Ohio | Bureau of Workers' Compensation

Application for Death Benefits and/or Funeral Expenses

Please reference page two of this form for information regarding evidence that you must submit with the application. You can submit this form and supporting documentation via fax to 1-866-336-8352, or send it to your local BWC customer service office.

Name of decedent	Social Security number	Claim number *if known*	Date of death
MICHAEL LOUIS PALUMBO, JR.	[redacted]2	17-142990(SF)	5/24/17

1 Check all that apply:

☐ I am applying for death benefits and, if applicable, funeral expenses (check one of the boxes below) and proceed to section 3.
- ○ For myself
- ☒ For myself and other dependents of the decedent
- ○ On behalf of dependents of the decedent

☐ I am only applying for reimbursement of funeral expenses or services related to the decedent's death. Proceed to section 2.

2 **This section is completed when only requesting reimbursement of funeral expenses or other services**

Complete this section and proceed to section 6

Name	Street address, city, state, ZIP code	Relationship to decedent *if applicable*
Christina M. Palumbo	810[redacted]	Spouse
Social Security number or Federal tax ID #	Cell/phone number with area code	Email address
[redacted]	[redacted]	

List all persons who were dependent on the decedent for support (attach sheet for additional dependents if needed)

First dependent

Name	Street address, city, state, ZIP code	Relationship to decedent
Christina M. Palumbo	[redacted]	Spouse
Social Security number	Cell/phone number with area code and email address	Date of birth
[redacted]	[redacted]	[redacted]

	Street address, city, state, ZIP code	Relationship to decedent
**PLEASE SEE ATTACHED SHEET AND EXHIBITS WITH INFORMATION REGARDING THE		
Social Security number	Cell/phone number with area code and email address	Date of birth
ADDITIONAL DEPENDENTS / 3 MENOR CHILDREN WHO RESIDE WITH THEIR MOTHER *** ATTACHED ***		

Name	Street address, city, state, ZIP code	Relationship to decedent
Social Security number	Cell/phone number with area code and email address	Date of birth

4 **Complete this section if you are the decedent's spouse**

Was the decedent residing with you at time of death? Yes ☒ No ☐ If no, please explain why you were living separately.

Were you previously married? Yes ☐ No ☒

5 **Decedent information**

Was decedent married more than once? Yes ☐ No ☒ Does the decedent have any children not listed in section 3? Yes ☒ No ☐ Attached

6 **Signature**

I am applying for death benefits, reimbursement of services related to the decedent's death and/or funeral expenses under the Ohio Bureau of Workers' Compensation Act for work-related injuries. I affirm that I elect to receive compensation and benefits under Ohio's workers' compensation laws for my claim, and I waive and release my right to file for and receive compensation and benefits under the laws of any other state for this claim. I request payment for compensation and/or benefits as allowable.

I certify the information on this form is true and correct to the best of my knowledge. I understand that any person who knowingly makes a false statement, misrepresentation, concealment of fact or any other act of fraud to obtain benefits and/or compensation as provided by BWC or self-insuring employers, or who knowingly accepts compensation to which that person is not entitled, is subject to criminal prosecution and may, under appropriate criminal provisions, be punished by a fine or imprisonment or both.

Person completing this form (please print)	Christina M. Palumbo	Date 6/14/17
Signature of person completing this form	[signature]	Cell/phone number [redacted]

BWC-1108 (Rev. March 17, 2016)
C-5

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17-142990

ADDITIONAL EVIDENCE FOR C-5
APPLICATION FOR DEATH BENEFITS AND FUNERAL EXPENSES

Widow / Dependent:

Christina Marie Palumbo Date of Birth: [redacted]
Phone: [redacted]
[redacted]
Concord OH 44077

Block 3:
List of Children / Dependent Children Residing with mother, Christina M. Palumbo

	Michael Louis Palumbo III	[redacted]	[redacted]
	Marisa Marie Palumbo	[redacted]	[redacted]
Dependent	Christian Thomas Palumbo	[redacted]	[redacted]
Dependent	Samuel Marcelino Palumbo	[redacted]	[redacted]
Dependent	Nicholas Benjamin Palumbo	[redacted]	[redacted]

EXHIBITS:

- Death Certificate of Michael Louis Palumbo, Jr.
- Birth Certificate of Michael Louis Palumbo, Jr. - Born: December 26, 1967
- Social Security Card Michael Louis Palumbo, Jr. 291-64-3912

