action. But PCP risks are different: there is no opportunity to learn about and adapt to them. If the catastrophe happened, the impact would be enormous and irreversible; the equilibrium point of the society would be lost, and the new one, when attained, might bear no relationship to the old one. One catastrophic event becomes one too many. The society is therefore forced to learn about risks without ever experiencing them. While some may argue that this has always happened in the past such as in the introduction of agriculture or the mass production of automobiles, both one way paths with uncertain consequences, there are now so many different and interrelated PCP's that the chances of catastrophe are becoming less and less remote.

(53) Ibid., pp. 229-300.

Another way to think about the magnitude of these catastrophes is by realizing that we cannot insure ourselves against them: the losses are too great. Insurance companies spread risks such that large numbers of individuals and businesses can pay a small premium to be protected against the possibility of a large loss. The costs of a dam rupture, a chemical plant explosion, or an airplane crash, all relatively small disasters compared to the typical PCP, can exceed the pooled resources of a group of insurance companies. (54) In such cases and more generally for PCP's, even the government, the insurer of last resort, may be unable to spread the risks over the whole population to yield an acceptably small per capita payment.

The Price-Anderson Act, passed in 1957, limits the aggregate liability for a single nuclear incident to \$560 million. This is a gross underestimate of the potential losses compared with the projected costs of a major accident. According to even the most conservative analysis, the property damages alone are some thirty times greater than this overall liability -- and this is before the costs of disease and death have been factored in. In early 1977, a Federal court in North Carolina found that the Price-Anderson Act was unconstitutional, violating the equal protection and due process provisions of the Fifth

(54) K. A. Solomon and D. Okrent, Catastrophic Events Leading to De Facto Limits on Liability, Report No. ENG-7732 (UCLA: School of Engineering and Applied Science, May 1977).

Amendment. (55) The Supreme Court struck down these objections in its June 1978 ruling. The unanimous opinion argued that the 1957 law was a "classic example of an economic regulation." Congress had designed it "to remove the economic impediments in order to stimulate the private development of electric energy by nuclear power while simultaneously providing the public compensation in the event of a catastrophic nuclear incident."

(56) The State of California had not been convinced that the federal government would in fact repay the injured parties after an accident. The State's Resources Agency had joined the suit against the Price-Anderson Act because it too believed that the government could not, or would not, adequately compensate it for its losses. (57)

In the particular case of nuclear power, there is an institution that is identifiable as the source of the disaster, the utility operating the reactor. In most other PCP's, responsibility is less easily affixed, either because the disaster is a result of diffuse social activities (carbon dioxide build-up and antibiotic resistance) or because the source of the disaster is not readily identifiable due to a delay in cause and

(55) Carolina Environmental Study Group, Inc. vs. U.S. Atomic Energy Commission, March 31, 1977.

(56) United States Law Week, June 27, 1978, p. 46 LW 485. In a concurring opinion, Justice Stewart went even further: "there has never been such an accident and it is sheer speculation that one will ever occur. For this reason I think there is no present justiciable controversy, and that the appellees were without standing to initiate this litigation." Ibid, p. 46 LW 4854.

(57) Amicus Brief on Behalf of the Resources Agency, State of California, February 15, 1978.

effect. (If a recombinant bacterium causes a pandemic, can we find out which laboratory created it?) In such cases, there is no law to challenge; the liability is simply limited by the society's ability and willingness to cover all the costs.

Thus the growing number of PCP's commit us to collective, involuntary, and non-adaptive risks, different from the conventional dangers which we have learned to struggle with. How much do we know about the nature and degree of these risks? As I shall show in the next chapter, not very much.

Chapter 3

UNCERTAINTY

The future is inherently unpredictable -- what is to come is, by definition, unknowable, and therefore uncertain. Beyond this somewhat obvious fact, the term uncertainty is used ambiguously: in some contexts, we use it to refer to what we will know in the future, and in others, about what we can never know. In this chapter, I develop a framework for thinking about the various kinds of scientific uncertainty. (58)

When a coin is flipped into the air, we do not know whether it will, on landing, reveal a head or a tail. If a person offers a bet to a friend, and the amount riding on the outcome of the toss is small, each accepts the odds as 50-50, both perceiving the uncertainty to be governed by chance. We have all tossed coins before, and we understand the process. We are uncertain about a single toss while being certain about tossing. As the potential gains and losses grow, we may become more circumspect: we may question whether the coin is "fair" -- are there in fact a head and a tail, and if so, are they equally probable? These concerns reflect a different kind of uncertainty: they involve what we do not know about the coin and the person offering the bet. We have made a transition from being uncertain about the future (the next toss) to uncertainty about the process of tossing. There could be further uncertainties. How would we react to a bet decided by the toss

(58) Early in the process of working on this chapter, I was influenced by Alvin M. Weinberg, "Science and Trans-Science," Minerva, 10 (April 1972), pp. 209-222; and Harvey Brooks's letter in reply to this article, ibid., 10 (July, 1972), pp. 484-486. (See also Weinberg in Science, 177 (July 21, 1972), 211 and letters, ibid., 180 (June 15, 1973), pp. 1122, 1124.

of a coin which would be tossed in another room: we could neither see nor test it. What if the coin is thick, and likely to land on its edge? And further: how could we even be sure there was indeed a coin in the other room and that someone there would toss it?

I shall try to sort out these various ideas by distinguishing among four types of uncertainty:

Type 1 Uncertainty: can be defined in terms of outcomes and their associated probabilities. Type 1 uncertainty is completely defined and is illustrated by a coin toss.

Type 2 Uncertainty: can be resolved by research, which, though feasible, has yet to be done. Type 2 uncertainty is illustrated by the unknowns which become interesting when the amount riding on the outcome of the coin toss increases; we want to test it before betting.

Type 3 Uncertainty: can be resolved by research which we cannot yet do. This type of uncertainty is like type 2, with the complication that we are unable to clarify the unknowns. We are denied access to the coin.

Type 4 Uncertainty: cannot be estimated. In this case, we are not only prevented from seeing the coin, we are also unsure of the process used to determine whether we will win or lose.

In addition to all the scientific and technical uncertainties catalogued above, there is an additional one which is present in the implementation of all policies: human error. The safest of machine systems can succumb to an inadvertent mistake: the New York City blackout of 1977 could have been avoided if the "right" button had been pushed. (59) A margin for error must always be allowed in the risk-benefit equation.

The prediction of risk is dependent on what is known about the forces at work. The four types of uncertainties catalogued here provide us with a means of indexing risks. Table 3-1 summarizes which information is available for each type of uncertainty.

(59) Victor K. McElheny, "Con Edison Admits its Operator Erred," New York Times, August 29, 1977.

Uncertainty

<u>Type</u>	<u>Theory</u>	<u>Parameter</u>
1: Expected Value	known	known
2: Resolvable Uncertainty	known	unknown
3: Unresolvable Uncertainty	partially known	partially known
4: Inestimable Uncertainty	unknown	unknown

Table 3-1

There are other kinds of uncertainty which I do not include in this framework. Uncertainties also arise out of political conditions. Terrorists may turn to sabotage as a strategy to promote their objectives. While such threats must be entered into the cost-benefit analysis, they involve strategic risks like those due to war. The uncertainties catalogued in the taxonomy that follows are scientific and technical; our ignorance may lead us to pursue what may otherwise have been inappropriate policies, but we do not expect nature to confound us by suddenly changing the laws of physics. Nature is not an active opponent, as a political antagonist might be. The risks arising out of political upheaval can be weighed, with different alternatives having different likelihoods for subversion. Commercial nuclear power brings the possibility of diversion of radioactive materials; if they are of bomb grade, the dangers are awesome. Blackmailers would be in a strong bargaining position if they simply threatened random contamination with stolen radionuclides. On the other hand, fossil fuels entail no such risks -- though terrorists could black out a region by attacking an electricity generating plant, a danger common to all large scale installations.

Type 1 Uncertainty: Expected Value

Expected value is a summary measure of the expected costs and benefits of a proposed action, and can be calculated if all the relevant outcomes and their associated probabilities are known. The particular result observed is uncertain, but the set of all outcomes is known.

As the number of past cases with similar characteristics grows, the confidence we place in the prediction of anticipated outcomes, and their likelihoods, grows. For example, there are many uncertainties associated with the planning and building of a new oil-fired power plant: What will be the future demand for electric power? Will the emissions from the power plant aggravate the region's air pollution problems? How accurate will the cost estimates for the construction be? Which of the possible sites for the plant will be selected by government officials? These uncertainties can be handled by decision theory: for each question, there is only a small number of possible outcomes or ranges of outcomes, and the planner should have had enough experience to anticipate them and assign a widely agreed probability to each.

Type 1 uncertainty, as in the siting example, is quantified from available information and knowledge, both theoretical and practical. While there remain formidable obstacles associated with type 1 uncertainties and the use of

statistical decision theory, there does, at least, exist a reasonable approach to the problem.

The essential feature of type 1 uncertainty is that even though a given set of events have not yet taken place, similar ones have in other instances. To the extent that each case is different, its specifics are uncertain; but to the extent the case is a member of a class for which there is a historical record, it may be represented by formal models. For the power plant, the uncertainty can be incorporated into the analysis through energy demand curves, air pollution dispersion models, experience with cost estimates for construction materials, and the performance of local decision making units. (60) Though not rote, the analysis does not require much originality or imagination: the models may be flawed, but they are available and useable.

Type 2 Uncertainty: Resolvable Uncertainty

Often there exists sufficient technical knowledge for a given type of research to be done, but it has yet to be carried out. No new theories or technical breakthroughs are required but directed research must be completed before the uncertainty can be

(60) It could be argued that the impact of a power plant is much less predictable than the outcome of a coin toss. Certainly, policy questions cannot be as completely defined as flipping a coin or spinning a roulette wheel. These exact systems are presented for their heuristic value in describing the limiting, ideal case.

resolved.

Chlorofluorocarbons (CFC's) are widely used as spray can propellants and refrigerants. Until 1974, scientists believed them "inert." Today, the scientists are no longer so confident; in particular, there is a growing consensus that CFC's indirectly catalyze the destruction of the stratospheric ozone, which acts as a protective shield, filtering out ultraviolet light (UV) from the sun's incoming rays. (61) Once released into the atmosphere, the CFC's diffuse slowly, taking years to reach the upper atmosphere, where photolysis generates chlorine atoms, the initiators of the depleting reaction. In order to gauge the potential threat of CFC's, their sources, sinks, and reactions in the atmosphere must be known. Are there major non-human sources of CFC's such that the contributions from spray cans and refrigerants are negligible? Could they be more reactive in the atmosphere than presently believed, and therefore never reach the ozone layer? Will the reaction path leading to ozone destruction be followed? Or will there be other, competitive reactions which compensate for the effects of CFC's? These questions (62) can be

(61) There are many Type 3 uncertainties concerning the effect of increases in the global exposure to UV radiation, such as the impact on incidence of skin cancer and the biosphere's energy budget which, for instance, regulates photosynthesis and the general climate: these, as I shall discuss later, are problems which go beyond our present knowledge. Nevertheless, there are some questions which can be resolved by research based on existing theories.

(62) For a list of some of the original uncertainties in the CFC's controversy, see "Critical Research Areas," Council on Environmental Quality, Federal Council for Science and Technology, Fluorocarbons and the Environment (Washington, D.C.: Government Printing Office, June 1975), pp. 13-17.

at least partially answered on the basis of our present knowledge of chemical kinetics. Once reaction rate constants and the lifetimes of chemical species are known, scientists will be better able to determine which of the many competitive reactions dominate. This and other research in physical chemistry and photochemistry will indicate whether there are other sources or sinks for these molecules.

The theories underlying these branches of chemistry and physics are firm enough to allow the research to proceed; scientists know how to design and execute the necessary experiments. None of this means that the work is trivial; each experiment requires painstaking work and the results of individual research must be integrated to present a comprehensive view of the problem, a task which would be impossible without the aid of a computer to model the complex interactions.

While both type 1 and type 2 uncertainties can be resolved with the aid of existing theories, type 2 requires a broad experimentation program before the theories can be applied to a specific problem. Specific parameters are unavailable though a general theory does give direction. Thus, one important characteristic of type 2 uncertainty is the time and cost necessary to quantify it into type 1 uncertainty. There is a time delay between the definition of the problem and the results of the research.

The many experiments on CFC's in the atmosphere will

themselves take years. (63) Sometimes there is an added delay for the construction of the equipment to conduct the experiment. One of the central uncertainties associated with the safety of nuclear power plants is the possible failure of the emergency core cooling system (ECCS). If the core of a nuclear reactor loses its coolant, a second supply of water must flood the reactor if a "melt-down" (a major catastrophe) is to be averted. At this writing no actual test of the ECCS has ever been performed on a full-scale reactor. A loss of fluid test (LOFT) facility was intended to have provided some data on the reliability of the ECCS. Construction for the LOFT began in Idaho in 1963, and was not completed until 1975; tests did not begin until 1977. (64) Even after the testing is finished, LOFT will only provide a rough estimate of what might actually happen in case of an accident. The LOFT facility is less than 1/50th of the size of a typical nuclear reactor. As the study group on light-water reactor safety of the American Physical Society concluded: "Our overall assessment is that LOFT cannot be regarded as a proof test of PWR (pressurized-water reactor) ECCS behavior." (65) Therefore, after waiting more than 15 years, we

(63) National Research Council, Halocarbons, Effects on Stratospheric Ozone, and Halocarbons: Environmental Effects of Chlorofluoromethane Release, (Washington, D.C.: National Academy of Sciences, 1976).

(64) Ralph Nader and John Abbotts, The Menace of Atomic Energy (New York: Norton, 1977), pp. 106-7. For a general discussion of the ECCS controversy see pp. 100-108.

(65) Report to the APS by the Study Group on Light-Water Reactor Safety, " Reviews of Modern Physics, 47 Supplement No. 1 (Summer 1975), p. S76.

still do not know much about the risks of some types of major nuclear accidents, and there is little hope that we will learn much more in the near future.

A further example of the delay in resolving type 2 uncertainty is the time required to test a compound's carcinogenicity. Since toxicologists do not understand the theory of carcinogenesis, they must resort to indirect techniques of assessing the threat posed by chemicals. If there is a latent history of occupational illness, long-term epidemiological studies would indicate a potential hazard. More often scientists simulate human exposure with animal feeding studies, experiments which cost hundreds of thousands of dollars and take many years to complete.

In 1973, the Environmental Protection Agency (EPA) discovered that there were "large concentrations" of asbestos fibers in Duluth's water supply, Lake Superior. At the time, there was ample evidence that asbestos fibers were carcinogenic under certain, but varied, circumstances. (66) Asbestos had been shown to be carcinogenic when administered to animals by inhalation or injection, into the muscle, lung, and abdomen. Also, workers exposed to asbestos have a higher incidence of cancer of the gastrointestinal tract, including the stomach, colon, and rectum. All this is in addition to asbestos-caused lung cancer and abdominal cancer (mesothelioma) contracted not

(66) National Research Council, Asbestos: The Need for and Feasibility of Air Pollution Controls (Washington, D.C.: National Academy of Sciences, 1971).

only by occupationally-exposed workers but by their families in indirect contact (for instance, with asbestos brought home on overalls). Yet, because the primary evidence of harm was due to breathing rather than drinking it, there was no quick way to determine the carcinogenic potential of fibers in the water supply. Indeed, there will probably be no scientific data implicating carcinogenicity of water-borne asbestos until the end of the decade. (67) Until then, EPA officials can only respond to anxious queries as they did in 1973: There is "no conclusive evidence" to show that the lake water is unfit for consumption but that "prudence dictates that an alternate source of drinking water be found for very young children." (68)

Type 3 Uncertainty: Unresolvable Uncertainty

There are many questions which we cannot answer because we are ignorant of the underlying processes at work. Scientists simply do not have enough information to form theories upon which to base directed research.

Returning to the case of asbestos, if scientists understood how cancer causing agents acted on human cells, long-term animal testing might be avoided, and chemical

(67) In addition to Dr. Irving Selikoff's studies showing cancer of the G-I tract, Dr. Sidney Laskin has found that 25 to 50% of the inhaled particles of asbestos are likely to be coughed up and then swallowed (the fraction is dependent on particle size). Jane E. Brody, "Conferees Study Asbestos Hazard," New York Times, November 25, 1973.

(68) William E. Farrell, "Duluth Stoically Awaits Tests for Cancer Risk in Its Water," New York Times, June 24, 1973.

carcinogens might be predicted before they are marketed. It will still be many years before a comprehensive theory of cancer is developed; until then, the key lies in basic research on cell function. (69) A leading scientist chastised the government for not perceiving these needs: "The National Cancer Institute program today primarily suffers from the illusion that there exists enough basic knowledge about cancer on which to build an applied program. It is as if there had been a huge government program to put man on the moon in 1920, before missiles were invented." (70)

Similarly, molecular biologists are uncertain about many of the underlying processes which determine the risks of recombinant DNA research. Robert Sinsheimer, the chancellor of the University of California at Santa Cruz, and the past head of the Division of Biology at Caltech, has catalogued some of the uncertainties inherent in splicing fragments of DNA, possibly those containing the genes for the growth of tumor viruses, into the E. coli bacterium, a resident on the human bowel, and the most intensively studied of all living organisms:

We are in considerable measure ignorant of the factors governing the ecology of the bowel... We are ignorant of the normal role of the bowel flora in human nutrition ... or in carcinogenesis... We are largely ignorant of the effects of plasmids, prophages

(69) Some hope for predicting carcinogens lies in the development of quick tests, like the Ames Test. Bryn A. Bridges, "Short Term Screening Tests for Carcinogens," Nature, 261 (May 20, 1976), pp. 195-200.

(70) Ruth Sager, letter, New York Times, March 24, 1977.

et cetera on the fitness of bacteria for bowel survival... We are ignorant of the ecology of E. coli outside the bowel, of the factors that determine its capacity to invade the intestinal wall, or to colonize the nasopharynx... We are in large measure ignorant of the range and frequency of gene transfer throughout the prokaryotic world... We are ignorant of the many aspects of the complex microbiological equilibria that truly underlie and maintain the entire world of life in its present form... We are grossly ignorant of the structural gene content of the eukaryotic genomes that were introduced so blithely into this E. coli... We are ignorant of the nature and mode of transmission of slow viruses. Could their ingredients lurk in those random bits of genome we now juggle... We remain largely ignorant of the factors that restrict the spread of viral species among different hosts... Except in the most general terms we are ignorant of the broad principles of evolution, of the factors that govern its rate and directions. We have no general theorems to account for the spectrum of organisms that we see and the gaps in between... We are ignorant of any absolute measure of adaptation. We are ignorant of the depth of security of our own environmental niche... Simply, we are in the end, ignorant of the extent of our ignorance." (71)

Some of these unknowns are type 2 uncertainties, but taken as a whole, they are beyond the scope of present theories.

Nuclear power entails type 3 uncertainties as well as type 2 uncertainties about reactor safety. The nuclear power industry increases the public exposure to low doses of ionizing radiation. An assessment of this cancer hazard is little different from that due to chemicals -- both are beyond our present theories. (72) Moreover, scientists are raising the possibility of a new radiation threat: weather modification

(71) Robert L. Sinsheimer, "Potential Risks," in Research with Recombinant DNA. (Washington, D.C.: National Academy of Sciences, 1977), pp. 75-78.

(72) See Chapter 10.

caused by electrical disturbances initiated by krypton-85, a chemically inert gas produced by nuclear fission explosions and reactors. One scientist assessed the long term outlook this way: "Our present understanding of atmosphere processes is insufficient to determine the extent of consequent weather changes and whether they would be beneficial or harmful...global changes may last decades." (73) Thus, the potential long-term hazard of low-level radiation is fraught with at least two kinds of type 3 uncertainty: the unknown interactions between radiation and biological systems and the unknown interactions between radiation and climate.

The disposal of nuclear wastes is another formidable issue. High-level wastes from nuclear reactors and reprocessing plants are active for as long as a million years, that is forever. Is it possible to assure long-level containment of highly toxic radionuclides over geological time spans? According to the present evidence, the answer appears to be "no." An official paper by White House scientists warns that there is a general consensus among technical experts that "the knowledge and technology base available today is not yet sufficient to permit complete confidence in the safety of any particular repository design or the suitability of any particular site." (74) This

(73) William L. Boeck, "Meteorological Consequences of Atmospheric Krypton-85," Science, 193 (July 16, 1976), p. 198. See also exchange of letters: H.L. Gjørup and W.L. Boeck, ibid., 196 (April 22, 1977), p 380-381, 460.

(74) Quoted in Luther J. Carter, "Nuclear Wastes: The Science of Geologic Disposal Seen as Weak," Science, 200 (June 9, 1978), p. 1135.

view is supported by a team at the U.S. Geological Survey who reported: "some key geologic questions are unanswered, and answers are needed before the risk associated with geologic containment can be confidently evaluated." Many of the interactions, between hot wastes, host rock, and possible water in the rock, they go on, "are not well understood, and this lack of understanding contributes considerable uncertainty to evaluations of the risk of geological disposal of high level waste." (75) The uncertainties are starting to hurt the future development of nuclear power. In 1976, California passed a law preventing the siting of any new nuclear plants until the Nuclear Regulatory Commission had demonstrated that waste disposal technology was available. (76) In the fall of 1977, Gus Speth of the President's Council on Environmental Quality urged that expansion of this country's nuclear power program be contingent

(75) J.D. Bredehoeft, et al., "Geologic Disposal of High-Level Radioactive Wastes -- Earth-Science Perspectives," Geological Survey Circular 779 (1978), p. 3; see also: Luther J. Carter, "Radioactive Wastes: Some Urgent Unfinished Business," ibid., 195 (February 18, 1977), pp. 661-666, 704. Resolution seems a long way off. A British royal commission on nuclear power observed in 1976: "The picture that emerges from our review of radioactive waste management is in many ways a disquieting one, indicating insufficient appreciation of long-term requirements ... a much more urgent approach is needed." Royal Commission on Environmental Pollution, Sixth Report: Nuclear Power and the Environment (London: Her Majesty's Stationery Office, September, 1976), p. 162. For a more optimistic view, see Bernard L. Cohen, "The Disposal of Radioactive Wastes from Fission Reactors," Scientific American, 236 (June 1977), pp. 21-31; but see letters, ibid., 237 (October 1977), pp. 9-12. On another front, see: Todd R. LaPorte, "Nuclear Waste: Increasing Scale and Sociopolitical Impacts," Science, 201 (July 7, 1978), pp. 22-28.

(76) Luther J. Carter, "Nuclear Initiative: Californians Vote 'No,' but Legislature Acts," Science, 192 (June 25, 1976), p. 1317.

on the ability to safely dispose of the wastes. (77)

Type 3 uncertainty arising out of incomplete understanding of atmospheric processes impedes our analysis not only of radiation PCP's, but also of a large number of other human activities which may perturb the weather and climate. As I discussed in the last chapter, there are many unresolved uncertainties with respect to the role of particulates, carbon dioxide, and ozone in the earth's energy flows. Two examples illustrate how much there is to learn. The first one concerns the relationship between atmospheric dust concentrations and temperature variations. Two interactions of the particles are simultaneously at work: with incoming solar radiation and with the earth's atmospheric radiation. Scientists are unsure about the net result of either process, and therefore also of the sum of the two. A review of past studies on the solar radiation effect led one team to conclude: "A sampling of results is highly ambivalent; many studies have predicted cooling trends from increases in atmospheric dust concentration, while many others have predicted warming trends." (78) In reference to the earth's thermal radiation, they state: "These studies have not been as equivocal as the solar radiation studies, as almost all of them have predicted a warming trend with increasing atmospheric dust concentration. However, they have not been completely

(77) David Burnham, "Carter Advisory Panel Urges Nuclear Power Delay for Safety Studies," New York Times, September 30, 1977.

(78) Sherwood B. Idso and Anthony Brazel, "Planetary Radiation Balance as a Function of Atmospheric Dust: Climatological Consequences," Science, 198 (November 18, 1977), p. 731.

unambiguous either, yielding somewhat different heating rates for different experimental conditions." (79) The next step is to allow for the interaction of these two unpredictable processes -- we are left with an even more complex, and therefore uncertain, system. Estimates of human contributions to atmospheric particulate concentrations are equally vague, ranging between 5 and 45 percent. (80) Present studies cannot forecast the effects of policies which would tend to increase dust emissions, such as an increased reliance on coal to ease the present dependence on foreign oil.

The second example concerns the analysis of carbon dioxide build-up. Any policy decision on this potential hazard rests on an understanding of the sources and sinks of this gas. Up to very recently the common scientific wisdom held that plants were a sink for carbon dioxide in the global carbon cycle. Indeed, some scientists even believed that if carbon dioxide concentrations increased, it would stimulate further oxygen production. Now, new evidence suggest not only that such enhancement is illusiory but the plants are a net source of carbon dioxide! (81)

(79) Ibid.

(80) Inadvertent Climate Modification Report of the Study of Man's Impact on Climate (SMIC), (Cambridge, Mass.: MIT Press, 1971) p. 192. The National Academy's estimate is 11 percent. National Research Council, Weather and Climate Modification: Problems and Progress, (Washington, D.C.: National Academy of Sciences, 1973), p. 154.

(81) G. M. Woodwell, et al., "The Biota and the World Carbon Budget," Science, 199 (January 13, 1978), pp. 141-146.

The ultimate example of a type 3 uncertainty about PCP risks was the first test of the atomic bomb in Alamogordo, New Mexico. In those early days of nuclear physics research, scientists had only a rudimentary understanding of reactions initiated by the release of huge amounts of energy. Given the unknowns in the theory and its inherent complexities, there remained a small chance that the atmosphere would be ignited by the atomic blast: would the earth's atmosphere become a fire ball of rapidly oxidizing nitrogen, cremating all life on the planet? Research could have, and finally did, resolve the uncertainties. But a war was continuing in the Pacific, and an immediate test was deemed necessary by the military. On June 15, 1945, the day before the test, Enrico Fermi, the noted physicist, wryly invited "bets against the destruction of all human life and second just that of human life in New Mexico." (82) The odds of atmospheric ignition were estimated to be three in a million, based on the chance that an error had been made in the complex calculations. (83) Nuclear scientists are now sure that there was no chance of a final holocaust but only with the benefit of today's theories and the 1945 experiments!

(82) Nuel Pharr Davis, Lawrence and Oppenheimer, (New York: Fawcett, 1969), pp. 233-234.

(83) Ibid. p. 165. See also: H. C. Dudley, "The Ultimate Catastrophe," Bulletin of Atomic Scientists, 31 (November 1975), pp. 21-24; H. A. Bethe, "Ultimate Catastrophe?" Ibid., 32 (June 1976), pp. 36-37, and various letters, ibid., 32 (January and March 1976).

Type 4 Uncertainty: Inestimable Uncertainty

This final type of uncertainty is reserved for those problems which cannot be estimated by research. Sometimes we are physically incapable of performing the necessary experiments, or we might not have ever perceived the presence of a potential risk. No one may have ever thought to pose the necessary questions to indicate an unresolved hazard. Chlorofluorocarbons (CFC's) are an illustrative example of the latter case. Prior to 1974, scientists did not know that these compounds degraded in the stratosphere, causing damage to the earth's ozone layer, because no one had asked the questions which today seem so obvious: "If these inert chemicals are released into the atmosphere, where do they go?" And if they build up: "What are their long-term effects?"

The exploration of the moon and the planets is another example of the difficulty of dealing with type 4 uncertainty. As I have already argued, the return of a pathogenic alien organism, known as "backcontamination," entails catastrophic risks. If we are to protect ourselves against such a potential threat, we must be able to recognize alien life. This is not a simple task.

Substances which may be purposefully or accidentally brought back from a space mission may be divided into two classes: living and non-living. A living system is defined as

one that metabolizes and self-replicates. (84) An inanimate material must be extraordinarily toxic to threaten a pandemic; an exploratory party would have to return and distribute large amounts to create substantial risk. Though such an outcome is possible, a catastrophe originating from a living system is comparatively riskier: just one unit of a self-replicating organism might "backcontaminate" the earth. (Two units are needed if it reproduces sexually by pairs.) If the necessary nutrients were available and the environment were tolerant, the organism might proliferate with unknown results.

Living beings can be further divided into those which are sufficiently similar to terrestrial life to allow easy identification and those which, based on different biochemical principles, would elude our tests. The prevailing opinion of biologists is that all life must be like our own; the experiments taken to Mars on the Viking landers were based on the conclusion that "wherever life arises in the universe it will most likely be based on carbon chemistry." (85) Is this a parochial view? Could there be life forms unlike those which evolved on earth?

(84) Joshua Lederberg, "Exobiology Approaches to Life Beyond Earth," Science, 132 (August 12, 1960), pp. 393-400. A subset of the group of living systems is composed of those forms which may be described as intelligent. If the alien life was not only alive but intelligent, we would face a strategic problem. I assume that any being which could use its powers of thought to hitch a ride on a returning spaceship, without making its presence known to the crew or ground control, would itself be aware of its own potential biological danger. The risk would therefore turn on whether the alien was friendly or not.

(85) Norman H. Horowitz, "The Search for Life on Mars," Scientific American, 237 (November 1977), p. 54.

Such speculation has long been the province of science fiction writers: their imaginations have conjured up life forms based on silicon, an element which, though heavier, is chemically similar to carbon. (86) Further, they have considered life independent of a solid structure. (87)

But even without relying on these admittedly improbable notions, there are reasons to believe that recognizing life may be problematic. Living systems might be in a suspended state, metabolizing at an imperceptible rate, or not at all. Spores may be lying on the surface of the planet, ready to "come back to life" when local conditions permit. Given the harsh environments on the moon and the planets in our solar system, dormant beings

(86) Horowitz discounts the possibility of silicon based life: "No other element, including that favorite of science-fiction writers, silicon, has the capacity carbon has to form large and complex structures that are so stable. It is no accident that even though silicon is far more abundant than carbon on the earth, it has only minor and nonessential roles in biochemistry. Biochemistry is largely a chemistry of carbon." Ibid. In contrast to Horowitz's view, a panel convened by the Space Science Board of the National Academy of Sciences to look into the possibility of other types of life forms began with a more open view: "It would be as great an error to omit consideration of non-Earth-like biochemical possibilities as it would be to fail to look for DNA," G. C. Pimentel, et al., "Exotic Biochemistry in Exobiology," in Biology and the Exploration of Mars, ed. by C. S. Pittendrigh, W. Vishiac, and J. P. T. Pearman (Washington, D.C.: National Academy of Sciences, 1966), p. 243. If Horowitz has over-stated his case, it would not be the first time: I shall review some of his conclusions on the possibility of terrestrial organisms surviving on Mars in Chapter 8. Certainly, Horowitz has the available facts on his side. But, as Erwin Schrodinger recognized over thirty years ago, the laws of physics do not explain the workings of life. Until they do, the possibility of other forms of life is ever present. E. Schrodinger, What Is Life? (Cambridge, U.K.: Cambridge University Press, 1967 [1944]).

(87) Fred Hoyle, The Black Cloud, (New York: Signet, 1957).

are more likely sources of life than actively metabolizing ones. They may be indigenous but in a state of suspended animation, left over from an earlier time when the environment was more hospitable. Or, they may have arrived due to panspermia, a natural migration of life forms from one planet to another or its artificial dissemination by alien visitors. (88)

Conclusion

We have much to learn about PCP's. What is clear is that many of the uncertainties are of types 3 and 4: we cannot predict when we will be able to handle them. And even for those type 2 risks for which we already have a firm theoretical base, it will take a great deal of time to do the necessary risk assessments.

(88) Lederberg, op. cit., p. 396.

Chapter 4

COMPLEXITIES

"There are more things in heaven
and earth, Horatio, Than are
dreamt of in your philosophy." Hamlet

The last two chapters give little cause for optimism about what the future may be like. Some will argue that the chances of catastrophe are so remote that we have no reason to be afraid. I disagree. These problems are much too complex and uncertain for us to believe that we can safely discount their risks. Too often such arrogance has backfired: many things which we once thought impossible now seem likely. We simply cannot predict where our technologies are leading us, especially when so many of the mysteries of science must still be unraveled.

In this chapter, I shall present four examples of how the unlikely and the unsuspected have occurred: (i) the discovery that a drug can exert a toxic effect long after it is administered; (ii) the convergence of apparently unrelated pollution problems into larger ones; (iii) the difficulty in identifying which chemical is responsible for which effect; and (iv) the danger that there is an unknown pathogen lurking in our common medicines. All highlight the dangers of dismissing PCP's.

A. One Generation's Actions Affect

All Subsequent Generations

Past and future are obviously inextricably related. How could the children that come from the bellies of their mothers be free of the world created by their parents? Yet we

are beginning to realize that we can have very profound effects on our children's lives which go beyond the amorphous generalities we usually associate with progress and destiny. (89)

In my earlier discussion of catastrophic risks, I noted the hazards of marketing a drug designed to alter the reproductive system of fertile women. The use of hormones involves tinkering, under huge uncertainty, with the process of continuing human life on the planet. There is some small chance that birth control pills could render the users sterile, or otherwise prevent a generation of women from bearing children. Little attention was given to such possibilities until quite recently; diethylstilbestrol (DES) has forced us to reevaluate the risks. In 1971, a team of Boston doctors reported that there was a significant association between the taking of DES during pregnancy and the later development of cancer in the vagina of the parents' daughters. (90) This was the first documented case

(89) Trying to define "progress" is in itself a hazardous occupation, and I will take a very cautious stand by defining its opposite: any action which threatens to break the thread connecting generations cannot be deemed to be progress. We must not do anything which arbitrarily limits the lives of our children. As Lifton has claimed: "Man requires a sense of immortality in the face of inevitable biological death," and it is partly through our children that we can achieve such a sense of continuity." Robert Jay Lifton, Boundaries (New York: Random House, 1969?), p. 21.

(90) Arthur L. Herbst, Howard Ulfelder, and David C. Poskanzer, "Adenocarcinoma of the Vagina," New England Journal of Medicine, 284 (April 22, 1971), pp. 878-881. A study of women taking DES has shown that they have a higher, though not statistically significant, incidence of breast cancer. Marluce Bibbo, et al., "A Twenty-Five Year Follow-Up Study of Women Exposed to Diethylstilbestrol During Pregnancy," ibid., 298 (April 6, 1978), pp. 763-767.

of treatment of one generation causing cancer in the next, still unborn, one. Had the use of DES been more widespread and its effects more crippling, the drug could have stopped the reproduction of the society.

DES is a synthetic estrogen prescribed to help prevent miscarriages. (91) Before 1971, between half a million and two million women were exposed to DES. (92) The Mayo Clinic estimated the incidence of vaginal or cervical cancer among DES exposed daughters could be as high as 4 in 1000. This particular kind of cancer is so rare that there is no "normal" incidence rate. One doctor put it in perspective this way, "This [rate] is equivalent to the annual death rate from heart disease in New York State, is 44 times higher than the annual incidence rate from leukemia, and at least ten times higher than the incidences of breast cancer and colon cancer." (93) In 1977 Albert Herbst, one of the researchers who first identified the inter-generation effects of DES, reduced the risk estimate to between 1 in 10,000 and 1 in 1,000. Nevertheless, by his own estimate from 1951 to 1969 some 1% to 10% of all pregnant women in the United States

(91) DES has also been used as a cattle feed additive and, surprisingly, as a "morning after" contraception pill.

(92) Barry Kramer, "How Cancer in Women Was Linked to a Drug their Mother's Took," Wall Street Journal, December 23, 1975. See also Olli P. Heinonen, "Diethylstilbestrol in Pregnancy," Cancer, 31 (March 1973), pp. 573-577.

(93) Statement of Peter Greenwald, Regulation of Diethylstilbestrol (DES), 1975, Joint Hearing before Subcommittees of the Senate Committees on Labor and Public Welfare, and the Judiciary, on S.963, 94th Congress, 1st Session, 1975, p. 82.

took DES. (94)

These new statistics are welcome, but they do not negate the important lessons that DES can cause cancer in someone who has not yet been born, and that unlike the thalidomide induced deformities which were apparent at birth (only a few months after use) DES deceived mothers, doctors, and children for twenty years into thinking that it was safe. DES was administered to pregnant women over two decades before its effects were understood. What if the incidence rate had been much higher? What if the women need not have been pregnant at the time of taking the drug for their children to become diseased? Could there be similar undiscovered second generation effects in the children of the 150 million women who have taken the birth control pill?

Less physically direct, but no less a threat to human survival are the potential time-delayed hazards associated with the build-up of carbon dioxide in the atmosphere and the injection of ozone-depleting chemicals into the stratosphere. In each case, members of the present generation may be mortgaging their children's future, threatening life on earth. A technological optimist might assume that our children's children will be able to stop the warming trend by cleaning the atmosphere of carbon. In the same way that carbon-based plastics are now derived from petroleum, future generations may have to turn to

(94) Joan S. Lublin, "New Study Cuts Estimate of Cancer Risk for Daughters of Women Who Took DES," Wall Street Journal, May 3, 1977.

the air for carbon and survival. Similarly, it may be possible to replenish the ozone layer. But the burden is on our descendants to undo our mistakes. They must work to find answers so that they can live a full life. If progress, then, is the bequest of the accumulated knowledge of past generations, we are changing the rules of the game. Our children's inheritance is what we might call the opposite of information, the necessity to find solutions to the problems we have created.

B. E Pluribus Unum

We have learned that the earth's natural cycles are interconnected so that any one disruption leads to perturbations throughout the system. Sometimes the resulting impact is more damaging than the original one. For instance, when refuse containing toxic materials is improperly buried, the chemicals will leach out of the dump and contaminate surface and ground waters. If the chemical is persistent (like DDT), it may be concentrated up the food chain; eventually, it will poison local populations. What had been a solid waste management problem will have become a water pollution problem and much worse, a public health disaster. This example is a simple one compared to those we must now confront. Our manmade systems are becoming complex, and when they interact with the complex ecological cycles, a further, unnatural, level of complexity is obtained.

Over the last few years, there has been a growing recognition that rain water is becoming more acidic. Sulfur

dioxide, sulfates, and nitrogen oxides, routinely emitted from man-made sources, are converted into sulfuric and nitric acids in the atmosphere, thereby increasing the acidity (lowering the pH) of rain. Ironically, it seems that particulate control systems and tall stacks, required by some air pollution control plans, are aggravating this problem. Sulfur dioxide is adsorbed onto, and precipitated out by, the particulates. "It would appear, then, that these trends in fuel consumption, fuel preference and population control technology (increasing the height of smokestacks and installing particle precipitators) have transformed local 'soot problems' into a regional 'acid rain' problem." (95) This was the first lesson in complexity: attempting to control one hazard, particulate emissions, led to the creation of a new, potentially much more serious one. Increasing the acidity of rain water could cause tremendous damage to all forms of life dependent upon a stable environment, as well as destroy valuable crops and materials.

A second lesson centered on the overuse of fertilizers, which are now suspected agents of ozone depletion. (96) We have

(95) Gene E. Likens and F. Herbert Bormann, "Acid Rain: A Serious Regional Environmental Problem," Science, 184 (June 14, 1974), p. 1177. See also Gene E. Likens, "Acid Precipitation," Chemical and Engineering News, November 22, 1976, pp. 29-44.

(96) Michael B. McElroy, et al., "Sources and Sinks for Atmospheric Nitrous Oxide," Review of Geophysics and Space Physics, 14 (May 1976), pp. 143-150; Deborah Shapley, "Will Fertilizers Harm Ozone as Much as SST's?" Science, 195 (February 18, 1977), p. 658; Harold S. Johnston, "Analysis of Independent Variables in the Perturbation of Stratospheric Ozone by Nitrogen Fertilizers," Journal of Geophysical Research, 82 (April 20, 1977), pp. 1767-1772; and letter to Science, 195 (March 25, 1977), p. 1280.

already seen that aerosol propellants and refrigerants are capable, on reaching the stratosphere, of dissociating and catalyzing the destruction of the ozone layer. Some researchers believe that the growing production of fixed nitrogen (97) might perturb the atmospheric nitrogen cycle, leading to further ozone destruction. To balance the cycle, the fixed nitrogen must eventually be returned to the atmosphere through "denitrification." Bacteria, living primarily in bogs, convert the bound nitrogen back into free nitrogen gas. These same bacteria which produce atmospheric nitrogen also yield nitrous oxide, some of which slowly diffuses up into the stratosphere, where it reacts to destroy the ozone. Many found this series of steps hard to believe, but further research has supported the early hypotheses.

