

DioxinSpin.com has been established to provide a source of dioxin-related information to Midland and Tittabawassee River residents. The website has a wide variety of important information related to human health, current dioxin exposure levels, historic Midland plant waste facilities and operations.

Much of what is presented on the website disputes The Dow Chemical Company's claims as to the risk to human health from dioxin exposure.

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Long Term Dioxin Contamination

Dioxins have been present in Midland soils and in the sediments and floodplain of the Tittabawassee River for more than 70 years.

Midland Soil Contamination

In the late 1930's, The Dow Chemical Company constructed its first chemical waste incinerator – the "Vertical Tar Burner". The VTB was a vertical, open top tank – much like a very large trash burning barrel. A coal fire burning beneath a metal grate in the bottom of the tank vaporized and ignited the chemical wastes. When the metal grate was "visibly hot enough", liquid chemical wastes were sprayed on the grate and the burning coals. Packaged wastes could also be placed on the grate and burnt.

The coal fired burner had no air pollution equipment and released large amounts of black smoke – a sign of incomplete combustion. The company continued to use the VTB into the 1950's and as a backup burner into the 1960's.

Some of the incinerators and tar burners that were built after the VTB were poorly designed, often overloaded and some became recognized as "sources of local air pollution".

- Newsletter Topics:**
1. Long Term Dioxin Contamination
 2. Epidemiology Concepts
 3. Mortality of Dow's Dioxin Exposed Workers

Some thirty years later, in 1968, the company constructed a greatly improved tar burner and, shortly thereafter, installed advanced air pollution control equipment on its rotary kiln incinerator, built 10 years earlier in 1958.

However, by the 1960's, severe dioxin contamination had already occurred in the Midland plant and in large portions of the city of Midland. It is believed that concentrations of dioxins in the plant and in the city may have peaked in the 1969 time period.

Table A: Estimated Dioxin Levels - parts per trillion, ppt - 1969

	TCDD	TEQ
Midland Plant (a)	11,120	89,903
City of Midland (b)	490	3,690

(a) Based on avg. TCDD, 1984

(b) Based on 3 highest congener levels, 2001

Tittabawassee River Contamination

Unknown quantities of dioxins were released each year as the company drained its organic chemical waste ponds to take advantage of "the self purification forces of the river during summer months."¹

By 1931, the Midland plant had more than 600 acres of chemical waste ponds in operation – a 150 acre salt brine pond and approximately 450 acres of chemical waste ponds.

In 1929, the city of Saginaw began to use the Tittabawassee-Saginaw River as its source of drinking water and a company publication indicated that the company was aware of the drinking water plant.

Eight years later, in 1937, the company constructed its first biological waste treatment plant and treated approximately 25% of the chemical wastes being discharged to the river.

During WW II, untreated chemical wastes to the river increased to 50 Million gallons per day. In 1946, the company constructed its second biological treatment plant.

1944 was a drought year and river flows in the summer months dropped to very low levels. In August, 1944, the company's flows of untreated and treated wastes made up approximately 28% of the river as it flowed past Mapleton, Freeland and Saginaw. It is not known if children continued to swim in the river or if riverside residents used river water for bathing, drinking, cooking and crop irrigation during 1944.

As the company began to construct waste incinerators with improved air pollution control equipment, the dioxins in the contaminated scrubbing water from the incinerators were partially treated in the biological treatment plant before being discharged to the river.

Higher levels of dioxins continued to be discharged with the treated waste water until a series of large settling ponds were constructed in the 1970's.

Contaminants continued to leak from the Midland site until the 1990's when a groundwater collection system was installed to collect contaminated groundwater. Even though the chemical waste ponds had been drained, filled and closed for several years, groundwater with dioxin levels greater than 7,400 ppt-TEQ was still being collected in 1998.

It is impossible to estimate the levels of dioxins that were discharged to the river before and after the treatment plants were constructed. It is also impossible to estimate the amounts of dioxins that "leaked" from the waste ponds for more than 60 years.

The annual springtime flooding results in a re-distribution of dioxins from the river sediments to the surface soils of the floodplain. When flooding is mild, sediments gently settle on the surface and bury the dioxins deposited the previous year. When flooding is severe, deeper layers of floodplain soils are suspended and re-distributed by the strong force of the flowing river.

