Exposing the myth of the 'danger' of DDT

by Dr. William E. Hazeltine

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To begin with, let me point out that it is impossible to disprove a myth. We can only look at the data that are used to support it and the data that are used to refute it, and see, in balance, whether the concept, the theory, the hypothesis, whatever it is, is consistent with all the data.

I want to talk about two or three examples, and look at some things in detail, regarding the DDT dilemma.

The Brown Pelicans' eggs

The first issue is the Brown Pelican on the coast of California. It was said that these birds were laying eggs that were 40% thin. This was discovered in 1968, shortly after the Santa Barbara oil spill. People went out to the Channel Islands of California and discovered thin eggs. The allegation was made that this thin-shelled condition was due to DDT or a metabolite of it, one of the breakdown products, called DDE.

It was observed that the eggs were thin. There were only a few eggs reported as present in the nests there the first year (1968), and the report was that there were only four young that survived and flew away from the island.

There were various mechanisms that were proposed, as part of the followup research, to allege that DDT was the cause of those thin eggs. Something was said about an enzyme being necessary to call up the calcium from the long bones in the bird. When a bird puts the eggshell on an egg, it's a very rapid process. You have to be able to have a store of calcium to call up, and there were proposals that DDE was blocking a certain enzyme in the parent bird, and that this was the cause of the thin shells. Those were the allegations that we were faced with.

Now let's look and see what the data say. First of all, ducks were one of the models used for bird egg-thinning. Ducks were fed a diet of DDT, at 40 parts per million, for a fairly long period of time, and the maximum thinning that could be achieved was about 15%—not the 40% seen in the pelicans. We also know that the residues in the fish that the pelicans were feeding on in 1969, were a little bit over 3 parts per million. In the following two years, the residues dropped down to about three-quarters of a part per million.

Next, there were some data published which alleged to show that there was a correlation between residues in the eggs, reflecting the residues in the parent bird, and the measurement of the shell thickness. As most of you realize, correlations do not prove cause and effect; they only give you a basis for suspicion, and you need to do some actual experimentation, such as a feeding study or something else, for proof. However, there was one set of data on residues and eggshells of pelicans that was provided by workers for the California Department of Fish and Game, and that data showed a *positive* correlation. As the residues in the eggs went up, the shells got *thicker*. I don't know how many of you are familiar with statistical techniques, but one guiding rule is, if you have a set of data that show an opposite trend, it signals that you do not have a supporting correlation. If shell thickness or thinness is correlated with pesticide residue, it should be consistent. If it is not consistently correlated, there is no cause and effect conclusion which can be drawn.

There were also some publications that showed that the California pelican residue/shell thickness data were not showing any particular trend. One worker tried to combine Florida, the Carolinas, and California—three clumps of data—and then draw a correlation line. As statistical workers will tell you, this will lead you to spurious conclusions. You just can't derive a correlation with that kind of data.