Unfortunately the ratio of nitrogen gas to nitrous oxide produced by the bugs depends on the acidity of the bog.

(98) The greater the acidity of the bacteria's home the greater their production of nitrous oxide at the expense of nitrogen gas replacement. Thus, two already complex PCP risks, acid rain and ozone depletion, have converged into a third risk. (99)

(97) Fixing nitrogen requires breaking the stable nitrogen-nitrogen triple bond, freeing the element for biological and chemical processes. Nitrogen is fixed during any high temperature combustion process. Burning oil and gasoline in power plants and automobiles are examples of nitrogen fixing through oxidation. Chemical fertilizers, are the major human source of fixed nitrogen.

(98) Johnston, op. cit., p. 1968.

(99) Michael B. McElroy, "Threats to the Atmosphere," Harvard Magazine, 79 (February, 1976), pp. 24-25. An opposing view holds

There is a long time delay before these dangers could be upon us. Before then, some other part of the ecosystem may disequilibrate, forcing revisions in the original risk estimates. If the rain water becomes too acidic, it might kill the denitrifying bacteria, or so endanger crops that fertilizers would be of little benefit. Here again the uncertainties, nested within the complexities, prevail.

If we continue on our present course, all of the earth's major cycles (the carbon, oxygen, nitrogen, and sulfur cycles) will be jointly disrupted. At that point, man will no longer know how to tinker with the process to make it right. Perhaps a new non-catastrophic equilibrium point will be reached, but there may be wide variations in the earth's environment before a calm is restored. One can only guess at what surviving through such a cataclysmic period would be like.

C. Who's on First?

Over the last few years, the threat of known toxic chemical substances has grown practically daily. Vinyl chloride, benzene, mercury, lead, PCB's, dioxin, and asbestos are the most commonly known, but the list goes on and on. There are now some 63,000 chemicals in common use -- an enormous number, but still a tiny fraction of the four million chemicals that have been

that while McElroy's scenario is a "logical theoretical extrapolation," it has "little support from available data." Hugh W. Ellsaesser, "Has Man Increased Stratospheric Ozone?" Nature, 270 (December 15, 1977), p. 592.

catalogued. (100) The chemical industry has become a major part of the U.S. economy: in 1975, the synthetic organic chemical industry alone produced 155 billion pounds of 8,000 compounds for a total value of \$25 billion. (101) Not all of these chemicals are toxic, but it is difficult to sort out which chemicals will have which effect even one at a time. The present approach to regulation is based on the assumption that a chemical is safe until proved otherwise. This innocent-until-proven-guilty standard is desirable for human crimes, but poses substantial risks when applied to chemicals; it condemns many of us to serve as guinea pigs. We are slowly remedying such regulatory failures. In 1976, Congress passed the Toxic Substances Control Act, which, among other provisions, requires manufacturers to notify the Environmental Protection Agency of an intent to market new chemicals and new uses of old chemicals. A strategy of prevention will be difficult to implement under the best conditions, but we are discovering that in some cases the hazardous substance is a product of common and relatively innocuous, chemicals acting together. In other instances, we do not know which chemical is responsible for which toxic effect.

Nitrosamines, a class of extremely potent carcinogens, illustrate the problem. Nitrosamines are formed by the reaction of amines with nitrous acid. Amines are constituents of

(100) Thomas H. Maugh II, "Chemicals: How Many are There?" Science, 199 (January 13, 1978), p. 162.

(101) U.S. International Trade Commission, Synthetic Organic Chemicals United States Production and Sales 1975 (Washington, D.C.: Government Printing Office, 1977), p. 3.

proteins, and are therefore ubiquitous. Nitrous acid is also common, being a product of the atmospheric reactions of nitrous oxide, a gas emitted from all high-temperature burning processes. When the two chemicals meet, they form nitrosamines. (102) Any attempt to control nitrosamines must be addressed to their precursors, nitrous oxide and amines, since no one releases nitrosamines directly into the environment. But amines are the stuff of amino acids, and amino acids are the building blocks of proteins, the ingredients of life itself; and nitrous acid is produced whenever one burns anything at a high temperature. Not surprisingly, nitrosamines are becoming pervasive; a recent study found them in urban air, soil, water, and sewage treatment waste.

(103) EPA has yet to devise an effective strategy to control these chemicals, and it will be a long time before our early warning system will be able to alert us of these kinds of synergistic toxic actions.

On another front, there are those chemicals which appear to be non-toxic, but, once inside the human body, are metabolized into cancer-causing agents. In early 1977, John Finklea and his associates at the National Institute for Occupational Safety and Health warned that two "relatively innocuous chemicals, N-pheny-B-naphthalamine (PBNA) and

(102) Adding nitrates and nitrites to color and preserve fish and meat (especially bacon) can also lead to the formation of nitrosamines.

(103) Deborah Shapley, "Nitrosamines: Scientists on the Trail of Prime Suspect in Urban Cancer," Science, 191 (January 23, 1976), p. 268.

2-nitronaphthalene, once ingested, could be metabolized into the known carcinogen, B-naphthalene (BNA). (104) Some 15,000 workers are exposed to PBNA alone, primarily in the rubber industry. Until we have a more detailed understanding of our bodies' various metabolic pathways, we will be unable to design comprehensive testing protocols.

Beyond the dangers of human toxicity are the myriad environmental effects, which might boomerang back to hurt us. I can think of no better example of the difficulties of predicting potential environmental harm than the case of fluorocarbons. As I have already noted, before 1974, these compounds were believed to be inert. By that time, total global production of the two principal, ozone-depleting chemicals had exceeded ten billion pounds. (105) Sometimes, the substitution of one chemical for a hazardous one can have unpredictable consequences. Methyl chloroform has been replacing trichloroethylene because the latter substance is carcinogenic and contributes to urban photochemical smog; now scientists have discovered that methyl chloroform could deplete stratospheric ozone. (106)

(104) Roscoe H. Moore, et al., letter, Science, 175 (January 28, 1977), p. 344.

(105) Council on Environmental Quality -- Federal Council for Science and Technology, Fluorocarbons and the Environment, (IMOS Report), (Washington, D.C.: Government Printing Office, June 1975), p. 83.

(106) J. C. McConnell and H. I. Schiff, "Methyl Chloroform: Impact on Stratospheric Ozone," Science 199 (January 13, 1978), pp. 174-177. See also letters, ibid., 200 (May 19, 1978), pp. 718-722.

D. A Shot in the Arm

Any mass inoculation program using a vaccine tested only for short term efficacy entails some risks. Mild reactions to all vaccines are expected in a small fraction of the population, and a much smaller group suffers more severe complications. The at-risk population for these effects is too small to show up on routine small-scale testing. Sometimes a batch of vaccine will be contaminated, causing severe reactions. As the number of persons to be inoculated grows, the special at-risk groups become more noticeable, and a new and qualitatively different risk emerges: the vaccine may be contaminated with a slow acting toxin, and before any remedial action can be taken, millions of people may have it injected into them.

Lest the possibility of inadvertently injecting some toxic agent into huge numbers of people seems too remote to be considered, precisely this type of accident occurred during the mass polio immunization drives of the mid-1950's. In 1955, soon after the vaccine was introduced, some of it was contaminated: ten people died and 192 others came down with polio. (107) High level Federal officials were forced to resign. Many years later a potentially much greater hazard was discovered. The polio vaccines were found to contain a virus now known as Simian Virus 40 (SV40). SV40 causes cancer in newborn hamsters. Thus, from

(107) Nicholas Wade, "Division of Biologics Standards: In the Matter of J. Anthony Morris," Science, 175 (February 25, 1972), p. 861.

1955 to 1961, when the contaminant was discovered and removed, tens of millions of people were inoculated with a polio vaccine containing a potential carcinogenic agent. (108) Happily, SV40, while able to infect humans, does not seem able to cause them cancer -- though one statistically significant Australian study showed that compared to controls, a larger number of children with cancer also had received a polio shot; other studies have reported no association between the pre-1961 polio vaccine and cancer. (109) Recent studies, however, have implicated the SV40 virus with chronic central nervous system diseases in humans. Dr. Andrew Lewis of the National Institute of Allergy and Infectious Diseases (NIAID) has urged those who work with SV40 to be cautious: "Until satisfactory studies evaluate the long-term effects of SV40 infection in humans and clarify the relationship between SV40 and SV40-related agents to chronic degenerative central nervous system disease in humans, it appears to [me] that the laboratory manipulation of SV40 involves some risks." (110)

The epidemiological evidence on possible effects of SV40 is fragmentary and far from conclusive. By 1957, some 60

(108) The Division of Biologics Standards, the government department with the responsibility for regulating vaccines, did not recall those lots of vaccine already on the market when the contamination was found, lest the public confidence in the vaccine be undermined. Nicholas Wade, "Division of Biologics Standards: The Boat that Never Rocked," Science, 175 (March 17, 1972), pp. 1224-1225.

(109) Andrew M. Lewis, Jr., "Experience with SV40 and Adenovirus-SV40 Hybrids," in Biohazards in Biological Research, ed. by A. Hellman, M.N. Oxman, and R. Pollack (Cold Spring Harbor, N.Y.: Cold Spring Harbor Laboratory, 1973), p. 99.

(110) Ibid., p. 106.

million people under the age of 50 had received one or more inoculations, and three quarters of them were under 20. (111) Estimating the amount of SV40 in the various lots of vaccines has been exceedingly difficult. One study found that of the approximately nine and a half million children between the ages of six and eight who received the vaccine between May and July, 1955, nearly four million received what was considered to be a high dose of SV40, and another three and a half million received a low dose. (112) Despite the huge number of people exposed, surveillance has been scattered: the records of only some 6,764 people have been examined during the intervening years. Reviewing the available studies in 1973, Dr. Lewis concluded: "there has been no systematic, in-depth follow up study of persons receiving SV40-contaminated poliomyelitis vaccines ... while there appear to be no untoward short-term effects from human infection with SV40... As it is now about 15-20 years since the general use of these contaminated vaccines, the time for the appearance of any untoward long-term effects may soon be at hand." (113)

The polio accident occurred because the vaccine was prepared in monkey cell cultures which harbored the SV40 virus. Could vaccines used today contain other possibly dangerous

(111) Joseph F. Fraumeni, Jr., et al., "An Evaluation of The Carcinogenicity of Simian Virus 40 in Man," Journal of the American Medical Association, 185 (August 31, 1963), p. 715.

(112) Ibid.

(113) Lewis, op. cit., pp. 99, 101.

contaminants? The answer is surely yes -- this being an example of type 4 uncertainty. It is hard to find something when you don't know what you are looking for.

Some precautions can be taken, however. The most probable sources of contamination are the cells used to grow the vaccines; they may contain bacteria which are themselves infected by viruses (bacteriophages). Some of these viruses may persist and be in the vaccine. (114) By studying the bacteria which are indigenous to the species in whose cells the vaccine is cultured, the researcher has a starting point for searching for possible viral contaminants.

(114) John C. Petricciani, et al., "Bacteriophages in Live Virus Vaccines," Proceedings of the Society for Experimental Biology and Medicine, 144 (December 1973), pp. 789-792.

PART II

RISK PERCEPTION AND DECISION MAKING

Chapter 5

A MODEL OF PCP RISK PERCEPTION AND DECISION MAKING

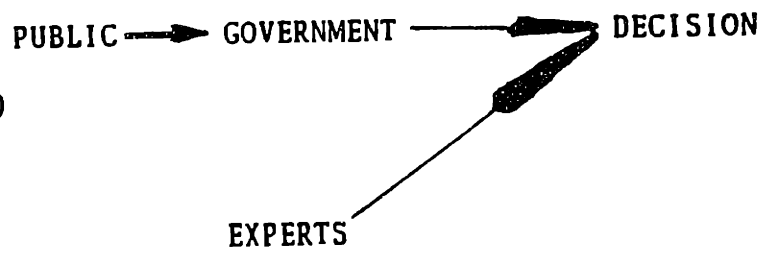
For my purposes, I shall simplify the social decision making process by assuming that there are three types of participants: individuals, experts, and government. In later chapters, I shall discuss how members of each type perceive, or more appropriately misperceive, potential PCP risks. Here I propose three models to show how these decision makers interact. The key variable is the flow of information. Since PCP risks are so obscure, individuals must depend on the experts to identify possible new sources of risk as well as specifics about those of which the public is already aware.

There are three possible models of interaction among the public, the experts, and the government in reaching social decisions on LPC's. (See Figure 5-1.)

Model 1: Individuals make their preference known to the government, which, working in consultation with the experts and the public, reaches some decision on how to deal with the risk in question. This model is based on a democratic view of social decision making. The government acts in the interest of its citizens and seeks to limit those risks which the public finds unacceptable. In this case the decision analyst and the expert play a secondary role, which is limited to clarifying the issues and modeling the policy implications of alternative actions. Thus, the government decision makers begin with the public's perceived risks and comparing these with those of the experts, deduce a level of acceptable social risk.

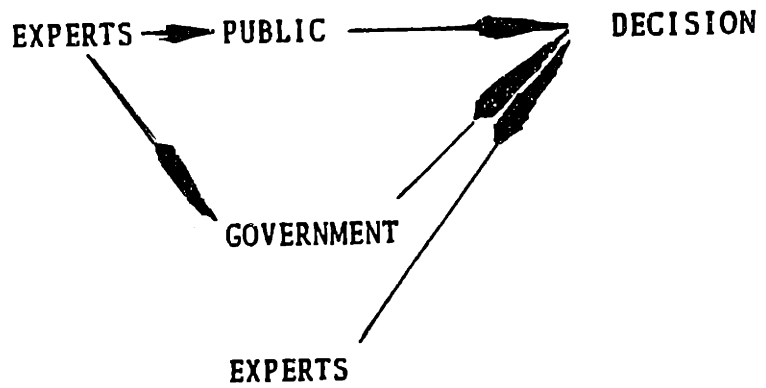
MODEL 1:

(DEMOCRATIC MODEL)



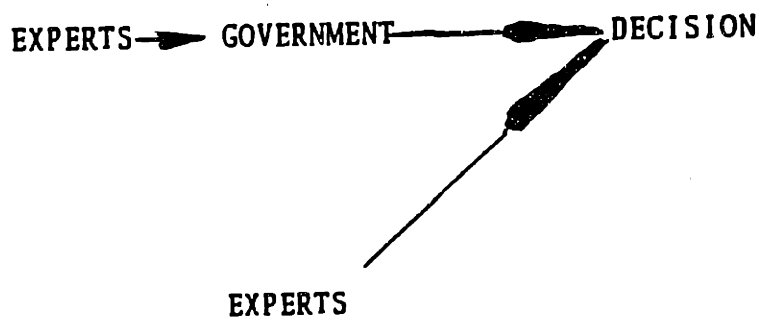
MODEL 2:

(EXPERT MODEL)



MODEL 3:

(GOVERNMENT-
EXPERT MODEL)



Flow of Information

Figure 5-1

Examples of risks whose intensity is determined according to Model 1 are airplane, automobile, and fire safety, as well as most natural hazards: See Table 5.1 -- (though the public has difficulty assessing the risks associated with rare, but severe disasters like some floods and earthquakes, see Chapter 6).

Types of Catastrophes

<u>MODEL 1</u>	<u>MODEL 2</u>	<u>MODEL 3</u>
Airplane Safety	Antibiotic Resistance	Comprehensive Swine Flu Immunization
	Atmospheric Particles	
Automobile Safety	Birth Control Pills	
Common Diseases	Carbon Dioxide Build-up	A-bomb Test at Alamogordo
Crime	Diethylstilbestrol (DES)	Microwaves
	Earthquakes (Large)	
Dam Safety	Fluoridation	Military Weapons
Economic Security	Food Additives & Chemicals & Drugs	
Most Natural Hazards	Nuclear power	
Thalidomide	Ozone Depletion	
	Planetary Quarantine & Backcontamination	
Zeppelins	Recombinant DNA Research	
	Running Out of Oxygen	

Table 5-1

We are all aware of such risks, and when we perceive them to be too great, we demand that additional precautions be taken. For instance, as soon as the teratogenic effects of thalidomide became apparent, it was forced off the market. Public reactions to crime, economic security, and disease also play a primary role in establishing government policy.

Model 2: The experts must notify the government and the public of a given risk. The three parties then work together to set the appropriate policy. Because of the technical nature of a growing number of public policies, most people are unaware of the risks each one may present. The burden then falls on the expert to publicize otherwise obscure dangers. Once an expert or a group of experts has generated an awareness of a potential risk, Model 2 reduces to a modified type of Model 1: the democratic process with the aid of extensive consultation with the appropriate experts determines the acceptable level of the new risk. Here, the government begins with the experts' estimates of the risk, and translates them into a social risk level by consulting the public and its own analysts.

Examples of this case abound: the experts have warned of the risks of recombinant DNA experiments, ozone depletion, carbon dioxide build-up, the return of a pathogenic alien organism, and growing antibiotic resistance in bacteria. See Table 5.1. Some of these issues have attained widespread public attention, while others have generated only scant publicity.

Model 3: This is a special case of Model 2, in which, either by design or default the public is not a party to the decision on the desired level of a risk. According to Model 3, decisions are made only by the experts and the government. Thus, only the experts' and the analysts' evaluations of a potential hazard enter into the risk-benefit equation. The public is bypassed when there is no time for open consultations, and, most often, in the interest of national security.

When influenza scientists predicted a swine flu pandemic in March 1976, there was too little time to establish a national consensus on the desirability of a full national immunization drive if the vaccine was to be produced and distributed before the 1976-1977 flu season. The government does not inform the public about the risks arising out of weapons programs (the first atomic bomb test at Alamogordo) or the ancillary effects of military hardware (the hazards of microwave radiation).

Chapter 6

INDIVIDUALS AND PCP'S

A. Individual Decision Making

Even in the best of circumstances, making decisions is troublesome. Our short-term memory is small, and we need time to transfer thoughts in and out of long-term memory. (115) Beyond the physical obstacles, simply thinking about decision problems is difficult. We only consider a small and manageable number of outcomes of a decision, artificially reducing the uncertainties. Such observations led Herbert Simon to devise a psychological theory of administrative decision making in the 1940's. (116) According to Simon's model, individuals choose courses of action which are "good enough," rather than searching for the best ones; they satisfice. At the heart of his theory is the principle of "bounded rationality": "The capacity of the human mind for formulating and solving complex problems is very small compared with the size of the problems where a solution is required for objectively rational behavior in the real world -- or even for a reasonable approximation to such objective rationality." Simon goes on: "If the principle is correct, then the goal of classical economic theory -- to predict the behavior of rational man

(115) For a general discussion of many of these issues see: George Miller, "The Magical Number Seven, Plus or Minus Two: Some Limits in Our Capacity for Processing Information," Psychological Review, 63 (March 1956), pp. 81-96; Herbert A. Simon, "How Big is a Chunk?," Science, 183 (February 8, 1974), pp. 482-488.

(116) Herbert A. Simon, Administrative Behavior, 2nd edition, (New York: Free Press, 1965), p. 81; see also pp. 242-242. There are many parallels between Simon's theory of "bounded rationality" as applied to individual decision making and Lindblom's theory of "muddling through" as applied to comprehensive-rational models of formal analysis: see Chapter 10.

without making an empirical investigation of his psychological properties -- is unattainable." (117)

If we do not follow an economically rational path, how then do we make decisions and perceive risks? Two distinct types of models have evolved to describe risk taking behavior. One school of thought holds that people perceive risk in terms of probability distributions over the possible outcomes. This has been called the "moments" approach: Individuals decide among gambles by comparing the moments of the distribution: expected value, variance, and skewness. (A person may use only one of these measures at any one time.) The alternative view stresses the importance of certain dimensions of the risks -- the probability of winning and losing, and the amounts which can be won and lost. In the "dimensions" approach, individuals choose among gambles by comparing one or more parameters of the risk. (118)

Both models of decision making behavior are consistent with the finding of limited cognitive skills. According to the "moments" model, we are acting in a manner similar to that described by the normative economic decision theories, but we do so "imperfectly." Non-optimality arises out of intrinsic limits in our ability to organize and structure the available information, and out of a lack of motivation to find the best

(117) Ibid., p. 199.

(118) John W. Payne, "Alternative Approaches to Decision Making Under Risk: Moments Versus Risk Dimensions," Psychological Bulletin, 80 (1973), pp. 439-453.

solution. The "dimensions" view holds that we are purposefully or haphazardly selecting out elements of the available information. Paul Slovic and Sarah Lichtenstein, two of the leading proponents of the dimensions school, stress the application of "importance beliefs" towards these parameters. Their hypothesis is simple: a person tends to pay "more attention to some risk dimensions than to others because he believes that these particular dimensions are most important for his present decision." (119) Of course, this is not very helpful for building a predictive model! Both models are still largely speculative. Among individuals, an inconsistent approach to risks is the rule rather than the exception. With regard to the dimensions model alone, experiments show that subjects give greater weight to the probability of winning than that of losing (120) while others have the opposite preference. (121)

One of the less credible aspects of the moments model is its assumption about the way people combine information to estimate the parameters which describe the distribution of probabilities over outcomes. Shepard's research confirms a limited ability to unify the risk parameters: "although a small number of attributes evidently can be combined according to a

(119) Paul Slovic and Sarah Lichtenstein, "Relative Importance of Probability and Payoffs in Risk Taking." Journal of Experimental Psychology, Monograph, 78 (November, 1.968), p. 1.

(120) Ibid., p. 14.

(121) Paul Slovic, "The Relative Influence of Probabilities and Payoffs Upon Perceived Risk of a Gamble." Psychonomic Science, 9 (1967), pp. 223-224.

simple or linear additive rules, non-linear or complex interactions between variables seem to offer great conceptual difficulty." (122) He concludes: "the confidence that we have tended to invest in our rational ability to weigh and combine many subjective factors appears to have been somewhat misplaced."

(123) Slovic and Lichtenstein's experiments provide added support: "The picture of the decision-making process that emerges ... is one of a person struggling to integrate several sources of information into a single choice or judgment." (124)

As I have already noted, psychologists know little about how people retrieve information from long-term memory. As a result, we can only speculate on the way people perceive the probabilities and outcomes of a decision. Nevertheless, we can safely assume that individuals rely on past experiences, logical deduction, and their imagination in estimating risks. Two psychologists, Amos Tversky and Daniel Kahneman, designed some elegant experiments to explain how they do it. They conclude that "people rely on a limited number of heuristic principles which reduce the complex tasks of assessing probabilities and

(122) Roger N. Shepard, "On Subjectively Optimum Selections Among Multi-Attribute Alternatives," in Human Judgments and Optimality, ed. by M. W. Shelley, II and G. L. Bryan, (New York: Wiley, 1964), p. 263.

(123) Ibid., p. 267.

(124) Slovic and Lichtenstein, op. cit., p. 15.

predicting values to simple judgmental operations," and "In general, these heuristics are quite useful, but sometimes they lead to severe systematic errors." (125)

Tversky and Kahneman offer three principal heuristics: representativeness, availability, and adjustment and anchoring. With the aid of the representativeness heuristic, people estimate the probability of one event by comparing it with another which they believe "represents" it, or is similar to it. They give the following illustration:

consider an individual who has been described by a former neighbor as follows: "Steve is very shy and withdrawn, invariably helpful, but with little interest in people, or in the world or reality. A meek and tidy soul, he has a need for order and structure, and a passion for detail." How do people assess the probability that Steve is engaged in a particular occupation from a list of possibilities (for example, farmer, salesman, airline pilot, librarian, or physician)? How do people order these occupations from most to least likely? In the representativeness heuristic, the probability that Steve is a librarian, is assessed by the degree to which he is representative of, or similar to, the stereotype of a librarian. Indeed, research with problems of this type has shown that people order the occupations by probability and by similarity exactly the same way. (126)

When a person assesses probability and frequency "by ease with which instances or occurrences can be brought to mind," he is using the availability heuristic. (127) In one study,

(125) Amos Tversky and Daniel Kahneman, "Judgment Under Uncertainty: Heuristics and Biases," Science, 185 (September 27, 1974), p. 1124.

(126) Ibid., see also Daniel Kahneman and Amos Tversky, "On the Psychology of Prediction," Psychological Review, 80 (1973), pp. 237-251; and "Subjective Probability: A Judgment of Representativeness," Cognitive Psychology, 3 (1972), pp. 430-454.

(127) Ibid., p. 1127.

subjects were asked whether the consonants 'K,' 'L,' 'N,' 'R,' and 'V' appeared more frequently in the first or third position in words taken from any random English text. (For instance as in king and lake.) Kahneman and Tversky found that "each of the five letters was judged by a majority of subjects to be more frequent in the first than the third position. ... These results were obtained despite the fact that all letters were more frequent in the third position." (128) Since, as they point out, it is "much easier to search for words by their first letter than by their third letter," (129) what probably happens is that people count words in each class, and assume that the ratio would be no different if the search were exhaustive.

The third heuristic, anchoring and adjustment, involves an estimation that starts "from an initial value that is adjusted to yield the final answer. The initial value, or starting point, may be suggested by the formulation of the problem or it may be the result of a partial computation." (130) They cite as an example their experiments on subjects who were asked to rapidly estimate the products,

8x7x6x5x4x3x2x1

or the same factors in reverse order,

1x2x3x4x5x6x7x8.

(128) Amos Tversky and Daniel Kahneman, "Availability: A Heuristic for Judging Frequency and Probability," Cognitive Psychology, 5 (1973), p. 212.

(129) Tversky and Kahneman, "Judgment Under Uncertainty," op. cit., p. 1127.

(130) Ibid., p. 1128.

The group offered the first set consistently judged the answer to be larger than those offered the second set of numbers: the median for the ascending sequence was 512, while that for the descending sequence was 2,250. The correct answer is 40,320. Once again, Tversky and Kahneman's explanation is appealing: the subjects begin by multiplying from left to right and, after a couple of computations, finding themselves out of time, adjust the answer up. (131)

Tversky and Kahneman's work shows that, left on our own, we assess probabilities in ways that are subject to gross distortions. Also, one can infer from their heuristics that individuals do not actually distinguish between an outcome of a decision and its associated probability. We fuse these two variables together. For example, if probabilities are determined by the availability heuristic, it is inappropriate, if not misleading, to assume that an individual first thinks about all the possible outcomes and then assigns a probability to each. For, in the process of listing outcomes he is also working out the likelihood of each happening.

Psychologists still have a long way to go before they will be able to predict how choices are made, even in the controlled environment of the laboratory. As one review summed

(131) Ibid.

it up:

it seems that the concept of risk is psychologically meaningful but highly elusive. Expected value, variance, number of independent plays, probabilities of winning and losing, and other transformations of two-outcome gambles all affect the perceived riskiness of gambles in one way or another, making the development of a satisfactory theory of risk a very difficult task. (132)

Thus, I have no comprehensive theory of decision making upon which to base an analysis of individual attitudes towards PCP's. Nevertheless, some of the experiments cited above have some general implications about judgments on complex questions reached under conditions of uncertainty. I turn to this problem now.

B. Uncertain and Complex Risks

People have a difficult time making sound, or logically consistent decisions in the simplest of situations, but they do even worse working with the highly complex, uncertain, and incomplete information characteristic of PCP's.

Tversky and Kahneman suggest that "in evaluating the probability of complex events only the simplest and most available scenarios are likely to be considered." (133) Specifically, they deduce that when estimating the probability of complex events people show "unwarranted optimism in the evaluation of the likelihood that a plan will be completed on

(132) Amon Rapoport and Thomas S. Wallsten, "Individual Decision Behavior," in Annual Review of Psychology, 23 (1972), p. 145.

(133) Tversky and Kahneman, "Availability," op. cit., p. 229.

time" on the one hand, and on the other, "tend to underestimate the probabilities of failure of complex systems." (134) In the first case, success depends on completing each of a series of tasks, and in the second, failure may occur in any one of many components. Thus, at the same time, individuals tend to be too optimistic about the chances of finishing a nuclear plant on schedule, and too sanguine about its safety. Moreover, this false sense of optimism is compounded by the more general finding supported by a variety of investigators that individuals tend to place too much confidence on the reliability of their judgments. (135)

To the extent that we base probability assessments on past experience using the availability heuristic, we tend to underestimate the probability of rare events. Research on human response to natural hazards, especially Robert Kates's studies, supports this view. (136) For instance, in 1962, Kates attributed the difficulty in achieving better flood control to people's inability to conceive of floods which were larger than those they knew from personal experience. (137) Kunreuther's

(134) Tversky and Kahneman, "Judgment Under Uncertainty," op. cit., p. 1129.

(135) Paul Slovic, Baruch Fischhoff, and Sarah Lichtenstein, "Behavioral Decision Theory," Annual Review of Psychology, 28 (1977), p. 6.

(136) For an excellent review, see Paul Slovic, Howard Kunreuther, and Gilbert F. White, "Decision Processes, Rationality, and Adjustment to Natural Hazards," in Natural Hazards: Local, National, Global, ed. by G. F. White, (New York: Oxford University Press, 1974), pp. 187-214.

(137) Cited in "Decision Processes," op. cit., p. 194.

recent field survey of attitudes toward flood insurance is consistent with Kates's earlier findings; he concludes that "individuals find it too difficult to assess the probability of [low probability] events on which to estimate the potential losses." (138) Overall, it is hard to argue with Kenneth Boulding's observation that "we make our decisions, and quite reasonably so, by regarding a very small probability, for all practical purposes, as zero. We all know when we take a plane that there is a certain small probability that we won't come back, but the probability is so small that we ignore it." (139)

When people have something to associate with, they can magnify rare risks and make sense, correctly or not, of obscure risks. Those who have lost a close relative or friend in a plane crash will probably perceive the probability of another plane accident as being greater than those who have not experienced such a loss. An illustration of this process for speculative risks occurred in Britain right after a group of microbiologists publicized the uncertain and mysterious hazards associated with recombinant DNA research. The British government responded

(138) He also notes: "The expected utility model, as traditionally used by economists, provides relatively little insight into the individual choice process regarding the purchase of insurance." Howard Kunreuther, "Limited Knowledge and Insurance Protection," Public Policy, 24 (Spring 1976), p. 250. Zeckhauser agrees: "Much of the difficulty [in the assessment of low probability health hazards] arises because neither individual citizens nor policy makers are well equipped to assess their personal evaluation of low probabilities." Richard Zeckhauser, "Procedures for Valuing Lives," Public Policy, 23 (Fall 1975), p. 444.

(139) Kenneth Boulding, "The Importance of Improbable Events," Technology Review, 78 (February 1976), p. 5.

quickly: it set up a committee to assess the risks, making England the first country to issue a government report of these biohazards. One of the reasons attributed for the speedy reaction was that a year earlier, a laboratory technician at the London School of Hygiene and Tropical Medicine had infected two outsiders with smallpox, both of whom subsequently died. So when the recombinant DNA hazards were publicized, they met a British public which was already apprehensive about other risks related to laboratory research. Later scientists would try to make sure that there was no public confusion between the predictable hazards associated with "natural" laboratory pathogens and the unpredictable hazards associated with recombinant DNA experiments. (140)

Risk assessment is further hampered by an individual's desire to avoid uncertainty. Daniel Ellsberg, who became a national celebrity by releasing the "Pentagon Papers" to the press, has shown that, when offered a series of choices, people act to reduce what he calls "ambiguity," something that is related to the "confidence" one has in the estimates of relative likelihood. (141) Subjects consistently act "'as though' the worst were somewhat more likely than [their] best estimates would indicate." (142) A propensity to avoid uncertainty has also been

(140) "Forever Amber on Manipulating DNA Molecules?" Nature, 256 (July 17, 1975), p. 155.

(141) Daniel Ellsberg, "Risk, Ambiguity, and the Savage Axioms," Quarterly Journal of Economics, 75 (1961), p. 657.

(142) Ibid., p. 667.

observed in business decision making and governmental policy making. (143)

C. Factors That Influence Risk Perception

Chauncey Starr has divided risks into two types: "voluntary" and "involuntary." (144) This dichotomy makes intuitive sense: as he puts it, "we are loathe to let others do unto us what we happily do to ourselves." (145) Economic pressures have clouded the definition of what is in fact voluntary: a person may hate flying, but may not have the time to take a train across country to a business meeting. I prefer the less ambiguous terms, "active" and "passive" risks. (146)

Lowrance has offered a list of factors which he believes influence safety judgments. (See Table 6-1.)

(143) Paul Slovic, "Limitations of the Mind of Man: Implications for Decision Making in the Nuclear Age," in Risk vs. Benefit: Solution or Dream, ed. by Harry J. Otway, (Springfield, Virginia: National Technical Information Service, February 1972), Report No. LA-4860-MS, p. 47.

(144) Chauncey Starr, "Social Benefit Versus Technological Risk," Science, 165 (September 19, 1969), p. 1237. He claims that the "public is willing to accept 'voluntary' risks roughly 1,000 times greater than 'involuntary' risks" and that "the statistical risk of death from disease appears to be a psychological yardstick for establishing the level of acceptability of other risks."

(145) Ibid., p. 1235.

(146) S. A. Goudsmit, letter, Science, 183 (February 22, 1974), p. 702.

An Array of Considerations Influencing Safety Judgments

Risk assumed voluntarily	Risk borne involuntarily
Effects immediate	Effect delayed
No alternatives available	Many alternatives available
Risk known with certainty	Risk not known
Exposure is an essential	Exposure is a luxury
Encountered occupationally	Encountered non-occupationally
Common hazard	"Dread" hazard
Affects average people	Affects especially sensitive people
Will be used as intended	Likely to be misused
Consequences reversible	Consequences irreversible

Table 6-1 (147)

(147) W. W. Lowrance, Of Acceptable Risk: Science and the Determination of Safety. (Los Altos, California: William Kaufman, 1976), p. 87.

And I too have my own list: in addition to the already discussed difficulties in assessing complex and rare risks, five other characteristics play a major role in influencing the way we perceive PCP risks: (i) time delay, (ii) affected group, (iii) benefits, (iv) responsible group, and (v) age.

(i) Time Delay

There are two time variables: a possible delay between a decision and its impact (now-later), and how long it takes for the impact to be manifest -- from an instant to generations (bang-fizzle). The longer the time delay and/or the actual catastrophic event, the smaller the perceived threat. The "now" catastrophe projects a greater danger than the "later" one; and a "bang" PCP appears more risky than a "fizzle" PCP. Table 6-2 gives examples of PCP for each of the four types.

	<u>NOW</u>	<u>LATER</u>
<u>BANG</u>	Nuclear Reactor Accident	Backcontamination From a Mission to Mars
<u>FIZZLE</u>	Cancer Pandemic	Skin Cancer Due to Ozone Depletion

Table 6-2

There is little experimental evidence on the nature of risk-taking behavior for delayed effects. As a recent review noted: "It is surprising that, with the exception of a few studies using gambles as stimuli, the determinants of perceived risk remain unexplored... [an] untested notion is that hazards with delayed consequences (e.g., smoking) are discounted." (148)

(148) Paul Slovic, Baruch Fischhoff, and Sarah Lichtenstein, "Cognitive Processes and Societal Risk Taking," in Cognition and Social Behavior, ed. by J. S. Carroll and J. W. Payne, (Hillsdale, N.J.: Lawrence Erlbaum, 1976), p. 180. Psychologists have shown that individuals will choose riskier alternatives if there is a delay between the decision and the consequences, but the time delay in the experiments ranged only from 30 minutes to 3 hours, too short to apply to PCP risks: the effects of ozone depletion, for example, do not become apparent for decades. Edward E. Jones and C. Anderson Johnson, "Delay of Consequences and the Riskiness of Decision," Journal of Personality, 41 (1973), pp. 613-637.

Nevertheless, we are more apt to take risks if the adverse outcomes, should they occur, are in the future, especially if the benefits are collected in the present. "I'll cross that bridge when I come to it," and "we'll have thought of some way to solve the problem long before then" are common responses to "later" risks.

The willingness to assume that we can assure the containment of radioactive wastes over hundreds of centuries is the most obvious example of a confidence in future innovations. However perilous the disposal problem may be, at least we are constantly reminded of it as the wastes accumulate. Other PCP's offer no such warnings: sometimes the first manifestation of the potential catastrophe is itself delayed. Today's policies on chlorofluorocarbons (CFC's) will determine the extent of ozone depletion at the beginning of the next century -- the gases diffuse slowly into the stratosphere, where the catalytic destruction itself takes time. The environmental effects of increasing UV radiation may also be delayed. Twenty-first century scientists may discover a way of replenishing the ozone layer, or perhaps some still unknown, ozone-producing reaction will take over when the ozone concentration falls below some minimum level. Whatever the outcome, the people who decide the extent of CFC's regulation will not witness the catastrophe if the emission restrictions prove to be insufficient and no other solutions are found.

That people attach more importance to one large accident (bang) than to many small ones in which the total number

of deaths is the same (fizzle) is a widely known yet little understood social phenomenon. Such a preference may be the product of the communication media: newspapers and television give more-than-proportional coverage to large accidents. Is this a reflection of social concerns or the determinant of them? No one has yet resolved this chicken-and-egg problem. Nevertheless, examples of our distaste for multiple death incidents abound: a plane crash killing a hundred people is front page news, but an equal number is killed on highways each day and goes unreported (each fatality is only local news). (149)

(11) Affected Group

How a catastrophe is perceived depends on whom it would affect. There are three important factors to consider: first, is what I shall call the "us-them" dichotomy. Members of the "us" group are both the assessors of the risk, and may or may not also be members of the at risk population; the "them" population is left in the passive role, having no opportunity to decide about the risks. In the limiting case the us group makes decisions for the them group, for instance: the executive's (us) decision on the degree to which the company's workers (them) are exposed to a carcinogenic chemical, the bureaucrat's (us) decision in Washington, D.C., on the extent to which dams across the country should be inspected to safeguard the lives (them) of those in the

(149) Joseph Ferreira, Jr. and Louis Slesin, Observations on the Social Impact of Large Accidents, Report No. 122 (Cambridge, Mass.: MIT Operations Research Center, October 1976) p. 33. See also Chapter 10.

path of a possible deluge, and my own decision on whether or not to throw a burning cigarette into the woods from a moving car, placing hikers at risk of fire. Clearly, when the decision maker is not in danger, he will perceive the risks to be smaller than when he is. (150) The difference in perceived risk between the two groups is, in part, the same as in the now-later risk dichotomy. A disaster which does not occur until late in the future will not hurt anyone now alive. Replacing "us" for "now" and "them" for "later," the two perceptual biases are equivalent.

The second variable is what I shall call the "namedness" of the affected population. The term is an adaptation of the distinction between a "statistical" and a "named" life, and is best explained by Thomas Schelling's description of the effort we make to save an identified individual's life:

Amelia Earhart lost in the Pacific, a score of Illinois coal miners in a collapsed shaft, an astronaut on the tip of a rocket or the little boy with pneumonia awaiting serum sent by dogsled -- even the heretofore anonymous victims of a Yugoslavian earthquake -- are part of ourselves, not a priceless part but a private part that we value in a different way, not just quantitatively but qualitatively, from the way we measure the incidence of death among a mass of unknown human beings, whether that population includes ourself or not. If we know the people, we care. (151)

(150) Sometimes when the risks are obscure, they may pass unnoticed by those in danger. Then the expert is expected to warn and protect those at risk.

(151) T. C. Schelling, "The Life You Save May Be Your Own," in Problems in Public Expenditure Analysis, ed. by Samuel B. Chase, Jr., (Washington, D.C.: Brookings Institution, 1968), pp. 129-130. Emphasis added. See also: Charles Fried, An Anatomy of Values, (Cambridge, Massachusetts: Harvard University Press,

While the us-them dichotomy identifies whether the perceiver of the risk is a member of the affected group, namedness describes his relationship to the group, if he is not at risk. In contrast to Schelling's individuals who are either named or statistical, the namedness variable spans the spectrum from a close relative or friend through acquaintances and celebrities, to an obscure individual mentioned in a news report. The greater the namedness of the group, the greater the perceived risk.

The third property of the affected group which can influence the way a risk is estimated is the perceived fragility of the at risk population. No risk is believed to be more outrageous than that which makes victims of "innocent" children, or pregnant women.