- Social Security Card Christina M. Palumbo [redacted]
- Social Security Card Christian Thomas Palumbo [redacted]
- Social Security Card Samuel Marcelino Palumbo [redacted]
- Social Security Card Nicholas Benjamin Palumbo [redacted]

- Marriage Certificate of Michael Louis Palumbo, Jr. / Christina Marie Knight - Palumbo

- Birth Certificates:

			Born:
*	Christina Marie Knight (Palumbo)		[redacted]
*	Nicholas Benjamin Palumbo	10	[redacted]
*	Samuel Marcelino Palumbo	15	[redacted]
*	Christian Thomas Palumbo	20	[redacted]
	Marisa Marie Palumbo	21	[redacted]
	Michael Louis Palumbo III	23	[redacted]

- Paid invoice from Monreal Funeral Home & Cremation Service $13,594.48
- Paid invoice from Catholic Cemeteries Association - Diocese of Cleveland $5,830.00

- 2014 W-2 statements from City of Beachwood and City of Willowick
- 2015 W-2 statements from City of Beachwood and City of Willowick

Pg 2 of 6

Ohio Department of Health - Vital Statistics

CERTIFICATE OF DEATH

State File No. 2017052228

Registrar's No. 4300 2017 000804

1. Decedent's Legal Name: MICHAEL LOUIS PALUMBO JR	2. Sex: MALE	3. Date of Death: MAY 24, 2017
4. Social Security Number: 291-64-3912	5a. Age: 49	8. Birthplace: CLEVELAND, OHIO
9a. Residence State: OHIO	9b. County: LAKE	9c. City or Town: CONCORD
9d. Street and Number: 8100 BUTLER HILL DRIVE	9f. Zipcode: 44077	9g. Inside City Limits? YES
9. Ever in US Armed Forces? NO	10. Marital Status at Time of Death: MARRIED	11. Surviving Spouse's Name: CHRISTINA KNIGHT
12. Decedent's Education: ASSOCIATE DEGREE (E.G. AA, AS)	13. Decedent of Hispanic Origin: NO	14. Decedent's Race: WHITE
15. Father's Name: MICHAEL LOUIS PALUMBO SR	16. Mother's Name: BARBARA MARKMAN	
17a. Informant's Name: CHRISTINA PALUMBO	17b. Relationship to Decedent: SPOUSE	17c. Mailing Address: 8100 BUTLER HILL DRIVE CONCORD, OHIO 44077
18a. Place of Death: DECEDENT'S HOME		
18b. Facility Name: 8100 BUTLER HILL DRIVE	18c. City or Town, State and Zip Code: CONCORD, OH 44077	18d. County of Death: LAKE
19. Funeral Service Licensee: JANET SRNICK MONREAL	20. License Number: 008425	21. Name and Complete Address of Funeral Facility: MONREAL FH 35400 CURTIS BLVD EASTLAKE, OH 44095
22a. Method of Disposition: BURIAL	22b. Date of Disposition: JUNE 01, 2017	
22c. Place of Disposition: ALL SOULS CEMETERY	22d. Location: CHARDON, OH	
23a. Signature: *Mariann Rusnak*	Date: MAY 30, 2017	
23b. Name of Person Issuing Disposition Permit: RUSNAK, MARIANN	23c. Date Disposition Permit Issued: *May 30, 2017*	
25. Time of Death: *4:57 pm*		
26. Signature and Title of Certifier: *M. Lazbin* MD	License Number: 35.066100	
27. Name and Address of Person who Completed Cause of Death: MARINA LAZBIN, 17876 ST CLAIR AVE, MENTOR, OH 44110		

28. Part I. Immediate Cause: *Glioblastoma multiforme* — Approximate Interval Between Onset and Death: *18 month*

Due to (or as Consequence of):

Due to (or as Consequence of):

Due to (or as Consequence of):

29a. Was An Autopsy Performed? ☒ Yes ☐ No

29b. Were Autopsy Findings Available Prior To Completion Of Cause of Death? ☐ YES ☐ NO ☐ Not Applicable

30. Did Tobacco Use Contribute to Death? ☐ Yes ☐ Unknown ☒ No ☐ Probably

31. If Female: Not pregnant within past year; Pregnant at time of death; Not pregnant, but pregnant within 42 days of death; Not pregnant, but pregnant 43 days to 1 year before death; Unknown if pregnant within the past year

32. Manner of Death: ☒ Natural ☐ Accident ☐ Suicide ☐ Homicide ☐ Pending Investigation ☐ Could not be determined

33a. Date of Injury (Mo/Day/Year) | 33b. Time of Injury | 33c. Place of Injury | 33d. Injury at Work?

