PESTICIDE RESIDUES – A MEDICAL APPRAISAL

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New analytical procedures make it possible to detect pesticide residues in food at a fraction of the level previously detectable. The possibility exists that products formerly thought to have no residues will now be shown to have them. Fear has been voiced that pesticide residues may cause disease. If pesticide residues in food are shown to be more widespread than formerly believed, this fear of danger may be further stimulated. Investigations among the human population have failed to reveal any deleterious effects from pesticide residues in food. Nor is there any other positive evidence of effect on the human population resulting from pesticide residues. Analysis of mortality statistics tends to show many more likely reasons than the introduction of pesticides for changes in causes of death. There is no reliable evidence that the leading causes of death have been influenced by pesticide exposure in food or otherwise. Despite this absence of positive information there is no doubt that we need quantitative investigations to determine the actual exposure of the population to pesticide residues and long term, carefully controlled clinical investigations to determine whether or not injury actually occurs.

There has been developed recently a new analytical method which may well revolutionize the analysis of organic chemical residues in food (1). This new method, gas liquid chromatography with electron capture enables the analyst to detect dieldrin, for example, at a level of 0.0001 ppm (2). What is more he can make this analysis in one hour instead of the three days previously required. The introduction of such a powerful analytical tool reduces the concept of zero to an absurdity. What is a practical zero? What is the meaning of zero?

This question is asked because the current concept of protection of the public health requires that there shall be zero quantity of certain pesticides in our food. This requirement has been particularly vexing, as is well known, in connection with the production of milk but has also been a problem in meat production and has become a problem wherever cultivated crops requiring pesticide application are grown in close relationship to pasturage. What is the basis for the zero tolerance of pesticides in milk and the generally low tolerance limits in other foods?

As a general rule we wish to limit to the lowest amount possible the man-made chemicals in our daily diet. This rule is followed on the general premise that man has been able to adapt to natural stresses over the millennia but that he may not have the ability to adapt to the stresses imposed by unusual quantities of naturally occurring inorganic materials or man-made organic materials. The validity of this generalization has never been tested on a population-wide basis but there has been sufficient confirmation of the general premise to accept its essential validity.

THE ZERO TOLERANCE

As our analytical techniques become more acute the earlier zero becomes a finite number. The fact that zero today is a positive number tomorrow, requires that we fully understand that nothing has changed except our ability to measure. Measurement that is impossible below a 0.1 ppm today, and is therefore labeled zero, may become possible tomorrow and becomes 0.001 ppm instead of zero. The actual quantity of pesticide that is present remains the same.

PESTICIDE CONCENTRATION IN THE HUMAN POPULATION

The most widely used pesticide is DDT. It has been used throughout the world for more than 15 years. There is no doubt that DDT is present as a residue on many crops and has been present as a residue for many years. The concentration of DDT in human fat has been shown to range from one to ten parts per million in the investigation reported by the Public Health Service (3). At this concentration no effect has ever been detected.

No pesticides are legally allowed in milk sold for human consumption. However, Lange (4) found 0.1 to 0.15 ppm of DDT in human milk without any known effect on the nursing child. Thus we have a situation in which the child nursing at his mother’s breast may receive more DDT than the child raised on cow’s milk.

Accusations are heard frequently that quantities of pesticides, such as described above, may result in harmful effects. Is there any evidence that the pesticide residues found in the American dietary has resulted in harm or may result in harm to the population?

ANSWERS TO THE QUESTION

There are a number of ways in which we can look for an answer to our question. Each has been tried and found wanting in some respect but, neverthe-
Animal Experimentation

Before any pesticide is introduced into use it must undergo extensive testing on animals. Feeding experiments lasting as long as two years are required with many compounds. Though two years is a short period in the life span of a man, it is the major part of a lifetime of a rat and a very significant portion of the life span of the dog. Deleterious effects resulting from such feeding experiments rule out the material as a commercial possibility. Lack of effect in animals is no guarantee of lack of effect in humans but it is an indication; it is a direction signal.

