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Michael Gough, PhD
Office of Technology Assessment
Congress of the United States
Washington, DC 20510

before the
Subcommittee on Investigations and Oversight
Committee on Science and Technology
United States House of Representatives
April 23, 1985

I am Michael Gough, and I am employed in the Office of Technology Assessment (OTA). Since December of 1979, my colleague Hellen Gelband and I have been responsible for OTA's Congressionally mandated (Public Law 96-151) oversight activities regarding Agent Orange. We have been observers on the White House's Cabinet Council Agent Orange Working Group since 1980 and have followed developments in the efforts to understand the effects of dioxin on human health.

In years past, some chemical industry workers were exposed to dioxin during the manufacture of herbicides and bactericidal products. In addition, widespread spraying of dioxin-contaminated herbicides exposed the workers who sprayed them and anyone who entered sprayed areas or lived near them. Now that the herbicides are no longer manufactured, those routes of exposure have been closed, but others remain. Residuals of former manufacture, chemical plant wastes, deposited in dumps and mixed wtih oils and sprayed in horse arenas and along roadways in several Missouri towns and other places are sources of exposures now and, perhaps, for years to come. Moreover, as unavoidable synthesis of dioxin as a byproduct of chemical manufacture declined and essentially ceased, we became aware that it is also produced by burning of certain kinds of municipal wastes.

I will use the term "industrial exposure" to refer to worker exposure and "environmental exposure" to refer to exposure of residents near waste disposal sites. Clearly, many people have been exposed to dioxin and, probably, many more will be. Exposure must be accepted as a given; it has happened. The political, legal, scientific, and public health debates are about whether the exposures have caused or are likely to cause adverse health effects. Little information is available about environmental exposures, which typically are to low dioxin concentrations over very long periods of time. More data are available about industrial exposures, often marked by exposures to high concentrations over short periods of time. In both environmental and industrial situations, we are largely unsure of exposure levels.

Interpretation of the data is further complicated by the absence of consistent findings. With the exception of the skin disease chloracne, which has been found in many highly exposed populations, excesses of other diseases have not been consistently found. That means that studies of dioxin-exposed human populations remain "fishing expeditions," with efforts made to look for any and all diseases; we cannot yet focus on a disease, except chloracne, that is a "dioxin disease." It is important to remember, however, that animal studies, which first alerted us to dioxin's potential for causing birth defects and cancer, provide many suggestions of possible associations between dioxin and disease. But the applicability of those data to making estimates of human risk is not always obvious. For example, it is difficult to know what implications a rat study has for humans when the rat results disagree with those from a similar study done in mice.

Thick books have already been published about possible effects of dioxin on human health, and anyone talking about the subject must pick and choose from the abundant available information. I will focus on studies that

have examined the effects of exposure to relatively high levels of dioxin. Those studies are important because there is no doubt that people were exposed and, in many cases, sufficient time has elapsed since the exposure to allow for the development of any dioxin-related diseases. Taken altogether, I think those studies strongly suggest that environmental exposures to dioxin are not likely to cause dire effects in humans.

One well-studied industrial population was exposed to dioxin on March 8, 1949, at a Monsanto plant in Nitro, West Virginia. Excess pressure blew open a safety valve and burst pipes, and a black powder and dark brown tarry substance spewed out. We know that dioxin spewed out too. Unfortunately no attempt was made to analyze the materials, and we do not know how much dioxin they contained, but there was enough to cause human health effects.

Immediately after the explosion, efforts were directed at cleaning up the mess, and workers from throughout the plant were recruited to assist. And the workers got sick. Many complained of skin, eye, and respiratory tract irritation during the time they worked in the contaminated building; headaches, dizziness, and nausea were common. Also within a week or two of the explosion, workers broke out in chloracne.