33e. Location of Injury: (Street and Number or Rural Route Number, City or Town, State)

33f. Describe How Injury Occurred

33g. If Transportation Injury, Specify: ☐ Driver/Operator ☐ Pedestrian ☐ Passenger ☐ Other

HEA 2724 Rev.

VOID

MARIANN RUSNAK, REGISTRAR

MAY 30 2017

Mariann Rusnak

Brown 17-142990 (SZ) Williams 17-142995

Dunn Coroner

35 of 44

REBY CERTIFY THIS DOCUMENT IS AN EXACT COPY OF THE RECORD ON FILE WITH THE OHI [redacted]

Cleveland
55 Public Square, Suite 2222
Cleveland, Ohio 44113
Phone: (216) 861-0804
Fax: (216) 861-5322
Toll-free: (888) 684-0804

Columbus
2291 Scioto Harper Drive
Columbus, OH 43204
Phone: (614) 276-8959
Fax: (614) 276-9132
Toll-free: (844) 410-9442

January 18, 2019

Fax: (216) 787-5289
Industrial Commission of Ohio
Cleveland Service Office

ATTENTION: STAFF HEARING OFFICER

In re: Claimant: Michael Palumbo
Claim No: 17-142990 / City of Beachwood
17-142995 / Willowick
Hearing: Friday 1/25/2019 at 10:00 - Room 1

PRESUMPTION APPLICATION REQUEST

Dear Sir or Madam:

Attached please find the C-265 previously filed. Claimant is requesting that the presumption apply under Ohio Revised Code 4123.68(X) for the allowance hearing in both claims 17-142990 and 17-142995.

Thanking you for your cooperation, I remain,

Very truly yours,

Bradley E. Elzeer II

BEE /lmp

cc: Meghan Delaney, Esq.
Fisher & Phillips
via fax: (440) 838-8805

Lisa Gattozzi, Esq.
Dinsmore & Shohl
via fax: (216) 413-3839

PLEVIN & GALLUCCI CO., L.P.A.

The Illuminating Building, Suite 2222, 55 Public Square, Cleveland, Ohio 44113-1901
216/861-0804 Fax 216/861-5322
Toll Free 888/684-0804

FAX COVER SHEET

FAX: (216) 787-5289

DATE: December 5, 2018

TO: Industrial Commission of Ohio
Cleveland Service Office

CC: Meghan Delaney, Esq.
Fisher & Phillips
Via fax: (440) 838-8805

FROM: Bradley E. Elzeer II

RE:

Claimant:	Michael Palumbo
Claim No.:	17-142990
Employer:	City of Beachwood
Date of Hearing:	12/10/2018

MEMO: Enclosed please find the information regarding Jeremy N. Rich, MD, MHS which provides his specialties that we respectfully submit for your consideration at the above captioned hearing.

Thank you.

Total number of pages including cover: 4

Please contact this office if the total number of pages are not received.

« Search Results « Previous |

Jeremy N. Rich, MD, MHS

Neuro-oncologist

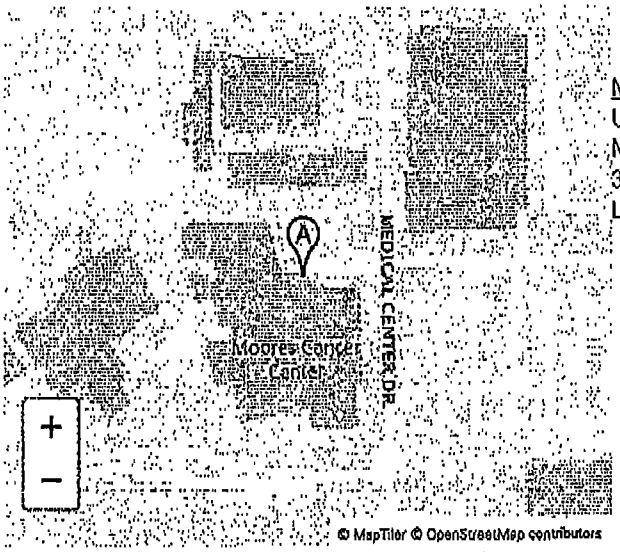
Professor of Medicine

Cancer *(Primary Specialty)* | *Neurological Oncology*
Neurology

17-142980
17-142995

Michael Palmer
DOB

Practice Locations



Moores Cancer Center
UC San Diego Health - La Jolla
Moores Cancer Center
3855 Health Sciences Drive
La Jolla, CA 92093