(iii) Benefits

As the benefits associated with a risk increase or appear to increase, the public's perception of the risk diminishes. Tversky and Kahneman's work on the availability heuristic is particularly relevant: if a person associates a policy with its positive attributes instead of its dangers, the risk will not loom as large. Starr, an engineer, agrees with the two psychologists: "The social acceptance of risk is directly influenced by public awareness of the benefits of an activity, as determined by advertising, usefulness, and the number of people

1970), Chapter 12: "The Value of Life." Schelling's observation is simply a restatement of the old farm rule: "if you're going to kill it and eat it, don't name it."

participating." (152) Starr is careful to point out that the benefits may be real or imagined. Thus he acknowledges the possibility of contriving public acceptance through advertising, producing "a fictitious benefit-risk ratio -- as may be the case for smoking." (153)

CFC's are used both as aerosol propellants and as refrigerants. Spray cans may be convenient but, except for a small number of medical uses, few people would claim that they improve our standard of living. Refrigerators, on the other hand, are essential parts of modern life. Who would argue that CFC's should be banned as a coolant, at least until a substitute is on the market? Today, CFC-based aerosols are being phased out, but there has been practically no public discussion of the risks of continuing their use in (and eventual release from) refrigerators.

The strategy of emphasizing the benefits of a potentially risky policy to make it more attractive has not been overlooked by advocates of genetic research. At a public hearing of Senator Adlai Stevenson's subcommittee on the risks and benefits of recombinant DNA experiments, the first witness was Philip Handler, the President of the NAS. He used the occasion

(152) C. Starr, op. cit., p. 1237. Starr has concluded that "the acceptability of risk appears to be crudely proportional to the third power of the benefits (real or imagined)." Ibid. Others have contested the validity of this cubic relationship; see Harry J. Otway and J. J. Cohen, Revealed Preferences: Comments On the Starr Benefit-Risk Relationships, Report No. RM-75-5, (Vienna, Austria: International Institute for Applied Systems Analysis, March 1975).

(153) Ibid.

to announce "a scientific triumph of the first order:" (154) scientists had made E. coli bacteria produce a protein, somatostatin, normally only found in animal and human brains. For the first time, microbiologists had been able to harness genes to suit their purposes. Word of this as yet unpublished breakthrough dominated the news reports of the hearing. The headline in the following morning's New York Times read: "Substance Usually Made in Brain Grown in Bacteria," (155) This article did not mention the dangers of recombinant experiments (the objective of the hearing), as described by others who testified that morning.

(iv) Responsible Group

Risks appear to increase as the number of identifiable groups or individuals responsible for them decreases. If a single corporation is marketing a product which can have catastrophic consequences, the risk will be perceived to be larger than if we are all contributing to the potential for disaster. Once a source of harm can be pinpointed, it is natural to affix "blame," and to seek a reduction of the risk.

The most dramatic illustration of this effect took place in a Texas courtroom in the early 1970's where a jury was hearing the case of Anita Reys, a victim of paralytic polio. In

(154) Statement of Philip Handler, Regulation of Recombinant DNA Research Hearings before the Senate Subcommittee on Science, Technology, and Space, 95th Congress, 1st Session, November 2, 1977, p. 13.

(155) Harold M. Schmeck, Jr. New York Times, November 3, 1977.

the spring of 1970, during a local poliomyelitis outbreak, Anita Reyes's parents took her to a county health clinic to be immunized. Fourteen days later, the child was diagnosed as a polio victim. The Reyes family sued the manufacturer of the vaccine, Wyeth Laboratories, for damages, claiming that the vaccine had caused the polio and that they had not been properly warned of the risks of immunization. At the trial, a professor from Johns Hopkins University estimated that the odds of catching polio naturally from the community at the time of the outbreak were 1 in 3,000. In contrast, there was undisputed testimony that the chances of catching polio from the vaccine were 1 in 5.88 million. Nevertheless, the jury found that the child's polio was caused by the vaccine and ordered Wyeth Laboratories to pay the family \$200,000. (156) The Supreme Court refused to hear the case. (157) Ascribing motives to juries can be a foolhardy exercise, but surely the jury preferred to rule against the drug company because otherwise the child would receive nothing -- and they would then be responsible for leaving the family destitute. In order to do so, the jurors had to choose the alternative which was 2,000 times more unlikely!

For many PCP's there is no single individual or

(156) William J. Curran, "Public Warnings of the Risk in Oral Polio Vaccine," American Journal of Public Health, 65 (May 1975), pp. 501-502.

(157) Reyes v. Wyeth Laboratories, 498 F.2d 1264 (5th Cir. 1974), cert. denied, 419 U.S. 1096 (1974). See also: Marc A. Franklin and Joseph E. Mais, Jr., "Tort Law and Mass Immunization Programs: Lessons from the Polio and Flu Episodes," California Law Review, 65 (July 1977), pp. 754-775.

institution to blame for the risks -- we are all contributing to the threat of disaster. People add to the concentrations of atmospheric carbon dioxide and particles whenever they drive their cars and burn oil to warm their houses. Also the steel plants which supply the raw material for cars and the boilers are major sources of such air pollutants. When we take antibiotics to guard against bacterial infections, we increase the chances of breeding antibiotic-resistant pathogens. In both PCP's the risk associated with any single person's actions is infinitesimal, but the aggregate impact of millions of people burning fuels and popping pills cannot be so easily discounted. Because we are all involved, we tend to perceive the dangers as being less severe than if the responsibility was focussed in just a few places.

(158)

The first step in tackling such diffuse threats is to set up an institution with a mandate to protect the population. In 1977, when the NAS expressed concern over the build-up of carbon dioxide from burning fossil fuels the Energy Research and Development Administration established the Office of Carbon Dioxide Effects, Research, and Assessment (now within the

(158) The formal model of this process is known as "The Tragedy of the Commons," or the "Prisoner's Dilemma." Garrett Hardin, "The Tragedy of the Commons," Science, 162 (December 13, 1968), pp. 1243-1248. For an economic analysis of the model see Mancur Olson, The Logic of Collective Action, (Cambridge, Mass., Harvard University Press, 1971). Olson is interested in the special problem of group provision of public goods. Since many of the PCP's involve the degradation of public goods such as the air we all breathe and the water we share, one of his conclusions is especially noteworthy: "the larger the group, the farther it will fall short of providing an optimal amount of a [public] good." p. 35.

Department of Energy). The carbon dioxide group is still doing research (159) -- when it comes time for action, their proposals will no doubt meet considerable opposition. One need only look at FDA commissioner Donald Kennedy's attempts to restrict the routine addition of antibiotics to cattle and poultry feeds to support this prediction. Faced with regulations on penicillin and tetracycline, farmers deny responsibility for the rise of antibiotic resistance among bacteria and point to the medical uses of antibiotics as the principal culprit. (Such a strategy is particularly effective for PCP's where many of the arguments are riddled with uncertainties.) At the same time, they warn of the dire economic consequences of government regulations. (160) As the number of contributors to a given risk grows, so does the likelihood that when confronted, one of them will point to the others.

The importance of having someone to blame is typified by what happened with CFC's. While all owners of refrigerators and users of spray cans are potential contributors to ozone depletion, there are in fact only a small number of chemical companies that produce CFC's, with Du Pont at the head of the list. As the evidence implicating CFC's grew, the public held Du

(159) "DOE Sets Interagency [Carbon Dioxide] Research Priorities," Science News, 112 (December 3, 1977), p. 375. See also note (430).

(160) For example, see the exchange of letters in the New York Times: Richard Novick of the Public Health Research Institute of the City of New York (November 21, 1977); Richard McDougal of the National Cattlemen's Association (December 19, 1977); and Werner Maas of New York University Medical Center (January 3, 1978).

Pont responsible, especially when it, trying to protect its market, began an advertising campaign to rebut those who raised the alarm. (161)

(v) Age

As risks age, we adapt to them. Sometimes we adjust by changing the way we live, reducing the probability and the severity of the hazard. Otherwise, we may block out the danger, and pretend it does not exist. The second reaction is especially worrisome -- denial can stop us from taking precautionary measures. A study of the attitudes of San Francisco residents towards earthquake risks found that the inhabitants gave the hazard a low priority. But the more interesting result was the resistance the interviewers met from potential respondents. The authors suggest that people may have not wanted to answer the questionnaire because it was long, or they didn't like to answer surveys, or perhaps they were suspicious of the interviewer. But the researchers give "denial of the earthquake problem" as the most important reason:

The introductory statement used to recruit the respondents mentioned the topic of earthquakes, which possibly discouraged a significant proportion of those who refused from participating. Common responses included "What do you want to know about that for?"; "Earthquakes don't bother me"; "Let it rock"; "We have no earthquake problem"; and so on. The tone of most such refusals suggested that many people are worried about the earthquake hazard but do not allow themselves

(161) "You Want the Ozone Question Answered One Way or the Other So Does Du Pont," Science, 190 (October 3, 1975), pp. 8-9, and letters, ibid., 190 (December 12, 1975), pp. 1038-1042.

to think about it. This may well be minimization of the earthquake threat through a process of dissonance reduction. (162)

Dissonance reduction is the centerpiece of Festinger's theory of a cognitive dissonance: people try to reduce dissonance, a psychologically uncomfortable condition, by avoiding information and situations which place them in a state of psychic conflict. (163) One of Festinger's favorite examples is cigarette smoking in the face of horrifying claims made by medical scientists: individuals suppress the information rather than control the urge.

As a policy ages, psychological mechanisms that discount risks are joined by parallel economic motives. The longer a policy has been in effect, the larger the financial investments which corporations and government will seek to protect. (Large institutions are already organized and can safeguard their interests more easily than individuals.) Whether the motives are profits or job security, the result is similar: new information on risks is disregarded or suppressed lest it jeopardize the economic status quo. In San Francisco, the individual reaction is due less to economic factors than to a desire to avoid moving and other risk-mitigating actions.

(162) Edgar L. Jackson and Tapan Mukerjee, "Human Adjustment to the Earthquake Hazard of San Francisco, California," in Natural Hazards: Local, National, Global, op. cit., p. 163.

(163) Leon Festinger, A Theory of Cognitive Dissonance (Stanford, California: Stanford University Press, 1962). His theory is partly an outgrowth of a case study of a group of people who predicted the destruction of the world. Leon Festinger, Henry W. Riecken, and Stanley Schachter, When Prophecy Falls (New York: Harper and Row, 1964 [1956]).

Nevertheless, if avoidance of earthquake risks became a primary national objective, the costs of relocating industry and reinforcing structural weaknesses would be enormous.

Others have raised these same issues in a more general context. Donald Schon writes of "selective inattention" to "data which would upset our current ways of looking at things." (164) Arthur Koestler speaks of "filters" which screen out unwanted information. Koestler decries government or dogmatic doctrines. Sardonicly, he detects an as yet unidentified interest group in the "Society for the Prevention of Cruelty to Dead Horses" (S. P. C. D. H.):

The German Government during the war killed six million civilians in its death factories. This was at first secret; when the facts seeped through, the S. P. C. D. H. took the line that to keep harping on them and bringing those responsible to trial was unfair and in bad taste -- flogging a dead horse. (165)

PCP's are especially susceptible to distortion over time. The uncertainties and gaps in knowledge associated with all PCP's are so large that it is easy for the mind to manipulate the data in order to construe them in the least threatening terms. The issues themselves, large-scale catastrophes, are perfect subjects for suppression. Surely there can be no more dissonant thoughts than these!

(164) Donald A. Schon, Beyond the Stable State, (New York: Norton, 1973), p. 14.

(165) Arthur Koestler, The Ghost in the Machine (Chicago: Henry Regnery, 1971), p. 349.

Conclusion

The perception of risk is a complicated and haphazard affair. While I can discern trends that influence individual judgments, it is impossible to predict the interplay among them. There is no model that describes how an individual will react to a given risk, especially complex and uncertain ones typical of PCP's.

What is clear is that untrained people have a difficult time evaluating any but the most common hazards: there are too many distortions at work. We cannot, and should not, rely on a Model 1 approach to PCP's. The public is therefore forced to rely on the experts (Models 2 and 3) in each of the PCP disciplines to explain which threats are indeed dangerous. The next questions are: What is the nature of the learning process between the public and the experts? And should the public trust the pronouncements of the experts? I shall address these in the following two chapters.

Chapter 7

LEARNING FROM THE EXPERTS

In the first part of this study, I emphasized the uncertain and complex nature of PCP risks. Whichever potential hazards one might wish to investigate, a skilled and trained mind is needed to sift through the mounds of technical information. The usual reaction on facing such a task is to ask the appropriate expert to evaluate the hazard -- often the person who notified the public that the risk existed in the first place. The expert finds himself the only one qualified to evaluate the hazards of his own work.

A. Inaccessibility

In each subject, the experts have evolved their own arcane language to describe their hypotheses and theories. In the case of recombinant DNA research, the medical and biological details are inaccessible to the uninitiated. A first glimpse of the jargon-filled journals is overwhelming: one is greeted by a cast of genomes, eukaryotes, phages, restriction endonucleases, nucleotides, and plasmids among others. The words act as hurdles to discourage further reading. With sufficient stamina and a glossary in hand, one can push forward, only to be met by other, theoretical problems: How do genes recombine? Could genetically altered E. coli be pathogenic, and if so can they be rendered harmless? How does the scientist know which genes are in a particular strand of DNA? And, which genes will recombine? These are not simple questions -- often the researchers themselves are unsure of the answers. One need only peruse a

standard text on microbiology or genetics to realize how much must be learned to speak with the experts.

Often, behind this barrier of words are concepts which we can all understand. But the evolution of science is accompanied by the development of obscure jargon, and risk assessment is thereby limited to the hardy few who dare to speak to the scientists in their own tongue. Nor do the scientists welcome outsiders. The recombinant DNA controversy has brought the scientists together in opposition to those who wish to regulate their research. After having called public attention to the hazards of recombinant DNA research, the scientists began to regret their decision. Mark Ptashne, a professor at Harvard, balked when his research project was threatened by the Cambridge City Council's vote for a moratorium on all high containment recombinant DNA experiments: "Increasing community control of science, which I used to support, now worries me." (166)

Recombinant DNA is only one example of the more general and pervasive problem. There are now many tribes of experts, each speaking in an arcane dialect, and each initiating programs with PCP risks. In addition to the microbiologists who join strands of DNA, there are virologists who predict the emergence of new strains of pandemic illness; there are the endocrinologists who formulate sex hormones as birth control pills and for menopausal "stress"; there are the nuclear and chemical engineers, who design atomic power stations and LNG

(166) Arthur Lubow, "Playing God with DNA," New Times, (January 7, 1977), p. 61.

tankers; there are the geophysicists, who assess the reliability of radioactive waste containment and the possibility of predicting earthquakes; there are the toxicologists, who monitor the chemical substances that may cause cancer or otherwise affect our genetic inheritance; there are the astrobiologists who guess the risks of returning life from another planet to earth; and there are the climatologists who predict the build-up of atmospheric carbon dioxide or the depletion of stratospheric ozone. The list could go on with every specialty playing a part. It is as if the catastrophic policies against which we seek protection have a built in defense mechanism of their own: their uncertainties and complexities do not allow our cursory analysis. Yet most people have heard about these PCP's, and have opinions about their risks. How did the public penetrate these barriers of inaccessibility? And how well do they understand the issues involved?

B. The Making of a PCP

The public does not become aware of complex problems overnight. A period of public learning is needed as the issues evolve: the process takes time. (167) As information about the

(167) Sociologists have long written about the "life cycle" of a social problem. Various types and stages of development have been proposed over the years, but no general theory has ever won wide acceptance. See: Richard C. Fuller and Richard R. Myers, "The Natural History of a Social Problem," American Sociological Review, 6 (1941), pp. 320-329; Malcolm Spector and John I. Kitsuse, "Social Problems: A Re-Formulation," Social Problems, 21 (Fall 1973), pp. 145-159; and Anthony Downs, "Up and Down with Ecology -- The 'Issue-Attention Cycle,'" Public Interest, Number 28 (Summer 1972), pp. 38-50. My discussion of the emergence of a

risks is channeled to interested parties, gradually filtering down to the public, there is ample opportunity for the issues to become fuzzy and distorted. People are often left with an incomplete and faulty view of the potential hazards.

Any new social problem might arouse immediate interest, but the first flurries of concern soon wane if they are not reinforced by more publicity. Unless the issue is continuously newsworthy or touches a raw nerve -- a highly unlikely proposition in a society that has accustomed itself to world poverty, disease, and war -- it will fade before any corrective action is taken. Attracting an interested public is not easy; pressure groups have a hard time tapping the communication networks without extensive financial backing. It is hard to mobilize a population that is already overcommitted and usually less than overjoyed on hearing about a new hazard.

As for PCP's, the development process is even more haphazard because the subject matter is so remote. Once told that a certain policy may have disastrous consequences, people are left in the unenviable position of being unable to gauge the risks for themselves. Very few people outside the appropriate community of experts know enough to be able to distinguish the sensational and impossible risks from those which present real threats. Indeed, for some PCP's the experts themselves are

PCP as a public issue (using the recombinant DNA controversy as an example) is loosely based on the life cycle model, with the diffusion of information as the dominant variable. See: Everett M. Rogers, Diffusion of Innovations, (New York: Free Press, 1962); and Donald Schon, Beyond the Stable State, (New York: Norton, 1973), Chapter 4: "Diffusion of Innovation."

unable to make categorical statements about what is or is not possible.

I shall use recombinant DNA research as an example of the development of a technical public issue. Before I turn to it, let us be clear about what recombinant DNA research is. Every living organism contains DNA (except some viruses which have only RNA); the DNA contains the master plan for its growth and differentiation. Recently, a number of technical breakthroughs has allowed scientists to take strands of DNA from different organisms and literally splice them together. (168) Much like editing a series of sequences into a film, DNA's may be joined together to give a new genetic blueprint. Two DNA's, one from the recombinant laboratory and the other from nature, would be indistinguishable if they contained the same nucleotides, the building blocks of DNA. The analogy between film and DNA must not be carried too far: while a film editor can screen all the frames within a sequence, in all but a very few cases, the scientist does not know exactly what he is joining to what. He can recognize where the splices are, but not the instructions coded within the joined segments.

(168) For a description of how such a recombinant DNA experiment is actually carried out in the laboratory, see chapter 2, "Creating New Molecules" in June Goodfield's Playing God (New York: Random House, 1977) pp. 12-21.

Recombinant DNA Research (169)

Recombinant DNA research has become a public issue. Stories about the ongoing controversy hardly need an introduction: each new incident evokes a rash of newspaper, magazine, and television stories. Genetic research is now a favorite of fiction and non-fiction writers alike. Unlike the dissent over nuclear power, there are no emissions of radiation, no threatening bombs, no radioactive wastes, and no risks of core meltdowns. Nevertheless, recombinant DNA research may soon challenge nuclear power's preeminence as a social issue. How then, did the public learn to be wary of, or delight in, such an arcane subject when most people do not know what a gene is, or what the recombination process is? At a time when so many advocates are searching for a constituency to attend to other risks, why recombinant DNA?

Phase 1: The Beginning

Public awareness of recombinant DNA has its seeds in the writing of novelists and luminaries, who speculated about a future without disease and hunger. In those days, no one paid much attention to how such wonders would come about. It would

(169) The following brief survey of the emergence of recombinant DNA controversy is based on my Recombinant DNA Research: A Chronology, Occasional Paper No. 2, Environmental Design Program, School of Architecture and Planning, MIT, November 10, 1976. I have omitted all citations here.

have something to do with biological engineering; it was never more precise for as everybody knew it was only science fiction. Not all saw a rosy future however; some, like Aldous Huxley warned of a cloud within the silver lining. Progress towards refining the generalities into specifics was slow. Ten years passed between the discovery that heredity was governed by DNA, and the determination of its structure in 1953. Watson and Crick's double helix structure was a turning point: the scientists now knew where to focus their attention. The visions were still dreams, but some experts realized that their work would soon have implications for society as a whole.

The period of speculation came to an abrupt end in 1965 when a strange discovery was made at the Oak Ridge National Laboratory in Tennessee. Scientists detected what they believed was the first evidence of genetic intervention. Workers at the Laboratory had abnormally high concentrations of a particular enzyme, arginase, in their blood. The technicians' genes had somehow "learned" how to make that specific enzyme. The best guess was that a virus had infected the workers giving them some of its genes, including the one that could order the production of arginase. Now there was an indication of what might be one day be possible: a special virus could be programmed to cure genetic diseases by transferring missing genes.

The nation's geneticists met at Rockefeller University to consider the Oak Ridge "accident." They decided to play the whole thing down, and not even publicize the fact that they had been concerned about the risks of biological research. They

would continue their work for, as they told themselves, if they were not qualified to go on, who was? Some ten years later, many of them would meet again, but the next time they would not be able to sweep the whole matter under the rug so easily.

Phase 2: Breakthroughs in Technology

The next few years were a period of outstanding technical development. Researchers all over the country were making scientific history: at Caltech they synthesized biologically active DNA; at Harvard they isolated a gene (a specific, self-contained segment of DNA), and at MIT they synthesized a gene. Then in the fall of 1972, a team led by Paul Berg of Stanford announced that they had successfully joined DNA from different sources. Scientists had created a new combination of genetic material.

The experiment was thought to be an isolated success when more news came from Stanford: a class of enzymes, now known as restriction enzymes, could break the DNA molecule in such a way that different DNA's could be spliced together. Recombinant DNA experiments were now easy to do.

As the techniques of recombinant DNA became more versatile, the community of scientists split into two. Members of one group, aware of the potential, though uncertain, risks of their work, spoke out for an assessment of the hazards and greater public involvement. The others warned of exaggerating the dangers, for this might lead to a curtailment of their work.

Meanwhile intellectuals in unrelated fields, on hearing about the breakthroughs and the emerging controversy, were drawn to microbiology. Philosophers, lawyers, psychologists, sociologists, theologians, and public policy analysts studied what the scientists were doing. Genetics was attracting a wider audience -- though the absolute number of people involved was still small. Little nuclei of interdisciplinary research evolved, creating new lines of communication among professionals, who otherwise hardly ever spoke to each other. For the lay public, a number of books appeared that described the kind of society the new biology would bring. They dealt with generalities and not specifics.

Phase 3: To Asilomar and Back

The war against cancer was on, and the microbiologists intensified their research on tumor viruses. Not surprisingly, the first recombinant DNA experiments involved manipulating genes from such cancer-causing viruses (e.g. SV40) and inserting them into common bacteria (e.g. E. coli). To some, the risks seemed obvious: what if the bacteria could learn how to make cancerous tumors? What if they escaped from the laboratory?

In January 1973, the leading scientists met at the Asilomar conference center in California to discuss the potential hazards of biological research. Ironically, while fear of the uncertain dangers of joining foreign pieces of DNA was one of the motivating factors for the meeting, these risks were barely

mentioned. Most of the sessions were devoted to the general risks of working with tumor viruses.

By the following summer, recombinant DNA experiments were at the center of attention. Scientists were more and more worried about the implications of joining pathogenic and benign genes. At the Gordon research conference, they expressed their "deep concern" about the unpredictable biological activity of new DNA combinations. They would send two letters, one to the National Academy of Sciences and the National Institute of Medicine, asking them to study the problem; the other was for publication in Science, the leading interdisciplinary journal in the country. All those centers of interest were now formally advised of the risks. The NAS-NIM letter set the wheels of the scientific establishment turning: Paul Berg was asked to set up an advisory committee. Apart from those already watching the biologists the reaction was minimal.

The experiments were becoming more and more sophisticated, and the number of possible genetic combinations was now unlimited. As the permutations mounted, so did the concern over their effects. The most promising experiments were also the most hazardous: many believed that tumor viruses held the key to the cause, and therefore the cure, of cancer. The fear of casual experiments with these and other toxic genes led the Berg committee to ask for a voluntary moratorium on some particularly dangerous experiments -- those which involved certain manipulations of tumor virus or antibiotic resistance genes. The scientists also called for an international conference to explore

the implications of the new recombinant techniques. In February 1975 they met once again at Asilomar.

An important shift had occurred in the two years between the Asilomar conferences: while scientists were still apprehensive about working with tumor viruses, their attention was now directed towards the recombinant technology itself. Also this second time, the world was watching what the scientists were doing at Asilomar. The call for a suspension of research was unprecedented, and recombinant DNA was fast becoming a national issue.

Phase 4: Towards Regulation

At Asilomar, the scientists negotiated a set of protocols for recombinant DNA experiments with the objective of lifting the self-imposed moratorium. Safety would be assured by a combination of physical and biological containment. In addition to stringent laboratory practices, dangerous experiments would use new, specially designed, bacteria that would self-destruct outside the laboratory (a technique known as biological containment). Before work could continue, however, a new committee of the National Institutes of Health (NIH), set up just before the conference, would have to approve the rules. It took the NIH a year and a half to release the research guidelines -- in that time the recombinant DNA issue turned into a bitter scientific controversy, watched by a suspicious and confused public.

The two opposing factions of scientists became increasingly polarized as the NIH struggled to balance the benefits of continued research with the need to protect public health. The stringency of the guidelines seesawed, and with each change in the balance of power, the losing group became more militant. By 1976, most of the scientists were tired of waiting: practically all those who had first spoken out about the risks now thought the guidelines provided enough safety. It was time to go back to work. They resented the delays, after all, they argued, the community of microbiologists had acted in the public interest, and the public should trust them. The critics, now definitely a minority of the original group, protested the lack of public involvement in the regulatory process, in addition to the laxity of the standards.

If the early controversies had attracted a variety of professional studies, the worsening polarization among the experts stirred even greater curiosity. The other disciplines, already seeded with students of biology, now acted as secondary centers for the growing professional and public interest in recombinant DNA. The microbiologists were no longer the only source of information -- others were watching and interpreting the scientists' deliberations. Each new center attracted its own following. The recombinant DNA issue flourished, and with its expanding audience, a host of ancillary issues surfaced. The original problem of biohazards was complemented by questions about freedom of inquiry, public control of science, corporate exploitation of genetics, creation of new life forms, patent

rights for new organisms, applications to chemical and biological warfare, the production of scarce drugs (e.g. insulin), the potential for self-fertilizing crops, and genetic engineering on humans. There was something for everyone.

Public awareness of recombinant DNA spread as hearings were held all over the country. At every level of government, committees looked into the need for new legislation and regulations. The greatest public involvement came when universities planned new laboratories that could satisfy NIH safety guidelines. In Ann Arbor, Cambridge, and many other college towns, people questioned whether they wanted the scientists to do potentially hazardous experiments in their communities. Often the meetings turned into confrontations -- good for eliciting headlines, but hardly conducive to reaching consensus. The hearings assumed some of the qualities of a travelling road show with the same outside experts appearing to testify on each of the local initiatives. Little was ever resolved, though now recombinant DNA was a national issue.

C. Public Understanding or Misunderstanding?

From the beginning, the scientists have been in control of the recombinant DNA debate. In 1977, when a number of legislative proposals were before Congress, the scientists again took the offensive, this time charging that the risks had been exaggerated. The NIH regulations, in force for over a year, were sufficient, they told everyone, and perhaps even too stringent. Tabitha Powledge of the Hastings Center has observed: "if there

is one factor that has been characteristic of the recombinant DNA arguments from the beginning to the present, it is that scientists have been in charge all along, and that the public has been relying on them for all its information, pro and con. The subject was raised by scientists, the guidelines were devised by them, and the new intrasigence is led by scientists and vindicated on the basis of work done by scientists." (170) This is not surprising given how obscure the subject matter is. Yet, on the one hand, the biologists acted on our behalf, while on the other, as some critics have correctly charged, "the public was neither informed, consulted, nor educated." (171) Despite the lack of formal instruction, however, today many people not only have heard about recombinant DNA research, they also have opinions on whether or not the work should continue. Should the public perceptions of the risks be trusted? Or put another way, if there were a general referendum on the subject, what would be the significance of the outcome?

The public learned about recombinant DNA from a mixture of fantasy writers and secondary sources, as well as the fallout from a polarized and heated controversy. Some may perceive the experiments as the first step toward realizing Dr. Frankenstein's dream, with many fearing that today's scientists will do no

(170) Tabitha M. Powledge, "Recombinant DNA, Backing Off On Legislation," Hastings Center Report, 7 (December 1977), p. 10.

(171) "Gene Implementation: Hazards of Genetic Engineering," Science for the People, 8 (March 1976), p. 10.

better controlling their handiwork. (172) As Nobel laureate James Watson told one reporter: "The risk of genetic engineering has captured the popular imagination because it extrapolates so easily into a science-fiction doomsday scenario." (173) Also, some believe the scientists are "playing God," and thereby evoking a sense that they are violating ethical and moral taboos. Because the techniques of recombinant DNA are so hard to understand, the public may be confusing many different issues commonly lumped together in the catchall term, genetic engineering. Most people are probably not distinguishing between gene splicing on the molecular level with test tube babies, cloning, experimentation with human subjects, and genetic screening programs. And no doubt, many think recombinant DNA research is risky because the scientists originally told us it is.

Of course, it is hard to say exactly how public attitudes towards genetic research were formed, or what the public perceives to be the main risks of biological research. People talk of cancer plagues -- an allusion to the possibility of breeding contagious or transmissible tumor viruses -- and other irreversible toxic effects of engineered microorganisms gone amok, both scenarios loosely based on the warnings of some

(172) Willard Gaylin offers the term "Frankenstein Factor" for those anxieties which are beyond the calculus of risk-benefit analysis. "Sounding Board: The Frankenstein Factor," New England Journal of Medicine, 297 (September 22, 1977), pp. 665-667.

(173) Arthur Lubow, op. cit., p. 63.

scientists. Yet other, related PCP's have been overlooked. Natural recombinants between tumor and more benign viruses and the growing ability of pathogenic bacteria to become resistant to antibiotics also pose serious health hazards. Both risks can arise independently of the technology of recombinant DNA: both involve the natural recombination of genes. Viruses can recombine without restriction enzymes, though in such cases, nature and not the scientist controls which fragments are spliced together. (174) And resistance to antibiotics is caused by the exchange of antibiotic resistance genes among bacteria. This process is being accelerated by a combination of abuse of prescription and over the counter antibiotics and their routine addition to animal feeds. Interestingly, the "artificial" manipulation of tumor viruses and antibiotic resistance gene are the two types of experiments which Paul Berg's committee asked scientists to defer when it called for a moratorium. (175) Thus, while both risks have received attention in the context of planned recombinant DNA experiments, the public is generally oblivious to them as natural byproducts of human actions. That people could be so close to a set of potential risks without recognizing them, or having them pointed out for them, underscores the superficiality of the public's familiarity with biohazards, and the failure of the present modes of interaction

(174) Natural recombination of influenza viruses will be discussed at length in Chapter 9.

(175) See Type 1 and Type 2 experiments, P. Berg. et al., letter, Science, 185 (July 26, 1974), p. 303.

between the public and the experts.

Natural Recombination of Tumor Viruses

Since the 1960's, virologists have known that the SV40 virus and the adenovirus can recombine naturally to yield viruses which contain a mixture of the two sets of genes. SV40 is a tumor virus, capable of causing cancer in some animals, though probably not in humans. It does infect people, however, and has been implicated in chronic central nervous diseases. (176) Adenoviruses --there are many different types-- cause mild respiratory ailments like the common cold; they can remain latent in tonsils and adenoids for many years, periodically reactivating according to some unknown schedule. In 1971, Dr. Andrew Lewis of the National Institute of Allergy and Infectious Diseases was breeding such recombinant viruses. He began to worry about the risks (177) : could SV40-adenovirus recombinants have the worst properties of each virus? Could they become established in the general population and years later cause an epidemic of cancerous tumors? Lewis expressed his concern about these viral combinations at both Asilomar conferences.

The public knows next to nothing about the risks of

(176) See "A Shot in the Arm" in Chapter 4.

(177) Andrew M. Lewis, Jr., "Experience with SV40 and Adenovirus-SV40 Hybrids," in Biohazards in Biological Research, ed. by A. Hellman, M. N. Oxman, and R. Pollack, (Cold Spring Harbor, N. Y.: Cold Spring Harbor Laboratory, 1973), pp. 96-113; Michael Rogers, Biohazard (New York: Knopf, 1977) pp. 34-35 and 69-71; and Nicholas Wade, "Dicing with Nature: Three Narrow Escapes," Science, 195 (January 28, 1977), p. 378.

naturally occurring recombinant viruses. Lewis's colleagues are at least partly responsible: when he first raised questions about the potential hazards of working with these viruses and urged voluntary control of their use and distribution, he won only marginal support. In fact, some researchers threatened him with Congressional and administrative action unless he made his viruses freely available. Attitudes have now at least partially changed -- three of those who threatened Lewis later joined Paul Berg in calling for the voluntary moratorium and the second Asilomar conference. (178) Data on Lewis's work and concerns are freely available, and the NIH now requires those working with these kinds of hybrid viruses to meet some minimum laboratory standards and are not to distribute them without authorization. (179) But none of this has filtered down to the public. Today as some people argue for a new moratorium on all applications of recombinant DNA technology, there is no awareness of the risks of naturally occurring recombinants. Ironically, because adenovirus-SV40 recombinants do not require the use of restriction enzymes, the NIH guidelines do not apply to them. Two scientists constructing the same hybrid virus, one tapping the virus' natural tendency to recombine and the other using restriction enzymes, would be subject to different safety requirements. The experiment with restriction enzymes would have

(178) Rogers, op. cit., and Wade, op. cit.

(179) Memorandum of Understanding and Agreement for the Transfer of Adenovirus 2-SV40 Hybrid Viruses from One Laboratory to Another," National Institutes of Health, Bethesda, Maryland.

to be carried out under stricter procedures in a high containment laboratory.

The risks associated with tumor viruses originally dominated the discussions of biohazards -- before the discovery of restriction enzymes. This is no longer the case. A set of more general fears, some legitimate others not, are now the subject of the controversy. In some ways this is not surprising given the number of issues that have arisen in the last few years. What is surprising, and disconcerting, is that a risk so closely linked to the original one, has not joined the many others in the public debate.

Antibiotic Resistance

The proliferation of antibiotic resistance among bacteria has not become a public issue. This too is surprising because, unlike many of the hazards associated with recombinant DNA experiments, the risks are not speculative. As I pointed out in Chapter 2, a host of disease agents now have the ability to withstand the anti-bacterial drugs which used to kill them. As a result, deadly epidemics have occurred in many parts of the world. In the United States gonorrhea strains have been growing resistant to antibiotics for a long time: once 200,000 units of penicillin were enough to cure a patient, today the recommended dose is 4.8 million units! (180) More recently some gonorrhea

(180) Barbara J. Culliton, "Penicillin-Resistant Gonorrhea: New Strain Spreading Worldwide," Science 194 (December 24, 1976), p. 1395.

bacteria have "learned," by the natural recombination of genes, how to become immune to even such massive doses by producing penicillinase, an enzyme capable of breaking down penicillin. These new kinds of strains have been detected in 21 American states as well as 15 other countries. (181)

Many scientists have been outspoken about the seriousness of the problem -- some believe naturally bred antibiotic resistance is much riskier than that artificially created by a planned recombinant DNA experiment. Soon after the Berg committee's letter was published in 1974, E. S. Anderson, a British researcher on bacterial infections, responded that he felt "little inclination to respond to the call for a moratorium" on recombinant DNA experiments involving the manipulation of genes which conferred antibiotic resistance. (182) (He agreed about the need for caution when working with tumor viruses.) He argued:

"A sense of proportion must be retained in considering these matters. The widespread and indiscriminate use of antibacterial drugs in man and animals has exerted immeasurably more pressure on the bacterial population than could be wielded by all the research workers in this field put together. These drugs are used prodigiously in human and animal medicine, and as animal feed additives. The long-predicted results, now realised, were the spread on a gigantic scale of transferable drug resistance [genes] in the ordinary intestinal bacteria such as Escherichia coli; and the emergence of epidemic drug-resistant and lethal lines of enterobacterial pathogens: Shigella dysenteriae 1

(181) Morbidity and Mortality Weekly Report, 26 (May 13, 1977), p. 153.

(182) E. S. Anderson, "The Indiscriminate Use of Antibiotics Has Exerted More Pressure on the Bacterial Population Than Could Be Wielded by All Research Workers in the Field Put Together," Nature, 250 (July 26, 1974), p. 279.

(Shiga's bacillus) in Central America, the typhoid bacillus in Mexico, India, South Vietnam and Thailand, and, and S. typhimurium and other salmonellae with massive drug resistance and apparently increased virulence in many countries. This has been the direct consequence, not of [recombinant DNA experiments] but of commercial pressures and administrative and professional irresponsibility." (183)

Stanley Falkow also thinks the risks associated with antibiotic resistance are greater than those arising from most recombinant DNA experiments. As a leading authority on drug resistance, he was one of the original members of the NIH advisory committee on recombinant DNA research and worked on writing the NIH guidelines. In July 1976, he went to Cambridge to testify before the City Council which was holding hearings on Harvard's application to build a high containment laboratory. There he told Mayor Vellucci why he had resigned from the NIH committee: "I wanted to work on a committee that was dealing with antibiotics and animal feed, which I considered, in my personal opinion, to be a greater biohazard than the experiments that you are talking about today." (184)

Antibiotic resistance has not been ignored by

(183) Ibid.

(184) Hearing on Recombinant DNA Experimentation, official transcript, Cambridge City Council, July 7, 1976, pp. 165-166. Harvard had asked permission to build a "P3" laboratory. P3 refers to the third most stringent level of physical containment dictated by the NIH guidelines. Note that more hazardous experiments than those planned by Harvard had to be done in a P4 laboratory, and at that time, many of the most dangerous experiments were still banned. Elizabeth Kutter of Evergreen College, the head of one of the NIH subcommittees which designed the guidelines, has told me that she too believes the spreading resistance to antibiotics among bacteria is a more serious social hazard than recombinant DNA research. Personal communication.

newspapers and magazines. There have been many articles on this subject, and even television programs. In 1976 PBS televised a BBC produced show on the abuse of antibiotics, describing among other things the 1972 Mexican typhoid epidemic in which some 14,000 people died. (185) Nevertheless the issue has never sparked the public's imagination. One can hardly argue that the concept of disease because our drugs are no longer working is too remote for the public to understand. In the course of human history, plagues have killed more people than any other single cause, yet the idea that we are losing the very means scientists have developed to fight and prevent them cannot take hold.

Conclusion

In the last chapter I concluded that it is impossible to predict how, left on their own, individuals will react to PCP risks. On the basis of the recombinant DNA experience, I further conclude that the expert-public interaction does little to improve the chances that individuals approach the balancing of risks and benefits in an intelligent and sober manner. They are never given enough information in an accessible form that they can weigh the risks for themselves. Possibilities for confusion and distortion abound. The relevant and the irrelevant have as much chance of influencing public perceptions. Worse, there is a

(185) NOVA, "The Overworked Miracle," first televised March 21, 1976.

natural reaction to mimic the experts by taking up extreme positions. The polarization of the experts leads to the polarization of the public -- people feel pressured to pick a side, and respond to a complicated problem with a nod or a shake of the head. Indefinite PCP facts are like Rorschach inkblots which elicit a person's innate feelings. An individual's opinion of a PCP is more likely to be determined by a visceral reaction to its risks or benefits than the available facts. For instance, the single most important factor influencing such an appraisal may not be the probability of a catastrophe, however high or low it might be, but on whether he is a technological optimist or pessimist. (186)

The recombinant DNA PCP does illustrate how those factors which I cited in the last chapter influence the public's perception of risks. While it is difficult to pinpoint the precise source of the public's anxiety over recombinant DNA experiments, much can be learned by comparing the three PCP's: restriction enzyme based recombinant experiments, natural recombinations with tumor viruses, and antibiotic resistance among bacteria.

It is easy to dispose of the question of why there is

(186) A recent study of the public's perception of the hazards of nuclear power reached a similar conclusion: "the distrust of nuclear power rests in part on its social history; in part on its unique combination of hazards; and in part on the special way it has been managed and regulated. Furthermore, the public distrust of nuclear power is significantly amplified by the rancorous debate in a polarized expert community." Christoph Hohenemser, Roger Kasperson, and Robert Kates, "The Distrust of Nuclear Power," Science, 196 (April 1, 1977), p. 30.

so little public interest or understanding of natural recombination with tumor viruses: the experts never publicized these risks. Andrew Lewis met with massive opposition, and some antagonism, when he tried to generate concern for such hazards. A PCP can follow many paths to the public spotlight: the recombinant DNA development path is but one route. None is quicker than that catalyzed by a major accident! In the absence of a disaster, the backing of the expert community is a necessary condition for public notice. There is no point expecting PCP risks to emerge according to Model 1, we are dependent on the experts to publicize PCP risks, either according to a Model 2 or 3 process.