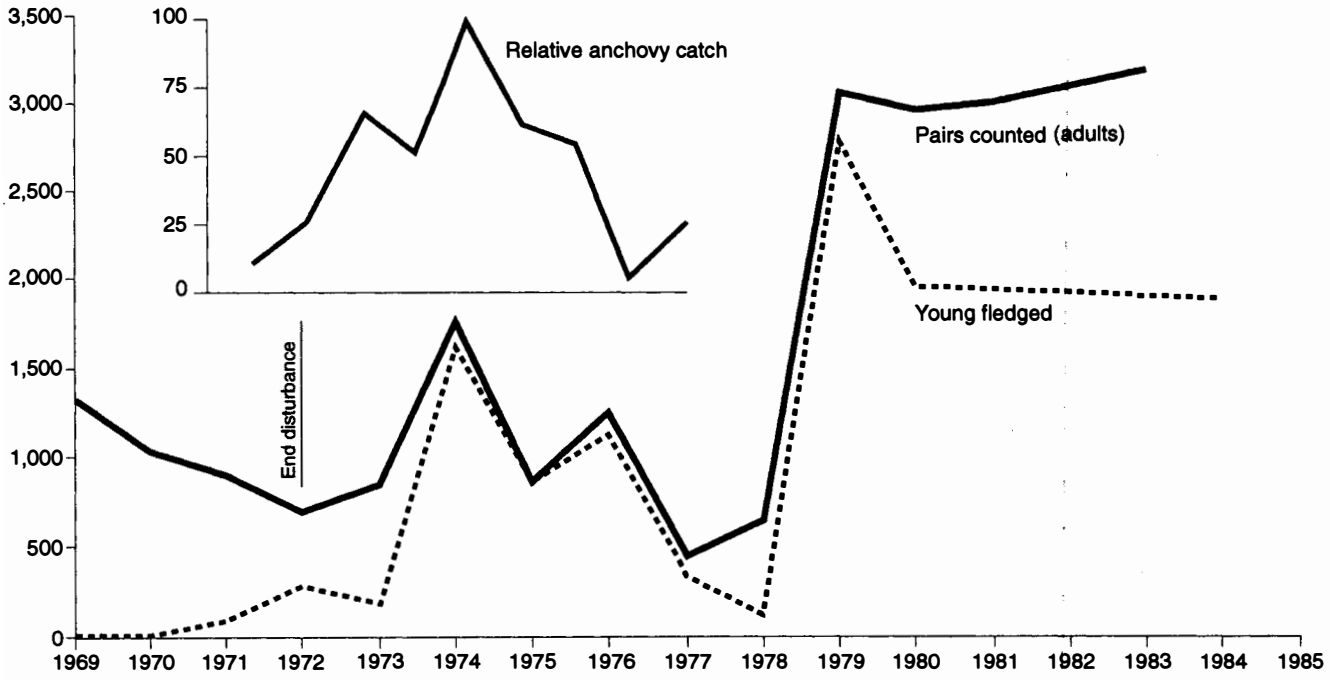
Another interesting sidelight: At the time that the DDT issue arose, it was suggested that the DDT was having adverse effects on birds. A worker at the University of Missouri decided that there were too many pigeons on campus, so in order to reduce the number of pigeons, without having dead birds falling out of the air and exciting the students, he decided that he was going to slip the birds a dose of DDT, and cause diminished reproduction: They just wouldn't produce young, and he could control the population that way. He concluded that the experiment was a miserable failure, because he could *not* control the pigeon population by dosing those birds.

There's another factor that most people do not want to admit. That is that on the Channel Islands there was a disturbance factor. Some of the workers, including Eddie Albert and a few others, who went to the islands after the oil spill, looking for adverse effects of that Santa Barbara oil spill, were the ones that found the thin eggs. The records show that in 1967 there was no nesting, as well as the following year, 1968.

It's also interesting to go back in history. There were records of observations in 1898 and again in 1910. People went to the islands, and the following year they went back. In 1898, there was nesting, but none the following year. In 1910, they observed 500 nests on the island; the following year, there was

FIGURE 1

Brown Pelicans on the Anacapa Islands



no nesting. Obviously in 1910, there was no DDT.

If you look at the disturbance factor, in 1969, after the thin eggs were discovered, there were at least six trips to the island. Scientists collected 54 eggs, which was apparently about 95% of the total egg production on the island, but they only reported data on 23 of those eggs. They shot five of the birds. In Florida, people observed that the pelicans incubate those eggs between their feet, and if you flush the bird, the wings start flapping and the feet contract, and mashed eggs are usually the consequence. In 1970, there were five young produced, and there are records of disturbance through 1972 at least, and maybe through 1973.

Figure 1 shows the numbers of adult birds and young on the islands. Beginning around 1974, there was a very rapid increase in numbers of birds nesting plus the successful fledging of young. Even more telling from this graph, is that there was another crash in that population, after the DDT residues had gone down. That, to me, is one of the most telling bits of evidence to suggest that the idea of eggshell thinning and DDE, as being the cause of the pelican problems, is really unsustained. It really is refuted.

So first of all, correlations of egg residues and shell thickness do not fit, and improper statistics have been used. Residues in female birds do not fit. Workers analyzed the two female birds that were shot in 1969. Interestingly, the higher-residue female had thicker eggs than the lower-residue female.

Now, it turns out that the abundance of anchovies was probably the most important cause of the thin pelican eggs. Workers in the Department of Interior showed that you can account for approximately 80% of the variation in productivity in pelicans, by the abundance of anchovies. If birds don't get their proper nutrition, you're not going to get egg production. So looking at the anchovies, and accepting this as probably the most likely cause for the problems, and recognizing that the data on anchovies were produced by some of the same people who testified at the public hearing that DDT was supposed to have adverse effects, it seems to me that we have some real problems of credibility. And the issue before us today really is credibility.

DDT residues in soil and water

Take a look at a different topic now, the issue of ocean residues and soil residues. The commonly held idea is that DDT lasts forever, or an exceedingly long time; this is very popularly believed.

There are some data produced by the Department of Interior. One of the people who was a witness at the public hearing on DDT, was in charge of the laboratory where this work was done. Without going into a lot of details, let me tell you that the experimentation was to put DDT in sea water, seal it, periodically take samples, and look at not only the DDT, but also the degradation products. The experiment showed that in about a 38-40 day period, in sea water, there

was about a 90% loss of DDT. That pretty well undoes this idea that the residues last forever.