Contact Numbers

New Patient Registration:
(800) 926-8273

Direct Clinic Line - Moores Cancer Center:
(858) 822-6100

About Jeremy N. Rich, MD, MHS

Bio

Jeremy Rich, MD, MHS, MBA, is a certified neuro-oncologist and renowned physician-scientist who specializes in diagnosing and treating brain tumors, including gliomas (e.g. glioblastoma, astrocytoma, brainstem glioma, ependymoma, and oligodendroglioma) and brain metastases. As part of a multidisciplinary approach, Dr. Rich works closely with neurosurgeons and medical and radiation oncologists to provide comprehensive treatment plans unique to each patient.

Dr. Rich has made seminal contributions to the brain tumor field understanding how brain tumors grow and resist current treatments, through the prism of stem cell biology. He is a professor in the Department of Medicine's Division of Regenerativ Medicine, where his research focuses on developing new techniques for treating aggressive brain cancers. Dr. Rich is also active in mentoring medical students, residents and fellows at UC San Diego School of Medicine.

A recognized expert, Dr. Rich is an invited speaker at many national and international conferences and meetings. He has published over 200 peer-reviewed articles and serves on the editorial board for numerous medical journals, including *Science Translational Medicine*, *Clinical Cancer Research*, *Neuro-Oncology*, and *PLoS Biology*, among others.

Prior to joining UC San Diego Health, Dr. Rich served as chair of the Department of Stem Cell Biology and Regenerative Medicine at Cleveland Clinic and as co-director of the National Center for Regenerative Medicine at Case Western Reserve University School of Medicine.

He completed a fellowship in neuro-oncology at Duke University Medical Center (Duke University School of Medicine) and a residency in neurology at the Johns Hopkins Hospital (Johns Hopkins School of Medicine), where he was elected as chief resident. Dr. Rich earned his medical degree and a Master of Health Sciences in clinical research from Duke University Schoo of Medicine. He also holds a health care Master of Business Administration from Baldwin Wallace University in Berea, Ohio. Dr. Rich is board certified in neurology and certified in neuro-oncology from the United Council for Neurologic Subspecialties.

He is a member of many professional organizations, including the Association of American Physicians, the American Society for Clinical Investigation, the American Academy of Neurology, the Society of Neuro-Oncology, and the American Association for Cancer Research, among others.

Dr. Rich is the recipient of many awards, including the 2015 National Cancer Institute (NCI)'s Outstanding Investigator Award, which supports accomplished leaders in cancer research.

Outside of work, Dr. Rich enjoys playing soccer, running and spending time with family.

UC San Diego Health Links

For more information, see Brain Tumors and Moores Cancer Center.

Gender

Male

Date Joined Staff

Education

Fellowship
Duke University, School of Medicine, Durham, NC

Master's Degrees
Baldwin Wallace University, Berea, OH
Duke University School of Medicine, Durham, NC

Medical Degree
Duke University, School of Medicine, Durham, NC

Residency
Johns Hopkins School of Medicine, Baltimore, MD

Board Certifications
Neurology
Neuro-Oncology

Clinical Expertise Areas
Astrocytoma
Brain Metastases
Brain Tumors
Brainstem Glioma
Ependymoma
Glioblastoma
Glioma
Oligodendroglioma

Treatments and Conditions
Brain Tumors

Jeremy Rich Lab

17-142995

17-142980

Jeremy Rich

Dr. Jeremy Rich, MD, MHS, MBA is Professor of Medicine, Director of Neuro-Oncology, and Director of the Brain Tumor Institute of UC San Diego Health. Previously, he served as the inaugural Chair of Stem Cell Biology and Regenerative Medicine at Cleveland Clinic and Co-Director of the National Center for Regenerative Medicine. He has received numerous awards, including named as one of the inaugural NCI Outstanding Investigators, as well as induction into the American Society for Clinical Investigation and American Association for Physicians. His work has been published in over 200 original research reports. He serves on several editorial boards and the NCI Board of Scientific Counselors.

17-N2998
17-N2990

Jeremy Rich • 3rd

Professor at University of California San Diego; Director of Neuro-Oncology

Beachwood, Ohio

Message ...