Why were the microbiologists willing to publicize the new enzyme based experiments and not the naturally occurring recombinant research? There is no doubt that the new enzymes entailed greater opportunity for catastrophe than did the natural recombinants. As I shall argue in the next chapter, the scientists spoke up to protect the public and themselves. The public was spared from the possibility of a terrible accident, and the researchers gained because they did not have to carry out the riskiest experiments, which might possibly bring the greatest rewards, in the competitive atmosphere that pervades molecular genetics. Once the high risk experiments were regulated -- and having witnessed the public's reaction to the recombinant DNA issue -- the scientists reacted to the two types of PCP's in a very similar fashion: by trying to deny the risks.

Contrasting antibiotic resistance to recombinant DNA,

we can see why one risk has emerged while the other has not:

Time Delay: While both PCP's could bring a "bang"-type disaster (an epidemic), antibiotic resistance is perceived as a "later" event as compared to the "now" potential of DNA manipulation.

Affected Group: Though we might all suffer the ravages of a plague, however it might be caused, people living near a high-containment laboratory felt more at risk and thus voiced more opposition when those research centers sought permission to do the risky experiments. Antibiotics are too widely administered to have a locus of concern.

Benefits: At the beginning, before progress was made towards the production of somatostatin and insulin, the benefits of recombinant DNA were still seen as speculative. The benefits of antibiotics were unchallengeable: they protect us all from disease.

Responsible Group: We all take antibiotics -- admittedly at times our doctors prescribe them unnecessarily -- and we are therefore all responsible for the growing resistance to them. The responsibility for a recombinant DNA accident, on the other hand, would lie with a readily identifiable group: the microbiologists. (The farmers and drug companies are also responsible for the growing resistance because they add antibiotics to livestock feeds, but I believe that the human, medicinal applications of these drugs are perceived as their primary use.)

Age: Although both issues were new to the public, recombinant DNA experiments represented a totally new technology -- something

straight out of science fiction. The concept of resistance had arisen before in connection with the ability of pests to become immune to DDT and other pesticides.

Thus practically every factor encouraged recombinant DNA experiments to be perceived as a greater threat than growing antibiotic resistant strains of bacteria. The experts had been the teachers but the public's perception of hazard followed a predictable course; much of this is because the experts are not disinterested parties who simply provide the facts for the policy debate.

Chapter 8

THE EXPERTS AND PCP'S

In the last two chapters, I showed that individuals have a distorted view of PCP risks, and that the process by which they learn of new risks from the experts does little to improve this situation, and often it leads to further misperceptions and misunderstandings. If the public's appraisal of PCP's, with or without expert assistance, is unreliable, the next question is whether or not the experts themselves may be relied upon to gauge the nature and extent of PCP risks. While there is no doubt that they know more than the untrained observer, there are compelling reasons to believe that they too misestimate the dangers of PCP's.

Whether the PCP threatens ozone depletion, carbon dioxide build-up, pathogenic recombinant organisms, nuclear accidents, or a new influenza virus, the public needs expert guidance (Models 2 and 3). Without help, people will not know which outcomes are possible and what their probabilities are. But can we confidently begin our deliberations with the list of outcomes and probabilities presented by the experts? Certainly an expert is knowledgeable about his discipline. His work is constantly being judged by the community of scientists of which he is part; his success is reflected by his ability to win grants, to pursue his research, and to rise within the community's hierarchy. But, the peer review system, while providing internal checks and balances within the profession, has the more general failing that the experts may, individually, and

as a group, overestimate how much they know about an issue, and thereby be insensitive to some of the potential risks. Like the rest of us, the experts are faced with the intrinsic uncertainties and complexities of the PCP's. They are better able to ask the relevant questions and design experiments to test hypotheses which may lead to more precise risk estimates, yet they too stumble along, if only because the task before them is so difficult.

A. Research at the Frontiers

Studies on carcinogenicity, genetic structure and function, atmospheric equilibria, and life on other planets all entail uncertainties which can neither be resolved nor estimated (types 3 and 4). Experts working on such PCP related research are at the frontiers of knowledge, as I argued in Chapter 3, they have no theoretical base on which to build reliable and predictive models. Their task is like trying to do a jigsaw puzzle without the benefit of the picture that usually comes on top of the puzzle box. Imagine that many of the pieces are a hazy blue, and that they may be part of the ocean or the sky. The scientific method involves proposing and testing of hypotheses followed by a lot of painstaking work, careful deductions, and a measure of guesswork. Experiments will support some hypotheses, and others will have to be discarded. Hypotheses follow hypotheses, and slowly a general theory begins to emerge. Only once there are many islands of interlocking

pieces can the relationship among them be deduced, and an outline of the theory or puzzle can be recognized. This is the way of science and mistakes must be expected. Some hypotheses will be accepted, only later to turn out to be wrong. The rub is that the experts' estimate of the risk for PCP's must depend on such working hypotheses -- for there is nothing more substantial at hand. The danger is that the experts lose sight of their ignorance, and in so doing, prematurely reduce the uncertainties, thereby distorting the potential risks.

In the pages that follow I shall cite some examples of models, which though supported by experimental results, presented an incomplete picture of the world -- forcing later corrections. The failure to allow for the potential complexities, lulled the scientists into a false state of confidence about the adequacy of the models, and leads me to wonder about the accuracy of their estimates of the risks of recombinant DNA research and the hazards of planetary containment, and more generally about all PCP's.

The Central Dogma of Molecular Biology

The central dogma concerns the flow of genetic information for protein synthesis. The instructions are contained in the DNA molecule, which acts as a template for the manufacture of proteins. The DNA guides the production of RNA, which in turn controls the production of proteins. Before 1970, the prevailing belief among geneticists was that the flow of

information could only go in one direction: from DNA to RNA to proteins. Information could only flow according to the arrows in Figure 8-1. The central dogma dictated that neither RNA nor proteins could affect DNA; only DNA could change another DNA. The implications were enormous. If the dogma did not hold as to proteins and DNA, then the integrity of all genetic materials whether they were contained in the smallest bacterium or a human cell, could be disrupted upon eating most foods. Our essential nutrients would also upset our body biochemistry; indeed, under such rules, life as we know it could never have evolved. Looking at the previous step in the information transfer scheme, we face the more subtle question of RNA interaction with DNA. If RNA could affect DNA, certain pathogens, like RNA viruses, could alter human genes. If cancer is seen as DNA instructions run amok, research into RNA tumor viruses might lead to an understanding of the causes of cancer. A correspondent for Nature, writing in 1970, described the central dogma as the "cardinal tenet of molecular biology." (187)

(187) "Central Dogma Reversed," Nature, 226 (June 27, 1970), p. 1198.

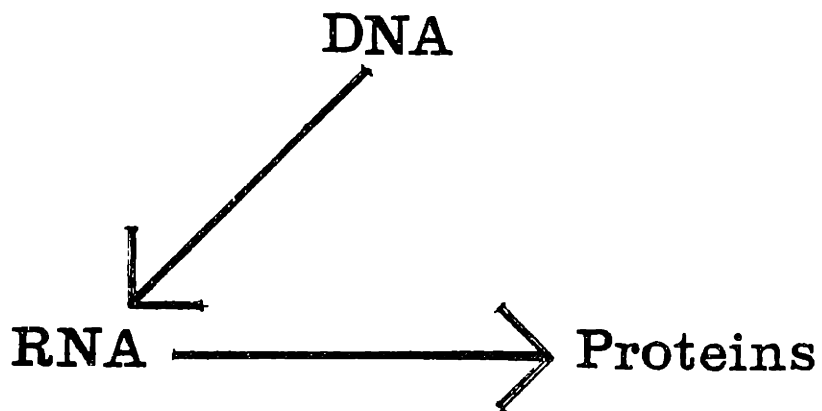


Figure 8-1 (188)

(188) Adapted from Francis Crick, "Central Dogma of Molecular Biology," Nature, 227 (August 8, 1970), p. 561.

In 1970, David Baltimore of MIT, who would later play a pivotal part in the recombinant DNA debate as a member of the Berg Committee, and Howard Temin, of the University of Wisconsin, independently showed that the RNA in certain tumor viruses could itself act as the template for the synthesis of DNA. (189) The headline in Nature proclaimed in large black type, "CENTRAL DOGMA REVERSED." (190)

Francis Crick, the first advocate of the central dogma, who, with James Watson, had deciphered the structure of DNA, quickly replied that a clear reading of the theory allowed for the transfer of information from RNA to DNA. (191) Perhaps Crick's qualifiers had not received sufficient notice. As Temin would later describe it: "Although Crick's original formulation contained no proscription against a "reversed" flow of information from RNA to DNA, organisms seemed to have no need for such a flow, and many molecular biologists came to believe that if it were discovered, it would violate the central dogma." (192) In fact, Gunther Stent, one of this country's leading molecular

(189) D. Baltimore, "Viral RNA-Dependent DNA Polymerase," Nature, 226 (June 27, 1970), pp. 1209-1211; H. M. Mizutani, "RNA-Dependent DNA Polymerase in Virions of Rous Sarcoma Virus," ibid., 226 (June 27, 1970): pp. 1211-1213.

(190) "Central Dogma Reversed," Nature, 226 (June 27, 1970), p. 1198.

(191) Francis Crick, "Central Dogma of Molecular Biology," Nature, 227 (August 8, 1970), pp. 561-563.

(192) Howard M. Temin, "RNA-Directed DNA Synthesis," Scientific American, 226 (January 1972), pp. 24-33. Reprinted in The Chemical Basis of Life, Readings from Scientific American (San Francisco: Freeman, 1973), pp. 356-365.

biologists, writing in 1970, noted that "an essential feature of the central dogma is the one-way flow of information,

DNA  RNA  protein

a flow which is never reversed." (193)

Temin's belief that some types of RNA's could alter DNA, the "provirus hypothesis," was largely ignored from the time it was first offered in 1964 until 1970, in large part because it conflicted with the central dogma. (194) Baltimore and Temin's experimental achievement was soon rewarded, confirming the importance of revising one of the cornerstones of modern biology. In 1975, they shared the Nobel Prize with Renato Dulbecco, in whose laboratory both had trained. There was no doubt that they were being honored for their discovery of what is now known as reverse transcriptase, the enzyme that could violate the central dogma of molecular biology.

The upheaval caused by Baltimore's and Temin's experiments was only two years old when Janet Mertz and Ronald Davis discovered restriction enzymes, the breakthrough which, more than any other, opened up the possibility of planned recombinant DNA experiments. The theory of genetic information transfer was corrected, a Nobel prize was awarded, and the research continued.

(193) Gunther S. Stent, "DNA," Daedalus, 99 (Fall 1970), p. 926.

(194) Howard M. Temin, "The DNA Provirus Hypothesis," Science, 192 (June 11, 1976), p. 1076 (Temin's Nobel lecture), and Walter Eckhart, "The 1975 Nobel Prize for Physiology of Medicine," ibid., 190 (November 14, 1975), p. 714.

One Gene -- One Protein

In late 1976, it was England's turn to rock the foundations of molecular biology. British scientists showed that one sequence of DNA could code for two different proteins, violating what Scientific American called "[p]erhaps the most basic dogma [of molecular biology] expressed by the phrase 'one gene, one protein.'" (195) An organism, the bacterial virus PhiX174, was found to have overlapping genes. (A bacterial virus is more commonly called a bacteriophage.)

DNA's instructions for the manufacture of proteins are contained in long sequences of nucleic acid bases. There are four possible bases: adenine (A), cytosine (C), guanine (G), and thymine (T), and they can be arranged in any order. DNA is a ladder of bases, and proteins are chains of amino acids. The relationship between them is known as the genetic code: a set of three bases, a codon, specifies an amino acid. For instance, the triplets TCT and AAG code for the amino acids serine and lysine respectively. There are $4 \times 4 \times 4$, or 64, possible codons, and each plays a role in the manufacture of proteins. With 64 condons and only 20 amino acids, there is some redundancy; different codons can signal the production of the same amino acid. For instance, TCT, TCC, TCA, TCG, AGT, and AGC all code for serine.

(195) "Science for the Citizen: Amended Dogma," Scientific American, 236 (May 1977), p. 50; see also Roger Lewin, "Virus Violates an Important Biological Law," New Scientist, 72 (October 21, 1976, p. 148.

The sequence

...GCGGAAGGAG...

can code for a protein containing the amino acids alanine, glutamic acid, and glycine in a row. Before the PhiX174 revelation, it was universally believed that proteins were manufactured according to the order of the DNA bases, each DNA sequence, or gene, coding for only one protein. Genes were arranged sequentially and discretely along the DNA helix. This theory of gene arrangement became the orthodox view.

What happened in 1976 was that scientists working at the Laboratory of Molecular Biology in Cambridge, England, found that one sequence of bases could contain duplicate instructions, that is, it could code for more than one protein at the same time. By shifting the starting point for reading the triplets one base over, a whole new sequence of amino acids is obtained. Thus, the sequence of bases given in the last paragraph

...GCGGAAGGAG...

was read

...GCG/GAA/GGA/G...

but if the starting point is shifted one base to the right, the instructions would begin with the "C" base, and a new set of codons would result,

...G/CGG/AAG/GAG/...

coding for the arginine, lysine, and glutamic acid sequence of amino acids. A further or two-base phase shift yields a third set of triplets,

...GC/GGA/AGG/AG...

The sequence of bases cited here is not hypothetical, but one actually found in the bacterial virus PhiX174, and both sequences of amino acids are produced by the virus. (196) While the third set of codons are not synthesized by PhiX174, one of its close relatives, bacteriophage G4, can accomplish such an incredible feat. Along one stretch of its DNA, all three possible sets of codons are read simultaneously. (197) Only small segments of the whole PhiX174 and G4 viral DNA's have overlapping genes; most of them code for only one protein at a time. Yet the fact that double and triple coding occur at all is truly remarkable.

The scientists had deciphered part of the genetic code without allowing for a second or third layer of complexity, ones which do not allow any redundancy. At some point researchers must have considered the possibility of overlapping genes, but ignored them in the face of their staggering improbability. Double and triple coding are highly unlikely since they remove so much of the flexibility of evolution: random mutations in an overlapping segment of DNA have a double or triple impact, making viable improvements even more unlikely. That is, a change in only one DNA base could at once change two or three proteins.

(196) B. G. Barrell, G. M. Air, and C. A. Hutchinson, III, "Overlapping Genes ϕ X174," Nature, 264, (November 4, 1976), p. 37; see also F. Sanger, et al., "Nucleotide Sequence of Bacteriophage ϕ X174 DNA," ibid., 225 (February 24, 1977), pp. 687-695.

(197) D. C. Shaw, et al., "Gene K, a New Overlapping Gene in Bacteriophage G4," Nature, 272 (April 6, 1978), pp. 510-515; Maria Szekely, "Triple Overlapping Genes," ibid., p. 492.

Most chance mutations in non-overlapping genes are destructive, thus the odds that a mutation in an overlapping DNA segment will cause a beneficial change in more than one protein are extremely small.

Overlapping genes may be very rare; they have yet to be identified in a bacterium, a more complex organism than a virus. The PhiX174 and G4 phenomena are not isolated ones however: the SV40 virus, a small virus with an ability to infect a wide range of organisms, including humans, also has overlapping genes. (198) If this trend continues, the implications would be enormous. As one reviewer noted, "If it occurs in bacteria, chances are that it occurs in higher organisms as well. Now that the idea that genes may overlap is becoming respectable, some molecular biologists believe that many more examples of this effect will be found in the near future. The way in which the concept of gene overlap will affect present ideas of gene regulation is uncertain. But if this phenomenon is widespread, the very concept of a gene will have to be redefined and the study of bacteria will enter a new era." (199)

So the highly unlikely does occur, contradicting what many people believe to be possible or impossible. What

(198) V. B. Reddy, et al., "The Genome of Simian Virus 40," Science, 200 (May 5, 1978), pp. 494-502.

(199) Gina Bari Kolata, "Overlapping Genes: More than Anomalies?" Science, 196 (June 10, 1977), p. 1188. An opposite view holds that the overlapping genes arise due to the PhiX174's limited amount of DNA, and that double coding is unlikely in higher organisms because they have an excess, rather than a 'shortage' of DNA. John C. Fields, "The Nucleotide Sequence of Viral DNA," Scientific American, 237 (December 1977), p. 65.

probability would scientists have assigned to the discovery of overlapping genes? A million to one? Whatever the odds we can only wonder how they compare to some of the current probability estimates of the risks associated with recombinant DNA experiments.

These two illustrations of fundamental revisions in the laws governing molecular biology are rare but not unique. The field is new, and much must still be learned; undoubtedly there will be many new surprises. For example, researchers have found that bacterial genes are capable of moving in, or among, chromosomes. (200) These so-called "jumping genes" are forcing the scientists to change their static models of DNA into dynamic ones. More recently another finding upset widely held beliefs about the nature of information transfer by RNA. Researchers have discovered that some of the messenger RNA (mRNA), which carries the instructions for protein synthesis, of two viruses (adenovirus and SV40) are coded by non-contiguous segments of its DNA. (201) Thus it is not enough to know the sequence of DNA bases to deduce the corresponding sequence of amino acids and the resulting proteins because some of the bases may be skipped over

(200) Gina Bari Kolata, "Jumping Genes: A Common Occurrence in Cells," Science, 193 (July 30, 1976), pp. 392-94.

(201) Jean L. Marx, "Viral Messenger Structure: Some Surprising New Developments," Science, 197 (August 26, 1977), pp. 853-855, 923.

in the process of translation. Similar, though perhaps unrelated, findings have been identified in the mRNA of higher organisms. One of the genes of rabbits is "interrupted" by a long strand of DNA which is not transferred to the mRNA. (202) Which bases "count" for the production of proteins and how and why such intervening sequences are ignored remain mysteries.

All these new findings contribute to the conclusion that many of the current theories are still in their infancy. Only 25 years have passed since Watson and Crick proposed a double-helical structure for DNA. In this short time scientists have made remarkable progress towards understanding the workings of cells and their genes, but few would dispute that the scales of knowledge are tilted toward ignorance. As Lewis Thomas, author of Lives of a Cell, put it in a column on the recombinant DNA controversy: "The only solid piece of scientific truth about which I feel totally confident is that we are profoundly ignorant about nature. Indeed, I regard this as the major discovery of the past 100 years of biology." (203)

Given such basic uncertainties regarding how information is coded in DNA, how it can be transferred, and how it moves around, it is difficult to understand how the scientists can be so certain about their predictions of some gene splicing experiments. The question of hazard turns on whether or not

(202) Bob Williamson, "DNA Insertions and Gene Structure," Nature, 270 (November 24, 1977), pp. 295-297.

(203) Lewis Thomas, "Notes of a Biology-Watcher: The Hazards of Science," New England Journal of Medicine, 296 (February 10, 1977), p. 327.

planned or accidental joining of DNA fragments will result in a sequence of nucleotides which, in some fashion, could prove hazardous. Is this, as some might suggest, similar to the risk of starting a nuclear war by placing monkeys at the control consoles of Minuteman missiles with their firing dependent on the monkeys typing, without prompting, the first few lines of the Gettysburg Address, "Four score and seven years ago our fathers brought forth...?" Because they are sitting at typewriters, the text which the monkeys produce is composed of numbers and letters but there is no guarantee, indeed, it is unlikely that intelligible words will result. (204) On joining strands of DNA, the biologists are one step ahead of the monkeys, for the pieces they are splicing together are already mostly words and sentences, though often they are unaware of their meaning. As Robert Sinsheimer warned at the National Academy's forum on recombinant DNA: "The DNA introduced in [E. coli.] are in no sense random sequences of nucleotides. They have been, most often, selected by nature to code for proteins that achieve a function, very often a catalysis. The action of such proteins upon indigenous components of E. coli might split off polypeptides with unfortunate sequences or might convert normal metabolites into undesirable products." (205)

(204) For a fascinating new look at this old problem, see W. R. Bennett, Jr., "How Artificial Is Intelligence?" American Scientist, 65 (November-December 1977), pp. 694-702.

(205) Robert L. Sinsheimer, "Potential Risks," in Research with Recombinant DNA, (Washington, D.C.: National Academy of Sciences, 1977), p. 76.

Under the current NIH regulatory scheme, the level of physical and biological containment for each type of recombinant DNA experiment increases with the presence of known hazardous fragments. Thus, after the initial moratorium was lifted in 1976, the prohibition against using DNA for some carcinogenic viruses or potent toxins (e.g. diphtheria) continued. (206) Other carcinogenic viruses, classified as "low risk," could be used even though virologists do not know how they act: SV40, for instance, causes cancer in newborn hamsters and can infect humans, but is not a human carcinogen. Precautions will be taken, although not on a scale commensurate with Sinsheimer's concerns. (207) The nagging question, therefore, persists: Are the existing models and hypotheses sufficiently well developed to protect against the small possibility of a catastrophic accident?

In contrast to formal scientific hypotheses, there are less rigorous and more intuitive opinions derived from experience. Lewis Thomas has articulated his working philosophy that an organism's pathogenicity is dependent on a malfunctioning symbiosis between it and its host. He believes that: "To be a

(206) National Institutes of Health, "Recombinant DNA Research Guidelines," Federal Register, 41 (July 7, 1976, pp. 27,915 and 49,601. By the end of 1977, NIH had relaxed the total prohibition, suggesting that under special circumstances these experiments might be allowed. "Recombinant DNA Research: Proposed Revised Guidelines," ibid., 42 (September 27, 1977), p. 49,601.

(207) See Sinsheimer's letters to Donald Fredrickson, director of NIH, February 5 and 12, 1976, reprinted in Office of the Director, NIH, Recombinant DNA Research Volume 1: Documents Relating to "NIH Guidelines for Recombinant DNA Molecules" February 1975 -- June 1976 (Washington, D.C. Government Printing Office, August 1976), pp. 436-438 and 443-445.

pathogen, and especially to be a pathogen for us, really takes a long period of interliving and a lot of familiarity," (208) and therefore he perceives infections as "the misreading of signals between the invader and the host... For the signals to be misread, they must be read. If a signal is too strange, it will simply be ignored." (209) With such a view and despite the fact that he is willing to admit how little he and the expert community know about the intricacies of biology, Thomas can at once discount both the hazards of recombinant DNA experiments and of contaminating the earth with a microbe from the moon or Mars. (210) Novel forms of life can do us no harm, he believes, for the very reason that they are new and different. Thomas is by no means alone. Professor Bernard Davis of Harvard Medical School regrets the absence of infectious disease specialists among those evaluating the dangers of recombinant organisms, with the obvious implication that such experts could have told the others that there is nothing to worry about. Davis even believes that tens of millions of dollars were "wasted" on the lunar quarantine program. (211) One can only wonder how many people who are not members of the elite club of researchers would be so cavalier

(208) Jeremy Bernstein, "Biology Watcher: Profile of Dr. Lewis Thomas," New Yorker, January 2, 1978, p. 39.

(209) Ibid., p. 41.

(210) For instance, he finds the risks of recombinant DNA as being "very largely imaginary." ibid., p. 36.

(211) Bernard D. Davis, "The DNA Research Scare," Wall Street Journal, April 5, 1978. See also Davis's "The Recombinant DNA Scenarios: Andromeda Strain, Chimera, and Golem," American Scientist, 65 (September-October 1977), pp. 547-555.

about dismissing such complex and uncertain risks, when the dangers however small, would be so catastrophic.

Life on Mars

The issue of planetary contamination further illustrates how the experts can overgeneralize their experimental results. I have already mentioned the hazards of backcontamination, the return from space of pathogenic organisms which could invade the earth. The parallel and opposite problem is that of contamination, the deposition and growth of terrestrial organisms on alien worlds. Apart from the possibility of destroying some as yet unrecognized life on another planet, the principal fear, first expressed by a National Academy Sciences resolution in February 1958, is that early space research projects will compromise future "critical scientific experiments." (212) An analysis of the potential for contaminating Mars with a terrestrial organism depends in part on its ability to survive and multiply once deposited on the planet, with the numerical probability of growth, $P(G)$, based on some model of the Martian ecology. The best way of learning about an alien planet is by going there, but in so doing one risks its contamination. Space research, like many other PCP's, has a built in catch-22: the most rewarding experiments often entail the greatest risk. While scientists can make some useful

(212) "Development of International Efforts to Avoid Contamination of Extraterrestrial Bodies," Science, 128 (October 17, 1958), p. 887.

observations from afar, the power of long distance research is necessarily imprecise. Given the impossibility of experimenting on Mars, a group of biologists opted for the second best alternative; they looked for a place on earth most like the red planet. They picked the dry valleys of Antarctica. There, they simulated the ability of microbes to live in the harsh and inhospitable Martian environment. Their studies led them to conclude that: "It is evident that the fear that terrestrial microorganisms could multiply and contaminate the planet is unfounded." (213) Whether or not the biologists were correct about what would actually happen on the Martian surface is still an open question. But, three years later, in 1975, a second set of experiments, also staged in the South Pole, yielded contradictory results. Walter Sullivan of the New York Times reported: "Discovery of bacteria in all 24 samples from one of the driest, coldest regions on Earth has raised fears that microbes carried to Mars may proliferate there, eliminating any chance of learning whether that planet had any life of its own."

(214) Subsequent studies have shown that there is indigenous microbial life in those same Antarctic dry valleys. Blue-green algae live inside rocks, which lie on the valley floors. The rocks are porous and the algae can grow in their interior air

(213) N. H. Horowitz, R. E. Cameron, and J. S. Hubbard, "Microbiology of the Dry Valleys of Antarctica," Science, 176 (April 21, 1972), p. 244.

(214) Walter Sullivan, "Experts Fearful for Mars Mission," New York Times, April 1, 1975.

spaces. (215)

In 1976, the two Viking spacecraft landed on Mars, and began to send spectacular pictures of the planet's rock strewn terrain back to earth. Mars seems to have withstood the assault of Viking -- the planet does not appear to have been contaminated. One of the main reasons for the Viking mission was to settle one of man's oldest questions: Is there life on Mars? For reasons similar to those that pointed away from contaminating the planet, the odds of finding anything alive were thought to be tiny. The first signals from the Viking experiments to reach the Jet Propulsion Laboratory in Pasadena, California astonished the scientists: they indicated biological activity. During those uncertain times, one scientist is reported to have revised his probability estimate of finding life on Mars from a million-to-one to fifty-fifty. (216) Today, careful examination of all the Viking data suggest that the planet is indeed without life, though this is hardly a certain conclusion.

What are we to make of these estimates (guesses?) of the probability of unlikely events? How could the first group of biologists have cited their conclusions on the chances of contamination so confidently? The researchers failed to keep in mind the complexities and uncertainties. Most students of

(215) E. Imre Friedman and Roseli Ocampo, "Endolithic Blue-Green Algae in the Dry Valleys: Primary Producers in the Antarctica Desert Ecosystem," Science, 193 (September 24, 1976), pp. 1247-1249.

(216) Timothy Ferris, "The Odyssey and the Ecstasy: The Viking's Search for Life on Mars," Rolling Stone, April 7, 1977, p. 64.

ecology have focussed on those regions of the earth where the majority of people live, and it is not surprising that so little is known about the desolate areas of Antarctica. The biologists did not allow for the possibility that even in the extreme conditions of the South Pole, there could be significant environmental variations, and therefore variable odds of microbial survival and growth. The fact that they made the wrong inferences does not make them blameworthy, for that is not uncommon on the frontiers of scientific research. What is disconcerting is the ease with which they and other experts overestimate their knowledge and neglect the uncertainties. When it comes to PCP's, there is always the chance that the result will be disastrous.

B. The Experts as an Interest Group

There is a parallel between the individuals' and the experts' reaction to PCP's. On one level, there are the individuals who are unable to fathom the intricacies of the subject, and who tend to misperceive PCP risks. On another level, there are the experts who are technically knowledgeable, but who fall victim to perceptual distortions when evaluating risks. For each, the distortions are different, and each's resultant bias may or may not be in the same direction -- to magnify or diminish the risk. The effect is that neither can be trusted to give a balanced view of PCP risks. In order to

understand the distortions at work in the minds of the experts, one must begin by seeing them as members of an interest group.

Like all pressure groups, the experts seek to pursue their interests -- for them the objective is to promote their work. Roger Noll, an economist at Caltech, identifies the experts' appetite as no different from that of other members of the society who seek to satisfy their particular needs and pleasures: "Whether the specific project is unraveling the genetic code, searching for life on Mars, discovering the essence of physical matter, or comprehending more completely the behavior of complex social systems, the act of expanding the frontiers of human understanding is, to some at least, of considerable interest in its own right. Research is, then, a form of consumer good." (217)

Scientists therefore are motivated to align their estimates of risk with their perceived research interests; of course, the extent to which this occurs varies from case to case.

(218) Research as a goal should not be construed too narrowly: scientists also want to have their work applied. The microbiologists study genetics with the hope of curing genetic diseases and breeding high yield crops; influenza specialists study virology in the hope of preventing future pandemics; and

(217) Roger G. Noll and Paul A. Thomas, "The Economic Implications of Regulation by Expertise: The Guidelines for Recombinant DNA Research," in Research with Recombinant DNA (Washington, D.C.: National Academy of Sciences, 1977), p. 265.

(218) My focus here is the distortions stimulated by professional interests -- there may be other, more mundane, factors at work: for instance, the corrupting influences of direct financial gain.

seismologists study geophysics with the hope of predicting earthquakes. Most experts, however esoteric their work may seem, would like to see social benefits derived from their efforts. Nor should the research objective be oversimplified: there are rivalries within and among the various communities of experts. Scientists will compete with each other for the glories of being the first to make a discovery or propose a new theory. Within each community, there is a hierarchy and advancement brings both larger research grants and honors such as membership in the National Academy and, for a select few, a Nobel prize. To the extent that most research monies now come from the federal government, with the overall degree of support proportional to the relative standing of the community as a whole, members of each group work together to enhance the status of their particular profession. Recently, earthquake prediction captured Congress' imagination (no doubt partly because Frank Press, an earthquake specialist, is Carter's science advisor); the seismologists' gain will no doubt cost another group some part of the research money pie.

At first, the recombinant DNA controversy appears to contradict these propositions for the scientists themselves were the ones to focus world attention on the hazards of the experiments. If they were so self-interested, why were the members of the Berg Committee willing to "entail postponement or possibly abandonment of certain types of scientifically worthwhile experiments," on the basis of "potential rather than

demonstrated risk?" (219) The call for the moratorium is not as paradoxical as it might seem. Without any doubt, some of the possible experiments were hazardous, as supported by the fact that, as I noted earlier, a few experiments are still banned. The biologists wished to continue their research but not at the risk of a catastrophe, especially one for which they would be blamed. The catch was that the most interesting and possibly most rewarding experiments were also the most dangerous. Research with cancer viruses might bring a breakthrough but might also lead to the escape of a disease organism which could thrive outside the laboratory. And if such a calamity were to occur, experience with other risky policies indicated that strict government intervention would soon follow. The scientists found themselves in a classic prisoner's dilemma: if they did not cooperate voluntarily to restrict some of the experiments, they might all lose much more. But it wasn't good enough for only some of them to hold off on the experiments, for then the others would have an advantage. If some had to wait, they all had to wait; hence the call for a moratorium, and the NIH guidelines. Surely it was better to regulate themselves through an advisory committee at NIH, than to risk an independent regulatory agency later promulgating much more stringent rules. NIH was, after all, the source of research funds and the chief promoter of research. When the AEC had played the dual role of promoter and regulator of nuclear power (before it was split into two separate

(219) Paul Berg, et al., letter, Science, 185 (July 26, 1974), p. 303.

bodies), the nuclear industry had thrived. The molecular biologists were buying insurance: give up a little now to protect against a later, potentially huge loss.

The experts were not prepared for the public's reaction. Once advised of the risks, many people were apprehensive; they wanted to be protected from what they perceived to be awesome dangers -- that they did not really understand all the details did not stop them from voicing their concerns. The scientists were offering self-regulation, not seeking public regulation. As quickly as they had brought the hazards into public light, they sought to cover them up. The NIH guidelines was all the biologists wanted. With the threat of strong legislation pending in Congress, especially the possibility of a law allowing each state to design its own, stricter research rules, the scientists counterattacked. Meeting in June 1977 at the Gordon Research Conference on Nucleic Acids (the same forum from which, four years earlier, the scientists first publicly warned about the risks of recombinant DNA, leading to the establishment of the Berg Committee (220)), some of the biologists concerned with the threats to free inquiry by legislation and regulation, wrote an open letter to Congress which said, in part: "We feel that much of the stimulus for this legislative activity [under consideration by Congressional, state, and local authorities] derives from exaggerations of the hypothetical hazards of recombinant DNA research that go far

(220) Maxine Singer and Dieter Soll, letter, Science, 181 (September 23, 1973) p. 1114.

beyond any reasoned assessment. This meeting made apparent the dramatic emergence of new fundamental knowledge as a result of application of recombinant DNA methods. On the other hand, the experience of the last four years has not given any indication of actual hazard. Under these circumstances, an unprecedented introduction of prior restraints on scientific inquiry seems unwarranted." (221) By the end of the year, most of the "dramatic" new knowledge had yet to appear in the scientific journals. At an NIH meeting convened for public discussion of some revision to the research guidelines, Robert Sinsheimer found it difficult to comment, given the "extensive reliance on unpublished data." (222) Having protected the public and themselves from the most serious risks, the molecular biologists wanted to resume their research. But in order to do that, the scientists began to take the position that there were no risks -- it suddenly seemed as if the public had made the whole thing up. The letters to Science and Nature warning of potential dangers were forgotten, as was Asilomar. By 1978 recombinant DNA research was proclaimed safe much like President Nixon had once proclaimed that the fight to reclaim the cities had been won. David Baltimore, an original member of the Berg Committee, chastized the author of one of the many books on genetic engineering for failing to demonstrate "that there are any

(221) Walter Gilbert, letter, Science, 177 (July 15, 1977), p. 208.

(222) Nicholas Wade, "Gene-Splicing Rules: Another Round of Debate," Science, 177 (January 6, 1978), p. 31.

hazards to recombinant DNA work; [the author] merely accepts quotable remarks of some of the fearmongers who would rather listen to their own imaginations than to think through the questions rationally." (223) Suddenly there were no more risks!

The issue of recombinant DNA regulations as a threat to freedom of inquiry has become one of the rallying cries of the research community. Lewis Thomas has asked: "Is there something fundamentally unnatural, or intrinsically wrong, or hazardous for the species, in the ambition that drives us all to reach a comprehensive understanding of nature, including ourselves?"

(224) On the contrary, he argues, the greater danger is that we try to suppress our curiosity. But Thomas has missed the point: no one seeks to curb inquiry for its own sake, only the means of that inquiry. (225) Gene-splicing is a research technology; it is not the only avenue leading to an understanding of genetics, although in certain cases it may be the quickest.

Just as the microbiologists, once threatened with restrictions on their research, minimized the risks of recombinant DNA experiments, other groups of experts have acted in their professional interests to increase the perceived risks

(223) David Baltimore, "Genetic Scaremongering," Nature, 272 (April 27, 1978), p. 767. Baltimore was reviewing Robert Cooke's Improving on Nature: The Brave New World of Genetic Engineering, (New York: Quadrangle-New York Times Books, 1977).

(224) Lewis Thomas, "The Hazards of Science," New England Journal of Medicine, 296 (February 10, 1977), p. 328.

(225) David Baltimore has followed the same misleading line of reasoning, see "Limiting Science: A Biologist's Perspective," Daedalus, 107 (Spring 1978), pp. 37-45.

of other policies. It was a group of scientists who sought and gained the adoption of strong quarantine measures against the contamination of the Moon and the planets. The risks of ruining future experiments, one of the most feared consequences of accidentally releasing microbes on an alien planet, was most eloquently phrased by Carl Sagan: "biological contamination of the Moon would represent an unparalleled scientific disaster, eliminating promising approaches to such problems as the early history of the solar system, the chemical composition of matter in the remote past, the origin of life on Earth, and the possibility of extraterrestrial life." (226) A month after the 1958 NAS resolution which first recognized the contamination problem, a Committee on Contamination by Extraterrestrial Exploration (CETEX) of the International Council of Scientific Unions (ICSU) met at the Hague and recommended that a "code of conduct be drawn up" so that early experiments do not spoil subsequent research. (227) The then newly-organized NASA accepted its responsibility to protect scientific investigations into space, and the reputation and integrity of the United

(226) Carl Sagan, "Biological Contamination of the Moon," Proceedings of the National Academy of Sciences, 46 (1960), p. 376. See also: Joshua Lederberg and Dean B. Cowie, "Moondust," Science, 127 (June 27, 1958), p. 1475.

(227) "Contamination by Extra-Terrestrial Exploration," Nature, 183 (April 4, 1959), p. 926. CETEX took a conservative position: "In view of the great uncertainties which face space research, all operations which are not capable of conveying meaningful scientific data are to be discouraged even if they do not appear to carry with them a known source of contamination. Risks with the unexpected must be taken, as otherwise no space exploration is possible; but such risks must be justified by scientific content of the experiment." Ibid.

States, establishing as official policy that "payloads which might impact a celestial body must be sterilized before launching." (228)

The NAS's concern with contamination in 1958 was six years before the NAS first officially considered the hazards of backcontamination. (229) (Remember: backcontamination is polluting the earth with alien organisms, and contamination is the opposite hazard.) To some scientists, this precedence of contamination over backcontamination is logical because we were able to go to the moon and planets before we could return. Further, they may contend that we know there is life on earth which might contaminate other worlds, but we have no evidence that that the solar system harbors other forms of life. Neither of these views is persuasive; fears of backcontamination, if sufficiently strong, could be the principal force in the development of a space exploration program. (230) More important, whatever the threats of contamination may be to future scientific experiments, the threat of backcontamination is the continuation of life of earth! The hiatus between the

(228) Letter of Dr. Abe Silverstein, director of Space Flight Programs at NASA to the affiliated laboratories, October 1957, quoted in Charles R. Phillips, The Planetary Quarantine Program, Origins and Achievements, 1956-1973, (Washington, D.C.: NASA, 1974), p. 10.

(229) Space Science Board, Conference on Potential Hazards of Back Contamination from the Planets 29-30 July 1964, (Washington, D.C.: National Academy of Sciences, revised February 19, 1965).

(230) For an example of how such considerations might affect missions to Mars, see Richard S. Young and Donald L. De Vincenzi, "From Mars w'ith Love," Science, 186 (November 8, 1974), p. 495-501.

examination of these two PCP's is partly because the contamination "disaster" might directly affect the work of a group of scientists, while the other would prove to be a more diffuse hazard -- that is a problem for us all.

Conclusion

Experts have attained a new and extremely important role in social decision making. Theoretical and linguistic barriers place them beyond easy review; the public cannot keep them accountable.

Diverse groups of experts are becoming the sole estimators of potentially very serious types of risk. Each community of scientists can have enormous impacts on society as a whole if they misjudge the hazards of their work. In the next chapter, I will take a close look at one such community: I will show how, in 1976, the influenza experts convinced the country to embark on a national swine flu immunization program on the basis of a combination of premature models, a touch of hysteria, and a genuine desire to do good. Here again it will become apparent that the flu scientists were promoting themselves and their work; they were acting in their own interests, and there was no one who could stop them.

Chapter 9

THE SWINE FLU IMMUNIZATION PROGRAM OF 1976

On March 25, 1976, President Ford asked Congress for \$135 million for the "production of sufficient vaccine to inoculate every man, woman, and child in the United States" against swine flu. (231) He had consulted many of the best doctors in the country, and was convinced that he was making the right decision. After all, he now believed that the "scientific community essentially understands what we are dealing with" and had developed an effective vaccine against it. While he acknowledged that the seriousness of the threat was in doubt, he would not risk the health of the nation because, as the President did not fail to note, during its last visitation in 1918-1919 the swine flu had caused the death of over half a million Americans and some 20 million people worldwide. (232) Congress soon enacted the special budget request, and on April 15, President Ford signed it, initiating the largest immunization drive in United States history.

The chain of events which ended with Ford's signature on the appropriation bill began only 61 days earlier when in the midst of a A/Victoria flu epidemic, a soldier, private David

(231) "Swine Flu Inoculations," Weekly Compilation of Presidential Documents, 12 (March 29, 1976), pp. 484-485. (Emphasis added.)

(232) Ibid.

Lewis, collapsed and died at Fort Dix, New Jersey. Ten days later, government scientists in Atlanta identified the cause of death as a virus similar to the swine influenza virus, the agent long believed to be the cause of the 1918-1919 influenza pandemic. In the fifty remaining days, health officials designed and endorsed a national plan to provide free flu shots for all Americans before the swine virus returned, as they predicted, the following winter. The plan breezed through the Federal bureaucracy, and was approved by Congress. With surprising speed, diverse groups joined hands to promote what would soon become one of the most embarrassing chapters in the annals of preventive medicine. By the year's end, the swine-like virus had failed to make a re-appearance, and the immunization drive had flopped -- on December 16th, the program was suspended.