There are two examples of soil residue loss to consider, one in forest soils, and one in the marshes of Long Island. It turns out that some of these data were deliberately biased, according to one of the witnesses under oath. They were looking for the *highest* residues that they could find. One of these workers was singled out by the hearing officer at the public hearing on DDT as an incredible inaction, where workers found out that their data were faulty, and they never bothered to correct them.

A worker tried to duplicate the findings on forest soil, so he went into the forests of New Brunswick, but could not duplicate the residue level that had been published, until the workers told him that the man took his samples on the air strip, and these were alleged to be the forest soil samples. Well obviously, they had calibrated the aircraft there, they had loaded and mixed the materials there, and sure enough, he looked at the airport soil and found high levels.

These are the problems that we get into if the data are not looked at clearly and objectively, and evaluated in detail.

Effects of DDT on humans

I want to talk about some human effects, because this is the area of greatest concern. What about us, or people, in relation to DDT?

First of all, it is important to look at the basic issue, that most of the expected adverse effects on people are extrapolated from test animals, mice, rats, dogs, whatever. It is important to consider this, particularly with the issue of cancer, since this is the biggest issue that will cause concern on the part of people.

Let me point out to you that with DDT, mice are not a predictor for human impact, and they do not meet the protocol of the cancer research people. Mice and rats have different physiologies. The control of mice, in the old days, used to be with DDT. Control of rabid bats, even today, is with DDT. Mice do not have a capacity to degrade DDT in the liver. That's where the major degradation begins.

I asked one of the researchers at the public hearing, "How can you continue to cite mouse cancer data, when it violates your protocol? The protocol says that you do not use a test animal that has a physiology different from humans, if you're going to try and extrapolate that information to predict problems in humans." Rats have a physiology similar to humans, and so do dogs. But with mice and DDT, you can't "turn on" their liver enzyme, and liver enzymes are the garbage disposal system, or the foreign chemical disposal system. Therefore, the issue of looking at mice data, as compared to rat data, is an important issue to consider when looking at DDT and cancer risk.

It is interesting to look at a very nice study that was done with rats, on rates of DDT degradation. In this experiment, a worker used excised rat tissue. It was a very well-done study. The study used not only DDT, but all of its breakdown products, to treat rat tissue, and to do analysis of residues over time. What was found out, is that the first two-thirds of the metabolism is in the liver, and the last third in the kidneys. The experiment also provided some information so that we can extrapolate the speed of degradation. If rats were used, without preconditioning, they found that over a 24-hour period there was about a 15% metabolism excretion rate. So the next day you would expect to find only 85% of that original dose. If those animals were pre-dosed, or preconditioned, by giving small doses of DDT ahead of time, then the rate of loss was something like 25%, and this was within a 12-hour period. They found DDA, and other breakdown products, showing that metabolism had occurred to cause this loss.

In another study, a 17-year-old had jaundice, and they had been treating him with barbiturates. This was in England, where they have a greater tendency to experiment. They then treated that teenager with DDT, at 90 mg a day for six months. They were able to build up a liver residue of something like 200 parts per million at the end of the experimental period. But they controlled his juvenile jaundice, and the physicians who examined him said that he had better liver clearance than if they had been using the barbiturates as a therapy.

It's also interesting to consider the human impact of DDT at very low levels. We're told that this "might have some adverse effect." But it equally well might have a beneficial effect, such as with juvenile jaundice, if the unborn infant is exposed to very low levels of materials that can turn on their liver. Researchers say the mother causes the same liver response with alcohol as well as with DDT, but with alcohol they recommend not drinking too much, because "you don't want to get the fetus drunk," and you don't want to start some addiction problems later on.

On the issue of cancer, it would seem to me that it is important that we very clearly do *not* accept or continue to accept the "DDT—what ifs," nor allow them to continue. If we allow possibilities to set policy, we will find that we will continue to lose very beneficial tools, such as pesticides, for agriculture and public health.

Let me tell you an experience. At the time I was employed, I asked for the use of about 50 pounds of DDT, to treat a particular mosquito problem. The request was denied, on the basis that this mosquito was only associated with dog heartworms; we could not produce data to show that it was connected with encephalitis or one of the other viral diseases of people. As a consequence, the use was denied. This to me makes no sense.

It leads me to the conclusion that the decisions on DDT were largely political rather than scientific. If any of you want to discuss this afterward, or want to look at the mass of information that is available to refute the hypotheses that DDT causes all of the alleged effects, I would be happy to discuss it with you or to show you the published studies.