In 2001, the DEQ sampled 22 residential properties that are subject to frequent flooding. The table shows the average dioxin concentration by depth of soil.

Table B: Dioxin Concentration by Depth, (2001), ppt-TEQ

Depth	ppt-TEQ
1"	320
3"	522
6"	516
15"	835

As can be seen, the 3" and 6" dioxin concentrations are approximately the same while the average concentration at 15" is almost 3 times the surface concentration. The amounts of dioxins that might be buried in deeper levels is unknown.

Flooding in the Tittabawassee River is generally mild for many years in a row – followed by a single year of extreme flooding.

During 1948 to 1959, there were ten (10) years in which flooding was not severe. Large amounts of dioxins may have accumulated in the river sediments only a short distance from the Dow plant. The flood of 1959 (approximately equal to the flood of 2004) re-suspended and re-deposited the settled dioxins further downstream and higher in the floodplain.

From 1938 to 1946, the amounts of dioxins that were released grew dramatically due to the higher production levels during WW II. During these years, the company was only treating approximately 25% of its waste flows to the river.

After 1946, the company began to treat all of its waste flows to the river, but only about 80% of the organic chemicals were removed before discharge to the river. Based on the long half-lives of the dioxins, it is possible that only a small

amount of dioxins were removed in the treatment plants.

Dioxins will degrade with time, the only question is how fast or how slow. The Midland site's biological treatment plants treat a mixture of chemical and sanitary (human) wastes. The influence of natural bacteria and human-based nitrogen fertilizers on dioxin degradation in the river is not known.

Table C: Severity of Flooding

Extreme Flooding	Less Flooding	Extreme Flooding
1938	39, '40, '41	1942
1942	43, '44, '45	1946
1948	49, '50, '51, '52, '53, '54, '55, '56, '57, '58	1959
1960	61, '62, '63, '64	1965
1967	68, '69, '70, '71, '72	1973
1974	'75	1976
1976	77, '78, '79, '80, '81, '82	1983
1983	'84	1985
1986	87, '88, '89, '90	1991
1992	93, '94, '95, '96	1997
1997	98, '99, '00, '01, '02, '03	2004

Some very preliminary modeling of dioxin levels found at the 15" depth has been carried out in 20 year increments and are shown in Table D.

Table D: Dioxin Concentration - Floodplain, 15" depth

	2002	1982	1962	1942
TEQ, ppt	835	5,286	647,499 (647 ppb)	4,011,285 (4 ppm)
TCDD, ppt	161	569	2,011	7,107

The real value of calculating dioxin levels is to demonstrate that dioxin levels were **much, much higher** in the floodplain and in the river sediments in prior years than they are today – how much higher is subject to debate.

With higher levels of dioxins in the upper soils, riverside residents will ingest higher amounts of dioxins and dioxin blood serum levels will increase substantially.

The properties along the river have only been sampled in a few locations and there is a low probability that a complete understanding of dioxin levels along the river has been achieved.

The EPA has indicated that **30 locations per acre** must be sampled to achieve a 95% confidence level that all areas of high dioxin contamination have identified.

Each springtime flood will re-suspend and re-distribute dioxins on riverside properties. Severe flooding that occurs in some years may cut into deeper depths of the floodplain and may carry dioxins into portions of the floodplain that are a further distance from the low water mark.

Just to be certain that dioxin exposures are within the allowable range, **all riverside properties should be sampled and analyzed for dioxin levels after each flood.**

Each flood may bring additional dioxin exposure. Dow has agreed to clean up areas of newly deposited sediments. However, cleanup can not be done simultaneously on all riverside properties. It is doubtful that many residents, especially children, will be willing to stay indoors when warm springtime weather replaces the harsh winter.

Forcing residents to remain inside to minimize dioxin exposure until cleanup occurs may be another aspect of a "taking of property rights."