Things went wrong quickly. In early June, Parke Davis, one of the four drug companies producing vaccine, announced that it had made two million doses of the wrong vaccine. (233) (The actual number would turn out to be closer to six million doses.) (234) Then in close succession, European health experts intensified their criticism of the American effort, (235) the insurance companies began to cancel the drug companies' liability

(233) "Two Million Doses Made of Wrong Swine Flu Vaccine," New York Times, June 3, 1976.

(234) U.S. General Accounting Office (GAO), The Swine Flu Program: An Unprecedented Venture in Preventive Medicine, (Washington, D.C.: GAO, June 27, 1977), pp. 51-52.

(235) Walter Sullivan, "Experts in Europe Question U.S. Plan for Mass Flu Shots," New York Times, June 9, 1976.

policies, and the vaccine failed its first trial for persons under the age of 23.

July followed June's example. As the Congress continued to look for a solution to the liability problem, editorial writers at the New York Times, apprehensive about the program from the beginning (236) urged President Ford to scale down "the frenzied emergency program that the scaremongers foisted on the nation." (237) Albert Sabin agreed: although he had stood alongside other health experts and the President the afternoon Ford had committed the nation to the fight against swine flu, he now defected, favoring a limited immunization drive aimed at only "high risk" groups (238) -- the type of program which had long since become standard practice. Massachusetts, a maverick once more, adopted such suggestions as official policy; only the old and the chronically ill would be encouraged to have a shot. (239) But the most damning news came from England where tests showed that volunteers infected with the New Jersey virus suffered only mild reactions. The virus did not seem to have any of the dread properties which had brought death and havoc at the

(236) See: "Flu Vaccine," New York Times, April 6, 1976, and "Swine Flu False Alarm?" ibid., June 8, 1976.

(237) "Swine Flu Scare," ibid., July 3, 1976.

(238) Matt Clark and Mary Lord, "The Swine-Flu Snafu," Newsweek, July 12, 1976. See also: Albert B. Sabin, "Swine Flu: What Happened?," The Sciences, 17 (March/April 1977), pp. 14-15, 26-30.

(239) Loretta McLaughlin, "Massachusetts Approves a Limited Swine Flu Immunization Program," Boston Globe, July 8, 1976.

close of World War I. (240)

The drug companies continued to make vaccine, but without insurance they refused to ship any out until the government guaranteed them protection from the suits they believed would grow out of a massive drive with a partially tested vaccine. They would accept responsibility for any negligence on their part, but not for the myriad possible side-reactions unrelated to their product -- especially when there was no chance for patient and doctor to weigh the risks and benefits of immunization. As Ivan Husovsky, the President of Merrell-National Laboratories, another of the vaccine producers, testified somewhat prophetically to a House subcommittee: "There are going to be circumstances ... where, by pure co-incidence, people just happen to die on the same day when they get vaccinations." (241) The summer was almost over and the program was stalled. Attributing the delay to a Congressional deadlock on the assignment of liability, seemed to be an excuse for a deepening skepticism about the whole vaccination venture.

Just when the insurance bill appeared doomed, it was revived by a mysterious assault on an American Legion convention in Philadelphia. Many Legionnaires were developing fevers, aches, and respiratory problems and an alarming number were

(240) A. S. Beare and J.W. Craig, "Virulence for Man of Human Influenza-A Virus Antigenically Similar to 'Classical' Swine Viruses," Lancet, II (July 3, 1976), pp. 4-5.

(241) Swine Flu Immunization Program, Supplemental Hearings before the House Subcommittee on Health and the Environment, 94th Congress, 2nd Session, 1976, pp. 42-43.

dying. During the first days of August, there was a possibility that so-called Legionnaire's disease was swine flu, and as the death toll grew, so did the odds of passing the bill. By August 6, the Center for Disease Control (CDC) in Atlanta, had ruled out a connection between the two diseases, leaving an eerie specter of sudden and uncontrollable death. Some of the advocates of the flu program were not as willing as the epidemiologists to separate the two. In a message to Congress urging immediate action on the indemnification proposal, Ford used the Philadelphia outbreak to his advantage: "I am greatly relieved of course that these tragic deaths were not the result of swine flu," he said, "But let us remember one thing: they could have been." (242) With more than twenty unexplained deaths and scores of other victims in the hospital, the horror stories of the 1918-1919 pandemic came to life. Feeling pressure from all sides, Congress voted to protect the drug companies and absolve itself from the responsibility of a national plague. On the 12th of August, President Ford signed the National Swine Flu Immunization Program of 1976, (243) and the race to deliver vaccine before the flu season was on again.

The program resumed, but a cloud continued to hover over it. Sixty million forms to be signed by those receiving shots were printed and then judged inadequate. Revisions had to

(242) "Swine Flu Immunization Program," Weekly Compilation of Presidential Documents, 12 (August 9, 1976), p. 1249.

(243) Public Law 94-380, August 12, 1976.

be made. (244) Vaccine production was behind schedule, and the gap between proposed and actual delivery dates steadily widened. The Department of Health, Education, and Welfare (HEW) had scheduled two hundred million doses for distribution, by the end of October; the manufacturers now proposed to have eighty million delivered by October 1. When the first vaccination centers opened their doors on October 1, only twenty-five million doses had been shipped out. (245) Even the inauguration day had to be delayed until October because of an ambiguity in the insurance law. The program was running about six weeks late.

That the vaccine would be late was not unexpected; the surprising and disconcerting news was that something had gone wrong during production and part of the vaccine was inactive. A flu shot is supposed to induce the body to make antibodies against the virus. Antibody molecules are complementary to the antigens which dot the surface of the virus; on meeting, the antigens and antibodies essentially interlock, and the virus is neutralized. The influenza virus has two types of antigens, hemagglutinin and neuraminidase, but the new vaccine would stimulate the production of only hemagglutinin antibodies. The importance of neuraminidase antibodies is a matter of debate, though their presence would of course have been preferred. And, as if a final blow was needed, it was discovered that the millions of doses of the "wrong" vaccine produced by Parke Davis

(244) GAO, "Swine Flu," op. cit., pp. 23-25.

(245) Ibid., pp. 48-50.

that spring, had the missing component, and might have provided greater protection than the "right" vaccine! (246)

Throughout that summer, while CDC and HEW officials were planning the upcoming campaign, it was winter and therefore flu season in the southern hemisphere. The World Health Organization (WHO) was coordinating a global search for the new virus. If the swine flu was ready to start a new pandemic, it would probably emerge below the equator as it waited for the right conditions, cold and damp weather, to return in the north. In WHO's collaborating laboratories from Buenos Aires to Johannesburg to Hong Kong, the testing continued but no trace of the swine virus could be detected. Every viral sample turned out to be related to the A/Victoria strain, the virus that had brought the flu at epidemic levels throughout the United States and the world that past winter. (247)

The day before Ford signed the insurance bill, Dr. H. Delano Meriwether, the director of the immunization program, told a Washington Press Club news conference that more than a million Americans would have to be vaccinated each day to meet the campaign's goals. (248) By the time the program was nine days old, on October 9th, only some 681,000 doses had been

(246) Philip M. Boffey, "Swine Flu Vaccine: A Component Is Missing," Science, 193 (September 24, 1976), pp. 1224-1225.

(247) See selected issues of Weekly Epidemiological Record, June-September, 1976.

(248) Harold M. Schmeck, Jr., "Million A Day to Get Flu Shots In Drive Starting in Early Fall," New York Times, August 12, 1976.

administered. (249) Then, two days later, on the 11th, three people, each over 70, died after receiving a shot at the same Pittsburgh clinic within an hour of each other. (250) The program, besieged by inefficiency and public apathy from the start, now turned into a national body hunt. By the 13th, CDC had identified some 14 people in nine states who had died within 48 hours of vaccination -- the AP counted 24 deaths, and UPI 25. Lines waiting outside vaccination centers disappeared, and many states suspended local programs until the causes of deaths could be established. (251)

CDC officials insisted that people, especially old people, died all the time with or without a flu shot. If you looked for bodies, you would undoubtedly find them, they argued. The Pittsburgh deaths became a statistics puzzle: were they a coincidence? Was something wrong with the vaccine? Or was something else going on? (252) The deaths seem to have been caused by a tense situation, receiving a shot, made more stressful by poor planning -- everyone regardless of age and

(249) Harold M. Schmeck, Jr., "More Deaths Reported After Shots But No Link to Flu Vaccine Is Found," ibid., October 14, 1976.

(250) Morbidity and Mortality Weekly Report, 25 (October 22, 1976) p. 331.

(251) Schmeck, "More Deaths Reported After Shots," op. cit.

(252) Philip M. Boffey, "Swine Flu: Were The Three Deaths in Pittsburgh a Coincidence?" Science, 194 (November 5, 1976), pp. 590-591, 648; see also Mitchell Gail, "Mass Vaccination Probability of Three Sudden Deaths," (letter) ibid., 195 (March 11, 1977), pp. 934-936, and Mark Kac and Sol I. Rubinow, "Probability of the Pittsburgh Deaths," ibid., 196 (April 29, 1977), p. 480.

health had to wait in long lines, with some worrying whether there would be enough vaccine to go around. The program's critics could not accept the coincidence. The New York Times called for a halt to this fight against a "nonexistent disease." (253) Despite the public outcry and slackened demand, the program continued, though now old people were advised to get their shots in the offices of their personal doctors instead of in public clinics.

Soon after the tally of vaccinations passed the 25 million mark in mid-November (254) there came the news the health officials were waiting for: a case of swine flu. Larry Harrison, a Missouri telephone lineman, showed signs of having had swine flu. Investigative teams were sent out from Atlanta, and samples were taken from his family and neighbors, but there was no trace of the virus itself. Nor was his flu contagious. (255) Harrison was the only one to have had swine flu, and in fact, it had not been a bad case: "I've been much sicker with the regular flu," he told one reporter. (256) A swine virus would not be isolated until December when throat washings from a Wisconsin farmer yielded the elusive specimen. Once again, this was not the sign

(253) "Phantom Flu," New York Times, October 14, 1976.

(254) Morbidity and Mortality Weekly Report, 25 (December 3, 1976), p. 383.

(255) Ibid., and ibid., 25 (November 26, 1976), p. 376. Lawrence K. Altman, "An Apparent Case of Swine Flu is Found in Missouri," New York Times, November 23, 1976, Harold M. Schneck, Jr., "Experts Widen Study of Flu in Missouri," ibid., November 24, 1976.

(256) Paul Delaney, "Swine Flu Victim, Back on Job, Asserts Regular Flu Was Worse," New York Times, November 24, 1976.

of an upcoming scourge; the Wisconsin man's pigs also had the flu. No doubt he had caught it from them, and he had not transmitted it to anyone else. (257)

Then on the 16th of December, the program suffered its final setback; this time it would not recover. A number of cases of an obscure paralyzing disease, Guillain-Barre syndrome, were showing up disproportionately among those receiving vaccinations. (258) The chances of coming down with the disease were minuscule, and the odds of succumbing were even smaller. (259) Yet no one understood the illness, or its cause. With still no sign of swine flu, Guillain-Barre closed the whole operation down. There was considerable irony that a program designed to save millions was stopped by a disease whose attack rate is measured in the million to one range.

The swine flu program was an unprecedented endeavor. Health officials at all levels of government had had little time to do the meticulous planning required of such a massive effort. When it was over, some of them would claim that, if nothing else,

(257) Morbidity and Mortality Weekly Report, 25 (December 10, 1976), p. 392, and (December 17, 1976), p. 394.

(258) Lawrence K. Altman, "Swine Flu Shots Halted in Nation; Paralysis is Cited," New York Times, December 17, 1976; and Philip M. Boffey, "Guillain-Barre: Rare Disease Paralyzes Swine Flu Campaign," Science, 195 (January 14, 1977), pp. 155-159.

(259) Morbidity and Mortality Weekly Report, 50 (December 24, 1976), pp. 401-402, and subsequent issues.

the exercise had taught them a lot about how to organize an immunization campaign. Yet the significance of what happened has less to do with insurance liability, Guillain-Barre syndrome, presidential politics, the drug industry, and even the influenza virus itself. Rather the whole episode is an example of a more pervasive problem: the role of experts and their interaction with the rest of the society in setting public policy.

Small groups of experts are in a position to dictate policy on complex and uncertain public issues, leaving those without training in the particular discipline nothing more than a back seat in what might turn out to be a bumpy, and possibly disastrous, rollercoaster ride. Whether the issue is recombinant DNA research, nuclear power, ozone depletion, or chemical carcinogens, government decision makers and the public alike must bow to the advice of experts. In the case of swine flu, the community of influenza scientists advocated a national campaign on what appears to be a combination of premature models, a touch of hysteria, and a genuine desire to do good. The trouble was that no one, with the exception of WHO, some foreign scientists, and a few lone Americans, was in a position to challenge the basis for the experts' prediction. The details were too arcane for the uninitiated, and there was no time for indecision. The scientists were telling everyone that the virus was on its way, and that it would bring a pandemic on a scale unknown since 1918-1919.

II

There was no shortage of people to blame for what has become known as the "swine flu fiasco" or the "swine flu snafu." Many accused Ford, a weak President seeking reelection, of using swine flu as a campaign issue. Others held the drug companies responsible for promoting a plan that would bring them added revenues and profits. Still others charged the public health officials at the CDC in Atlanta with trying to enhance their prestige -- a successful program would put CDC on the map as the nation's preventive health capital. And there were those who blamed the press for scaring the public with sensational propaganda detailing the 1918-1919 deaths.

Everyone appeared to have something to gain. Who would vote against a man who protected us from a coming plague, especially if the plague was not due until after the election? Who could not believe the drug companies were lusting after new markets, and therefore increased profits? Who still had faith in the CDC officials after the way they seemed to have botched the Legionnaire's disease investigation? And surely sensational headlines and stories about the coming influenza pandemic sell newspapers? While the answers appear to incriminate all the principal participants, a detailed look reveals a number of troublesome inconsistencies. It was highly unlikely that President Ford was doing anything but following the advice of his health advisors. He could see political advantage in the program, but he would never have endorsed it on his own. The

drug companies might make some money from the program, but large corporations seek long-term and assured markets for their products. Massive one-time sales are less desirable than constant, steady sales. There is some profit in annual vaccination drives, but this market was secured: high risk groups were already receiving yearly shots. With an anxious Congress watching over the program, the companies would have little opportunity to reap much profit, particularly if there would be no repeat performances in later years. CDC would gain the most -- a victory over swine flu would bring more money for other programs, and a larger role in national health policy. But if there was to be a pandemic, the CDC should mobilize to protect the American public: this was the Center's job. The newspapers covered the issue with many front page stories, and this helped their circulation, but the press hardly accepted the pronouncements of the government and health officials without question. The New York Times, the New York Daily News, the Boston Globe, the Chicago Sun Times, and the Christian Science Monitor, for instance, were often critical of the program. (260)

(260) See for instance: Judith Randal's three-part series, "The Big Swine Flu Snafu," New York Daily News, August 22-24, 1976; "Flu Fears Exaggerated," Boston Globe, editorial, July 24, 1976; Richard Knox, "Rumors About Swine Flu Worse than the Disease," Boston Globe, July 30, 1976; "Doubts About Flu Campaign," Christian Science Monitor, July 27, 1976; and letters on Swine Flu Campaign to the Columbia Journalism Review 16 (September/October 1977), pp. 72-74 (in response to David M. Rubin's "Remember Swine Flu?" ibid, 16 (July/August, 1977), pp. 42-46.) The Village Voice and the National Enquirer, two weekly papers were also extremely critical. Alexander Cockburn and James Ridgeway, "Is Death at the Doorstep? Journal of the Plague Scare," Village Voice, October 25, 1976; and Chuck Michelini and

In fact, with the exception of a few members of the public interest community and a few doctors, such newspapers were the only voices of dissent. (261)

It is more appropriate to say that no one had anything to lose from the campaign. Only CDC had much to gain. Yet none of this explains the incredible speed with which the program was adopted -- just two months after the identification of the virus, a national plan to immunize the entire country had been set in motion. The speed of approval holds the key to an understanding of the reasons for the program. (262)

At the time the soldier at Fort Dix died, influenza experts were already waiting for a new virus to appear, indeed some were even expecting the swine virus itself. They had a theory to explain the circulation of flu viruses and a plan for battling any upcoming scourge. The scientists had long dreamed of being able to control influenza, and they believed they would

Dan Schwartz, "Hidden Scandal Behind Swine Flu," National Enquirer, November 2, 1976.

(261) See Statement of Dr. Sidney Wolfe and Anita Johnson of Public Citizen's Health Research Group, House Hearings, June 28, 1976, op. cit., pp. 134-143; Sidney M. Wolfe, "Alarmists Overstate the Need, Ignore the Risks," Los Angeles Times, April 11, 1976; Philip M. Boffey, "Vaccine Imbroglia: The Rise and Fall of a Scientist-Critic," Science, 194 (December 3, 1976), pp. 1021-1025; and Kenneth D. Rosenberg, "Swine Flu," Health Pac Bulletin, no. 73, (November/December 1976).

(262) A further explanation is that everyone was simply doing what Charles Lindblom calls "muddling through." (See Chapter 10.) Arthur Visellear, "A Short Political History of the 1976 Swine Influenza Legislation," in History, Science, and Politics: Influenza in America, 1918-1976, ed. by June E. Osborn (New York: Prodist, 1977), p. 51. The fact that the national campaign was approved so quickly does not support such a contention.

soon be prepared for the arrival of a new pandemic strain. On the same day that CDC identified the Fort Dix strain to be swine-like, an article on the op-ed page of the New York Times warned of a new global influenza pandemic. Dr. Edwin D. Kilbourne, a noted researcher at Mount Sinai Medical Center in New York, writing on behalf of the community of flu scientists and independently of any knowledge of Private Lewis's fate, predicted that within a short time a new and especially lethal strain of influenza would emerge; he urged the government to prepare for it now. Committees had to be set up to ensure adequate surveillance of new virus types, and to guarantee the production of enough vaccine to immunize the population. (263)

So, the scientists were already mobilized, and now they were taking their case to the public. What they did not know was that this drive to influence public opinion was not for some future effort -- for they would soon believe the virus had arrived. The incident in New Jersey was the first skirmish in a new campaign in the long war against influenza. The virus had, as predicted, reared its head; now it was the scientists' turn and they were ready.

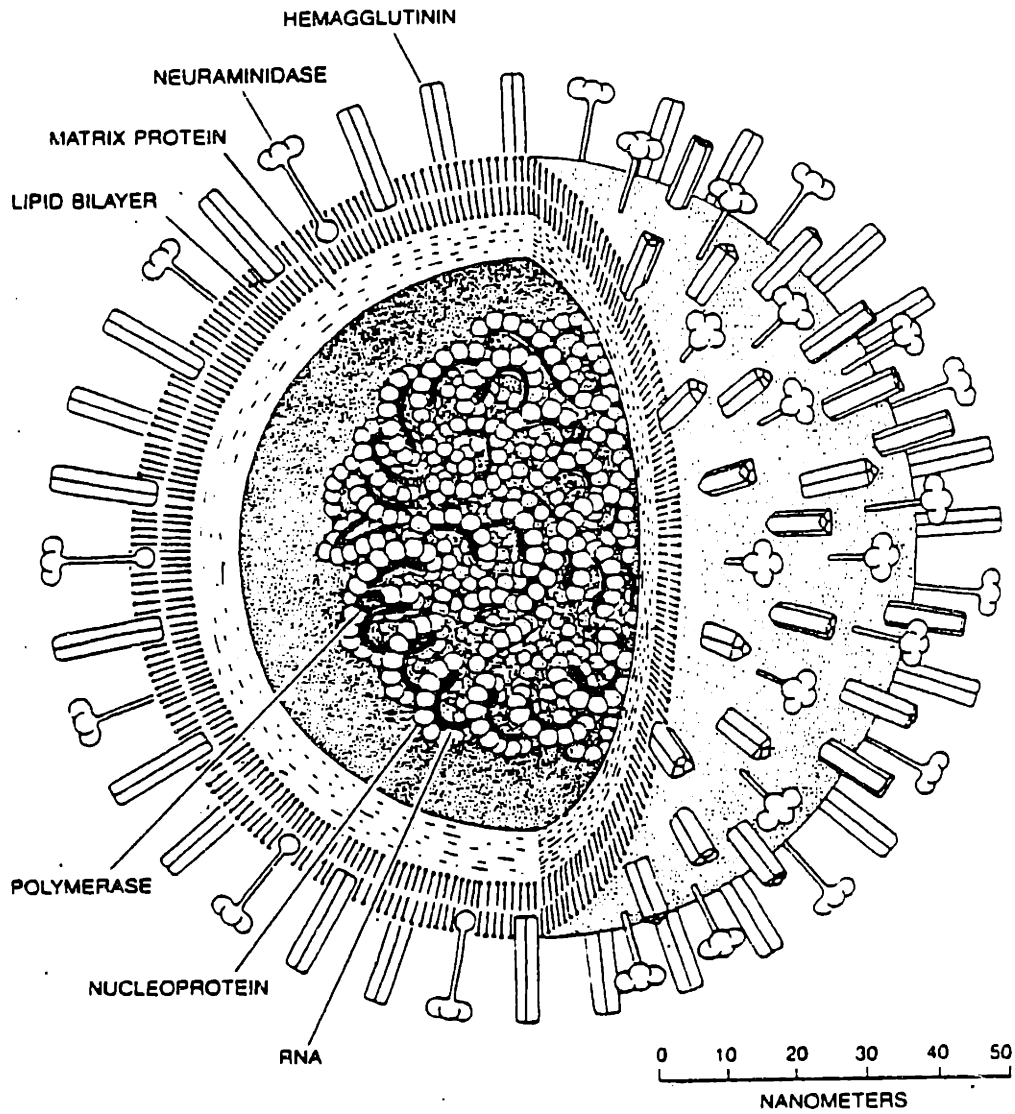
(263) Edwin D. Kilbourne, "Flu, to the Starboard! Man the Harpoons! Fill 'Em With Vaccine! Get the Captain! Hurry!" New York Times, February 13, 1976, and letter, ibid., April 14, 1976.

III

In order to understand how influenza scientists thought they could predict the coming of a new pandemic strain, I must explain the nature of the influenza virus, and the curious way people react to its infection.

The flu virus is always changing. Unlike scores of other pathogens which have yielded to medical science, the influenza virus has resisted human control by constantly mutating. Each time people develop enough protective antibodies, either by catching the flu or through vaccination, the virus has changed and they are defenseless once more.

The influenza virus is an envelope of protein whose exterior is dotted with two types of antigens, hemagglutinin (H) and neuraminidase (N). (See Figure 9-1.) Inside the protein envelope is a supply of RNA, the virus' genetic material. While the genes of human beings and other living beings are contained in DNA, some viruses, including influenza, have the information coded in RNA. The influenza virus has eight genes arranged along its single-stranded RNA (DNA is double-stranded). Two of the genes code for the H and N antigens, and the other six contain the instructions for making the rest of the



1 nanometer = 1 billionth of a meter

Figure 9-1 (265)

virus and for directing its reproduction. (264)

The antigens are the best understood bits of the virus partly because they are on its exterior, but primarily because they leave "negatives" of their structure, the antibodies, in the blood of those infected. When antigens and antibodies meet, the antibodies cover the active sites of the virus, thereby deactivating it. Different antigens generate different complementary antibodies, so a victim's blood can be used to identify the virus. As the surface antigens change, new antibodies are needed to neutralize the virus. Two types of changes have been identified: antigenic "drift" and antigenic "shift." Drift, as the name implies, involves less change than shift. Drift occurs through the random, and constant, mutations in the H and N genes. If the antigens are seen as spikes, drift may be thought of as small variations in the contours of those spikes. As the number of mutations grows, the antigens are in turn altered, and the virus can partially evade the antibodies: antigens and antibodies no longer completely interlock. Shift, on the other hand, involves a sudden and pronounced change in the structure of an antigen; the new spikes have a different shape, and the antibodies are unable to neutralize them. The body

(264) T. Barrett, "Sorting Out the Flu Genes," Nature, 267 (June 16, 1977) pp. 580-581; Peter Palese, "The Genes of Influenza Virus," Cell, 10 (January, 1977), pp. 1-10; Purnell W. Choppin and Richard W. Compans, "The Structure of Influenza Virus," in E. D. Killbourne, ed., The Influenza Viruses and Influenza, (New York: Academic Press, 1975), pp. 15-51; Charles H. Stuart-Harris and Geoffrey C. Schild, "The Antigens of the Influenza Viruses," Influenza: The Viruses and the Disease, (London: Edward Arnold, 1976), pp. 51-77.

cannot defend against a shifted virus, and when a whole population lacks protective antigens, an influenza pandemic often results. While drift is understood in terms of mutations, shift has no commonly accepted explanation.

One of the interesting features of the influenza virus is that when two of them meet their genes can rearrange themselves since each of the virus' eight genes is distinct and independent. After two viruses have recombined, an offspring virus can have either one of each of the "parent" genes; thus there are $2 \times 2 \times 2 \times 2 \times 2 \times 2 \times 2 \times 2$ or 256 possible variants. (265) (See Figure 9-2.) A virus with H1 and N1 genes (that is H1N1) and another with H2 and N2 genes (H2N2) can theoretically join and recombine to give any of the four antigenic types: H1N1, H1N2, H2N1, and H2N2. Similarly, there are variations in the other genes which determine other traits such as virulence, infectiousness, transmissibility, and survivability. Much less is known about which genes code for these characteristics. (It is possible that the antigenic genes affect these viral properties also.)

Influenza viruses are identified by their H and N antigens. Viruses having five major kinds of H (H0, H1, H2, H3, and Hsw1) and two kinds of N (N1 and N2) antigens have been found to infect humans. Of these ten possible combinations (e.g., H0N1, H0N2, H1N1...) five have been isolated. (See Table 9-1.)

(265) Martin M. Kaplan and Robert G. Webster, "The Epidemiology of Influenza," Scientific American 237 (December 1977), pp. 88-106.

ANTIGEN TYPE	FIRST ISOLATION	
	Place	year
H0N1	England	1933
H1N1	Australia	1946
H2N2 (Asian)	Singapore	1957
H3N2 (Hong Kong)	Hong Kong	1968
Hsw1N1 (Swine)	New Jersey	1976

OBSERVED INFLUENZA ANTIGEN TYPES

Table 9-1 (adapted from (265))

H2N2 is better known as "Asian flu," H3N2 as "Hong Kong flu," and Hsw1N1 as "swine flu." There is no quick or easy way to categorize changes in the non-antigenic genes, and they tend to receive less attention. (266) In addition to the human antigens, many other antigens have been found in animal influenza viruses. Pigs, horses, chicken, turkeys, and ducks are all susceptible to the flu, and each has its own types of antigens. Birds have the greatest antigenic diversity with nine different H subtypes and six N subtypes. (267)

The ability of the genes to recombine and the large stock of animal viruses suggest an explanation for antigenic shifts. New human viruses arise from the interaction of human and animal flu viruses; antigens (as well as other genetic characteristics) are exchanged and new infectious strains evolve. There are two possibilities: a new human strain emerges either directly from a mutant animal strain, or from the recombination of human and animal strains. (268) The two theories may be

(266) Some progress is being made in the recognition of the other genes. See Palese, op. cit., and Peter Palese, et al., "Genetic Composition of a High-Yielding Influenza A Virus Recombinant: A Vaccine Strain Against 'Swine' Influenza," Science, 194 (October 15, 1976), pp. 334-335. Peter Palese and Jerome L. Schulman, "RNA Pattern of 'Swine' Influenza Virus Isolated from Man Is Similar to Those of other Swine Influenza Viruses," Nature, 263 (October 7, 1976) pp. 528-530.

(267) Kaplan and Webster, op. cit.; see also: B.C. Easterday, "Animal Influenza," in The Influenza Viruses and Influenza, op. cit., pp. 449-481.

(268) A third possibility is that shifted viruses, like drifted viruses, arise from multiple mutations. This last possibility has been generally discounted. Robert G. Webster and W. Graeme Laver, "Antigenic Variation in Influenza Virus: Biology and Chemistry," Progress in Medical Virology, 13 (1971), p. 325; see

simply variations on the same theme since there is accumulating evidence on the transmission of flu viruses from animal to man and vice versa. (269) Whichever the specific route, the animals most probably serve as reservoirs for flu viruses, allowing strains to constantly circulate. A pandemic virus is created only when the shifted strain has the "right" combination of properties, that is, genes, which endow it with the ability to attack humans, to be transmitted from one person to another, and of course, to be virulent.

Given the 256 possible descendants from any two viruses, the availability of a large number of different animal and human viruses, and the constant potential for random mutations, the diversity of influenza viruses is assured. There is a huge number of possible viruses and not surprisingly, sometimes only a few of the necessary pandemic characteristics occur together. The Missouri telephone lineman and the Wisconsin farmer both caught some type of swine flu but neither virus was contagious, and the Missouri variety was not especially troublesome. Nor were such cases new: in 1974, a post mortem on a young boy who had died of Hodgkin's disease at the Mayo Clinic showed that he had been infected with a swine-like influenza

also Webster and Laver, "Antigenic Variation of Influenza Viruses," in The Influenza Viruses and Influenza, op. cit., pp. 269-314; and Kaplan and Webster, op. cit.

(269) "Influenza Viruses of Lower Animals and Birds," Stuart-Harris and Schild, op. cit., pp. 78-95; see also Edwin Kilbourne, "Recombination of Influenza A Viruses of Human and Animal Origin," Science, 160 (April 5, 1968), pp. 74-76.

virus. (270) The following year, an eight year old boy from Sheboygan, Wisconsin had a similar infection. (271) Both boys had had contact with pigs, and neither virus was contagious. With more surveillance, perhaps many more cases could have been found.

The agent responsible for the great flu pandemic of 1918-1919, known as the Spanish flu, has never been positively identified since the first human influenza virus was not isolated until 1933 -- though Richard Shope first isolated the swine virus from a pig in 1931. (272) If the pandemic began in 1918 and the virus was not recognized until the 1930's, how do we know that Spanish flu was indeed swine flu? While there can be no certainty about this, the pattern of human response to flu infection makes such a hypothesis credible. In a 1936 experiment, Shope found that blood serum from adults neutralized the swine virus but that from children under 12 did not. Only the adults who had been alive during or right after the great pandemic had antibodies to the swine flu. He inferred that the swine virus had been responsible for the 20 million deaths in 1918-1919, and that some ten years later, a new flu virus, the

(270) Thomas F. Smith, et al., "Isolation of Swine Influenza Virus from Autopsy Lung Tissue of Man," New England Journal of Medicine, 294 (March 25, 1976), pp. 708-710.

(271) Michael A. W. Hattwick, et al., Pandemic Influenza, the Swine Influenza Virus and the National Influenza Immunization Program, CDC, National Influenza Immunization Program Pamphlet No. 6, undated, p. 8.

(272) W. I. B. Beveridge, Influenza: The Last Great Plague, (London: Heinemann, 1977), pp. 1-10.

one isolated in 1933 (HON1) had replaced the swine virus -- it was to this new virus that the children's sera responded. (273) Some researchers were still skeptical, chief among them was Thomas Francis, Jr., who was on the way to becoming one of the world's most noted influenza specialists. (274) Francis had one piece of experimental evidence with which to counter Shope: repeated inoculations with HON1 strains induced immunity and antibodies to the swine virus. Francis believed that such multiple exposures stimulated a broad spectrum of antibodies, which could neutralize swine antigens. (275) It was as if the antibodies lost some of their specificity over time, and could accommodate more than a single kind of antigen. The failure of the young children's blood to defend against the swine virus could also be explained by Francis: the antibodies had not yet "broadened" and were still strain specific to HON1.

Francis would later be convinced that the 1918 and swine viruses were related by the mounting evidence for a new immunological phenomenon which he called the "Doctrine of Original Antigenic Sin." The first influenza virus to attack the human body governs all its future immune responses to influenza. Throughout a person's life, the body will make antibodies to all

(273) Richard E. Shope, "The Incidence of Neutralizing Antibodies for Swine Influenza Virus in the Sera of Human Beings of Different Ages," Journal of Experimental Medicine, 63 (1936), pp. 669-684.

(274) For a general history of these developments, see: Greer Williams, Virus Hunters, (New York: Knopf, 1960), pp. 200-227.

(275) Thomas Francis, Jr., "Influenza: The New Acquaintance," Annals of Internal Medicine, 39 (August 1953), p. 219.

flu viruses, but, as if the immune system cannot forget its first bout with the flu, it always makes more antibodies against that first virus than any later ones. (276) Therefore, according to the Doctrine, each person's blood contains a historical record of past flu strains, whichever antigens the blood serum best neutralizes indicates the dominant strain circulating during that person's youth. (Of course, the blood only reveals information about the antigenic types.) Now Shope's findings were no longer considered to be coincidental -- they fit into the larger immunological framework specified by the Doctrine. With the Doctrine, it was possible to venture even further: antibody histories showed that the HON1 strain, though first isolated in 1933, had actually emerged in the late 1920's. Because no pandemic had marked the virus' debut, the specific year remains ambiguous. (277) The influenza community now accepted the 1918-1919 pandemic as a swine related viral disease.

Many years later, in 1969, there would be new doubts about whether or not the Spanish and the swine flu viruses were related. Paul Brown, D. Carleton Gajdusek, and J. Anthony Morris chanced upon the island of Fais in the Caroline group in the western Pacific, which was so remote that the islanders had

(276) Thomas Francis, Jr., "On the Doctrine of Original Antigenic Sin," Proceedings of the American Philosophical Society, 104 (December 1960), pp. 572-578.

(277) Kilbourne gives both 1928 and 1929 as the date of the introduction of the HON1 strain -- he qualifies them by saying the actual date is "speculative," Edwin D. Kilbourne, "Epidemiology of Influenza," in The Influenza Viruses and Influenza, op. cit., p. 493.

escaped all global influenza pandemics except for one in 1924. The researchers found evidence that the attacking virus was a descendant of the 1918 strain. Antibody tests revealed a surprising, and to some experts, disconcerting discovery: the Fais islanders better neutralized the H0N1-type viruses than the swine virus. They concluded: "our results indicate that the virus circulating in the 1918 pandemic was more closely related to human type A strains circulating in the middle 1930's than to other known influenza virus strains, including the virus of swine influenza." (278) In their support, they cited Francis's earlier finding that older people could neutralize the swine flu virus because of a broadening of the H0N1 antibodies over time after multiple infections. With only one attack the islanders had experienced no such broadening. While they could not refute the Doctrine of Antigenic Sin, the three scientists had found a unique historical record which challenged the validity of holding the swine flu virus responsible for the 1918-1919 pandemic.

The Fais findings have never been satisfactorily explained by those who support the 1918-swine virus hypothesis. (279) As Edwin Kilbourne put it: "I can't ignore or discredit it

(278) Paul Brown, D. Carleton Gajdusek, and J. Anthony Morris, "Virus of the 1918 Influenza Pandemic Era: New Evidence About Its Antigenic Character," Science, 166 (October 3, 1969), pp. 118-119.

(279) In contrast to the Fais results, evidence from Alaska supports the 1918-swine virus connection. Stuart-Harris and Schild, op. cit., p. 136.

but I never understood or accepted that paper." (280) Since the report was published, Gajdusek has won a Nobel prize for his work on unconventional viruses, and Morris, a persistent critic of the FDA throughout the 1970's was fired from the FDA's Bureau of Biologics in 1976, because, he claims, he opposed the swine flu immunization drive. (281)

As early as 1952 Francis hypothesized that there are only a certain number of flu antigens, and that they keep circulating. In 1955 he even predicted that the next pandemic virus would be swine related! (282) Though wrong about swine flu, Francis was right about the recycling process -- he just did not know enough about all the possible antigenic types. When the Asian flu (H2N2) battered the world in 1957, blood serum studies indicated that the Asian virus was related to late nineteenth century viruses, possibly the 1889-1890 so-called "Asiatic," pandemic. (See Table 9-2.) Two Dutch scientists, J. Mulder and N. Masurel showed that some elderly people already had antibodies to Asian flu. They surmised that the Asian virus was not new, and that, consistent with the recycling hypothesis and the Original Sin Doctrine, one virus type was responsible for

(280) Thomas O'Toole, "Why the Swine Flu Program Failed," Washington Post, January 30, 1977.

(281) Boffey, "Vaccine Imbroglia: The Rise and Fall of a Science-Critic," op. cit.

(282) Francis, "Original Antigenic Sin," op. cit., p. 577.

both pandemics. (283)

(283) J. Mulder and N. Masurel, "Pre-Epidemic Antibody Against 1957 Strain of Asiatic Influenza, Lancet, I (April 19, 1958), pp: 810-814.

MAJOR

MINOR

1800-1802

1830-1833

1847-1848

1857-1858

1889-1890 (Asiatic)

1918-1919 (Spanish)

1957-1958 (Asian)

1968-1969 (Hong Kong)

1836-1837

1850-1851

1873-1875

1946-1947

MAJOR AND MINOR INFLUENZA PANDEMICS SINCE 1800

Table 9-2 (284)

(284) Beveridge, op. cit., pp. 26-37.

After the 1968-1969 Hong Kong (H3N2) pandemic, antibody profiles better supported an association between the Asiatic and Hong Kong viruses. Some experts countered that the presence of both antibody types could be explained by a 1899 Asian-type virus followed by a Hong Kong-type virus around 1900. (285) But there was no record of a turn of the century epidemic. A few researchers continued to believe that the Asian and Hong Kong flus had arrived in the same order in the 19th and 20th centuries; either there had been a mild epidemic in 1900, or if the 1889 virus had been of the Hong Kong variety, cyclical history could be preserved by believing that the 1857-1858 or the 1873-1875 epidemic had been caused by an Asian virus. (286)

Francis had predicted the return of swine flu partly because it was the oldest virus known to him, yet his eagerness to pick it reveals the dominance of the Spanish-swine flu pandemic in the minds of influenza scientists. Macfarlane Burnet, a contemporary of Francis and Shope and a Nobel prize winner for his work in immunology, has explained: "I should define the essential objective of influenza virus research as the understanding of the conditions for pandemic influenza of the 1918 type -- and the establishment of the conditions necessary to

(285) Nic Masurel and William M. Marine, "Recycling of Asian and Hong Kong Influenza A Virus Hemagglutinins in Man," American Journal of Epidemiology, 97 (1973), pp. 44-49; see also Stephen C. Schoenbaum, et al., "Epidemiology of Influenza in the Elderly: Evidence of Virus Recycling," ibid., 103 (1976), pp. 166-173.

(286) Masurel and Marine, op. cit., p. 48; and Kilbourne, "Epidemiology of Influenza," op. cit., pp. 515-517.

prevent its reappearance." (287) The prevention of another swine flu pandemic has always been the driving force of the profession. When the first papers on the possible connection between the 1957-1958 Asian and the 1889-1890 Asiatic epidemics were published in 1958, the editors of the noted British medical journal, Lancet, asked: "What has happened to the virus of 1918-1919 pandemic? Will it reappear, and if so will it be as virulent as before?" (288) In 1970 after Francis's death, the polio pioneer Jonas Salk, who would later become a strong advocate of the 1976 campaign, concluded his eulogy to Francis by wondering whether there was finally enough knowledge to prevent another 1918-type pandemic which killed 500,000 Americans among 20 million people around the world. (289)

The virology review is nearly over -- two recent findings will complete the picture. First scientists are detecting similarities between animal and human antigens, beyond that of the swine and 1918 viruses. Among all animal viruses, there are some eleven different H antigens and eight different N antigens. Of these, a horse H antigen is related to the H3 Hong Kong antigen, a duck H antigen is similar to the H2 Asian antigen, and a turkey N antigen is related to the N2 of both the Hong Kong and Asian viruses. In fact, for each type of human H

(287) Williams, op. cit., p. 210.

(288) "Influenza 1889 and 1957," Lancet, I (April 19, 1958), p. 833.