University of California San Diego

Duke University School of Medicine

See contact info

402 connections

Experience

Professor
University of California San Diego
Jun 2017 – Present • 1 yr 2 mos

Professor
Cleveland Clinic
Sep 2008 – Jun 2017 • 8 yrs 10 mos

Department Chair
Cleveland Clinic
Sep 2008 – May 2016 • 7 yrs 9 mos

Department of Stem Cell Biology and Regenerative Medicine

Fellow
Duke University Health System
Jul 1997 – Jun 1998 • 1 yr

Neuro-Oncology

Education

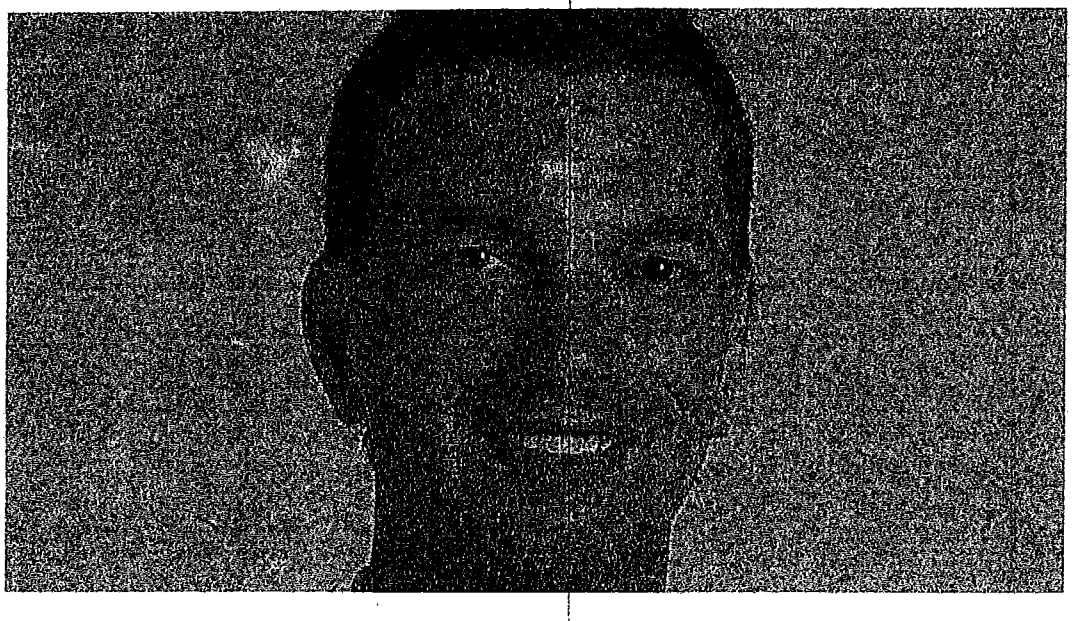
Duke University School of Medicine
Doctor of Medicine (M.D.), Medicine
1989 – 1993

Washington University in St. Louis
Bachelor's of Science, Electrical Engineering
1985 – 1989

17-14295

17-14290

UC San Diego hires renowned brain cancer expert Jeremy Rich



Cleveland Clinic brain tumor expert Dr. Jeremy Rich has been recruited by UC San Diego. (Cleveland Clinic)

By **Gary Robbins**

MAY 31, 2017, 3:40 AM

UC San Diego has recruited Dr. Jeremy Rich, a renowned Cleveland Clinic physician-scientist who grapples with the notoriously difficult problem of understanding and treating brain tumors.

Rich, 49, has been serving as chair of the Department of Stem Cell Biology and Regenerative Medicine at the Cleveland Clinic in Ohio, which is ranked among the best neuroscience centers in the U.S.

supportive tissue in the brain. The cancers include . Experts estimate that more than 12,000

Glioblastomas have killed prominent figures such as U.S. Senator Ted Kennedy, singer-actress Ethel Merman and retired baseball player Gary Carter.

17-142995

In 2015, Rich received the National Cancer Institute's Outstanding Investigator Award and was given nearly $7 million for research, some of which is expected to be transferred to his lab at UC San Diego.

17-142990

Rich said he is moving to San Diego because La Jolla is a mecca of neuroscience, especially when it comes to efforts to turn discoveries in the laboratory into new and better treatments.

"Moving the field forward requires the seamless collaboration between clinical and research arenas," Rich said. "At UCSD and partner institutions, there are some of the best researchers in the world in neuro-oncology, as well as research into the brain and cancer, more generally.