Except for the chloracne, many of the early symptoms went away within one to two weeks, only to be replaced by others. Aches and pains in their legs incapacitated some workers, requiring hospitalization. Others were affected by severe muscle pain in their shoulders and chests, fatigue, nervousness and irritability, insomnia, decreased libido, and sensitivity to cold. The muscle aches and pains persisted for months in the more severely affected workers.

A 1949 clinical examination revealed that some workers had enlarged and sensitive livers, changes in blood levels of certain chemicals that were consistent with liver damage, and that the pain in their arms, legs, hands, and feet resulted from irritation or damage to nerves. When examined four years later, in 1953, the workers' conditions were much improved; the symptoms associated with liver and nervous system damage had disappeared in some men and subsided in others.

There can be no doubt that the Nitro workers were exposed to substantial amounts of dioxin. Furthermore, they were exposed so long ago that enough time has passed for long-term effects to have been expressed. Two studies of the Nitro workers were published in 1984: one by Dr. Raymond Suskind[1], who had been employed by Monsanto; the other by Dr. Marion Moses and her colleagues[2] from the Mt. Sinai School of Medicine at the request of the union that represents many Nitro workers. Their findings were remarkedly congruent. Although chloracne persisted in 60 percent of the men and some biochemical abnormalities were seen, there was no excess of life-threatening or debilitating diseases in men who had ever had chloracne or who had been exposed to dioxin.

Another study carried out by Drs. Zack and Gaffey[3] examined causes of death among Nitro workers. There were no excess deaths from cancer. Although the heart disease death rate was higher than expected when compared to national averages, it was in line with expectations for the county in which Nitro is located. Obviously, either comparison can be made: a person who believes that dioxin is responsible for heart disease is likely to favor a

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1. Suskind, R.R. and V.S. Hertzberg. Journal of the American Medical Association 251:2372-2380. 1984
2. Moses, M. et al. American Journal of Industrial Medicine 5:161-182. 1984.
3. Zack, J. and W.R. Gaffey in Human and Environmental Risks of Chlorinated Dioxins and Related Compounds. (eds) Tucker, R.E., A.L. Young, and A.P. Gray. Plenum Press:New York. 1983.

comparison with the national average; a person who believes otherwise will favor comparing to the county average.

Although dioxin has been suggested as a cause of many diseases, it is most often associated with cancer. Dioxin has been fed to laboratory rats[4] and mice[5] and applied to the skin of mice.[6] It caused cancer in all those studies, and those data are sufficiently convincing that dioxin is a presumed human carcinogen. For a variety of reasons that are well described in the literature[7], dioxin is generally regarded as being important in the second stage of a two-stage model for cancer induction. If that is the case, the fact that it causes cancer in laboratory animals only at doses close to those that cause acute toxic effects may mean that it would be carcinogenic in humans only at exposure levels that cause other ill effects also. I do not want to dwell on that point; the problems of extrapolating from animal data to make predictions about human effects are too great.

If dioxin has caused human cancer, the most likely place to detect it is in studies of the humans who were most extensively exposed, chemical production workers. At least eight papers have reported the number of cancers seen in industrial workers exposed to dioxin through explosions, such as at Nitro, or through leaks during production [see table 1]. No statistically significant excess of total cancers was seen in any of the populations. Furthermore, no specific cancer was unusually common in the populations.

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4. Kociba, R.J. et al. Toxicology and Applied Pharmacology 46:279-303. 1978 and National Toxicology Program. Technical Report Series. No. 209..
5. National Toxicology Program. Technical Report Series. No. 209.
6. National Toxicology Program. Technical Report Series. No. 201.
7. Rodricks, J.V. in Human and Environmental Risks of. Chlorinated Dioxins and Related Compounds (ed) Tucker, R.E., A.L. Young, and A.P. Gray. Plenum Press:New York. 1982. pp 629-633 and Longstreth, J.D. and J.M. Hushon in same volume pp 639-664.