(289) Jonas Salk, "The Restless Spirit of Thomas Francis, Jr., Still Lives," Archives of Environmental Health, 21 (September 1970), p. 275.

antigen there is a related avian antigen. (290) Though the nature of the interaction between animal and human flu viruses -- for instance recombination between the two -- is hypothetical, it is now more appropriate to think of all the viruses as members of one family instead of two. Second, careful analysis has shown that the H0 and H1 strains are not different antigenic types as once believed; they are members of the same shifted type. (291) It is more accurate to think of the H1 strain as a drifted variation on the H0 virus than as a shifted virus. This discovery has enormous implications because the evidence now indicates that no new shifted virus had emerged between 1928 and 1957, a hiatus of nearly 30 years. The already very approximate eleven year cycle, 1918/1928(9)/1946/1957/1968, now has a gaping hole in its middle. Further, it suggests that a drifted virus can be more virulent than its predecessor; the 1928/1946 change may have had less to do with the virus' antigens and more to do with its other genes. This provides an explanation for the sudden and mysterious increase in mortality during the 1918-1919 pandemic. While most reports refer to the Spanish flu pandemic as a single plague, there were in fact three distinct waves of sickness. The first wave in the spring of 1918 was quite mild, with a death rate not unusual for a flu outbreak. It was during the second wave, in the fall of 1918, that the mortality rate soared and the flu terrorized the world (the third wave in 1919

(290) Stuart-Harris and Schild, op. cit., pp. 88-91.

(291) Ibid., p. 62; and Beveridge, op. cit., p. 33.

was less severe than the second). (292) The frightening thing was that the virus could knock out young, healthy men and women. Those stricken were not necessarily the old and infirm. Could the 1918 virus suddenly have mutated into some incredibly more virulent form during the summer of 1918? If nothing else, the changes in severity in 1918 and in 1928/1946 made it clear that one of the crucial determinants of risk lay inside the virus, and at least for the present, out of the scientist's reach.

Years of careful research in laboratories all over the world were paying off. New information was constantly surfacing which led to a stream of hypotheses about the virus. Influenza had a life cycle: parts of which were now clear -- others were still uncertain. A shifted virus would emerge often bringing a global pandemic; in subsequent years it would give rise to a number of drifted strains, all variations on the same antigenic theme, each causing lesser outbreaks of illness. People suffered, and as they recovered they became immune to a family of antigens. Then the virus would shift and drift anew, as the cycle repeated itself. (Table 9-3 summarizes the data on flu pandemics and virus types.) Past pandemic viruses did not disappear; they lay dormant in animal populations, mutating and recombining at random until a new combination of genes allowed the virus to emerge and attack humans once more.

(292) Beveridge, *op. cit.*, pp. 30-33, and Alfred W. Crosby, Jr., Epidemic and Peace 1918, (Westport, Connecticut: Greenwood Press, 1976).

<u>YEAR</u>	<u>ANTIGENIC TYPE</u>	<u>PANDEMIC SEVERITY</u>
1857 or 1873	H2N2 (???)	Major
1889	H3N2(??) Asiatic	Major
1918	Hsw1N1 (?) Spanish	Major
1928 (?)	H0N1-H1N1	None in 1928; Minor in 1946
1957	H2N2 Asian	Major
1968	H3N2 Hong Kong	Major
1976	Hsw1N1	None

INFLUENZA PANDEMIC AND ANTIGENIC TYPES

Table 9-3

In a laboratory at the Mount Sinai Medical Center in New York, a team led by Edwin Kilbourne was harnessing the virus' ability to recombine in order to grow influenza vaccines. In the same way that nature breeds flu strains that cause pandemics, Kilbourne was developing flu strains that reproduced quickly and easily in the laboratory but were designed to be generally benign. Genes for attenuated virulence and enhanced growth were joined with the desired H and N genes, yielding a vaccine which could protect against any new antigenic type -- as long as that type had already been isolated. (293) Once administered the vaccine induced the body to produce antibodies, but the virus itself would be relatively harmless since it was little more than a carrier for the antigens. By the early 1970's Kilbourne had reduced the minimum time for making a new vaccine from six months to four weeks with an accompanying huge increase in yield. (294) Progress was being made on a number of different vaccines, and confidence was high. Some experts hoped that we need never suffer another influenza pandemic. (295)

(293) Edwin D. Kilbourne, "Future Influenza Vaccines and the Use of Genetic Recombinants." Bulletin of the World Health Organization, 41 (1969), p. 643.

(294) Thomas H. Maugh II, "Influenza (II): A Persistent Disease May Yield to New Vaccines," Science, 180 (June 15, 1973), p. 1159.

(295) Ibid., p. 1215.

The flu experts were convinced that a new pandemic was coming. In 1971, the National Institute of Allergy and Infectious Diseases began sponsoring a series of workshops on influenza to share information and consider possible preventive action. (296) Which strain would be next and when would it arrive? If the scientists could predict the upcoming virus, a vaccine could be prepared, and the population immunized.

But there were many uncertainties: was there a limited number of antigenic types? If so, had all of them appeared since 1918, or were there others? Just as Francis could not have known of the pre-1918 viruses, perhaps there were other nineteenth century viruses, still unknown to us, waiting to reemerge. Should most of the attention be focussed on the H antigen, or did the N antigen also play a primary role? The Asian pandemic had been much more serious than the Hong Kong flu, and this was partially attributed to a shift in both antigens in the 1957 virus, from H1N1 to H2N2; in 1968 only the H antigen had shifted, from H2N2 to H3N2. (297) A second factor was that the older generation, a high risk group, already had some protective antibodies from the related 19th century outbreak. (298) The

(296) John R. Seal, David J. Sencer, and Harry M. Meyer, Jr., "A Status Report on National Immunization Against Influenza," Journal of Infectious Diseases, 133 (June 1976), p. 715. Summaries of the workshops were published in this same journal.

(297) Kilbourne, "Epidemiology of Influenza," op. cit., p. 493.

(298) Walter R. Dowdle, Marion T. Coleman, and Michael B. Gregg,

interior of the virus was even more mysterious: would the next strain necessarily be virulent (might the antigens be new, but the insides weak)? Would a virus mutate in the midst of an outbreak, perhaps becoming more lethal, or conversely, more benign? While the odds were that the next strain would be a repetition of a past virus, there was a large number of animal antigens which might adapt to a human host. The historical evidence for a cyclical pattern was circumstantial at best. The eleven year cycle had only begun in 1957 after decades of variability. There was only meager data to support the contention that the 1957/1968 outbreaks were repetitions of the 1873/1890 or 1890/1900 outbreaks, thereby implicating the swine virus as the next pandemic strain. (299) If there was a pattern to the rotation of shifted viruses, it was controlled by a force that no one yet understood.

The only sound conclusion was that the next virus would not have the H2 antigen since it had recently circulated on the Asian virus, and enough people had antibodies to resist a new outbreak. That left the swine and the H0/H1 strains as well as variations on the other animal antigens as possible surface markers. Then there were the virus' other properties, governed by the six remaining genes; they also seemed to be selected at random. The H0/H1 strains had not been very dangerous, while the

"Natural History of Influenza Type A in the United States, 1957-1972," Progress in Medical Virology, 17 (1974), p. 119.

(299) Masurel and Marine were predicting a return of the swine-like virus by 1985-1991, op. cit., p. 48.

second wave of the 1918 virus had been a killer -- no one knew why. With no predetermined sequence, as far as anyone knew the next pandemic virus to emerge would be governed by chance: whichever virus first acquired the "right" properties would be next. It was as if slot machines with eight independent wheels were constantly in play all over the world -- the virus was mutating and recombining and someday the "winning" combination would hit. On that day many people would lose.

As for when it would strike, W.I.B. Beveridge of Cambridge University offered the most sober view: "The only conclusion we can reach is that pandemics have occurred at very irregular intervals averaging between 12 and 24 years," and "experience has shown that predictions about [the virus' emergence] have seldom proved correct." (300)

(300) Beveridge, op. cit., p. 36.

IV

During the winter of 1975-1976, the flu was on a rampage; the cause was the A/Victoria virus, a variant of the 1968 Asian flu (H3N2). In late January, an especially violent wave of sickness struck the army camp at Fort Dix, New Jersey. Seeking confirmation of the virus' identity, army doctors took throat washings and sent them to the state laboratory for analysis. A/Victoria was there as expected, but there were four specimens that did not match. They were forwarded to CDC in Atlanta, where in mid-February, they were identified as a variant of the swine virus. (301) One of those infected by the so-called A/New Jersey/76 strain was private David Lewis. Lewis suffering from flu-type symptoms was ordered to his quarters for two days rest; instead, the following morning he joined the other recruits for a five mile march to the rifle range. After a long and tiring day at shooting practice, he collapsed on the journey back to the base. He died soon afterwards. Colonel Joseph Bartley, chief of preventive medicine at Fort Dix, speculated that if Lewis had obeyed orders and stayed in bed, he would still be alive. (302)

In all, the swine-like virus was isolated from five

(301) Philip M. Boffey, "Anatomy of a Decision: How the Nation Declared War on Swine Flu," Science, 192 (May 14, 1976), pp. 636-641.

(302) Ibid., p. 638; and Lawrence Wright, "Sweating Out the Swine Flu Scare," New Times, June 11, 1976.

soldiers, including Lewis. Seven others showed signs of recent infection, and some 500 others of the 12,000 at Fort Dix had swine antibodies, suggestive but not proof of infection. (303) Meeting followed meeting as health officials tried to decide what to do next. Was this an isolated outbreak, or was this a trial outing of a new pandemic virus -- new types of viral infection might occur quite frequently, but pass unnoticed? Indeed, according to Colonel Bartley, the new virus had nearly been missed at the Fort. (304) The World Health Organization (WHO) in Geneva was unimpressed with the outbreak: "The virus is not easy to characterize in the laboratory," it warned, "and some strains may have been missed in the past." (305) In any case, there were precedents: isolated antigenic changes, that is new viral combinations, had been identified during the interval between the Asian and Hong Kong outbreaks. Such outbreaks were small and had not spread. (306)

(303) Seal, et al., op. cit. p. 716. Some of those whose blood appeared to neutralize the swine virus, may have, as Francis speculated years before, "broadened" antibodies, instead of specific swine antibodies. Also, swine virus antigens were included in U.S. military vaccines from the late 1950's to the mid 1960's; these could have been responsible for some of the antibodies. "The New A/New Jersey/76 Influenza Strain (Memorandum)" Bulletin of the World Health Organization, 53 (1976), p. 3.

(304) Boffey, "Anatomy of a Decision," op. cit., p. 637.

(305) Weekly Epidemiological Record, 51 (February 20, 1976), p. 65.

(306) Dowdle (1970) cited in Sabin, op. cit., p. 27. Albert Sabin reports that Edwin Kilbourne called this fact to the President Ford's attention at a White House meeting on March 24, 1976 -- just before the President announced his decision to go ahead with the flu campaign to the press. Ibid.

But the question everyone was asking was whether this was the same virus as the one that killed twenty million people in 1918-1919, including some 500,000 Americans. No one knew the answer. At a March 10 meeting at the CDC, Edwin Kilbourne reminded his fellow experts that they only had information about the antigenic genes -- they knew little about the other six genes. (307) The available data indicated that the swine virus was not a strong contender for the next pandemic strain; it had been unable to compete with A/Victoria at Fort Dix. If the deaths of otherwise healthy people associated with the second wave of the 1918 pandemic were not to be blamed on secondary infection because of the absence of antibiotics -- Fleming discovered penicillin in 1929 -- the virus had to be able to devastate the lung. (308) It had no such powers. The A/New Jersey virus had died out while A/Victoria thrived, making its way west after sending thousands to bed on the east coast. On the other hand, the swine virus, like the Asian virus, had been the product of a double antigenic shift, H3N2 to Hsw1N1, and could therefore be more dangerous than the 1968 single shift to the Hong Kong strain. And to further complicate the problem, the shift may not be permanent. The virus might still mutate into a more dangerous or benign form. It could now be transmitted from person-to-person, perhaps a small change would make it deadly,

(307) Harold M. Schmeck, Jr., "Flu Experts Soon to Rule on Need of New Vaccine," New York Times, March 21, 1976.

(308) Charles Stuart-Harris, "Swine Influenza Virus in Man," Lancet, II (July 3, 1976), pp. 31-32.

or it might simply revert back to a virus that attacked only pigs.

Whatever the facts, the connection between A/New Jersey and 1918 could not be suppressed. Another pandemic was due, and this could be the big one -- for many scientists every potential outbreak after 1918 could be the swine flu. Sixty thousand Americans had died as a result of the 1957-1958 Asian virus and thirty thousand as a result of the 1968-1969 Hong Kong virus.

(309) Now the experts finally had their chance to beat the flu virus. For them everything fit. It had to be the swine flu: the cycle, as some had predicted, was continuing. The timing was right: it was due anytime according to the approximate, though hardly supportable, eleven year cycle. And best of all, the virus had shown its hand in time for vaccine to be produced before the next flu season. The scientists had never been able to have a vaccine ready before a pandemic because each vaccine had to be grown from last year's shifted or drifted strain. Before disease and control had always been at least one year out of sync; now they had advance notice of what was coming. All the uncertainties gave way. Those experts who believed in the cycle joined those who could not resist the chance to be one step ahead of the virus. This time they were going to be the victors. At the March 10th meeting in Atlanta, after considering a number of "options," the Advisory Committee on Immunization Practices decided that swine flu vaccine should be produced and made

(309) Seal, et al., op. cit., p. 715.

generally available to the entire public. (310) For Harry Meyer, Jr. of the Food and Drug Administration's Bureau of Biologics it was a "choice between gambling with money or gambling with lives." (311)

By choosing a comprehensive campaign, health officials rejected a number of less ambitious alternatives: adding a swine flu component to the next season's vaccine for high risk individuals, stockpiling the the vaccine and delaying inoculations until the virus reappeared, and encouraging immunization for other than high risk groups without attempting to inoculate all Americans. (312) The decision turned on a number of factors but none was more important than the efficacy and safety of the vaccine -- a controversial subject within the preventive health community. (313) In 1972, the U.S. General Accounting Office (GAO) reported that many of the influenza vaccines released by the government in the late 1960's had been sub-potent -- sometimes at a level of one percent of the

(310) Ibid., p. 717. See also: Hattwick, et al., op. cit.

(311) Schmeck, "Flu Experts Soon to Rule on Need of New Vaccine," op. cit.

(312) See Hattwick, et al., op. cit. and memorandum of Dr. Theodore Cooper, the Assistant Secretary for Health to the Secretary of HEW, prepared by David Sencer, CDC, March 13, 1976.

(313) Other variables to be factored into the cost-benefit equation are the costs of vaccine and its delivery, the costs of a pandemic and the acceptance rate of the vaccine. See Stephen C. Schoenbaum, Barbara J. McNeil, and Joel Kavet, "The Swine-Influenza Decision," New England Journal of Medicine, 295 (September 30, 1976), pp. 759-765.

established standard. (314) As for efficacy, the GAO cited CDC's own studies: "influenza vaccines at standard dosage levels had little, if any, effectiveness and that even very large doses of the vaccines did not approach the high degrees of effectiveness which had been achieved with other virus vaccines." (315) In a 1974 paper, three CDC experts hedged on the potential benefits of flu vaccines: "There is no doubt that properly constituted aqueous inactivated vaccines can provide some measure of protection. How much protection they afford is open to question." (316) Nevertheless, in 1976, the CDC was looking only at the high side of the efficacy scale. In a briefing pamphlet for the swine flu campaign, CDC officials (including Walter Dowdle, a co-author of the 1974 CDC paper) wrote: "efficacy rates of approximately 70 [percent] have been found," (317) and in another pamphlet addressed to a non-technically oriented audience the CDC exaggerated further: "at least 70 [per cent] of the individuals receiving the A/New Jersey influenza vaccine will be fully protected against that strain." (318)

If the vaccine provided only very limited protection,

(314) U. S. General Accounting Office (GAO), Problems Involving the Effectiveness of Vaccines, (Washington, D. C.: GAO, March 28, 1972) p. 20. Reprinted in Congressional Record, Senate, March 30, 1972, pp. 11073-11082.

(315) Ibid., p. 23, and Nicholas Wade, "DBS: Agency Contravenes Its Own Regulations," Science, 176 (April 7, 1972), pp. 34-35.

(316) Dowdle, et al., op. cit., p. 124.

(317) Hattwick, et al., op. cit., pp. 10-11.

(318) Rationale for Mass Immunizations in 1976, CDC, undated, National Influenza Immunization Program Pamphlet, No. 5, p. 3.

could it do any harm? Some adverse reactions were always expected. Sore arms, fevers, and general malaise were not uncommon. Beyond these discomforts, there was some uncertainty. In a crash effort, there was no time to test the vaccine for anything except short-term effects. (319) Yet a full-scale program entailed a different type of risk, the small chance of a major catastrophe. If the vaccine contained some latent pathogen, and everyone were to receive it, the repercussions might be dreadful. That possibility did not seem so remote to some after the polio immunization drive of the late 1950's. Between 1955 and 1961, tens of millions of people were vaccinated with a vaccine that contained a virus, simian virus 40 (SV40), which once discovered, was shown to cause cancer in newborn hamsters. Luckily, we seem to be immune to this particular virus, but the verdict is not yet final. Latent effects may show up in the next few years. (320) Unlike the polio vaccine which was cultured on monkey kidney tissues, influenza vaccine is grown in eggs, and there is less chance of serious contamination, yet the possibility remains. In 1976, as soon as a decision was made to protect everyone against swine flu, the risks changed and the improbable presented some disturbing possibilities.

No other country tried to duplicate the American

(319) Paul D. Parkman, *et al.*, "Summary of Clinical Trials of Influenza Vaccines," Journal of Infectious Diseases, 134 (July, 1976), pp. 100-107; and Philip M. Boffey, "Swine Flu Vaccination Campaign: The Scientific Controversy Mounts," Science, 193 (August 13, 1976), pp. 559-563.

(320) See "A Shot in the Arm," in Chapter 4.

effort. Like those at WHO, foreign experts were skeptical. (321) Canada and England decided to immunize only the high risk people, and stockpile some vaccine for a fraction of the rest of the population. (322) Most countries ignored the whole thing. E. Russell Alexander, the chairman of epidemiology at the University of Washington's School of Public Health, was the lone advocate of a wait and see strategy among American experts. (323) The general argument used against him was the lack of time for vaccinating everyone once the virus had struck. (324) Beyond such specific reasons, the Americans discounted the opinions of their foreign counterparts through a sense of technological superiority. As Delano Meriwether, the head of the national campaign, gloated: "No other country has the technical capacity to produce enough doses to meet its needs. We're the only ones able to do it." (325)

Why do it? Dr. Kilbourne summed it up this way for the New York Times:

(321) "Vaccination Against Swine Influenza," British Medical Journal, 2 (July 31, 1976), pp. 265-266; "Planning for Pandemics," Lancet, II (July 3, 1976), pp. 25-26; and Stuart-Harris, "Swine Influenza Virus in Man," op. cit.

(322) "Swine Influenza," Lancet I (June 5, 1976), p. 1252; and Robert Trumbull, "Provinces Had Doubts Epidemic Threatened," New York Times, October 17, 1976.

(323) Harold M. Schmeck, Jr., "Flu Experts soon to Rule on on Flu Vaccine Policy," New York Times, May 8, 1976.

(324) Louis Weinstein, "Influenza -- 1918, A Revisit?" New England Journal of Medicine, 294 (May 6, 1976), p. 1060.

(325) Boffey, "Swine Flu Vaccination Campaign: The Scientific Controversy Mounts," op. cit., p. 560.

Known: 1) we are nearing the end of a decade of prevalence of the Hong Kong subtype of influenza virus, and recently pandemics and major viral mutations have occurred every eleven years. 2) The disappearance of one virus subtype is followed by the emergence of another. 3) The world's population under fifty lack immunity to swine influenza virus. 4) The swine-like virus isolated at Fort Dix is transmissible from man to man and caused hundreds of infections and one death. 5) Influenza is essentially a winter disease, and absence of further cases of swine influenza now may reflect this fact rather than the true disappearance of the virus. 6) The Fort Dix virus is similar in its surface antigens to the probable cause of the 1918 pandemic. 7) Secondary bacterial pneumonia was probably the major cause of death in 1918, but it is also presently the major cause of death from influenza every winter despite the availability of antibiotics. 8) Influenza vaccines are 70 to 90 percent effective and have reaction rates of less than 1 percent, except in very young children. 9) Conferences with industry established the feasibility of producing the targeted 200 million doses. 10) In the relatively mild pandemic of 1968, the economic burden to the nation was nearly \$4 billion. 11) General immunization for next winter requires that the decision to produce and distribute vaccine be made now.

Unknown: 1) The Fort Dix virus may not produce epidemic disease next winter. 2) The virulence of the virus is unknown. 3) Victoria virus may persist. 4) Another mutant virus may emerge.

The alternative to the present proposal represents an abrogation of responsibility to the public health and a perpetuation of an indifference to the problem of influenza. (326)

Once the experts had decided what they wanted to do, few people could challenge them. All those who understood the intricacies of the virus and the epidemiology of the flu belonged to the same community of scientists. Some were at CDC, others were scattered at research centers across the country. The group's motives transcended any specific organizational

(326) Edwin D. Kilbourne, letter, New York Times, April 14, 1976. (emphasis Kilbourne's).

allegiance. The objective was clear: victory over the influenza virus.

David Sencer, the head of the CDC, wrote a memorandum endorsing the full scale immunization drive, and sent it to Theodore Cooper, the Assistant Secretary for Health. Cooper passed the decision document onto David Matthews, the Secretary of HEW. (327) By the time it reached President Ford and the Congress, neither had much choice. One White House Domestic Council staff member described it as a "no decision decision." (328) A national commitment had been made.

WHO, meanwhile, was still unconvinced: "It is entirely possible that this may have been a unique event in a military recruit population," it advised, "and will not lead to widespread epidemics as experienced in 1957-1958 and 1968-1969." (329)

(327) Memorandum of Assistant Secretary for Health to the Secretary of HEW, op. cit.

(328) Boffey, "Anatomy of a Decision," op. cit., p. 640.

(329) Weekly Epidemiological Record, 51 (April 15, 1976), p. 123.

During the months of preparation for the immunization drive, there was only bad news for those committed to the campaign. Many of the uncertainties began to be resolved: results from laboratories in the United States and Europe pointed away from a swine flu pandemic.

In England, volunteers exposed to the swine virus suffered only a mild reaction. The findings confirmed the virus' feeble performance at Fort Dix. The virus had none of the awesome properties that could kill healthy people. (330) From the Netherlands, Masurel found evidence that immunization with the Hong Kong (H3N2) yielded antibodies that could neutralize the swine virus. (331) The 500 cases of swine antibodies among army personnel at Fort Dix were no longer so convincing. Was this another case of antibody "broadening?" (332) The dimensions of the New Jersey outbreak shrunk, and the virus now appeared puny.

(330) Beare and Craig, op. cit.

(331) N. Masurel, "Swine Influenza Virus and the Recycling of Influenza-A Viruses in Man," Lancet, 11 (July 31, 1976), pp. 244-247.

(332) The available statistics from Fort Dix indicate that incoming recruits had low levels of antibodies to the swine virus, as did those who left the base before January 5. They do not detail the age distribution of those (25-63 of the men in the companies to which one of the twelve documented cases belonged) with swine antibodies. Army veterans would probably have received yearly inoculations of H3N2 vaccines which could prompt a swine antibody response, WHO, "The New A/New Jersey/76 Influenza Strain (Memorandum)" op. cit., p. 1.

In New York, two of Kilbourne's associates at Mount Sinai announced that, using a new technique for the analysis of the virus' RNA, they found that all eight genes were closely related to a non-contagious and non-virulent swine virus. Someone had finally managed to study the six interior genes, and the results confirmed the British experiment: the virus was weak. The New Jersey strain did not seem to be a product of a recombination of animal and human flu viruses. The researchers concluded: "we would regard the New Jersey 'swine' virus to be an unlikely candidate for the next influenza pandemic." (333) Their paper, submitted in late June, and published in early October, passed generally unnoticed. (334)

In late September when Philip Boffey of Science reported that the vaccine contained no N antigenic component, it began to look as if the country was preparing a useless vaccine for a non-existent disease. (335) CDC had known about the production error since June, but had not publicized it. Harry Meyer of the Bureau of Biologics saw no reason to believe the N deficiency would make the vaccine ineffective. (336) Yet in April 1974, at the fifth National Institute of Allergy and Infectious Diseases influenza workshop, Kilbourne had lectured on

(333) Palese and Schulman, op. cit.

(334) A later paper by Peter Palese, op. cit., was reported by Science News. 111 (February 19, 1977), p. 117.

(335) Boffey, "Swine Flu Vaccine: A Component is Missing," op. cit.

(336) Ibid., p. 1225.

the possibility of new vaccines which would induce the body to make only N antigens. In such a way, he and his collaborators believed that they could stop the growth and spread of the virus within the infected individual. (337) If they could stop the virus with only N antibodies, they would no longer have to worry about shifts in the H antigen. Now in 1976, on the eve of the immunization drive, Kilbourne could only lament that it would obviously be desirable to have a vaccine that raised antibodies to both H and N antigens. (338)

Despite all these new findings, despite WHO's inability to isolate another swine virus in any of its collaborating laboratories, the campaign went on. It seemed nothing could stop it -- until old people started dying in Pittsburgh. Then a million to one paralyzing disease put the program to rest.

Throughout the winter of 1976-1977, the only flu viruses to hit were A/Victoria and other H3N2 variants. There was no swine virus.

(337) E. D. Kilbourne, et al , "Influenza Vaccines -- Summary of Influenza Workshop V," Journal of Infectious Diseases, 129 (June, 1974), pp. 765-766.

(338) Boffey, "Swine Flu Vaccine: A Component is Missing," op. cit., p. 1225.

On December 7, 1977, another chapter of the long-running flu mystery ended: the Minister of Health of the Soviet Union informed WHO that a new influenza strain had been identified. (339) The new virus was related to the 1947 H1N1 strain. (340) In mid-January 1978, the A/USSR/77 virus struck a Cheyenne, Wyoming high school, sending many students to bed for fluids, aspirin, and rest.

(339) Morbidity and Mortality Weekly Report, 216 (December 16, 1977), p. 410.

(340) Ibid., 27 (February 3, 1978), p. 40.

Chapter 10

THE GOVERNMENT AND PCP'S

To contend that the influenza experts were wrong because the swine virus did not return would be unfair, for they themselves admitted that they were working with the possibilities of disaster, not certainties. (341) (As it turned out, a mild variant of the H1N1 strain surfaced, and will probably cause a minor epidemic during the 1978-1979 flu season.) Nevertheless, the basis for their prediction was extremely thin -- too thin to advocate anything more than preparing vaccine, immunizing high risk groups, and stockpiling some additional vaccine. The lesson of the flu campaign is that no one was in a position to challenge them. J. Anthony Morris, an FDA scientist spoke up, and he was fired. Sidney Wolfe and Anita Johnson of the Public Citizen Health Research Group warned that a mistake was being made, but Nader's organizations are rarely heeded until strong support for their position is in hand. WHO and foreign scientists had not believed the rationale for the program, but the Americans could discount their opinions too easily because the UN agency must respond to the more pressing needs of developing countries where poverty and malnutrition allow preventable diseases to turn every year into a replay of 1918. Who, then, shall monitor the experts? Because PCP's entail collective risks, the conscious choice to initiate, continue, or halt a PCP is usually made by

(341) Philip Boffey reports that according to his informal poll of experts, the probability of a pandemic varied between 2 percent to less than 50 percent, with point estimates at 10 and 35 percent. "Anatomy of a Decision: How the Nation Declared War on Swine Flu," Science, 192 (May 14, 1976), p. 637.

the government on behalf of the public. As we have seen, the government may act because of direct pressure from individuals (Model 1), on the basis of expert advice with public participation (Model 2), or solely in consultation with experts (Model 3).

Whether the actual decision making unit is the President, a member of the Cabinet, an administrator of a Federal agency (EPA, FDA, OSHA, or FAA), an independent regulatory commission (NRC, FPC, or CPSC) or the Congress itself, each relies on the decision analyst to simplify the policy choice. His charge is to write a non-technical document, outlining the pros and cons of each alternative. Blending the models of applied economics, political science, and planning, all elements of decision making technology, (342) the analyst outlines the various "options," searching for the "optimal" alternative. In the study of PCP's, he is guided by the expert who interprets the technical details. The risks of contaminating the planets may be framed in decision theoretic terms, but the analyst must consult those familiar with space travel machinery, the biology of terrestrial organisms, and the ecology of other planets. Similarly, computing the risks of recombinant DNA research requires, at a minimum, a knowledge of genetics, microbiology,

(342) The methods of decision making are part of what Ellul calls "technique." See Jacques Ellul, The Technological Society, (New York: Vintage, 1964).

and laboratory safety.

A. The Technology of Decision Making

The tools of the professional decision maker are known by many names: systems analysis, operations research, optimal resource allocation, and program evaluation, and they include a wide range of different descriptive and prescriptive modeling methods. My discussion will focus on two of the simplest techniques: cost-benefit and decision analysis. (343) Others have written comprehensive reviews of these methods, (344) so I will deal only with those issues of particular importance to PCP's. As in the case of individual decision making, the analyst must determine the possible outcomes and their probability of occurrence.

An illustration of the method is the representation of a simple decision to be made under uncertainty between two alternatives A and B as shown in Figure 10-1. Associated with each alternative are two possible outcomes: O(1) and O(2) for A,

(343) Other techniques include linear and non-linear programming, regression analysis, system dynamics, dynamic and integer programming, and input-output analysis.

(344) For instance, see E. J. Mishan, Cost-Benefit Analysis, (New York: Praeger, 1976); and Howard Raiffa, Decision Analysis: Introductory Lectures on Choices Under Uncertainty, (Reading, Mass.: Addison-Wesley, 1968); R. Duncan Luce and Howard Raiffa, Games and Decisions, (New York: John Wiley, 1957); Howard Raiffa and Robert Schlaifer, Applied Statistical Decision Theory, (Cambridge, Mass.: MIT Press, 1968); and Elizabeth M. Clark and Andrew J. Van Horn, Risk-Benefit Analysis and Public Policy: A Bibliography, (Cambridge, Mass.: Energy and Environmental Policy Center, Harvard University, November 1976).

and $O(3)$ and $O(4)$ for B. At the time of decision it is impossible to know what will result when either option is selected; the best one can do is assign probabilities to each of the possible outcomes. These are $P(1)$, $P(2)$, $P(3)$, and $P(4)$, that is $P(1)$ for $O(1)$, $P(2)$ for $O(2)$ and so on.

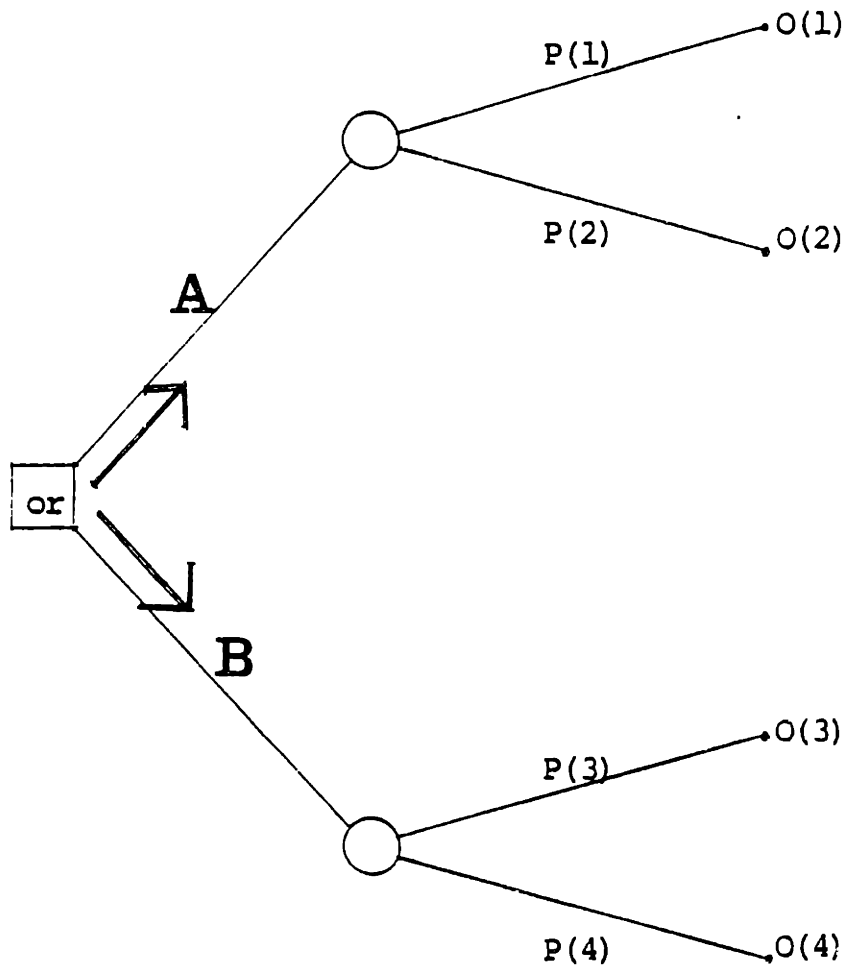


Figure 10-1

Once the outcomes are identified, cost-benefit analysis is used to compare them in some common units, usually dollars, ($\$0(i)$) or some richer measure like the subjective utility of the result. All the information is then brought together to give the expected value of each alternative action. For the option A in Figure 10-1, the expected value of the decision is:

$$P(1) \times \$0(1) + P(2) \times \$0(2)$$

similarly for option B,

$$P(3) \times \$0(3) + P(4) \times \$0(4)$$

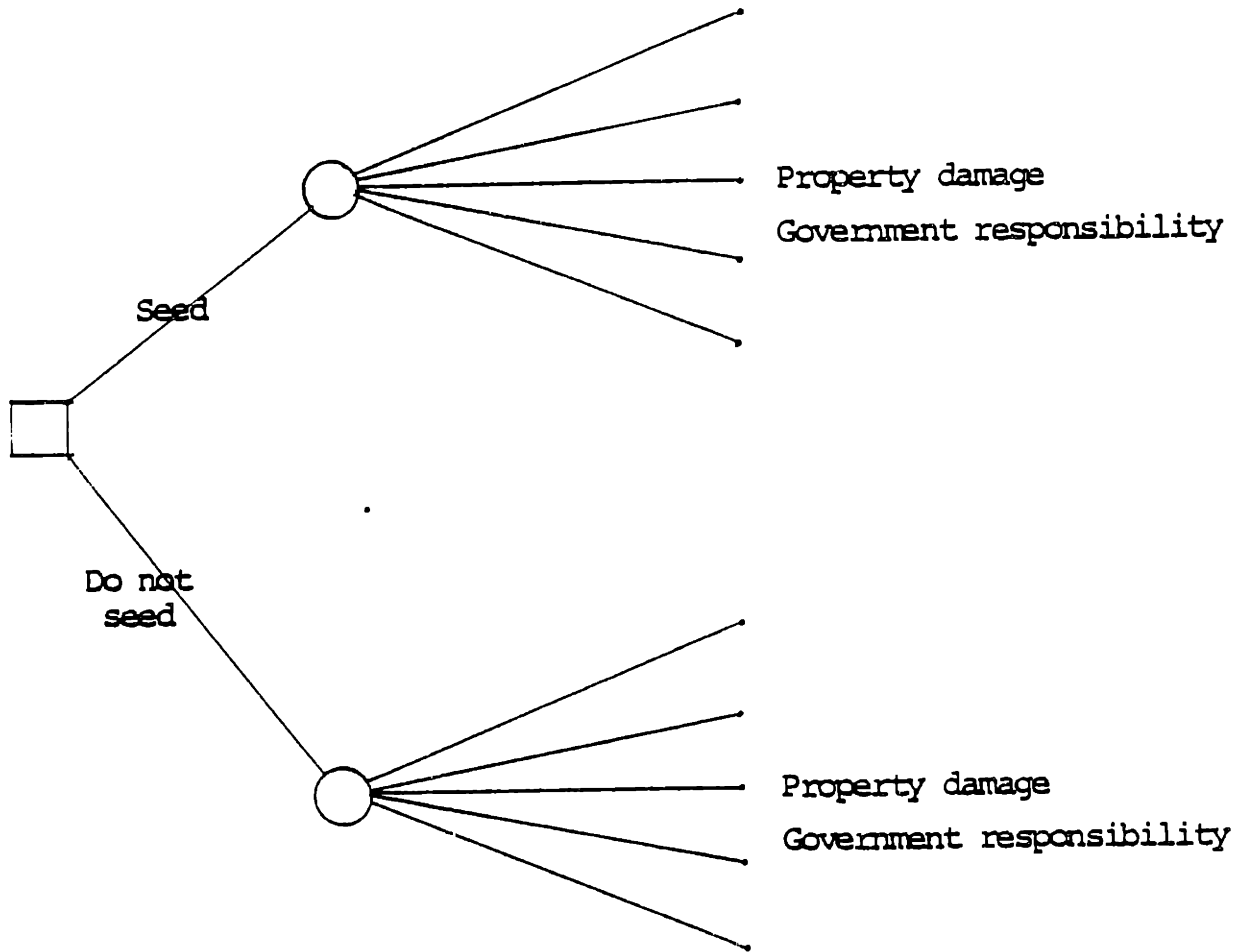
One of the best known applications of these techniques to a real world decision problem is the analysis of the government's policy on hurricane seeding by analysts working at Stanford Research Institute (SRI). Reduced to its essentials, the decision on whether or not to seed depends on the costs of property damage and government liability. The uncertainty revolves around possible changes in the hurricane's wind speed. Figure 10-2 shows the general shape of the decision tree.

Decision alternatives

Resolution of uncertainty

Consequences

(change in maximum sustained surface wind)

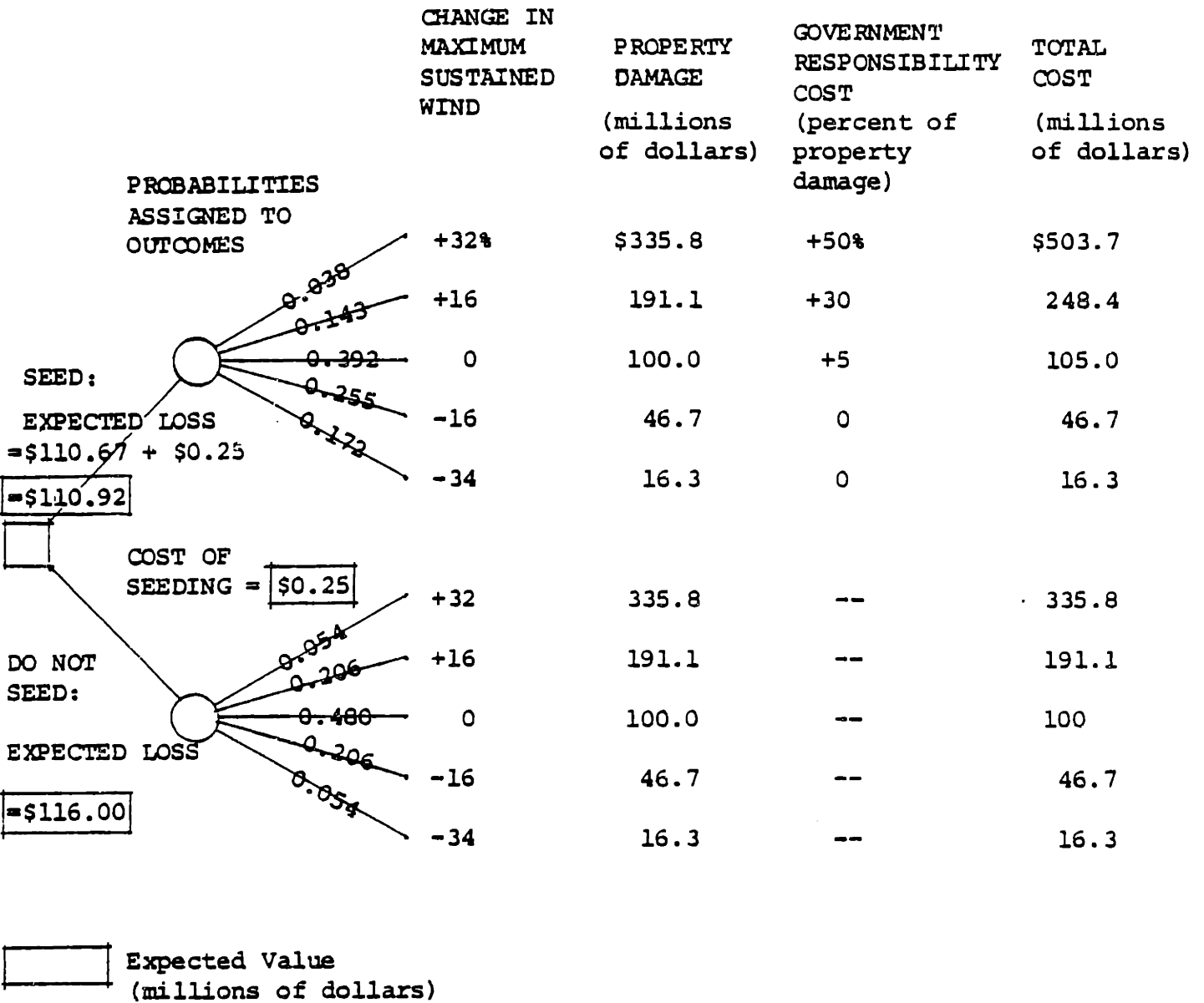


The seeding decision: decision tree.