References

1. The Dow Chemical Company, Dow Diamond, August 1938
2. Bond, GG, *et al.*, Cause-Specific Mortality Among Male Chemical Workers, A, Jour Ind Med, 12:353-383 (1987)
3. Cook, RR, *et al.*, Update of the Mortality Experience of Workers Exposed to Chlorinated Dioxins, Chemosphere, Vol. 16, No. 8/9 (1987)
4. The Dow Chemical Company, Dioxin Data, Nov 7, 2003
5. Ott, MG, *et al.*, Cohort Mortality Study of Chemical Workers With Potential Exposure to Higher Chlorinated Dioxins, Jour Occup Med, Vol. 29, No. 5 (May 1987)

Epidemiology Concepts

Epidemiology is the division of medical science concerned with defining and explaining the inter-relationships of the host (patient), the agent (dioxin), and the environment in causing disease.

The overwhelming majority of epidemiological studies that deal with the risk to human health from dioxin exposure are **mortality** studies in which the actual deaths in a cohort are compared to the expected deaths.

A **cohort** is a group of studied persons (both living and dead) that are linked by one or more common characteristics – exposure to industrial dioxins, living or having died between 1940 to 1982, a Dow dioxin worker, etc.

The size and, most importantly, the characteristics, of a cohort are extremely important in being able to prove or disprove a hypothesis – such as “does dioxin exposure result in an increase in cancer?”

A great deal of care is spent in selecting the cohort and **excluding or including** certain workers. One Dow study excluded 4,100 workers from a general mortality study – even though the workers clearly met the criteria of the study. When the morality of the excluded workers was finally disclosed ten years later, the group of workers had a **statistically significant increase in Mortality - All Causes and Mortality - All Cancers**. No explanation was ever given as to why the 4,100 workers were excluded from the initial study. It should be noted that including the 4,100 workers that were in poorer health would have affected the results of the initial study.

There have been cases in which companies may have falsified dioxin exposure results, but such cases are hopefully rare. Search “Kemner v Monsanto” on the Internet for additional information

The US Census Bureau and other organizations have compiled a great deal of statistics on the death rates of the US Population against a variety of factors - age, sex, disease, etc. A number of computer programs can be used to determine the expected death

rates for a group of US residents that are comparable to the studied cohort.

Actual deaths are labeled “**Observed deaths**” or simply abbreviated “**Obs.**”

“**Expected**” deaths are labeled “**Expected**” and are abbreviated as “**Exp.**” in a mortality study.

The “**Standardized Mortality Ratio (SMR)**” is equal to the “**Observed**” deaths divided by the “**Expected**” deaths times 100. An SMR = 100 is “normal”.

Each study has a specific way of reporting mortality statistics, but most will report **SMR, Obs.** and **Exp.** In some cases, only **SMR and Obs** will be given and the reader must calculate **Exp.**

In a mortality study, the two most important results are **Mortality, All Causes** and **Mortality, All Cancers**. The mortality from specific cancers and diseases are important, but of lesser importance.

In most mortality studies, the “expected” deaths are based on the US Population and the study will say so. In some studies, “expected” death rates are based on a comparison population, such as a group of age and gender similar employees at the same location. These comparisons are called “**Relative Risk**” studies. The most precise Relative Risk studies are when an exact match is found in the comparison group for every member of the studied cohort – a single match per studied subject.

However, if the comparison group is large enough, such as the non-dioxin employees of the Michigan Division, then a comparison is made using the mortality data of the entire comparison group. However, a study that uses too large of a comparison group may not be as reliable as a “one-to-one” comparison.

95% Confidence Interval (95% CI)

When mortality statistics are reported, a parameter that must always be reported is the Confidence Interval – in most mortality studies, the **95%** Confidence Interval.

In an unbiased study (one in which measurement does not effect results), a 95% CI indicates that there is a 95% chance that the confidence interval

includes the **true result**.

In a mortality study, **SMR's greater than 100** indicate that the number of Obs. Deaths greater than Exp. Deaths. In order for the elevation (SMR > 100) to be **statistically significant**, the lower range of the 95% CI must be greater than 100.

For example, if the SMR is equal to 120 and the 95% CI is 92 to 240, the elevation is not statistically significant since the true value can be less 100.

If on the other hand, the 95% CI is 105 to 240, then the SMR = 120 is statistically significant since the true value is at least 105.

Unfortunately, the calculation of the 95% Confidence Interval is complicated and the reader must rely on the integrity of the researcher. The method that was used to calculate the 95% CI is generally mentioned but the specific calculations are never given.