In contrast to the absence of excess cancer in production workers, Swedish lumberjacks who were exposed to dioxin-contaminated herbicides, were reported to have an excess of soft tissue sarcomas.[8] Other studies of herbicide applicators in New Zealand[9] have failed to find that association and directly contradict the Swedish results. That association took on more importance this year when a study done by the Massachusetts Department of Public Health found more deaths from soft tissue sarcomas among veterans who had served in Vietnam than would be expected from rates among other veterans and non-veterans who resided in that state.[10] A similar study of New York veterans found no excess of those tumors[11], and results of a third similar study in West Virginia are expected soon. It is important to remember that none of the veterans' studies has any information about dioxin exposure, and certainly no quantitative information about levels of exposure; all that *is* known is that some veterans served in Vietnam, some did not. The soft tissue sarcoma question is hot right now, and we can expect more information shortly because of the number of studies going on. It is far from clear at this point that there is any association. Nevertheless, the suggested association is better supported than any other between dioxin and cancer.

Besides lumberjacks and agricultural herbicide applicators, one other group that sprayed dioxin-contaminated herbicides has been well studied. The 1269 Air Force officers and enlisted men assigned to Operation Ranch Hand sprayed over 10,000,000 gallons of Agent Orange, which contained an average of two parts per million dioxin, in Vietnam. Medical examination of all the

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8. Hardell, L. and A. Sandstrom. British Journal of Cancer 39:711-717.
9. Smith, A.H. et al. Journal of the National Cancer Institute 73:1111-1117
10. Kogan, M.D. and R.W. Clapp. Massachusetts Department of Public Health. Typescript. January 25, 1985.
11. Greenwald, P. et al. Journal of the National Cancer Institute 73:1107-1109.

living Ranch Hands found no soft tissue sarcomas,[12] and no Ranch Hand has died from that cancer.[13] Table 2 shows the causes of deaths for Ranch Hands and a comparison population of Air Force officers and enlisted men who were not exposed to herbicides. There has been no excess of deaths overall nor excess cancer deaths in Ranch Hands.

Dioxin is often mentioned as a possible cause of birth defects. In fact, the original concern about it as a threat to human health stems from its causing cleft palates and kidney abnormalities in newborn mice when pregnant females were exposed.[14] The observation that dioxin causes birth defects when administered to pregnant mice has been made repeatedly, but several similar experiments in rats have failed to find birth defects[15], except for one report of kidney abnormalities.[16] In both rats and monkeys, the effect of dioxin administration to pregnant females is to cause fetal toxicity, not birth defects.[17]

One experiment tested the effects of dioxin on the reproductive function of male mice. The dioxin caused liver and thymus toxicity; there is no doubt that it made the mice sick. However, even those amounts of dioxin had no effects on sperm concentration, motility, or appearance even after eight weeks of exposure. To test the possibility that exposure of males could cause birth defects or spontaneous abortions, the male mice were mated

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12. USAF School of Aerospace Medicine. Project Ranch Hand II: An Epidemiolgic Investigationof Health Effects in Air Force Personnel Following Exposure to Herbicides. Baseline Morbidity Study Results. United States Air Force:Brooks Air Force Base, Texas. 24 February 1984.

13. USAF School of Aerospace Medicine. Project Ranch Hand II Mortality Update - 1984. United States Air Force:Brooks Air Force Base, Texas. February 1985.

14. Courtney, K.D. and J.A. Moore. Bull. Environ. Contain. Toxicol. 7:45-51. 1971.

15. Veterans Administration. Review of Literature on Herbicides, Including Phenoxy Herbicides and Associated Dioxins. Volume 1. 1981.