Observation of Human Subjects

Human beings cannot be controlled in the same fashion in which we control experimental animals with the result that human observation is difficult, time-consuming, and costly. Despite these handicaps several series of observations have been made on human populations and their exposure to pesticides.

A large number of persons exposed to lead arsenate either as consumers of fruit or workers in the orchards of Wenatchee, Washington, were examined by a team of Public Health Service personnel. No evidence was found that ill health was any more prevalent among the group exposed to the lead arsenate than among a similar group not so exposed. Nor was there evidence that any chronic disease had been caused or influenced by the exposure to the lead arsenate (5). There is underway at the present time an attempt to trace all of the people involved in that investigation of 25 years ago in order to gain additional information but such a job is obviously rather difficult.

Ortelee, reporting in 1958, found no correlation between prolonged, intensive occupational exposure to DDT and the frequency or distribution of clinical abnormalities (6). Hayes fed a diet containing a known quantity of DDT to a group of human volunteers and was unable to find any evidence of disease caused by the exposure to the pesticide despite the fact that the quantities of DDT were well in excess of that which might be found as a food residue (7).

Obviously, experiments of this type have not been used to screen all pesticides which might be found as residues in food. Those which have been done have produced uniformly negative results.

Studies of Population Groups

The statistical evaluation of reported illness and death offers yet another possible method of finding an answer to our original question. When large groups of the population are exposed to a new factor in the environment this method of approach is often best for obtaining an indication of possible ill effects. Detailed clinical studies can then attempt to confirm or refute the suggestion obtained by analysis of vital statistics.

Investigation of the effect of pesticide residues by this method is tremendously complicated by the simultaneous introduction into our environment of innumerable other chemical and physical agents. The problem is further complicated by the failure of official agencies to require the reporting of most illnesses and the changes in the official listing of causes of death during this Century. If we could show a significant difference in causes of death between 1900 and 1956, a difference which has some conceivable relationship to a change in the environment, we might then be able to design investigations to prove or disprove the validity of our assumption. Unfortunately, the difficulties in the way of such a comparison appear insurmountable.

Statistics Evaluated

Admitting that we are unable to make a valid comparison, let us look at the 1956 statistics and attempt to evaluate some of the changes in cause of death that have occurred over the years.

The ten leading causes of death of white males in the United States in the year 1956 and the number per 100,000 population are as follows:

1. Diseases of the heart
2. Malignancy
3. Vascular lesions of central nervous system
4. Accidents
5. Certain diseases of early infancy
6. Influenza and pneumonia
7. General arteriosclerosis
8. Suicide
9. Cirrhosis of liver
10. Congenital malformations

Note that the first three categories are for types of disease usually, but not exclusively, associated with aging. The increase in life expectancy has been accompanied by an increase in the degenerative diseases to a position as a leading cause of death.

It is impossible to single out any simple reason for an increase in a particular cause of death. Probably the most valid reasons for the shift that has occurred in this Century is the greater age at time of death and the decrease in infectious disease. Simultaneously accidental death has become a serious problem. Though accidents are now the fourth greatest cause of death in the United States it is probable that in actual fact deaths per 100,000 of population due to this cause have actually decreased. We are unable to muster any reliable evidence that the leading causes of death have been influenced by pesticide
Pesticide Residues

exposure in food or otherwise. For every shift in position of a particular cause of death there are a multitude of explanations most of them far more reasonable than the possible exposure to trace quantities of pesticides.

Malignancy is now the number two cause of death. If an item of diet were the cause of an actual increase in malignancy we would expect the increment to be divided equally between the sexes. We are aware that smoking habits differ between men and women but there is no obvious difference in types of food intake. Changes in the mortality from different types of malignancy have not been divided equally between the sexes. In some instances in recent years cancer mortality rates have decreased but by-and-large any changes in mortality rates are attributable to improved diagnosis and treatment. One exception to the previous statement should be noted. It is difficult to get comparable figures but it appears reasonably certain that there has been an absolute increase in leukemia since the turn of the century. At present there is considerable feeling that this increase is partially due to the increase in radiation exposure of much of the population. We have no evidence that other environmental factors have the same leukemogenic potentiality as ionizing radiation.