“True Lies”

It must be remembered that a great many dioxin studies are carried out by **US corporations** and it should be expected that some “spin” might be present in these studies. The most typical “spin” in when a significant result is discussed in the “results” or “discussion” portion of the study but then is not made part of the “**abstract**”.

Since many non-epidemiologists read only the abstract, “**True Lies**” – lies of omission – may not be apparent. It is extremely important to read and compare the “results-discussion” with the “abstract” in every dioxin-related study.

PR Summaries of Mortality Studies

In 2002 and 2003, the company provided an “Overview of Dow Worker Health Studies” on its Dioxin Data website. The summaries were provided for the convenience of the website reader. The Public Relations (PR) summaries contained varying amounts of “Dow-spin.”

The PR summary for **Cause-Specific Mortality Among Male Chemical Workers**² stated, "Comparison of observed mortality demonstrated lower mortality in the cohort from each of the major causes of death, including total malignant neoplasms." ***This is true.***

However, the PR summary failed to point out that study also reported, "Unique among hourly employees was **significant excess mortality** from in the categories of cancer from other lymphatic tissues, ... and both hourly and salaried nonexempt experienced **significantly higher mortality** from other and ill-defined cancers." In addition, "... a significant increasing trend in [hourly worker] mortality from **cancer of all sites combined** [All Cancers] with increasing duration of employment."

The PR summary for **Update of the Mortality Experience of Workers Exposed to Chlorinated Dioxins**³ reported, "No increased risk of cancer was found..." ***This is "spin".***

The study reported an elevation in eight cancer categories, including **one that was statistically significant.**

Following disclosure on DioxinSpin.com about these types of "errors", the company removed the PR summaries from its website.

More examples of "Dow-spin" can be found on the DioxinSpin.com web site. "Click" Mortality Overview on the left side of the Home Page.

References (continued)

6. Ramlow, JM, *et al.*, Ten-Year Update of a Cohort Mortality Study of Workers with Potential Exposure to Higher Chlorinated Dioxins, Unpublished, Jan. 25, 1996

- a. Brinewell, January 1961
- b. Brinewell, July 1959
- c. Dow Diamond, April 1967
- d. Brinewell, January 1970

Mortality of Dow's Dioxin Exposed Workforce

The most severe and confirmed impact on human health has been a significant increase in the number of deaths from a wide variety of cancers in persons exposed to high levels of dioxins.

The International Agency for Research on Cancer (IARC), a division of the World Health Organization of the United Nations has classified TCDD as a "**known human carcinogen**" affecting multiple sites and organs in the human body. The U.S. EPA, the U.S. Public Health Service, and the Institute of Medicine of the National Academy of Science agree with the assessment that TCDD is a carcinogen. The "**known**" classification is based on sufficient evidence of carcinogenicity found in **studies in humans.**

The IARC has concluded, "Overall, the strongest evidence for the carcinogenicity of 2,3,7,8-TCDD is for **all cancers combined**, rather than for any specific site ... There are few examples of agents which cause an increase in cancers at many sites; examples are smoking and ionizing radiation in the atomic bombing survivors ..."

Dow Chemical has stated, based on its own studies of the ~2,200 Midland plant employees that have been exposed to TCDD and the other dioxins, "These [Dow] studies show no convincing evidence of links between specific cancers and potential dioxin exposure."⁴

Dow Workers : Elevated Cancer Mortality

However, a close examination of Dow's mortality studies of its Midland Plant workers does show a very strong association between elevated cancer death rates and dioxin exposure.

The last page of this newsletter is a summary of the *Standardized Mortality Ratio's* (SMR's) taken from six epidemiology studies of Dow workers and one National Institute of Occupational Safety and Health (NIOSH) study of 5,172 dioxin workers from 12 U.S. companies.

The Dow Midland plant workers represent 42% of the total number of employees studied in the NIOSH report.

The "clustering" of increased deaths from specific cancers is very pronounced. **The Dow studies indicate a linkage between dioxin exposure and the higher death rates from cancer.**

Dow Legal Department Disputes Company's Claims

A letter written by the company's Legal Department seems to dispute Dow's claims that "... [Dow] studies show no convincing evidence ... between ... cancers and ... dioxin exposure."