16. Moore, J.A., M.W. Harris and P.W. Albro. Toxicology and Applied Pharmacology 37:146-147. 1976.

17. Veterans Administration, 1981. op cit.

repeatedly with unexposed females. There were no adverse effects: exposed males mated as frequently as unexposed males and fathered the same number of pups; there was no increase in birth defects nor early deaths among mice fathered by dioxin-exposed males.[18]

Reproductive health effects in humans have been important in policy decisions about dioxin-containing herbicides. A survey of spontaneous abortions among women who lived near herbicide sprayed areas in Oregon was instrumental in the Environmental Protection Agency's (EPA) regulating uses of the herbicides. However, the design and interpretation of that study has come under fire[19], and to my knowledge there is no other study that supports the idea that forest spraying caused miscarriages. The Centers for Disease Control (CDC)[20] investigated the possibility that Vietnam veterans were at increased risk of fathering children with major birth defects. No increase was found. CDC also attempted to estimate which veterans might have been exposed to Agent Orange, but as Hellen Gelband and I testified before the House Committee on Veterans' Affairs in October 1984,[21] those estimates are so inherently uncertain that the CDC study can tell us nothing about any possible relationship between Agent Orange and birth defects.

We, of course, do know that Ranch Hands were exposed to Agent Orange, and we have the results of asking them about their reproductive health. Ranch Hands and their wives more frequently reported minor birth defects, such as birth marks, than did a comparison group of Air Force personnel and wives. Those effects are not serious and might be related to Ranch Hands (who know

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18. Lamb, J.C., J.A. Moore, and T.A. Marks. National Toxicology Program:Research Triangle Park, NC. (publ. NTP-80-44). 1980.
19. Wagner, S.L et al. A Scientific Critique of the EPA Alsea II Study and Report. Oregon State University. 1979.
20. Erickson, J.D., et al. Journal of the American Medical Association. 252:903-912. 1984.
21. Gough, M. and H. Gelband. Testimony before the Subcommittee on Hospitals and Health Care of the House Committee on Veterans' Affairs. October 3, 1984.

they were exposed to Agent Orange) doing a better job of remembering minor birth defects. More importantly, Ranch Hands reported a significantly higher frequency of neonatal deaths, deaths that occurred within the first month after birth. The comparison population reported very few neonatal deaths, much below the national average, and the difference between the Ranch Hands and comparisons may be at least partly explained by good luck among the comparison population rather than a toxic effect in Ranch Hands.[22] xhe Air Force is now collecting hospital and medical records to verify the recollections of the Ranch Hands and comparions. That verification, which is routine in epidemiologic studies, will provide us more solid information about reproductive health effects, but overall the Ranch Hand study is reassuring that adverse effects are rare or nonexistant.

The residents of Times Beach, Missouri, are also known to have been exposed to dioxin. An examination of their health revealed no ill effects[23], but only a few years have passed since the exposure, and it can be argued that we have to wait to see if there are long term effects. A chemical plant explosion in Seveso, Italy, as well as the spraying of horse arenas in Missouri, exposed individuals to dioxin in sufficient amounts to cause chloracne.[24] Children who played in the horse arena also developed kidney disease that responded to treatment after the cause was discovered. Those episodes show that under special circumstances environmental exposures can be sufficiently bad to cause acute effects that resemble some of those seen in industrially exposed populations.

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22. USAF School of Aerospace Medicine, 1984. op cit.
23. Center for Environmental Health, Centers for Disease Control and the Missouri Division of Health. Missouri Dioxin Health Studies. October 16, 1983.
24. Wipf, H.K, and J. Schmid in Human and Environmental Risks of Chlorinated Dioxins and Related Compounds (ed) Tucker, R.E., A.L. Young, and A.P. Gray. Plenum Press:New York. 1982. pp 255-274.