"When I came to Cleveland Clinic in 2008, I was drawn to the opportunity to build a stem cell research enterprise. Now, I am moving to UCSD to catalyze an environment that I hope will make UCSD the destination for brain cancer patients regionally, nationally and internationally."

Rich's appointment was praised by Inder Verma, a cancer biologist at the Salk Institute for Biological Studies in La Jolla, which is part of the famed life-science mesa that's anchored by UC San Diego.

"Jeremy Rich is a great addition to the community because he brings with him a wealth of experience working on the deadly cancer glioblastomas. He has made seminal contributions in our understanding of how gliomas become invasive. He brings basic science and clinical expertise," Verma said. "I am quite excited that he will be a neighbor, and we are already discussing mesa-wide meetings and collaborations on glioblastomas."

Other stories you might be interested in:

UC San Diego booming as chancellor Pradeep Khosla finishes 5th year

La Jolla institute sending experiment into space to benefit astronauts

Twitter: @grobbins

gary.robbins@sduniontribune.com

This article is related to: Biology, Scientific Research, Stem Cell Research, Medical Research, Cleveland Clinic

Cleveland Clinic

Jeremy N. Rich, M.D. M.H.S., Neuro-Oncology
Brain Tumor & Neuro-Oncology Center
Mary E. Murphy R.N., BSN
Office: 216/444-445-7269
Fax: 216/444-0924

April 26, 2017

Re: Michael L Palumbo
DOB: 12/26/1967

Diagnosis: C71.9, glioblastoma

To Whom It May Concern,

Michael Palumbo is under my care at the Cleveland Clinic. I was involved and directed Mr. Palumbo's care, treatment plan, and evaluation imaging results beginning at the time of diagnosis, October 2015, until the present time. Unfortunately, Mr. Palumbo is currently off treatment and in Hospice care. It is in my professional medical opinion that Michael's work and job related duties as a firefighter, for many years, and the exposures to carcinogens in that role, contributed to and more likely than not caused his cancer.

Jeremy N. Rich, M.D., M.H.S

July 18, 2018

Judah Friedman, M.D.
Hematology and Oncology
Clinical Assistant Professor
University Hospitals Seidman Cancer Center

32340 Woodsdale Lane
Solon Ohio, 44139
judah.friedman@uhhospitals.org

Re: Claimant: Michael Palumbo, No 17-142990, Employer: City of Beachwood.

The patient was a 47 year old man who presented to the emergency room August 30, 2015 after experiencing confusion and disorientation. While in the emergency room he experienced a grand mal seizure, and required intubation for respiratory arrest. CT and MRI demonstrated multiple right sided masses in the brain. According to the records there was some initial concern these lesions could be metastatic, but full body imaging was normal. He was treated with surgical resection. According to records, pathology revealed glioblastoma multiforme. Following surgical resection, he was treated with chemotherapy and radiation through December of 2015. Due to disease progression he was treated with several other chemotherapy drugs as well as the Optune device (external alternating electrical currents), and anti blood vessel growth drug (Avastin). From the records it appears that he was seen and treated for his condition at Cleveland Clinic, University Hospitals Cleveland, and also sought additional opinions at National Institutes of Health and at a medical center in Boston, MA. Unfortunately, due to progression of his disease and clinical decline he was placed into hospice care and ultimately passed away from glioblastoma on May 24, 2017. His past medical history was notable only for hyperlipidemia and esophageal reflux disease (GERD). He is described in the chart as a never smoker and only occasional alcohol consumption. Family history notes that his grandparents suffered from pancreatic, colon and lung cancer.

Questions for Consideration:

1.Please define the term "latency period" as it relates to cancer and medicine.

The latency period for cancer is defined as the amount of time that elapses between the initial exposure to a carcinogen and the diagnosis of cancer. The latency period can vary tremendously depending on the particular carcinogen and the the dose of the carcinogen. The period may be short, such as with exposure to high doses of a radioactive substance as in the Hiroshima and Nagasaki atomic bombs, and the development of leukemias within a few months. Generally, cancer is a multi-factorial disease, and has a long latency measured in decades; it develops as a result of an accumulation of mutations over a long period time and is regulated by the individual's response to cell damage. Individuals who inherit a predisposition to cancer often have defects in the ability to repair cell damage, as in inheriting a mutation in the BRCA1 or BRCA2 genes. There are estimated to be hundreds if not thousands of genes involved in DNA

repair that may regulate the effects of damage of carcinogens on cells (DeVita, Hellman, and Rosenberg, 9th ed. 2011).