Figure 10-2 (345)

(345) From R. A. Howard, J. E. Matheson and D. W. North, "The Decision to Seed Hurricanes," Science, 176 (June 16, 1972), p. 1193.

The probabilities of various possible changes in wind speed, with and without seeding, are estimated and for each outcome, the costs and benefits are computed. (In this case, the benefits are measured in terms of costs avoided.) The results are presented in Figure 10-3. According to the calculation given here, the expected loss associated with seeding is over \$110 million, which is less than the expected cost of doing nothing. All other things being equal, one would therefore favor seeding.



The seeding decision for the nominal hurricane (government responsibility cost included).

Figure 10-3 (346)

Type 1 uncertainty is ideally suited to this kind of analysis, for commonly accepted values for the probabilities (P's), and the values of the outcomes (O's) are known or easy to calculate. For higher orders of uncertainty, the technique becomes more difficult to apply, especially for the calculation of the costs and benefits of catastrophic outcomes and the estimation of small probabilities.

Catastrophes as Outcomes

By definition, catastrophes entail huge impacts for they break the continuity of human affairs; these are non-marginal changes. Economic models assume only marginal, or incremental changes, and are therefore inappropriate for the study of discontinuous processes. In the conventional calculus of cost-benefit analysis, goods and services are measured by prices, which reflect their exchange value. But what are things worth after a sudden change? Prices determined in a stable market lose their meaning after the market has been ruined. Whole new pricing systems would evolve after a large-scale disruption. Without the "invisible hand" of the market to guide him, the analyst is unable to predict the nature, and thus the "value," of the outcomes.

The most often cited case is the erosion in the value of the German mark after the runaway inflation of the early 1920's. Barter became the only accepted form of trading. A more recent instance is the sudden drop in real estate prices in Saigon as the American troops withdrew. These examples fail to

capture the economic upheaval of a disaster. How many television sets would it take to "buy" a pound of beef after an escaped organism blighted the grain harvest? Whether the object is to measure the impact of a new plague, a change in climatic conditions, or a widespread contamination by a chemical, our economic models are not up to the task of predicting and measuring the values that would apply after a catastrophic upheaval.

An interesting metaphor for understanding sudden change is the recently developed mathematical "catastrophe theory." In contrast to the calculus, the mathematics of incremental change, catastrophe theory is the study of discontinuous change. Since it was first developed by Rene Thom, the new models of catastrophe theory have been applied to the study of cell division, aggression in dogs, buckling of buildings, and even a stock market collapse. (347) The utility of Thom's theory is the subject of an intense controversy, and I shall not attempt to sort out the various arguments. (348) At the least Thom has highlighted the differences between models needed to study continuous and discontinuous processes.

The large loss of life which inevitably accompanies a

(347) Ian Stewart, "The Seven Elementary Catastrophes," New Scientist, 68 (November 20, 1975), pp. 447-454; and E. C. Zeeman, "Catastrophe Theory," Scientific American, 234 (April 1976), pp. 65-83.

(348) Raphael S. Zahler and Hector J. Sussman, "Claims and Accomplishments of Applied Catastrophe Theory," Nature, 269 (October 27, 1977), pp. 759-763; and replies, ibid., p. 270 (December 1, 1977), pp. 381-384; and (December 22/29, 1977), p. 658.

catastrophe presents huge measurement difficulties for the analyst. Scholars, especially economists, have generated a vast literature on the valuation of human life, and no doubt this will continue. (349) Beyond the intractable issue of setting a dollar value on human life, there is the further complication of evaluating the loss associated with multiple death accidents. Are lives worth more when they are lost individually or in a group? An economist would probably claim that a life is a life and it should make no difference whether one dies alone or with others; the loss is the same. The question is not so simple, however. There is evidence to suggest that the social impact of accidents increases as the number of lives lost per accident goes up; one empirical study suggests that "a single 100-death incident has the social impact of one thousand 10-death incidents." (350) On another front, Richard Zeckhauser of Harvard's Kennedy School of Public Policy has argued that certain types of large accidents may have a smaller impact than one would expect. He writes: "If an explosion wipes out a community of 10,000 individuals, most of the people who would have placed a high value on the lives of those killed will have themselves been

(349) Richard Zeckhauser, "Procedures for Valuing Lives," Public Policy, 23 (Fall 1975), pp. 419-464; Thomas C. Schelling, "The Life You Save May Be Your Own," in S. B. Chase, Jr., ed., Problems in Public Expenditure Analysis, (Washington, D.C.: Brookings Institution, 1968), pp. 127-162; and Charles Fried, An Anatomy of Values, (Cambridge, Mass.: Harvard University Press, 1970), Chapter 12, "The Value of Life."

(350) Joseph Ferreira, Jr. and Louis Slesin, Observations on the Social Impact of Large Accidents, Technical Report No. 122, (Cambridge, Mass.: MIT Operations Research Center, October 1976.) p. 33.

killed. By contrast, if an additional 10,000 people are killed in auto accidents, most of the major externality sufferers will still be alive. Other things equal, concentrating the lives lost on a geographic basis reduces the externality loss per death."

(351) Thus, if a griever dies with the person he mourns, the cost would be less than that associated with the death of two persons whose mourners survive.

This line of reasoning while compelling in its originality is, I believe, incorrect. Large scale accidents are different from all others; disasters take on a life of their own.

(See the fizzle-bang discussion in chapter 6.) The incident is given a name, and is immortalized in the memory of even those not directly affected. (352) The San Francisco earthquake, the sinking of the Titanic, the Johnstown flood, the Blizzard of '88, and the Boston Cocoanut Grove fire are all still with us. They act as constant reminders of the dangers of natural hazards, fires, and travel. In such cases, as well as in Zeckhauser's lost community, we all become mourners, perhaps only because it reminds us of how perilous our own existence is. As Thomas Schelling, a colleague of Zeckhauser, has observed : "The pain associated with the awareness of risk -- with the prospect of death -- is probably commensurate with the costs of death

(351) Zeckhauser, op. cit., p. 444.

(352) This process is much like the namedness parameter discussed in Chapter 6.

itself." (353) We have a distaste for large accidents: no single American disaster in this century has claimed anywhere near 10,000 lives. In fact, the Texas City explosion of 1947 is the only domestic accident since 1940 in which more than 500 people lost their lives at the same time. (354) Not only is the value of individual lives ambiguous, but we cannot even state with confidence that losing ten people all at once is ten times as bad as losing one person!

In contrast to the problems of measuring the costs of a sudden catastrophe are those associated with irreversible losses over long periods of time. Future disasters, such as ozone depletion, carbon dioxide build-up, and nuclear waste accidents present a further set of methodological problems, better known as intergeneration effects. Allocating money in current budgets to safeguard the lives and resources of future generations can be politically unrewarding for government representatives: those who can now vote must be convinced to forgo benefits for others not yet born. Economists use discount rates to compute today's value of tomorrow's costs and benefits. The discount rate is a measure of the degree to which one would rather enjoy something now rather than later. (See the "now-later" risk perception dichotomy in Chapter 6.) The longer a cost or benefit is

(353) Schelling, "The Life You Save May Be Your Own," op. cit., p. 145.

(354) National Safety Council, Accident Facts, annually. On March 27, 1977, two jumbo 747's collided and burned in the Canary Islands killing 577 people in the worst accident in aviation history. One plane was operated by Pan American, the other by KLM.

postponed, the less importance it has in today's calculations. For private investments, the discount rate is reflected by the cost of borrowing money in the open market. The rate for government investments is not so easily settled, especially for those projects designed to promote long-term public benefits. Economists have long been arguing about the optimal social rate of discount. There are two schools of thought: (i) the government should use a discount rate that allows it to justify projects with long term payoffs which would not otherwise be undertaken by the private sector; (355) and (ii) the social discount rate should be the same as the private sector discount rate because "by and large, the future can be left to take care of itself." (356) When it comes to irreversible costs, however, even the free market school subscribes to government intervention: "If we poison our soil so that never again will it be the same, if we destroy the Grand Canyon and turn it into a hydroelectric plant, we give up assets which like Goldsmith's

(355) See for instance: Stephen A. Marglin, "The Social Rate of Discount and the Optimal Rate of Investment," Quarterly Journal of Economics, 77 (February 1963), pp. 95-111. These types of arguments are usually framed in terms of expensive programs like dams and nuclear reactors, whose benefits do not surface until long after the initial investment has been made. If long term social costs are substituted for the benefits, the argument is the same, for a cost avoided is simply a benefit gained. In addition to Baumol's paper cited below, for discussions from both schools, see: Gordon Tullock, "The Social Rate of Discount and the Optimal Rate of Investment: Comment," ibid., 78 (May 1974), pp. 331-336; Mishan, op. cit., and Robert Dorfman, ed., Measuring Benefits of Government Investments (Washington, D.C.: Brookings Institution, 1965).

(356) William J. Baumol, "On the Social Rate of Discount," American Economic Review, 58 (September 1968), p. 801.

bold peasantry, '...their country's pride, when once destroy'd can never be supplied.' All the wealth and resources of future generations will not suffice to restore them." (357) For many PCP's the risks include the peasant as well as his pride! One reviewer concluded this way: "While cost-benefit analysis may be a useful tool when considering projects with only short-run consequences, or marginal adjustments to a predetermined plan, it does not appear to be a satisfactory method for long-run planning when responsibilities towards future generations are taken into account." (358)

Small Probabilities and Uncertainties

The problem of assessing the frequency of rare events is no simpler. Techniques have been developed and applied to familiar phenomena (like changes in wind speed of a hurricane), but when there is little or no experience and knowledge to draw upon, the analyst's assigned probabilities become more speculative. (359) Nor can the number, once estimated, be easily

(357) Ibid. See also: Anthony C. Fisher and John V. Krutilla, "Resource Conservation, Environmental Preservation, and Rate of Discount," Quarterly Journal of Economics, 89 (August 1975), pp. 358-370; Anthony C. Fisher and John V. Krutilla, "Valuing Long Run Ecological Consequences and Irreversibilities," Journal of Environmental Economics and Management, 1 (1974), pp. 96-108 (Resources for the Future Reprint Number 117); and Talbot Page Conservation and Economic Efficiency (Resources for the Future; Baltimore: John Hopkins University Press, 1977).

(358) C. A. Nash, "Future Generations and the Social Rate of Discount," Environment and Planning, 5 (1973), p. 617.

(359) Richard Zeckhauser has also written about quantifying the potential hazards of low-level radiation: "Much of the difficulty arises because neither individual citizens nor policymakers are

tested. If a theory indicates that a certain catastrophe occurs with a frequency of once in a million years, the confirming experiment will hardly be able to validate the claim. PCP's routinely require the quantification of small probabilities. For instance, analysts at SRI offered the following general view of the problem in the context of estimating the probability of contaminating Mars with terrestrial organisms: "The assessment of probabilities on the order of 0.001 or less is at best a difficult task. The problem is that when asked to assess probabilities smaller than, say, 1/100, we all have difficulty conjuring up familiar reference events that we perceive to be of comparable likelihood. In many applications a probability of [0.01] or [0.001] can in fact be used as a working definition of impossibility. It might be argued that scientists are more comfortable than most people in working with numbers as small as [0.001]; however we are not convinced that even they are accustomed to using numbers of this magnitude to summarize their judgement about complex, unlikely events." (360)

Methods have been developed to simplify what seems to be an unmanageable task. In one of these, due to Selvidge, improbable events are divided into discrete, mutually exclusive

well equipped to assess their personal evaluation of low probabilities. Moreover, such probabilities are very difficult to assess on a scientific basis." Zeckhauser, op. cit., p. 444.

(360) Bruce R. Judd, D. Warner North, and Jacques P. Pezier, Assessment of the Probability of Contaminating Mars (Menlo Park, Calif.: Stanford Research Institute, 1974), p. 14. Note that this is very similar to what Kenneth Boulding has written, see Chapter 6.

initiating events and each's possible consequences. The advantage of her scheme is that "sub-events" occur more frequently, and presumably the analyst knows more about them.

(361) Selvidge's method is essentially the same as fault tree analysis, the quantitative technique used in the Rasmussen report (or Reactor Safety Study, RSS) on the likelihood and impact of nuclear accidents. (362)

The probability of a rare event (P) is thus assumed to be the product of the probabilities of more frequent sub-events:

$$P = P(1) \times P(2) \times P(3) \times \dots \times P(i)$$

where P(i) is the probability of one of the sub-events. Of course, the important assumption is that each of the events for which a probability, P(i) is given is independent of the others. The branch of the decision tree in Figure 10-4 shows how the probability of rare event O(i) is computed from the probabilities along its path.

$$P(O(1)) = P(1) \times P(2) \times P(3) \times P(4) \times P(5) \times P(6)$$

(361) Judith Selvidge, "Assigning Probabilities to Rare Events," presented at the Fourth Research Conference on Subjective Probability, Utility, and Decision Making, Rome, September 3-6, 1973.

(362) U. S. Nuclear Regulatory Commission, Reactor Safety Study [WASH - 1400] (Springfield, Va.: National Technical Information Service, October 1975). Appendix 11. Fault Trees. The RSS is commonly referred to as the "Rasmussen Report" after the study director, Norman Rasmussen, of the Nuclear Engineering Department at MIT.

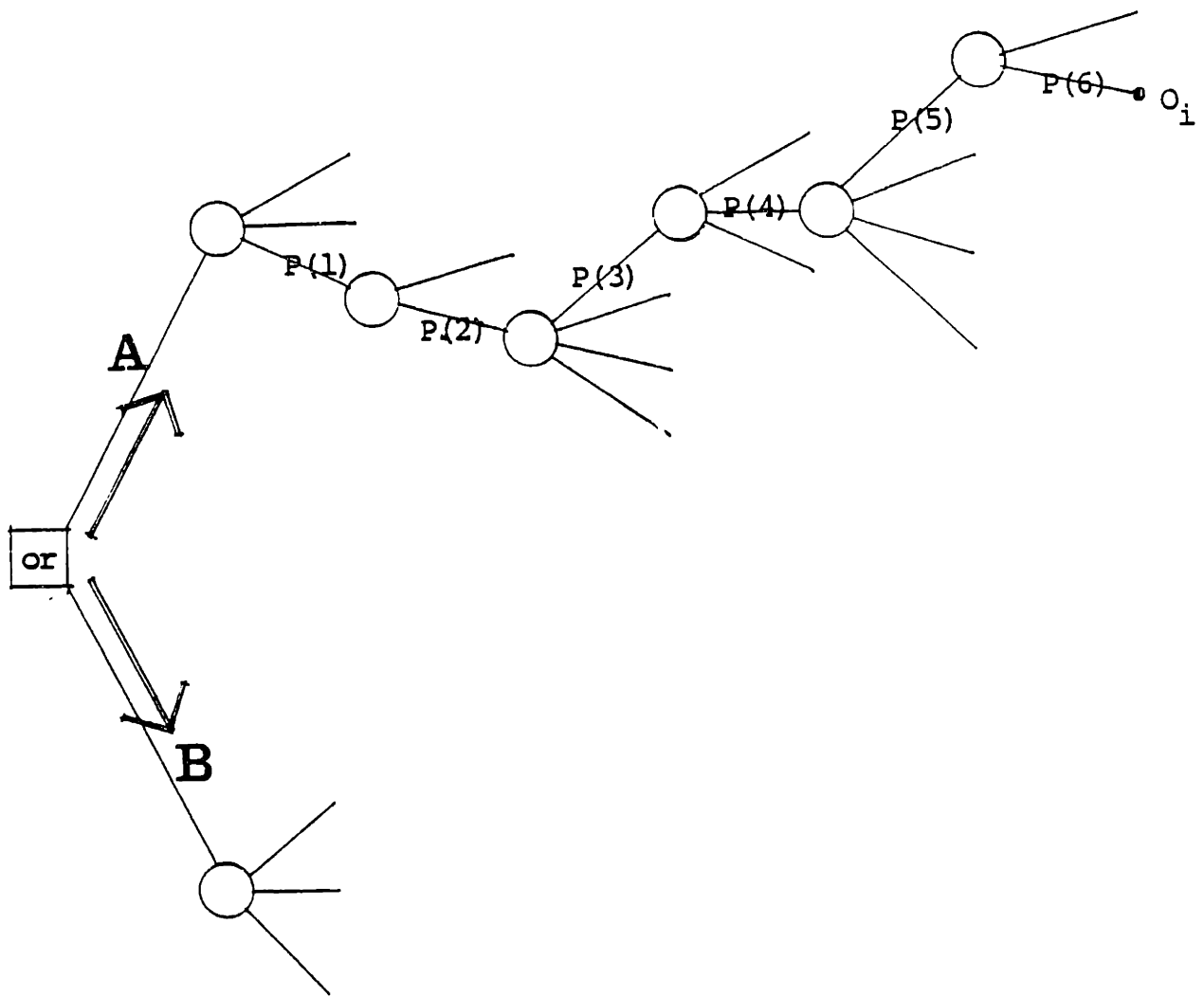


Figure 10-4

In the case of contaminating a planet from a single landing craft, the probability of contamination, P(C), is:

$$P(C) = P(N)P(R)P(G),$$

where P(N) is the probability of one organism in the lander, P(R) is the probability of release from the lander on reaching another planet, and P(G) is the probability of its growth and spreading there. (363)

The same model may be used for estimating the probability that a pathogenic organism will escape from a recombinant DNA laboratory and cause a cancer epidemic. One sequence of events (364) that would lead to such a disaster would be the creation of organisms containing cancer causing genes (P(1)). If so created, the bacteria might be accidentally spilled or swallowed, thereby releasing them from the laboratory (P(2)). If released, the organisms might be capable of surviving and proliferating in the human gut (P(3)), where "the fixed," dangerous genes may be transferred to another natural organism (P(4)). The new bacteria might then infect a new host (P(5)), and express its man-made genes, causing disease (P(6)), be quickly transmitted to other organisms (P(7)), and an epidemic would result. Therefore, the probability of an epidemic is:

(363) Morton Werber, Objectives and Models of Planetary Quarantine Program, (Washington, D.C.: NASA, 1975), p. 46.

(364) This example is adapted from Robin Holliday, "Should Genetic Engineers be Contained?" New Scientist, 73 (February 17, 1977), pp. 399-401.

$$P(\text{epidemic}) = P(1) \times P(2) \times P(3) \times P(4) \times P(5) \times P(6) \times P(7).$$

The most thorough analysis of this type is contained in the Rasmussen Report, on the risks of a nuclear reactor accident. The study cost four million dollars and required seventy man-years of effort; it attempted a rigorous approach to the problem of nuclear safety. The final report, issued in 1975 by the Nuclear Regulatory Commission, is thousands of pages long and covers eight volumes. Its first approximation of the worst case accident is based on a formula similar to the one cited earlier to describe planetary contamination or the escape of a pathogenic recombinant DNA molecule. Here, the compound probability is a product of the "sub-probabilities" (i) that something goes wrong at the plant (an initiating event, P(ie)) such as a loss of coolant in the reactor; (ii) that the safety systems fail (P(sf)), leading, for instance, to a core meltdown and the release of radioactive products; (iii) that the worst weather conditions are in effect (P(w)); and (iv) that the nearby population is distributed in the most vulnerable configuration (P(pop)). Thus, the probability of the worst accident is: (365)

$$P(\text{worst case}) = P(\text{ie}) \times P(\text{sf}) \times P(\text{w}) \times P(\text{pop})$$

(365) Norman C. Rasmussen, "The Safety Study and its Feedback," Bulletin of Atomic Scientists, 31 (September 1975), p. 28. See also RSS, Main Report, p. 183.

There are two ways of using these probability formulae. First, the analyst can estimate each of the probabilities and multiply them together; this is the approach taken in the Rasmussen Report and by Holliday in his much less ambitious study. In the Rasmussen Safety Study, the probability computed for the worst case is approximately one in a billion: (366)

$$P(\text{wc}) = 0.001 \times 0.001 \times 0.1 \times 0.01 = 0.000000001$$

For Holliday, a cancer epidemic will occur as a result of a recombinant DNA experiment once every hundred million years. A second method is to set a desired compound probability and then adjust the variables to limit those probabilities in the equation that are sensitive to human decisions. This was the approach adopted by those in charge of NASA's quarantine program. In the planetary contamination example, $P(G)$ is a property of a planet and should not be changed in the absence of new information. Having set a maximum value for $P(C)$, the probability of contaminating a planet, the analyst may only adjust $P(N)$ or $P(R)$; he can issue orders that there be fewer organisms in the landing craft or that those remaining be less likely to be released on impact. One can now see the importance of the South Pole experiments I discussed in Chapter 8: the smaller the likelihood that earth organisms could survive on Mars, the larger the probabilities of leaving an organism in the lander and of

(366) ibid.

releasing them on impact may be. The savings in the costs of designing and sterilizing the spacecraft could be in the millions of dollars.

The Union of Concerned Scientists (UCS) has been particularly critical of the application of fault tree analysis by the Rasmussen team. The UCS reviewers cite the example of the estimates of the reliability of the Apollo Service Module engine, which they believe to be a "simple system that represented an off-the-shelf technology," as compared to a nuclear power plant. In the case of the Apollo engine, they argue, fault tree analysis underestimated system failures by over 40 to 1 in the final product, and nearly 400 to 1 in the early tests. (367) UCS pinpoints the Rasmussen Study's assumption about the independence of sequential accidents as partially misleading: "For most of the RSS analysis failure of one component is deemed to be independent of failures of other components. To put it another way, RSS predicts random, as opposed to systematic failures. Accident sequences such as those that occur routinely, in which the failure of one component (for example, the explosion of a pump) causes the failure of another component (for example, damage to insulation by missiles generated by the pump explosion)

(367) Union of Concerned Scientists, The Risks of Nuclear Power Reactors A Review of the NRC Reactor Safety Study WASH-1400, (Cambridge, Mass: UCS, August 1977), pp. 11-12. In a report to Senator Gravel of Alaska, the General Accounting Office concluded that the Department of Defense and NASA experience "has limited usefulness in judging the validity of AEC's reliability predictions," and the "[r]eliability of major new systems cannot be accurately predicted because of the many variables -- materials, training, maintenance, and so forth -- that are involved." GAO Report reprinted in RSS, Main Report, p. 192.

are not adequately treated by RSS. The simple facts that components are in proximity to one another, or are interdependent in their functioning, are basic reasons why consideration of dependent failures is essential to satisfactory analysis." (368) The independent Ford Foundation-MITRE analysis of the Rasmussen Report agreed, finding that it "should not be used as a definitive guide for policy since it understates the uncertainties and has serious methodological deficiencies;" (369) and warns that some of the risks "could be low by a factor of as much as 500." (370) The UCS and Ford-Mitre criticisms are not surprising, for, as Zeckhauser has concluded: "The important point to recognize is that probability assessments may be substantially in error when compound events are involved." (371)

The requirement that sub-events be independent applies to all models using compound probabilities. (372) In Holliday's scheme, the necessary assumption is that the organism's infectiousness is independent of its ability to transfer genes,

(368) UCS, op. cit., p. 19.

(369) Nuclear Power Issues and Choices: Report of the Nuclear Energy Policy Study Group, (Cambridge, Mass.: Ballinger, 1977), p. 240. The study was sponsored by the Ford Foundation and administered by the MITRE corporation.

(370) Ibid., p. 241.

(371) Zeckhauser, op. cit., p. 445.

(372) The occurrence of unlikely sequences of events, such as accidents that led to the New York blackout of 1977, has directed interest in "cascading events." One possible hypothesis holds that the design of some of our complex systems may be so enmeshed in the web of natural processes that they are much more vulnerable than we suspect.

and that both are in turn independent of its carcinogenicity. Nevertheless the analyst must not overlook the possibility that the genes which code for one property may simultaneously trigger another. The genes may or may not be independent, we do not know. What is clear is that Holliday has oversimplified the problem.

While the same model has been applied to both nuclear power and genetic engineering risks, there are crucial differences between them: most of the recombinant DNA unknowns are type 3 uncertainties and those associated with reactor accidents (at least those studied by the Rasmussen team) are type 2 uncertainties. The UCS experts may disagree with the NRC experts about the extent of type 2 nuclear risks, but given a commitment of time and money, their arguments can be tested. On the other hand, most of the important questions about gene splicing are still beyond direct experimentation. Similarly, many of the unknowns in assessing planetary quarantine requirements are also for the present unresolvable and inestimable.

Nor are the probabilities for the more frequent sub-events easily assigned. We tend to believe that those trained in decision making techniques are less fallible than the rest of us when working with complex ideas and models. This is an overly optimistic view. Tversky and Kahneman, the two psychologists whose experiments I referred to in chapter 6, have found that the analyst is not immune to the cognitive biases which hinder most individuals: "Although the statistically

sophisticated avoid elementary errors, their intuitive judgments are liable to similar fallacies in more intricate and less transparent problems." (373)

One last example of the application of compound probability models shows how far astray one can go. The method has been used to estimate the number of extant civilizations (N) in the galaxy,

$$N = R(*) \times f(p) \times n(e) \times f(l) \times f(l) \times f(c) \times L,$$

where:

R(*) is the rate of star formation averaged over the lifetime of the Galaxy, in units of number of stars per year;

f(p) is the fraction of stars which have planetary systems;

n(l) is the mean number of planets within such planetary systems which are ecologically suitable for life;

f(l) is the fraction of such planets on which the origin of life actually occurs;

f(l) is the fraction of such planets on which, after the origin of life, intelligence in some form arises

f(c) is the fraction of such planets in which the intelligent beings develop to a communication phase;

(373) Amos Tversky and Daniel Kahneman, "Judgment Under Uncertainty: Heuristics and Biases," Science, 185 (September 27, 1974), p. 1130.

L is the mean lifetime of such technical civilizations.

(374)

Some of the variables may be estimated with current knowledge, others, such as $f(i)$, $f(c)$, and L can only be guessed at. Undeterred, a group of scientists organized a conference devoted to quantifying all these parameters. The objective was a value for N , the number planets populated by civilized beings. One reviewer of the conference proceedings, Alfred Adler, had some harsh words for this whole probabilistic endeavor: it was "a total fraud." (375) Adler's critique of how scientists estimate the inestimable is devastating. He has no need for mathematics to argue his case: "The fundamental issues of a search for extraterrestrial civilizations are, after all, not so arcane or so inaccessible to the non-scientist... Even though there is no a priori certainty that there exist any civilizations at all in the universe beyond Earth, it is reasonable to postulate that other civilizations exist, even that they exist in large numbers, and it does no harm, in any case, to proceed from such assumptions." (376) Undoubtedly, some experts believe the public is more easily convinced by equations than a straightforward

(374) Carl Sagan, ed., Communication with Extraterrestrial Intelligence, (CETI), (Cambridge, Mass.: MIT Press), 1973, p. 5. This formula is credited to Frank Drake from Cornell University. It is widely quoted. See, for instance, Timothy Ferris, "Seeking an End to Cosmic Loneliness," New York Times Magazine, October 23, 1977, p. 99.

(375) Alfred Adler, "Behold the Stars," The Atlantic, October, 1974, p. 109.

(376) Ibid., pp. 110-111.

argument. When mathematics are needlessly used, the public will often be misled for the abstract symbols suggest the experts know more than they do.

Completeness

In 1971, the National Academy of Engineering hosted a conference on benefit-risk decision making. Some of the participants pointed out "that from the public hazard point of view the most important events to be analyzed are those of extremely low probability but of unusual and severe consequences." (377) With reference to the study of nuclear reactor safety, they recommended: "One must be especially careful in such an analysis to include all possible failure modes that could lead to the main event; some would, perhaps, question that enough information is normally available to do so." (378) The multimillion dollar RSS did not meet the NAE's completeness standard. As Frank von Hippel of Princeton University has cautioned, the Rasmussen study omitted many potentially important initiating events, such as earthquakes and fires. (379) The RSS did not even consider sabotage, one of the specific "failure modes" cited by the NAE in need of "more attention." (380)

(377) National Academy of Engineering, Perspectives on Benefit-Risk Decision Making, (Washington, D.C.: NAE, 1972), p. 10.

(378) Ibid.

(379) Frank von Hippel, "Looking Back on the Rasmussen Report," Bulletin of Atomic Scientists, 33 (February, 1977), p. 44.

(380) NAE, op. cit., p. 11.

Indeed, there is a propensity to ignore precisely those kinds of low probability events which may bring the most serious repercussions. In a critique of the analysis of the decision to seed hurricanes, discussed earlier, Robert Kates of Clark University wrote: "There is no mention of the low-probability outcomes, for example, the potentially negative environmental impacts of large-scale injection of silver iodide particles into the atmosphere." (381) As I noted in chapter 2, the consequences of weather modification, both planned and inadvertent, may be huge. PCP risks due to a cloud seeding experiment may now be small, but if initial trials are successful and the techniques are adopted for use on a wide scale, in turn leading to other climate controlling technologies, the PCP risks may no longer be insignificant.

When to include long-term impacts in the decision making process is neither a new nor an easy question. The Court of Appeals of the District of Columbia had to decide precisely such an issue when the Scientists' Institute for Public Information (SIPI) sued to force the Atomic Energy Commission (AEC) to prepare an environmental impact statement on the AEC's entire liquid metal fast breeder reactor (LMFBR) program, not just on a specific breeder demonstration plant. If the pilot plant is successful, SIPI argued, the nation would commit itself to a program which would entail the widespread use of plutonium, an extraordinarily toxic substance, and the critical ingredient

(381) Robert W. Kates, letter, Science 179 (February 23, 1973), p. 744.

of nuclear weapons. The scientists concluded: "The great expense and long lead-times associated with the development of new energy technologies will give the LMFBR development program an irreversible momentum unless environmental hazards and alternatives to LMFBR are considered now." (382) The court found itself pulled in two directions:

"Statements must be written late enough in the development process to contain meaningful information, but they must be written early enough so that whatever information is contained can practically serve as an input into the decision making process." (383)

The Court of Appeals ruled in SIPI's favor, and the AEC was forced to comply. The impact study is at least partially responsible for our present awareness of the need to control plutonium both as an incredibly toxic substance and as an ingredient of nuclear weapons. President Carter has vigorously expressed his opposition to the breeder program. Nevertheless, studies of the breeder program are still woefully incomplete. An analysis for the Joint Economic Committee of Congress found that not one of five major cost-benefit analyses of the LMFBR (including three by the AEC), attempted to quantify the costs of

(382) "Another SST?," Environment, 13 (July/August, 1971), p. 19. [Emphasis SIPI's.]

(383) SIPI v. AEC, 5 ERC 1427, June 12, 1973.

(384) Mark Sharefkin, The Fast Breeder Reactor Decision An Analysis of Limits and the Limits of Analysis, Joint Economic Committee, 94th Congress, 2nd Session, April 19, 1976. Another Congressional study on the costs of nuclear power concluded: "The costs of virually indefinite radioactive waste storage and decomissioning of the nuclear plant remain essentially unknown, and in most cases, have not been factored into the price the

long-lived radioactive wastes. (384)

Human Error

However reliable a PCP technological system may be, one mistake, innocent or otherwise, may bring ruin. The fire at the Brown's Ferry nuclear station was started by a repairman looking for leaks with a candle; the New York blackout was precipitated by pressing the wrong button in the control room; and a large blowout at an offshore oil platform in the North Sea was caused by inexperienced workmen. Often, the chance of human error is greater than the chance of machine failure.

Roy Curtiss, an early advocate of caution with regard to recombinant DNA research, later became one of the leaders of research scientists who wished the work to go on as regulated by the NIH guidelines. Curtiss designed and built the "biologically safe" E. coli bacteria. While his calculations show that he believed the bacteria have only a very small chance of surviving outside of the laboratory, (385) he assumes no errors will be

present day consumer pays for nuclear-generated electricity," House Committee on Government Operations, Nuclear Power Costs, 23rd Report, 95th Congress, 2nd Session, April 26, 1978, p. 3.

(385) Others, including myself, would dispute the accuracy of Curtiss's estimates because the very process of modifying the genes of the bacterium in a recombinant DNA experiment might alter the ability of the bacterium to survive; also the bacterium may undergo further recombination, spreading the potentially toxic genes to "non-disarmed" bugs. As Robert Sinsheimer has countered, "The biological containment proposals are ingenious but suffer from a fatal flaw. They can all be circumvented by a simple recombination event in which the recombinant DNA is a passive participant. And regrettably organisms capable of initiating and carrying out recombination with E. coli abound in man (who will be doing the experiments) and his environs."

made by laboratory workers: "Although these total estimated probabilities for an occurrence of a biohazard are exceedingly small, it should be reiterated that non-compliance with use of standard required practices for physical and biological containment makes these low estimates meaningless." (386)

Human error may come into play not only from physical mistakes; the theoretician or the analyst could be at fault. In chapter 3, I noted that nuclear physicists working on the Manhattan project considered the possibility that they had made an error in the calculations, risking a global holocaust. John Holdren, a professor at Berkeley, looks at the possibility that the decision analyst is in error, with this simplified example: "Suppose an analyst says the probability of a certain outcome is one in a billion per year. If there is a 90 percent chance he is right, and a 10 percent chance he has made a mistake which, if discovered and corrected would make the probability one in a

Letter to Dr. Donald Fredrickson, Director of NIH, February 5, 1976. Furthermore, Sinsheimer has disputed Curtiss's estimates: "Nor am I persuaded that 'biological containment' is adequate. I do not rest easy on Roy Curtiss's 10^{-19} style numbers because I expect other events with which he has not reckoned will come into play at much higher probabilities (i.e. the original cloning culture will be contaminated; cells will lyse in a pharynx and the DNA will be taken up by another organism, etc. etc.)." Letter to Dr. Donald Fredrickson, Director of NIH, February 12, 1976. Both letters are reprinted in Recombinant DNA Research Volume 1: Documents Relating to "NIH Guidelines for Research Involving Recombinant DNA Molecules," February 1975 - June 1976, prepared by the Office of the Director, NIH, (Washington, D.C.: Government Printing Office, August 1976), pp. 436-438 and 443-445.

(386) Roy Curtiss III, "Genetic Manipulation of Microorganisms: Potential Benefits and Biohazards," Annual Review of Microbiology, 30 (1976), p. 527.

hundred thousand per year, then your best estimate of the probability of the outcome becomes:

$$0.90 \times 10^{-9} + 0.10 \times 10^{-5} = 1.0009 \times 10^{-6}$$

about 1,000 times higher than the analyst's figure." (387) Holdren has little faith in the utility of conventional analysis: "Nuclear power systems are so complex that the probability [that] the safety analysis contains serious errors (for example, that it omits failure modes more important than those included) is so big as to render meaningless the tiny computed probability of accident." (388)

Nor may the mistake be only accidental. The risk of sabotage may be larger than that of a technological malfunction. William Fairley illustrates this with an analogy to cheating at a poker table. The chance that one of four poker players holds a natural royal flush is 1/649,740: "Now suppose that cheating was given the probability of 1 in 10,000. What is the probability of a royal flush? It is equal to 1/10,000 plus 1/659,740, which is just a little over 1 in 10,000. The value of 1 in 10,000 is then a minimum value to an realistic estimate of the probability."

(387) John P. Holdren, "The Nuclear Controversy and the Limitations of Decision Making by Experts," Bulletin of Atomic Scientists, 32 (March 1976), p. 21.

(388) Ibid.

(389) William B. Fairley, "Criteria for Evaluating the 'Small' Probability of a Catastrophic Accident From the Marine Transportation of Liquid Natural Gas," in Risk-Benefit Methodology and Application: Some Papers Presented at the

(389) Fairley's conclusion is no different from the NAE's: "Estimates of 'the' probability of an accident must include, explicitly or implicitly, contributions from all of the possible sources of an accident." (390)

The Zero-Infinity Dilemma

Once all the information on probabilities and outcomes has been amassed, it is combined to give a summary measure of the "expected" risk. As I showed earlier, this expected value (or utility) of a policy is calculated by multiplying the probability of each outcome with its net costs or benefits, and summing the products for all the outcomes. For PCP's, the cost is enormous, and the probability of occurrence is correspondingly small. Combining small and large numbers in this way hides how great the potential losses of a decision may be. For instance, the "value" product for an accident which costs ten billion dollars but occurs with a frequency of one in a billion is:

$$-\$10 \times 10^9 \times 10^{-9} = -\$10$$

An expected loss of \$10 derived from a disaster is thus treated no different from a \$10 expected loss from miscalling a coin toss on which \$20 has been bet ($(-\$20) \times 0.5 = -\10). While the point

Engineering Foundation Workshop, September 22-26, 1975, Asilomar, California ed. by David Okrent, Report No. ENG-7598 (UCLA: School of Engineering and Applied Science, December 1975), p. 422.

(390) Ibid., p. 420 (emphasis Fairley's).

of this decision technique is to allow the analyst to choose among otherwise incomparable alternatives, in the case of disastrous risks, it does not provide a useful way of distinguishing between low probability catastrophes and small scale risks (391) Edward Teller has called this the zero-infinity dilemma -- a zero probability and an infinite loss.

The picture that emerges from this review of decision making technology is one of complexity, parallel to the intricacies of the subject matter under analysis. There are severe limits to the benefits of applying the methodology to PCP's. A recent Congressional report investigating the use of risk benefit analysis for environmental and safety policies (such as the advisability of requiring safety equipment in automobiles and controlling the use of potentially carcinogenic pesticides) concluded, on the basis of arguments similar to my own, that: "The limitations of the usefulness of benefit/ cost analysis in the context of health, safety, and environmental regulatory decisionmaking are so severe that they militate against its use altogether... In this regard the Subcommittee emphasizes that when subjective valuation is unavoidable, benefit/cost analysis

(391) If an accident is perceived as sufficiently catastrophic, a risk averse society would not take a chance on it happening. As I have repeatedly noted, however, such low probably events are usually ignored rather than allowed to be used as a reason for cancelling an ambitious program.

is neither neutral nor objective." (392) A similar conclusion was reached by a National Academy of Sciences committee investigating the control of toxic chemicals: "Highly formalized methods of benefit-cost analysis can seldom be used for making decisions about regulating chemicals in the environment." (393)

These criticisms of the utility of quantitative techniques for making decisions are addressed to problems simpler than the ones raised by PCP's. If some analysts are expressing strong reservations about applying them to conventional policies, surely their use for the more complex and uncertain issues so integral to PCP policies is even less advisable. (Nevertheless, it is not easy to recommend alternatives to benefit-cost analysis!)

There are other serious reasons to be wary of decision making technology. First, its complexity increases the public's dependencies by adding a new, and equally inaccessible, tier of analytical experts. Second, the impenetrability of the studies permits, if not encourages, the manipulation of the analysis to fit competing objectives.

(392) Subcommittee on Oversight and Investigations of the House Committee on Interstate and Foreign Commerce, "Use and Misuse of Benefit/Cost Analysis," Federal Regulation and Regulatory Reform, (1976), p. 515. The major themes of this chapter of the report are: (i) estimates may be grossly inaccurate or totally defy measurement; (ii) estimates may reflect the bias of their sponsors, benefit cost analysis being necessarily subjective; and (iii) critical factors may be neglected.

(393) National Research Council, Decision Making for Regulating Chemicals in the Environment, (Washington, D.C.: National Academy of Sciences, 1975), p. 44.