In a March 23, 1998 correspondence, a Dow attorney informed the U.S. EPA that Midland plant workers exposed to TCDD and other dioxins were found to have a statistically significant increase in **stomach cancer, prostate cancer and the very rare cancers grouped as "ill-defined or unspecified site cancers"**.

Workers with high levels of cumulative TCDD exposure had a risk of **stomach cancer** that was more than **4 times** that of unexposed workers, with risk from **prostate cancer** being almost **3 times** that of unexposed workers.

Michigan Division Employee Mortality

The company has pointed out that Michigan Division employees are generally "healthier" than the general US Population – perhaps subtly implying that dioxin exposure is not a risk to human health.

Company employees tend to be "healthier" than the US Population for several reasons:

- Only healthy job applicants are hired
- Employees have access to paid health care
- An anomaly of the US Population database.

The "**anomaly**" is that the US Population database includes persons too sick to work. Since the number of **Expected** deaths will be higher by including those too sick to work, it appears as if the group of Dow workers

is actually much healthier than they are. One Dow study ⁵ discounted the importance of a **lower** Mortality, All Causes, "The SMR for all causes of death was 93 ... but the ... [SMR] was not statistically significant (95% confidence limits, 84 to 104)."

In 1996, the company reported an update in the mortality of the dioxin exposed workers at the Midland plant. ⁶ The study also compared the death rates of workers exposed to TCDD against the death rates of Michigan Division employees **not exposed** to pesticide plant TCDD. The comparison is shown below in Table F.

Table F : Mortality of Dow TCDD Exposed Workers Vs. Dow Non-Exposed Workers

	<u>TCDD Exposed Chemical Workers</u> (1940 - 1994)		
	<u>Actual Deaths</u>	<u>Exp. Deaths (a)</u>	<u>SMR</u>
All Causes	655	637.1	102.8
All Cancers	168	161	104.3
Digestive organs	42	38.7	108.5
Stomach	10	5.7	169.5
Large intestine	16	14	114.3
Lung	48	56.1	85.6
Prostate	20	12.5	160.0
Lymphatic	21	15.8	132.9
Diseases of circulatory system	318	312.2	101.9
Non-malignant respiratory diseases	34	41.8	81.3

(a) Deaths in Michigan Division non-dioxin population

The dioxin-exposed workers have a **higher death rate** than do the Michigan Division workers that were not exposed to pesticide plant dioxins.

The information shown in Table F is extremely important since it clearly shows that TCDD exposure does have an adverse impact human health.

Mortality of Non-Chloracne Workers

The company has reported, a number of times, that dioxin-exposed workers that were diagnosed with chloracne are much healthier than the general U.S. male population. *This is true.*

Unfortunately, dioxin-exposed workers **without chloracne have higher death rates**. A comparison between chloracne and non-chloracne workers based on data provided in the

1996 study is shown below. For some reason, the study did not specifically address the increased mortality of the non-chloracne workers. The table is based on the calculated differences between the total cohort information and the chloracne worker data.

It is very apparent that workers that were **exposed to dioxins and did not develop chloracne** have a much higher cancer mortality than do Dow workers that were diagnosed with chloracne.

Table G : Mortality Comparison between Chloracne and Non-chloracne Dow Workers (1940 - 1994)

	<u>Chloracne Workers</u>			<u>Non-Chloracne Workers</u>		
	<u>Actual Deaths</u>	<u>Exp. Deaths (a)</u>	<u>SMR</u>	<u>Actual Deaths</u>	<u>Exp. Deaths (a)</u>	<u>SMR</u>
All Causes	51	86.5	59	645	720	90
All Cancers	10	20.3	59	158	150.7	105
Digestive organs	2	5.1	39	40	38.5	104
Stomach	0	0.7	---	10	6	167
Large intestine	0	1.9	---	16	13.7	117
Lung	0	0.1	---	54	62.7	86
Prostate	0	1.5	---	20	10.5	190
Lymphatic	0	1.9	---	21	14.5	138
All Other Cancers (b)	3	1.4	214	13	10.5	124
Diseases of circulatory system	25	41.1	61	293	313.7	93
Non-malignant respiratory diseases	7	6.2	113	27	44.2	61

(a) Expected deaths based on US male population
 (b) Very rare cancers without ICD category

DOW CHEMICAL RESPONSIBILITY

Dioxin contamination of Midland and the Tittabawassee River began in the 1930's – years before the presence of dioxins became known – years before the health effects from dioxin exposure became recognized.