2. Is there a latency period associated with glioblastoma?

Like most malignancies in adults, glioblastomas, occur later in life more commonly, and contains multiple genetic mutations, rearrangements, and chemical alterations (Wen et al NEJM 2008). The median age of diagnosis of glioblastoma is 64 years, with a peak incidence in the 75 to 84 year age category (Central Brain Registry US Report, 2012). Children treated with radiation for lymphomas or leukemias or atomic bomb survivors are noted to have an increased risk later in life for primary malignant tumors of the brain. The latency between radiation treatments and the development of brain tumors may be as short as five years or as long as many decades. In children exposed to ionizing radiation as part of treatment for childhood leukemia the latency period for high grade gliomas was a median of 9.1 years. Higher doses of radiation treatment are associated with both an increased risk of developing a brain tumor and a shorter latency period (Braganza MZ, et al. Neuro Oncol. 2012). Because glioblastoma is diagnosed at an older age, and because multiple, cumulative molecular defects are expected to occur over time leading to its pathogenesis, it is felt that glioblastoma has a long latency for its development, measured in decades.

3. Do you find any objective medical evidence supporting the diagnosis of glioblastoma?

Based on my review of the medical claim history, as described above, there is clear evidence the claimant suffered from a diagnosis of glioblastoma multiforme, a primary malignant tumor of the brain.

4. If the answer to Question No. 3 is yes, please explain in detail:

4.a. If there is evidence that Mr. Palumbo was exposed, outside the scope of his official duties as a firefighter for the city of Beachwood, to cigarettes, tobacco products, or other conditions presenting with an extremely high risk of the development of glioblastoma?

There is no evidence that Mr. Palumbo was exposed to ionizing radiation, either within or outside the scope of his official duties as a firefighter. Ionizing radiation is the only environmental risk factor reliably considered to increase the risk of brain tumors.

4.b. Whether or not those identified exposures and/or other conditions "probably a significant factor in the cause or progression" of the glioblastoma?

Many factors have been analyzed for their possible relationship to the development of brain tumors include trauma, medications, allergies, seizures, smoking and alcohol, and exposure to power-frequency electromagnetic fields. None of these factors, however, has been conclusively shown to be important (Wrensch M, Minn Y, Chew T, et al. Epidemiology of primary brain tumors: current concepts and review of the literature. Neuro Oncol 2002; 4: pp.278-299). Cell phone use has been extensively studied as a risk factor, but several large population studies found no evidence for causing brain tumors. Therapeutic doses of radiation are the only

environmental risk factor reliably considered to increase the risk of brain tumors. There is nothing from the medical chart to indicate a history of exposure to ionizing radiation.

There is an increased risk for primary central nervous system tumors in families with inherited cancer syndromes (familial) such as neurofibromatosis, Li-Fraumeni syndrome and Lynch syndrome as some examples. There is a family history of cancer in Mr. Palumbo's family, but this appears to be only in his grandparents generation, and not a first-degree relative. Thus there is not a strong suggestion of these syndromes in Mr. Palumbo's family.

5. If you can identify some other cause of Mr. Palumbo's condition, please identify it specifically. Please provide a detailed explanation of your opinion.

I can not identify any cause for the development of glioblastoma in Mr. Palumbo.

6. Is there competent evidence that the alleged exposure to the carcinogens identified by Mr. Palumbo (arsenic, inorganic arsenic compounds, asbestos, benzene, diesel engine exhaust, dioxin, formaldehyde, shift work that involves circadian disruption, and radio tower frequency) did not, or could not, have caused the alleged glioblastoma?

6.a. Is there competent scientific evidence linking any of the aforementioned carcinogens to the formation of glioblastoma?

Mr. Palumbo served as a firefighter for 24 years for the city of Beachwood from 1992 to 2016. The fire runs records included for review from 2000 to 2015, indicate cooking, vegetation, trash, passenger vehicle, and building fires. Given that Beachwood is not known to have a high industrial / factory - base and that most of the fire run records for Beachwood indicate vegetation, trash and passengers vehicle fires, it is difficult to claim a high exposure to industrial chemicals.