Illness is not reportable except for a number of specific diseases. The data available is insufficient to even attempt to correlate morbidity with changes in the chemicals available in the diet.

CAUSE AND EFFECT

Though each of the methods of approach which have been described has been found wanting in some respect, in the aggregate they offer no positive evidence that pesticide residues have caused any deleterious effect on the health of our population. To conclude that pesticide residues have resulted in disease, as has been frequently stated, calls for more evidence than presently exists.

Before we can design an experiment which will attempt to answer the question there are a number of preliminary questions which must first be answered.
1. How much pesticide residue is there in food?
2. What chemical form do these residues take?
3. How much of the residue is present after food preparation?
4. How much of the residue is ingested?
5. How much of the residue is excreted unmetabolized without effect?
6. How much of the residue remains in the human body?

The answer to question one is known. As we proceed down the list our knowledge becomes more and more fragmentary, yet if we wish to determine whether or not pesticide residues cause harm to people we must have some idea of how much they actually ingest, how much they retain, and what happens to the material in the body. Too much of the discussion of the effects of pesticides residues has proceeded on the assumption that the amount found on the farm or in the meat market or produce dealers shelf is the amount actually ingested. Food preparation may markedly alter the amount of residue. Habits of food intake may also affect the intake of pesticide residues. Pesticides which are primarily deposited in the fat may be ingested by the person who eats the fat on meat but would barely be touched by the person who trims away all visible fat.

In order to develop good clinical data we should set up two population groups. One group would consist of people with an aversion for fat and a second group would consist of people who eat the fat. We would have to insure that most other factors were identical and we would want analyses of duplicate samples of all food and drink in order to know exactly what the exposure has been. Of course we would have to analyze all excreta in order to gain an idea of how much pesticide has remained in the body. Following these two population groups for a period of at least 20 years might give us conclusive data. Assuming that new information during that interval didn’t invalidate our assumptions and methodology, we might by diligent clinical and laboratory examination determine whether or not pesticide residues had an effect. This approach is much more difficult than ex cathedra statements without a basis in controlled observation or experiment. Yet, we cannot be stumped into ill considered action by baseless hysterical statements.

In 1958 the Food and Drug Administration, after consultation with an advisory committee, ruled that no tolerance would be allowed for methoxychlor in milk and has since followed the rule that no tolerances would be allowed for pesticides in milk. The basis for this ruling was the unique place milk supposedly holds in the diet of certain groups of our population. In the present diet of the United States it might be questioned how unique a place milk actually occupies. It is questionable whether any group of the population, child or adult, now has an exclusive diet of milk for any prolonged period of time. Recognizing this, a more recent advisory group has reversed the earlier recommendation and has recommended that tolerances for pesticides in milk can be safely established.

In the light of all available evidence we must make judgements regarding our environment. If we are swayed by hysteria and the desire to return to the “good old days” of tetanus, diphtheria, wormy
apples, summer diarrhea, and swarms of flies in every household, we can take the approach that we do not wish to have pesticides in our environment and we do not want any pesticide residues in our food. If we wish to have the comforts, conveniences, and good health associated with today’s standard of living we may have to accept some residue of pesticides in our food. We can, however, reassure ourselves with the knowledge that there is at present no evidence that such residues have caused or will cause any deleterious effects on the human population.

Conclusions

1. No deleterious effect has been shown to result from pesticide residues in our food.

2. It should be possible to establish safe residue limits for all foods.

3. Quantitative investigations to determine the actual exposure of the human organism to pesticide residues would be highly desirable.

REFERENCES


NOTICE

Program and Reservation Cards

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