For many years, The Dow Chemical Company was unaware that the Midland plant was emitting large quantities of these extremely toxic chemicals. However, as analytical capability improved, the company did become aware of the dioxins being released in its emissions. Whether aware or unaware, the company must still be held responsible for its actions.

For many years, the company operated its waste facilities – the chemical waste ponds and tar burners and incinerators – on the **short term effects** of chemical exposure. The company relied on atmospheric dilution and river water dilution to reduce the acute (short term) toxicity of its waste emissions. For many years, “**dilution was the solution to pollution**”. With the chemical knowledge of the 1920's and 1930's, this was understandable. In later years, it was not.

In 1933, the company established its **Toxicology Department** to centralize research on the health effects from both short term and long term exposure to chemicals. Shortly thereafter, the company recognized that phenol and the chlorinated phenols in the river were responsible for the foul chemical taste being found in fish. In 1937, the company began the biological treatment of these wastes and the foul fish taste problem was corrected – until it reappeared in 1974. **Even at toxic levels, TCDD and the other dioxins may be tasteless.**

By the late 1930's, the concentrated organic wastes that had been dumped into the waste ponds (and then to the river) were being “incinerated” in a very crude tar burner. Dioxins began to be spread over large areas of the plant and the city. Subsequent burners and incinerators were also “sources of local air pollution” (visible black smoke) and contributed to the contamination.

Table H: Midland Plant Waste Incinerators

Unit	Years	Dow Comments
Vertical Tar Burner	1930's - 1960's	“air pollution problems due to poor combustion” (a)
Rotary Kiln Incinerator - No. 1	1948 - 1958	“stack effluent as a source of air pollution” (b)
Stationary Tar Burner - No. 1	1957 - 1968	“eliminate air pollution resulting from burning of [chemical] tars” (c)
Rotary Kiln Incinerator - No. 2	1958 - 2005	“a significant contributor to local air pollution” (c)
Power Houses	1960 - 1970's	“NT Power House ... frequently has blackened the air ...” (d)

By 1967, the company was incinerating more than **80 Million pounds per year** of chemical waste tars in units that the company recognized **were operating poorly and emitting large quantities of black smoke and soot.**

Exposure testing of laboratory animals to incinerator emissions was well within Dow's technical capability in the 1940's. It is not known if the company ever tested the black soot and fly ash being deposited on the community to determine if the emissions were toxic.

The company's analytical technology developed rapidly and, **in 1978**, the company reported that TCDD was found in fish taken from the river at the parts-per-trillion (ppt) levels. The EPA found that dioxin emissions from the Midland plant to the river posed a “substantial risk of injury to human health”.

Dioxins were not found in the river sediments or the floodplain at high levels **until 2001** when the DEQ “accidentally” found extremely high levels of dioxins more than 20 miles downstream from the Dow plant. Inadequate sampling and analysis of the river for dioxins from 1978 to 2001 was the fault of the EPA and the DEQ. Dow's responsibility is less clear.

Despite its analytical capability, the company did not test plant soils or Midland community soils for the presence of TCDD for many years. **In 1984**, the EPA (and Dow) finally tested Midland plant soils and city of Midland soils for TCDD – **high levels were found.**

There was ample evidence that the company recognized that TCDD was potentially harmful to human health. **In 1964**, Dow was concerned enough about the toxicity of TCDD that it shut down a new 2,4,5-Trichlorophenol plant for more than a year for decontamination and redesign at a cost of \$5 Million. **In 1978**, Dow's Toxicology Department reported that TCDD was carcinogenic in laboratory animals.

The company has, at great expense, gained knowledge about many of the aspects of dioxins. For some reason (or reasons), the company has been “less curious” about the residential dioxin contamination that it has caused.

Which is the greater error – testing for the presence of dioxins and then concealing the results – or not testing at all?

Future Newsletters:

Future Newsletters will be send via e-mail. If you would like to be on the distribution list, please e-mail newsletter@dioxinspin.com.

If you do not have access to the Internet, please call **1-888-832-0118** (Toll Free) and leave your name and mailing address. A copy of the Newsletters will be mailed to you.