In summary, dioxin has caused chloracne, and that disfiguring skin disease can last for more than 30 years and probably for a lifetime. It has also caused effects on the nervous system of industrially exposed workers as well as changes in blood chemistry. The larger question that remains is whether exposure to dioxin causes effects such as cancer or birth defects. There is no consistent or convincing evidence from industrially exposed populations for dioxin causing early deaths, or deaths from cancer or heart disease. The possibility that it causes a rare tumor, soft tissue sarcomas, is supported by some studies and not by others, and that issue is being investigated actively. So far as I am aware, there is no evidence for dioxin causing birth defects in humans and the evidence for its causing abortions is equivocal and disputed. Therefore, the human evidence suggests that dioxin has not caused death or life-threatening diseases. Furthermore, but more speculatively, since it has not produced significant effects in industrially exposed populations and in Ranch Hands, I think that it is unlikely that lower level environmental exposures are going to cause such effects. That conclusion must be qualified because environmental exposures might go on for a person's lifetime rather than the 8-hour a day workplace exposure, and children and elderly people as well as workers are exposed. Even with those qualifications, dioxin in the environment seems distinctly unlikely to be a major determinant of human disease, despite the fact that it can cause devastating effects in animals.

Table 1

Cancers in Trichlorophenol and 2,4,5-T Workers

Place of Exposure	Number of Workers Studied	Total Cancers[1] Expected	Observed	Most recent data
Dow, USA, trichlorophenol production 1964 and earlier	61 49 chloracne	1.6	3	1978
Dow, USA, 2,4,5-T production, 1964 and earlier	204 0 chloracne	3.6	1	1978
Monsanto, USA, trichlorophenol accident, 1949	121 all chloracne	9.4	9	1978
Monsanto, USA, 2,4,5-T production 1955-1977	58 deceased	10.9	9	1978
BASF, W. Germany, trichlorophenol accident, 1953	75 all chloracne	4.1 [0 stomach cancers expected]	7 [3 stomach cancers observed][2]	1979
Phillips-Dupbar, Holland, trichlorophenol accident, 1963	141 69 chloracne	6.9	8	1983
Coalite, England, trichlorophenol accident, 1968	90[3] all chloracne	NA[4]	0	1981
Spolana, Czechoslovakia, 2,4,5-T production, 1965-1968	55 53 chloracne	NA	2 lung	1981

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1. none of the differences between expected and observed total cancers is statistically significant.
2. the difference between expected and observed stomach cancers is significant. No excess of stomach cancers has been reported from other populations.
3. it is unclear how many of the 90 men are included in the statement that there has been no cancer in the population.
4. not available.

Table 2

Comparison of Specific Causes of Death Observed Among Ranch Hands and a Comparison Group of Air Force Personnel

Cause of Death	Number of Deaths in 1,256 Ranch Hands[1]	Number of Deaths in 6,171 Comparisons[2]	Ratio of the Frequencies of Deaths Ranch Hands[3] Comparisons
Accidental	19	94	94%[4]
Circulatory disorder	17	75	104%
Malignant neoplasms	6	43	68%
Digestive system disorder	5	13	192%
Suicide	3	16	94%
Homicide	2	4	250%
Respiratory disorder	0[5]	5	
Parasitic infections	0	4	
Uncertain neoplasms	0	2	
Endocrine system disorder	1	1	
Genitourinary disorder	0	3	
Mental disorder	0	1	
Nervous system disorder	0	2	
Ill defined	1	2	
All causes	54	265	100%

1. Air Force personnel who sprayed dioxin-contaminated Agent Orange in Vietnam.
2. Air Force personnel who flew and serviced aircraft similar to those used by Operation Ranch Hand but who were not exposed to herbicides.
3. frequency of deaths from indicated cause among Ranch Hands divided by frequency of death from the same cause in the comparison group, expressed as a percentage.
4. in this table, 100% means that the frequencies in Ranch Hands and Comparisons were identical. Less than 100% means that the frequency was lower in Ranch Hands than in comparisons. Greater than 100% means that the frequency was greater in Ranch Hands than in comparisons.
5. frequencies were not calculated for causes in which the number of Ranch Hand deaths was 0 or 1.

Source: adapted from Project Ranch Hand II Mortality Update - 1984. p. 14.