B. Technology and Dependencies

The application of this analytic technology as an integral part of decision making has further removed the process from public scrutiny. I have argued that the complexities of the subject matter have effectively stopped the public from participating in the evaluation of PCP risks -- that Model 1 does not apply to PCP's. Here, I conclude that the tools of decision making also hinder public participation; the technology discourages individual involvement even after a risk has been identified and described by the experts. The public's role in Model 2 is further restricted. As Jacques Ellul has observed: "The way in which [an individual] makes a decision is no longer the domain of the personal and voluntary; it has become the object of the techniques of 'operations research.'" (394)

We have seen that the individual has little choice but to accept the opinion of the expert on the nature of the risks. By using the experts' parameters in complex methodologies, the government and its surrogate, the analyst, accept their

(394) Jacques Ellul, The Technological Society (New York: Vintage, 1964), pp. 128-129.

evaluation: the experts' opinion becomes the social opinion. In Model 2, there is an opportunity for public comment, sometimes leading to dissent, as in the uproar over the banning of saccharin and laetrile. The public refused to accept the combined wisdom of the scientists and analysts; people demanded that they be able to buy both chemicals despite the expert/regulatory assessment. Under the conditions of Model 3, the public is totally excluded, further cementing the symbiotic relationship between the technical and policy experts: the public has no chance of speaking for itself.

If the subject is complex, must not the analysis be similarly complicated? To a degree, the answer is yes. But, however arcane the problem may be, the underlying issues can be presented completely and lucidly. Returning again to the Rasmussen report, we find that the risks were not fairly represented. As Joel Yellin of MIT has pointed out, many of the effects other than immediate deaths are discounted in the report: "In summary, the [RSS] relative risk analysis neither properly incorporates the latent effects -- radiation induced cancers and genetic disease -- which the study group itself predicts will heavily dominate nuclear accident consequences; nor does it treat the numerous, nonnuclear latent health risks with which modern society is deeply concerned." (395) By obscuring even the known hazards, the analysts did not present a document upon which

(395) Joel Yellin, "The Nuclear Regulatory Commission's Reactor Safety Study," Bell Journal of Economics, 7 (Spring 1976), p. 322.

a fair decision can be made.

In addition to downgrading the importance of some risks, the RSS failed to publish sensitivity analyses to support the study conclusions. Yellin asked the NRC to provide him with data on the uncertainty surrounding its estimates of the risks for all accident sequences studied in the RSS. In October, 1976, the NRC replied, "Upon searching through our records, we cannot locate this tabulation. This material had been placed in the public document room with other draft material of interest to the public. When this draft material was later discarded apparently the material you desire was also inadvertently discarded. We regret that we are not unable to furnish you with this material."

(396) Thus, the uncertainty analysis for the most detailed study of a civilian technology ever undertaken was accidentally thrown out. Not only did the NRC not deem it worthwhile to publish the sensitivity analysis in one of the report's eight volumes, the Commission was unable to hold onto it.

A reliance on sophisticated techniques for decision making should bring a responsibility to explain and clarify the details and implications of the analysis. But the Rasmussen Report experience indicates that the government made no effort to involve the public in the policy questions; instead it emphasized propaganda. Rather than indicate the myriad uncertainties of the analysis, ERDA chose to publish a brochure entitled, "How Probable is a Nuclear Plant Accident? 500,000,000,000/1,"

(396) The NRC letter is quoted in a letter from Frank von Hippel, Bulletin of Atomic Scientists, 33 (May 1977), p. 4.

without any qualifying footnotes or caveats. (397)

(397) UCS, op. cit., p. 109.

C. Conflicting Objectives

The way the government translates the experts' estimates of the risks and benefits into public policy depends on what the government is trying to do. (Its usual objectives are to protect the public health and welfare, to provide national security, and even to enhance national prestige -- these are all public goods.) It must balance one goal against another, with the result determined by who is deciding and with which objective. Once again, it becomes easy to ignore the small and uncertain risk in favor of what is perceived to be the public interest.

In promoting public health, the government spends billions of dollars on pure and applied medical research. Government grants and contracts nurtures the growth of many communities of experts studying the intricacies of medicine and biology. The problem is that when the government must evaluate the proposals of these experts, it has no way to distinguish between their shared explicit objectives of improving public health and the experts' implicit objectives of promoting themselves, their work, and their community. As I have already shown, the government sponsors many of the recombinant DNA and influenza scientists, and expects each group to act as impartial judges of their own work. As with the AEC and nuclear power, the NIH is cast in the dual role of supporting and regulating the experts, with the same group of experts making the decisions for

the public.

What can happen to the risk assessment process is illustrated by the AEC's review of the risks of nuclear power. Two years after the the release of the Rasmussen Report new evidence came to light suggesting that the "atmosphere and circumstances under which the Rasmussen study was conducted were anything but conducive to obtaining an impartial study." (398) Documents, obtained by the UCS under the Freedom of Information Act, show that the AEC was as interested in making sure that the study confirmed that nuclear reactors were safe, as in raising questions about them. Professor Rasmussen is himself a nuclear engineer, and a member of the community of nuclear experts. As AEC officials in charge of the project admitted, the agency wanted the study director to be someone who was familiar with both nuclear reactors and accident analysis, and nearly all such people are advocates of nuclear power; and he had to meet with the nuclear industry's approval otherwise utilities and manufacturers would have been unwilling to provide the essential information for the analysis. (399)

Sometimes the government's and the experts' objectives diverge, and the government will abandon their advice. I have already mentioned how the experts convinced the government to sterilize all interplanetary spacecraft in case they contaminated an alien planet with a terrestrial organism. Today after the

(398) Deborah Shapley, "Reactor Safety: Independence of Rasmussen Study Doubted," Science, 197 (July 1, 1977), p. 29.

(399) Ibid., pp. 29-30.

multiple successes of the Apollo program, especially the landing of a man on the moon in 1969, it is easy to forget the initial failures of the U.S. space program. In the early 1960's, the Ranger program to take television pictures of the moon experienced one failure after another. The first two Ranger rockets both flopped, and Ranger III, the first attempt to land on the moon missed it by 23,000 miles. Ranger IV, which took off in April 1962, did reach the moon, but went out of control and crashed on its far side; Ranger V was another miss. NASA still had no close-up pictures of the moon. (400) Up to that time, all the Ranger payloads had been sterilized, but with the mounting anxiety that the Russians would continue their superiority in space, the commitment to planetary quarantine began to waver. President Kennedy had made landing a man on the moon by the end of the decade a national goal, and unless something changed, it would never be achieved. By late 1962, the dry heat sterilization treatments of components for future Ranger vehicles was stopped -- this became the official NASA policy in September 1963. Even though no new experiments were cited to support the now accepted view that "Lunar conditions would mitigate [sic] against reproduction of known terrestrial microorganisms and that, of such sub-surface penetration of viable organisms were to be caused by spacecraft impact, proliferation would remain

(400) Charles R. Phillips, The Planetary Quarantine Program: Origins and Achievements, 1956-1973, (Washington, D.C.: NASA), p. 28.

highly localized." (401) Ranger VI also failed to send back TV pictures, but the next three missions were complete successes.

Of course, in this case the biologists were trying to make sure that the moon would continue to be their laboratory after the first man-made objects reached it, and there was no public risk in the policy reversal on sterilization. However the government was as quick to ignore the chance of catastrophe when the risk of contaminating the earth with lunar organisms conflicted with reaping the publicity of the successful landing on the moon. Earlier I quoted from the NAS report of a conference on backcontaminating the earth; one of its recommendations was that a three-week period of "strict" quarantine be imposed on all returning space missions. (402) The scientists specified that the "spacecraft itself should be received into an isolation environment on board the aircraft carrier immediately upon recovery from the sea." (403) These

(401) Ibid., pp. 30-31.

(402) The conference participants did not place great hope in the efficacy of quarantine. They reported: "The nature of extraterrestrial pathogens is unknown; it is conceivable that there would be no observable symptoms or that disease would not develop for a considerable period. It is impossible, therefore, accurately to predict the type or duration of the quarantine required." They did agree that it was necessary; the three-week period was a compromise with a minority favoring four or five weeks. Space Science Board, Conference on Potential Hazards of Back Contamination from the Planets, (Washington, D.C.: National Academy of Sciences, Revised February 17, 1965), p. 8. A second objective of the quarantine measures is the protection of returned samples from terrestrial contamination before they can be analyzed.

(403) Ibid., p. 10.

recommendations were later codified as official policy by NASA, (404) with a 21-day quarantine period for the first astronauts to walk on the moon. (405)

What happened on the morning of July 24, 1969, as Apollo 11 returned to earth from the moon? Within minutes of the splashdown, a frogman was in the water: the diver opened the hatch of the capsule to the earth's atmosphere and gave the three astronauts biological isolation garments. The hatch was closed while the astronauts put on their safety suits and reopened briefly to allow them to leave the capsule. Once they were aboard an inflated raft, the frogman swabbed down the resealed hatch area to kill any moon germs which might have escaped from the spacecraft. The spacemen were hoisted into a helicopter and flown to the recovery ship, Hornet. On the deck of the carrier they entered the mobile quarantine facility; as they walked along the flight deck, a disinfectant was sprayed behind them. Waiting for them aboard the Hornet were the President of the United States and the Secretary of State. The President welcomed and congratulated the astronauts as they peered through the window of the quarantine unit. (406)

(404) NASA Policy Directives, NPD 8020.13: "Backcontamination and Quarantine Containment Requirements for Manned Lunar Missions," April 14, 1963 and NPD 8020.14: "Extraterrestrial Exposure," July 16, 1969. (Reprinted in Federal Register, 34 (July 16, 1969), pp. 11,974-11,976.)

(405) NASA, "Extraterrestrial Exposure: Establishment of Quarantine Period," Federal Register, 34 (July 25, 1969), p. 12,305.

(406) Neil Armstrong, et al., First on the Moon, (Boston: Little Brown, 1970), pp. 355-362 and text under picture, p. 277.

Why did the hatch have to be opened to give the astronauts their isolation suits? Was the swabbing of the hatch necessary or useful? What was the efficacy of the disinfectant aimed behind the walking astronauts? By comparison with the technology that allowed men to walk on the moon and return safely, such techniques are primitive indeed, as if the quarantine protocols were an afterthought to the most carefully planned adventure in history.

According to Carl Sagan, (407) the Apollo 11 spacecraft was scheduled to be lifted out of the Pacific unopened, but at the last minute, this part of the quarantine procedure was abandoned. The Hornet's crane was discovered to be unsafe and could not be trusted to secure the capsule onto the carrier. Also, the ocean was rough that morning and the mission planners did not want to risk the astronauts' becoming seasick -- preferring to risk making the whole world moonsick. (408)

The official history of the quarantine program admits there was some disapproval of the lax procedures: "The public, as well as the scientists, detected several possible gaps in the quarantine procedure, and protests, particularly from the medical profession, were numerous, but the report continues, "these were blunted by the fact that all the gaps had been foreseen and were

(407) Carl Sagan, The Cosmic Connection, (Garden City, N.Y.: Anchor Doubleday, 1973), p. 161.

(408) The record at the Lunar Receiving Laboratory at the Houston Manned Spacecraft Center was not much better. There were reports that within 24 hours of its first use, air had escaped from the laboratory. Michael Rogers, Biohazard, (New York: Knopf, 1977), p. 124.

authorized by regulatory authorities outside of NASA." (409) What these authorizations were is not specified, but it is clear that the hazards of backcontamination were not going to rob the United States from basking in its technological achievement. Beyond its military importance the race to the moon represented a quest for national prestige, (410) and no improbable organism would douse the limelight.

Sometimes, the government does respond to public pressures concerning Model 2 risks, though the way it does so suggests that public decision makers are simply doing what Charles Lindblom has called "muddling through." Lindblom's thesis is particularly relevant: he believes that the techniques of policy analysis, like those described earlier, are impossible to apply to complex problems, for they assume "intellectual capacities and sources of information that men simply do not possess." (411)

The Delaney Clause requires the Food and Drug

(409) Phillips, op. cit., p. 32.

(410) Jerome Wiesner's "Ad Hoc Committee on Space" 1961 report to incoming President Kennedy listed national prestige as the first among five principal motivations for the space program. John M. Logsdon, The Decision to Go to the Moon, (Chicago: University of Chicago Press, 1976 [1970]), p. 72. Logsdon argues that Kennedy was particularly receptive to a prestige-oriented space program. Ibid., p. 134ff. A concurring view holds that the perceived loss of prestige associated with Yuri Gagarin's flight precipitated the Apollo decision: "It was a commitment to recapture national honor in space." Leonard Mandelbaum, "Apollo: How the United States Decided to Go to the Moon," Science, 163 (February 14, 1969), p. 652.

(411) Charles E. Lindblom, "The Science of 'Muddling Through,'" Public Administration Review, 19 (Spring 1959), p. 80.

Administration (FDA) to ban any food additive that is shown to cause cancer in animals. In 1976, after years of controversy, Alexander Schmidt, the commissioner of FDA, announced that he would ban Red Dye No. 2 under the Delaney Clause. While there had been many suggestive studies that the dye was a cancer hazard, the experiment which served as the final indicator of its carcinogenicity was particularly suspect. As one scientist scoffed: "It was the lousiest experiment I've seen in my life."

(412) Indeed it was not really a new set of results which forced Schmidt to ban red No. 2, but a public outcry against carcinogens. Philip Boffey has written, "Schmidt's decision was a panicky response to outside pressures. Without the barrage of criticism from the Naderites, the G.A.O., Senator Nelson and assorted editorial writers, it's unlikely he would have moved against Red No. 2. The safety evidence, even today, is subject to dispute." (413) The story has a further ironic twist for as the FDA was forcing industry to switch from Red Dye No. 2 to Red Dye No. 40, another color additive (about which even less is known), the Health Protection Branch of the Canadian Government reached exactly the opposite conclusion. Based on the same data, the Canadians refused to sanction the use of No. 40, while

(412) Philip M. Boffey, "Color Additives: Botched Experiment Leads to Banning of Red Dye No. 2," Science, 191 (February 6, 1976), p. 450.

(413) Philip M. Boffey, "Death of a Dye?" New York Times Magazine, February 29, 1976.

allowing No. 2 to remain on the market. (414)

For years the regulation of releases of low level radiation was dominated by a desire to protect the nuclear power industry. Growing out of the atomic blasts in Japan and the atmospheric testing of later nuclear weapons programs, the concern over the hazards of low level radiation re-emerged in the nuclear power debate. At high doses, radiation is known to cause leukemia and other types of cancers, but the risk associated with exposure to low doses of radiation is a point of fierce controversy. John Gofman and Arthur Tamplin argued for years that radiation exposure standards were too lax and that man-made additions to the natural background level should be minimized.

(415) In a review of their much criticized work, Robert Holcomb wrote in Science magazine, "most of the assumptions they use in making predictions can be neither proved nor disproved, but the consensus of their peers is that at least some of their assumptions are wrong. The inability to readily disprove their work on scientific grounds dramatizes the tenuous role that science plays in determination of radiation risk." (416)

These remarks were written in 1970. Since then scientists have progressed little towards solving the riddles of radiation's interreaction with biological systems. Nevertheless,

(414) Philip M. Boffey, "Color Additives: Is Successor to Red Dye No. 2 Any Safer?" Science, 191 (February 27, 1976), p. 832.

(415) John Gofman and Arthur Tamplin, Poisoned Power (Emmanus, Penn.: Rodale, 1971).

(416) Robert W. Holcomb, "Radiation Risks: A Scientific Problem?" Science, 167 (February 6, 1970), p. 853. Emphasis added.

in 1971, a little more than a year after Holcomb's report, the AEC proposed to sharply reduce the public's exposure to radiation from nuclear power plants -- the new standard was only one percent of the then current federal standard! (417) The scientific data base, on which the AEC acted, had not changed -- only its attitude towards it was different. (418)

Thus if there are dissident experts who are willing to speak out, like Gofman and Tamplin on radiation, UCS on nuclear reactors, and Science for the People on recombinant DNA, there is a chance that the government will be forced to reappraise their estimates of the risks. For Model 3 risks, however, there is no such opportunity. When there is no time for public debate, as in the swine flu campaign, or when officials can stamp documents "secret" in the name of national security, there is the danger that government and experts will evolve a symbiotic relationship, which is unaccountable to the public.

Paul Brodeur claims that the dangers of microwave radiation have been covered up by the Department of Defense and the companies that work for it under contract. (419) Russian

(417) Robert Gillette, "Reactor Emissions: AEC Guidelines Move Toward Critics' Position," Science, 172 (June 18, 1971), pp. 1215-1216.

(418) The authority to set radiation standards has since been delegated to the EPA, which has recently promulgated even stronger radiation standards. Federal Register, 42 (January 13, 1977), p. 2858.

(419) Paul Brodeur, The Zapping of America (New York: Norton, 1977); for an analysis of the relationship between the military and the private corporations which are contracted to do much of its work, see H. L. Nieburg, In the Name of Science (Chicago: Quadrangle, revised edition, 1970).

scientists believe that very low power densities of microwaves can cause behavioral and neurological effects -- in fact, the Russian occupational standards is 1000 times stricter than the equivalent American occupational guideline. With so many offensive and defensive missile systems, everything from today's radar tracking stations to tomorrow's "death rays," depend on the unchecked use of microwaves, defense officials have had no reason to encourage research which, if it should substantiate the Russian data, would dramatize the health risks, force the adoption of more stringent emission standards, and make electronic warfare much more expensive.

Conclusion

The reader will no doubt have realized that there are many similarities between the problems of risk analysis discussed here and those of individual risk perception in chapter 6. The analyst's task is to set normative rules to evaluate risks; that is, he must be able to decide when the public is misperceiving a given risk, and then correct for such false impressions. But as I have shown, too often there are no clear rules to indicate what is or is not a distortion in an individual's perception of risk, and therefore there can be no clear rules for correcting them!

The analyst uses discount rates to model the now-later variable and to make sure it does not overly bias societal priorities of what is an acceptable risk. Yet, the analyst is hardly in a better position for he must still set a numerical

discount rate: just as the individual does not know to what degree he is avoiding certain later risks, the analyst does not know which is the preferred rate of social discount. Similarly, in the fizzle-bang dichotomy, the analyst has no technique to compare a life lost in a multiple death accident with one lost individually.

Other examples of economists' nightmares abound: What is the appropriate level of analysis: whose benefits and costs should be considered? How does one balance equity and efficiency (us-them)? How can goods and services not traded in the market (externalities) be evaluated? Both the individual and the analyst often tend to ignore the low probability outcomes and are unable to gauge the consequences of a catastrophe. The analyst has formal models, but lacks ambiguous rules for applying them. We have thus come full circle: the analyst is in a quandry similar to that of the individual when it comes to evaluating PCP risks.

Also, like the individual, the government must defer to the experts for an assessment of the PCP risks, and therefore cannot check them independently. The government aggravates the problem of public confusion of the issues by burying its analysis in quantitative techniques that do more to obscure the hazards than to elucidate them. Like the public and the experts, it is easy for the government to ignore or downplay PCP risks, especially when they conflict with other objectives.

PART III

CONCLUSIONS

Chapter 11

CONCLUSIONS

A typical first reaction to the last ten chapters is to throw up our hands, and say that there is little we can do to protect ourselves from PCP's. After all, they raise a number of very difficult questions which many others have failed to answer:

-- Can the government slow the growth of technology that inevitably leads to PCP's? Should it?

-- Can the public be educated about PCP risks? How?

-- How will the public become sufficiently interested to enter into complex, technical debates?

-- How can the experts better communicate complex ideas to the public?

-- Who will regulate the experts? How?

-- How should social decisions be reached when the experts disagree?

-- What kinds of techniques can be developed to analyze PCP risks?

I do not have answers for these questions -- indeed each of these could be the subject of other dissertations.

Of course, all the participants in the PCP decision process --the public, the experts, and the analysts-- could all try to do better, but I am not sanguine about the chances of improvement. The problem is practically overwhelming, and will not yield to simple solutions. One need only to look at the environmental impact statement and the technology assessment experiences to see how difficult it is to make the government

bureaucracy accountable to a new set of social priorities. (420)

A. What Do We Do About PCP's?

First, we must acknowledge that the catastrophic, uncertain, and complex risks associated with PCP's are different from other kinds of public policies, and as such they must be treated differently. PCP's represent a new type of public policy problem. Whether a decision is about nuclear power, mass immunization drives, space travel, or biological research, the same generic issues of risk are involved.

Second, we must recognize that there is no practical way that we can be free of them. PCP's are part of the world we live in, and in the short run at least, they will not go away. Even if we could eliminate those PCP's we know about, there are others that are still unrecognized. The depletion of stratospheric ozone is a favorite example of a PCP that was recognized as such only after the chemicals had become widely used. Other PCP's are almost certainly lurking in unsuspected places. All present indications are that atmospheric oxygen concentrations are stable (421) -- could we be doing (or will be

(420) The Congressional Office of Technology Assessment does not have an encouraging record. One analysis of its efforts found that its "long-term early-warning" function has largely been ignored; and, with a few notable exceptions, OTA reports have been bland and superficial." Barry Caspar, "The Rhetoric and Reality of Technology Assessment." Bulletin of Atomic Scientists 34 (February 1978), pp. 20-31.

(421) Wallace S. Broecker has concluded: "in its natural state the oxygen content of our atmosphere is exceedingly well buffered and virtually immune to change on a short time scale (that is 100

doing) something that could upset this equilibrium? One such type 4 uncertainty is that the rate of deforestation might lead to a rate of oxygen depletion greater than now thought possible. We must acknowledge that we now live in a world with PCP risks, and we must adjust to them.

If PCP's represent a new type of policy problem and that they are here to stay, can anything be done about them? War, disease, and natural calamities have long dominated human affairs, bringing death and suffering on a global scale; undoubtedly they will continue to do so. It seems that PCP's must be added to that list as an inevitable consequence of modern technology.

Dealing with PCP's is a formidable task or as I have shown:

-- The expert communities know very little about the science that underlies PCP risks. What is known is riddled with time-delayed type 2 as well as type 3 and 4 uncertainties.

-- The public has no experience with evaluating PCP risks, and there is no way to gain such knowledge.

-- We live in a society in which we defer to the experts, and there is no quick way to reverse such dependencies.

-- There are no acceptable normative rules with which we can analyze PCP-type risks.

to 1000 years). "Man's Oxygen Resources," Science, 168 (June 26, 1970), p. 1537. See also, Leigh Van Valen, "The History and Stability of Atmospheric Oxygen," ibid., 171 (February 5, 1971), pp. 439-443, and letters, ibid., 174 (October 15, 1971). pp. 316-317.

So we are in a double bind: none of the principal actors is able to deal with PCP's on the one hand, and on the other, I can prescribe no rules to resolve the PCP predicament. Nonetheless, the very realization that PCP's are new and different adds force to some old advice (too rarely taken), and suggests new avenues for policy analysis to follow. I begin with some general observations, and follow these with some specific recommendations for handling the various types of PCP's.

Diversity of Viewpoints

The government is the main source of research and development funds, and as such responsible for the emergence of many PCP's: without the NIH there would probably be no recombinant DNA research or massive immunization drives, without the AEC, no civilian nuclear power, without the NSF, no earthquake prediction, and without NASA, no trips to the moon or Mars. Indeed without federal assistance, no commercial American SST was developed. It is ironic that the government in trying to promote socially beneficial programs has been so instrumental in the creation of a new and awesome type of risk. Government spending "creates" new interest groups that then advocate the continuation of their work, and therefore the risks. One of the intractable properties of PCP's is that there is a consistent bias towards taking such risks; that is, we accept more PCP risks that we would normally choose. There are insufficient incentives for not taking such risks.

Debates over PCP's become highly technical and too often acrimonious. Opposing factions quickly shun intermediate stands, and adopt extreme positions. Those who begin in the middle ground are soon unable to resist the pressure to join one of the adversary groups. PCP controversies become polarized. Relieving such tensions is contingent on breeding a diversity of interests in the various expert communities. Because of the government's instrumental role in all this, it has the responsibility to fill the now empty spectrum between the antagonistic factions with other voices that offer alternative opinions.

Frank Von Hippel and Joel Primack, two scientists long active in advancing public understanding of technical issues, want to create a corps of "public interest scientists," to provide such alternative views to the cadre of technical experts advising the executive branch of the federal government. (422) The trouble with advocating that people should promote the public interest is that it begs the questions: "Which public?," "Who shall represent it?," and "Who shall watch over those who promote the public interest?" Government policies on technology are no different from other political decisions: all are shaped by the actions of myriad and often confusing pressure groups. All voices must have the opportunity to be heard. When the government creates the interest groups by its funding programs,

(422) Joel Primack and Frank von Hippel, Advice and Dissent: Scientists in the Political Arena (New York: Basic Books, 1974), and von Hippel and Primack, "Public Interest Science," Science, 177 (September 29, 1972), pp. 1166-1171.

it has the obligation to give monies to other sources to ensure that a range of opinions are considered. (423) In fiscal year 1979, the federal government will spend 3.5 billion dollars for basic research alone; (424) the pie is huge and some pieces must be allocated in ways that promote a variety of viewpoints. Certain promising avenues may have to proceed more slowly under such a scheme, but over the long run, the quicker pace may bring costs that are now obscure. I am not suggesting the use of federal grants to support just anyone who applies with a different idea, nor that we give up the rigor of the scientific method. The last thing we need is to complement the communities of experts with astrologers. But for all those projects that are funded many others go begging. For years the government and other expert interests promoted nuclear power at the expense of other energy technologies. (425) Today as the funding priorities

(423) At a NAS Academy Forum, Jonathan King, a biology professor from MIT and a member of Science for the People complained that there was too little public participation at NIH hearings on recombinant DNA research: "I will tell you why there weren't members of the public there because they couldn't afford to go... a few of us called the NIH and said could you bring [some laboratory workers from MIT] down. You have paid for so and so to come in from the West Coast, how about paying for a couple of bottle washers to come down from Cambridge? No, they couldn't do that. So sure, we didn't have much in the way of public represented." Research with Recombinant DNA (Washington D.C.: National Academy of Sciences, 1977), p. 39. NIH money is buying only one group's opinions. The solution may not necessarily be the funding of all those who wish to testify, but NIH has a duty to hear a variety of views.

(424) "Carter Budget Tilts 'Back to Basics' for Research," Science, 199 (February 3, 1978), p. 509.

(425) Others have already concluded that the government should not develop specific technologies, such as the breeder reactor or the SST; they advocate instead a research policy of broad based

are changing, research in solar power is showing that it could be an attractive alternative.

Other examples of how one group of experts can triumph over competing interests at everybody else's expense are cancer research and microwave radiation. Those scientists who believed cancer to be viral virtually monopolized cancer research funds, leaving little for those who favored studying environmental and occupational carcinogens. Only today, hundreds of millions of dollars later are those who would strive for prevention as well as eventual cures receiving greater support! For years, American scientists insisted that microwave radiation was only responsible for heating effects and had none of the neurological, hematological, and behavioral effects described in the East European and Russian literature. The willingness to dismiss an entire body of research (admittedly some of the experiments were not very rigorous) at a time when investment in microwave technology was rising exponentially illustrates how segments of the scientific and defense communities can discourage dissent and diversity of opinion.

A last comment: setting up Arthur Kantrowitz's

support, allowing the market to sort out preferred designs. George Eads and Richard R. Nelson, "Governmental Support of Advanced Civilian Technology: Power Reactors and Supersonic Transport," Public Policy, 19 (Summer 1971), pp. 405-427. One set of the authors' conclusions is worthy of note: "We think this kind of centralization of R and D [that used to promote the breeder and the SST] is pernicious for two basic reasons. First, it is likely to be highly inefficient as well as costly. Second, governmental commitments to particular technologies and products pose an unusually difficult problem of public control." p. 423.

"science court" (426) would be a step in the wrong direction. Such a proposed adversarial system for resolving factual disputes would create a new and unmanageable bureaucracy -- the decentralized peer review and publishing systems are already in place and adequate to the task. The difficulty is not in establishing scientific facts. If my discussion has illustrated anything about PCP's, it is that there are few facts and much uncertainty. We must seek ways of elaborating on what we do not know as well as what we know.

Conflicts of Interest

The foregoing discussion points back to the old story of how the AEC was paralyzed by being simultaneously charged with promoting and regulating nuclear power. However trite the story now seems to be, its lesson has still not been learned: the NIH is assuming the same schizophrenic function with respect to recombinant DNA research. Indeed anytime the subject matter is too arcane for anyone other than those directly involved to understand it, such conflicts are bound to arise. There was no independent source of advice on the risks of the swine flu virus (except for WHO and a few lone researchers who suffered for speaking out): those who were studying the influenza virus were the same people who were saying that the country has no choice but to adopt a full scale immunization drive. It is difficult

(426) Arthur Kantrowitz, "Controlling Technology Democratically," American Scientist, 63 (September-October 1975), pp. 505-509
"The Science Court Experiment: An Interim Report," Science, 193 (August 20, 1976), pp. 653-656.

enough for scientists to be detached in doing their research, (427) no one should expect them to apply the same standards of objectivity when they are asked to judge the importance of their own work or to compare it to that of others.

Uncertainty

The uncertainties endemic to all PCP's can no longer be considered an unfortunate wrinkle to the formulation of efficient public policy; rather they have become the centerpiece of the PCP problem. As Skelly Wright of the Washington, D.C. Court of Appeals put it in his opinion on the hazards of lead in gasoline in the absence of a positive association between lead emissions from automobiles and the risk of lead poisoning in young children:

(427) The list of famous scientists who have been accused of manipulating their data include Mendel, Newton, and Ptolemy. Stephen Jay Gould of Harvard proposes the following view of Mendel's fudging: "I can easily picture the good abbot himself, walking down a row of peas, a bit worried (in the absence of statistical knowledge) because his running tally stands at five tall plants too many, coming on a specimen, obviously tall but slightly below most of the others in stature, and saying to himself, 'this one is not quite clear, so I'll skip it.' The point is this: unconscious finagling is probably the norm. We need not protect the great by fobbing off responsibility to a laboratory assistant. We measure greatness not by 'honesty,' but by insight. After all Newton and Mendel were right." "Morton's Ranking of Races by Cranial Capacity." Science, 200 (May 5, 1978), p. 504. See also: Nicholas Wade, "Scandal in the Heavens: Renowned Astronomer Accused of Fraud," ibid., 198 (November 18, 1977), pp.707-709; and Richard S. Westfall, "Newton and the Fudge Factor," ibid., 179 (February 23, 1973), pp. 751-758.

"Questions involving the environment are particularly prone to uncertainty. Technological man has altered his world in ways never before experienced or anticipated. The health effects of such alterations are often unknown, sometimes unknowable. While a concerned Congress has passed legislation providing for protection of the public health against gross environmental modifications, the regulators entrusted with the enforcement of such laws have not thereby been endowed with a prescience that removes all doubt from their decision-making. Rather, speculation, conflicts in evidence, and theoretical extrapolation typify their every action. How else can they act, given a mandate to protect the public health but only a slight or nonexistent data base upon which to draw?...

Undoubtedly, certainty is the scientific ideal -- to the extent that even science can be certain of its truth. But certainty in the complexities of environmental medicine may be achievable only after the fact, when scientists have the opportunity for leisurely and isolated scrutiny of an entire mechanism. Awaiting certainty will often allow for only reactive, not preventive, regulation. (428)

When one unfortunate outcome is a catastrophe, one is one too many; society has to develop new ways of weighing uncertain facts and theories and establishing who shall bear the burden of proof. (429)

(428) Ethyl Corporation v EPA, 8 ERC 1801-1802, March 19, 1976 (footnotes omitted). The Eighth Circuit of the Court of Appeals had earlier found the scientific and medical evidence on the risks of asbestos fibers in Lake Superior to be "on the frontiers of scientific knowledge." The judge went on:

"concepts of potential harm, whether they be assessed as 'probabilities and consequences' or 'risk and harm,' necessarily must apply in a determination of whether any relief should be given in cases of this kind in which proof with certainty is impossible."

Reserve Mining v U.S., 7 ERC 1635, March 14, 1975. See also: Industrial Union Department, AFL-CIO v Hodgson, 499 F. 2d 467, 474 (D.C. Cir. 1974).

(429) The Environmental Health Act of 1975, introduced by Senator Nelson and the late Senator Hart, shifted the burden of proof to those who wished to engage in a potentially hazardous commercial activity; they would have to show that it did not present an unreasonable risk. The Act was never passed. S. 841, introduced

Research

I have already argued for diversity as a means of dealing with uncertainty. Applied research is needed to reduce the orders of uncertainty associated with PCP's. In time, type 3 uncertainty will turn into type 2, and perhaps even to type 1. Handling type 4 uncertainty is a different kind of problem and underlines the importance of pursuing pure research. It is precisely because one cannot predict where it might lead, -- directly equivalent to not knowing when a type 4 uncertainty might lead to a PCP risk -- that pure research has become even more necessary.

B. Strategies for PCP's

In chapter 2, I divided the class of PCP's into four types. Each of these calls for a different set of policy strategies. Table 11-1 lists some PCP's by type.

February 25, 1975. See also Burdens of Proof in Environmental Litigation, Hearings Before the Subcommittee on Environment of the Senate Committee on Commerce, 93rd Congress, 2nd Session, November 19, 1974.

<u>GLOBAL</u>	<u>PERVASIVE</u>	<u>SELF REPLICATING</u>	<u>NATURAL</u>
Atmospheric Particles	Birth Control Pills	Antibiotic Resistance	Earthquakes
Ozone Depletion	DES	Planetary Quarantine & Backcontamination	Volcanic Eruptions
Oxygen Depletion	Drugs & Chemicals & Food Additives		
Carbon Dioxide Build-Up	Fluoridation	Recombinant DNA Research	
	Immunization Programs		
	LNG		
	Microwaves		
	Nuclear Power		

PCP'S BY TYPE

Table 11-1

1. Global Hazards.

The most important thing to do is to set up international institutions to monitor, and, if and when necessary, to act to prevent worldwide disaster. (430) These PCP's are decentralized threats: all peoples of the world contribute to the global risks, though some do more than others. I cannot imagine a system by which each nation could be assigned a permissible emission level of carbon dioxide or atmospheric particles, nor how such rules would be enforced. But one day such a program may be a necessity. The precedents set by the International Whaling Commission with fishing quotas and the U.N.'s seemingly endless sessions on the Law of the Sea Conference would appear to be the wrong models to follow -- though I admit I cannot suggest anything better to facilitate international haggling.

2. Pervasive Hazards

A policy becomes a PCP when an entire population is at risk: thus, the key to disarming the catastrophic potential is to make the risks less pervasive. Just as we must promote a diversity of opinions about PCP's, we must also encourage a

(430) One of the key strategies of the new Office Of Carbon Dioxide Effects Research and Assessment is "To begin the creation of a national and international focus for the carbon dioxide effects issue." A Comprehensive Plan for Carbon Dioxide Effects Research and Assessment. Part I: The Global Carbon Cycle and Climatic Effects of Increasing Carbon Dioxide, May 1978, Department of Energy.

diversity of technologies so that no single one can endanger the whole society.

For instance, as a drug or chemical becomes more ubiquitous, it should be tested more and more carefully. No uniform rules prescribe how to do a risk-benefit analysis -- in fact, as I noted in the last chapter, a committee of the NAS investigating the regulation of chemicals has recommended against the use of overly formal models. But whenever a drug or chemical enters a new, larger market, a new set of criteria should be applied to judge its safety in use. The Toxic Substances Control Act is a step in this direction: the 1976 law forces manufacturers to seek EPA permission before marketing a new chemical or putting an old chemical to a "significant new use." Unfortunately, EPA has essentially ignored this latter Congressional directive. The government has the regulatory authority to control drug and chemical PCP risks: we must use them, otherwise past experiences with DES, birth control pills, dioxin, asbestos, benzene, mercury, DDT, etc., may be overshadowed by even greater calamities.

Immunization programs provide the clearest example of the risks associated with pervasiveness: the leap from selective to universal immunization is enormous and should be taken only in the face of a known and certain danger. It is one matter to protect a specific at-risk population with a new vaccine: the danger of injecting a contaminant, though present, is overshadowed by the benefits of reduced susceptibility to disease. But it is a completely different matter to seek to

immunize the entire population. The latter policy jeopardizes social diversity, and the downside risks of contamination loom large. Many states now require all children to be immunized against a host of diseases before they can enroll in school. While I do not doubt that vaccines have brought great benefits over the years, this is no reason to ignore the social risks of massive immunization drives. For some diseases, like influenza and polio, selective vaccination can interrupt transmission of the pathogen, and therefore achieve the same goal as universal vaccination. (431) In times of epidemics it may be unrealistic to expect certain people to do without protection for the social good -- how many mothers would have denied their children a polio shot in the 1950's? But at other times, some kind of vaccination by lottery could be implemented; after all the winners (those who do not have to be vaccinated) could then enjoy protection from disease without suffering any of the possible side reactions of a shot.

Hardware technologies, like nuclear power, microwaves, and LNG, entail the same kinds of pervasive risks. While we cannot expect (or perhaps want) to soon give them up, we must learn more about their catastrophic hazards, and at the same time, the government should provide incentives for the development of alternatives. Perhaps these technologies should be taxed to guarantee a minimum investment in safety research --

(431) This is known as the "herd effect" and occurs for polio and influenza, among other diseases. Jonas Salk and Darrell Salk, "Control of Influenza and Poliomyelitis with Killed Virus Vaccines," Science, 195 (March, 1977), p. 835.

0.5 percent of total capital investment? The movement toward appropriate technology should be encouraged for it breeds regional diversity in our tools, ensuring that no single technology will achieve superiority over all others in a given sphere of economic activity, and therefore minimizing the possibility of PCP risks. I do not dispute that the policies I am suggesting would mean, according to some accounting schemes, the loss of economies of scale; nevertheless, we must trade such gains for protection from PCP risks.

3. Self-Replicating Hazards

These risks are qualitatively different because a single self-replicating organism can bring catastrophe -- though it might be more correct to think of the problem in terms of continuous pressure allowing a new disease organism to emerge as in case of antibiotic resistant bacteria. Once a new pathogen is introduced into a terrestrial environment, no one will be able to recapture it; we will be forced to hope that it will not survive and create an ecological niche for itself.

Such hazards, therefore, necessitate the strictest kinds of regulation. The government must not succumb to the pressures of the various interest groups: the drug companies and farmers for antibiotics, the microbiologies for recombinant DNA research, and NASA, defense contractors, and biologists for space research. Antibiotics should be prescribed only when a clear therapeutic value is assured. Recombinant DNA experiments with known pathogenic genes should be carried out in high containment

laboratories, and materials brought back from alien environments should be kept under strict quarantine.

4. Natural Hazards

Much research is underway to improve the prediction of natural disasters, but by all accounts it will be a long time before we can protect large urban populations with reliable early warning systems. In the meantime, the government must encourage greater contingency planning. For instance, the head of the federal Disaster Assistance Administration has reported that there is no plan for dealing with a large scale disaster. (432)

Conclusion

PCP's are new and they are a challenge to all levels of decision making. (433) Whatever reforms are selected, the single most important priority is to bring these risks into public discussion. They are social, collective, and catastrophic risks

(432) Roger Wilkins, "Handling Natural Disasters: U.S. Cities Are Vulnerable," New York Times, July 3, 1978; see also: "Report Calls Disaster Readiness Program Uncoordinated 'Monstrosity,'" ibid., May 9, 1977.

(433) There is a growing interest in PCP's: in late 1977, Senator Schmitt of New Mexico introduced a bill which would establish a National Science Policy Commission to investigate the risks and benefits of many types of scientific inquiry. Schmitt would have the Commission address PCP-type policies in general, including recombinant DNA, backcontamination, and chemical pollutants. Congressional Record, November 1, 1977, p. S18,344. [S.2267] See also Barbara J. Culliton, "Briefing: Schmitt to Seek Commission on All Hazardous Research," Science, 199 (January 27, 1978), p. 406-407.

and there can be no substitute for public debate and decision.

(434) It is too easy to delegate the decision to the experts and government analysts: recent controversies about saccharin and laetrile demonstrate both how little the public understands about what a carcinogen is and what role the FDA plays in regulating drugs and food additives. The public is confused and has lost confidence in the institutions designed to make these kinds of policy decisions.

Perhaps it is too late to ask the average person to weigh the risks and benefits of every PCP, but it is not too late to redistribute the responsibility to a wider public. There is simply too much at stake to do otherwise.

(434) H.L. Nieburg has warned us to be wary of the way the government exploits science and technology, especially in the pursuit of defense and space objectives. His conclusions calling for more citizen participation mirror my own: "The role of science and technology has made the conventional wisdom of democratic politics more pertinent than ever. No elixir of success is to be found in science and technology, or in the skills of any group of experts, which can relieve the citizen of his responsibility to know, to participate, to criticize and if necessary, to demonstrate." The trouble is that individuals are without sufficient organization and financial support to challenge the experts, the corporations, and the government. H.L. Nieburg, In the Name of Science, (Chicago, Illinois: Quadrangle, Revised Edition, 1970), p. 5.