Future Topics:

- 1. Dioxin Exposure in Children and Nursing Infants.**
- 2. Dioxin Blood Serum Levels - Cancer Impact**
- 3. Midland County Cancer Rates**
- 4. Rare Cancers in the Midland Plant**

CHEMICAL WORKER MORTALITY FROM DIOXIN EXPOSURE
STANDARDIZED MORTALITY RATIO'S (SMR) ≥ 110
(SMR = Actual Deaths / Expected Deaths X 100)

Report Number	1	2	3	4	5	6	7	
Author	NIOSH	DOW	DOW	DOW	DOW	DOW	DOW	
Chemical Exposure	TCDD	TCDD	TCDD	TCDD	H/OCDD	General	General	
Number of Employees Studied	5,172	2,189	2,192	2,026	770	37,682	42,076	
Period Covered	1942-1987	1940-1979	1940-1982	1940-1982	1940-1989	1940-1982	1940-1994	
Cause of Death								
All causes (000-999)	99	91	93	97	94/104*	95	109	
All malignant neoplasms (140-209)	115/146*	96	102	106	95/105*	119	111	
Cancer of buccal cavity and pharynx (140-149)	329							
Cancer - digestive organs (150-159)	140					119		
Esophagus (150)	200							
Stomach (151)	138	156	158		176	214		
Small and large intestine (152-153)	178		141			159		
Rectum (154)	115					111		
Liver and biliary passages (155-156)	116					138		
Cancer of respiratory system (160-163)	142					120	185	
Larynx (161)	268							
Lung (162-163)	139					123		
Cancer of bone (170)	521		250					
Soft-tissue and connective tissue (171)	568	330	250	500				
Cancer of skin (172-173)	155							
Cancer of prostate (185)	152	188	190		188	196		
Cancer of testis (186)	149	158	167		158	188		
Cancer of bladder (188)	159							
Cancer of kidney (189)	140				300			
Cancer of brain & CNS (191-192)						148		
Lymphatic-hematopoietic cancer (200-209)	125	167	150	177	132	129	129	
Lymphosarcoma & reticulosarcoma (200)	142			231		147	124	
Hodgkin's disease (201)	276			111		132	132	
Non-Hodgkin's lymphoma (200-202)	137	238	192	210	592			
Leukemia - aleukemia (204-207)	126		121	171		176	138	
Cancer-other lymphatic tissue (202-203,208)	161			200	200	135		
Multiple myeloma	262		200		250			
Cancer of other and unspecified sites	196	243	261	192	161	140		
Diabetes mellitus (250)					111		125	
Diseases of circulatory system (390-458)								
Arteriosclerotic heart disease (410-413)				110				
Non-malignant respiratory diseases (460-519)						133		
Emphysema (492)						174		
Gastric and duodenal ulcer (531-534)		208	185		556			
Cirrhosis of liver (571)			263		154			
Mesthelioma (est)	2755					1000	2687	
Shaded Areas = 3 studies or more with SMR's > 110		* 94/104 - second SMR is with 15-20 yrs of latency						
(1) NIOSH; Cancer Mortality in Workers Exposed to 2,3,7,8-Tetrachlorodibenzo-p-Dioxin; Fingerhut MA, et al; > 1 year of exposure; >20 years latency								
(2) Dow Chemical; Evaluation of the Mortality Experience of Workers Exposed to Chlorinated Dioxins, Cook RR, et al								
(3) Dow Chemical; Cohort Mortality Study of Chemical Workers with Potential Exposure to the Higher Chlorinated Dioxins; Ott MG, et al								
(4) Dow Chemical; Evaluation of Mortality Patterns Among Chemical Workers with Chloracne; Bond GG, et al; > 15 years after hire								
(5) Dow Chemical; Mortality in a Cohort of Pentachlorophenol Manufacturing Workers, 1940 - 1989; Ramlow JM, et al; > 15 years of latency								
(6) Dow Chemical; Cause-Specific Mortality Among Male Chemical Workers; Bond GG, et al; >20 years after hire								
(7) Dow Chemical; Cause-Specific Mortality Among Michigan Employees of a Chemical Company: 1940 to 1994; Burns CJ, et al; >20 years after hire								
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