12 records 1992 - 2000 (sic)

It is widely accepted that firefighters are exposed to many potential or suspected carcinogens. Many studies reported in the scientific literature have examined the relationship between occupation of firefighters and cancer risk; some have concluded there is a modest increase risk for brain, digestive, genitourinary, and lymphoma/leukemia malignancies (LeMasters GK et al. Cancer risk among firefighters: A review and meta-analysis of 32 studies. J Occup Environ Med 2006; 48:1189-202). In addition, the International Agency for Research on Cancer (IARC), after a review of 42 studies, concluded that firefighter exposure were possibly carcinogenic to humans (Group 2A) (IARC working group on the evaluation of carcinogenic risks to humans. Painting, firefighting, and shiftwork. IARC Monogr Eval Carcinog Risks Hum 2010; 98:9-764). Daniels et al. performed the largest review of cancer risk in career firefighters, reporting on the mortality of 30,000 firefighters from 1950 through 2009, serving in fire departments in San Francisco, Chicago, and Philadelphia. Compared with the US population, they found small to moderate increases in risk for several cancer sites, and for all cancers combined, stemming mostly from excess malignancies of the respiratory, digestive, and urinary systems. There was a two fold increase in mesothelioma presumably from asbestos exposure. However, it is important to emphasize with regards to Mr. Palumbo's claim, cancer of the brain, was not increased in career firefighters in this large study (Daniels RD, et al. Occup Environ Med 2014; 71:388-397).

With regard to cancer risk of the above mentioned chemicals, many cohort studies have examined the link to cancer risk in humans. Generally speaking the link is very strong in laboratory experiments, but proving the association with cancer in humans is much more difficult and complex. This is primarily due to the fact that each individual has different response to environmental toxins and exposure levels. Exposure to arsenic has been linked to bladder, kidney, lung and digestive cancers. Benzene may increase the risk for leukemias and other blood disorders. Formaldehyde may cause leukemias and cancers of the nasopharynx. Asbestos has been linked to lung cancer and mesothelioma. Vinyl chloride exposure is associated with increased risk for liver cancer as well as leukemias and lymphomas. Dioxin, commonly known as agent orange, is a complex benzene-like compound that has been linked to multiple cancers including kidney, esophagus, multiple myeloma, lymphoma and soft tissue sarcoma (NCI.gov)

However, there is lacking any conclusive evidence that occupational exposure to industrial chemicals lead to the development of brain tumors. Some of the chemicals that can induce brain tumors in laboratory animals, such as polycyclic aromatic hydrocarbons, can do so only when administered by direct contact or transplacentally, but not by inhalation or dermal contact; the latter two modes of exposure are more relevant in the occupational setting, such as firefighting. Specific chemicals that have been examined include organic solvents, arsenic, polyvinyl chloride, pesticides, polycyclic aromatic compounds to name a few. While these can induce tumors in laboratory animals, epidemiology studies have failed to show an association in human (McLaughlin JK, and Lipworth L: A critical review of the epidemiologic literature on health effects of occupational exposure to vinyl chloride. J Epidemiol Biostat 1994; 4: pp. 253-275).

IARC has identified working a night shift as a probable human carcinogen (Lancet Oncol 2007; 8(12): 1065-1066). This classification was based on evidence from experimental animal models but limited human studies. The majority of these studies looked at breast cancer risk in female employees. 2001 study from the Fred Hutchinson Cancer Research Center study showed nurses who worked night shift on a regular basis were more likely to be diagnosed with cancer (J Natl Cancer Inst 2001; 93 (20): 1557-1562). Proposed mechanisms of action include suppression of melatonin and oxidative stress repair, disruption of circadian clock–related gene expression and loss of control of cell proliferation and apoptosis, and sleep deprivation that alters immune function. However, a review of the medical epidemiology literature could find no link between circadian rhythm disturbance and glioblastoma in humans.

6 b. Is there competent scientific evidence linking any carcinogens and/or environment risk factors to the formation of glioblastomas?

Ionizing radiation is the only environmental risk factor reliably considered to increase the risk of brain tumors. Ionizing radiation is defined as any radiation capable of caring enough energy to free electrons from atoms. It is usually made of atomic particles, such as electrons or protons, moving at high speeds or high-energy electromagnetic waves such as gamma rays or x-rays. Exposure to ionizing radiation causes damage to DNA in cells. Cumulative damage to the DNA can over time result in cancer. Although, if given at high enough doses radiation can result in cell death. Humans can be exposed to ionizing radiation either from medical radiation treatment,

and diagnostic imaging, or from nuclear radiation from nuclear power plant failures or nuclear weapons.

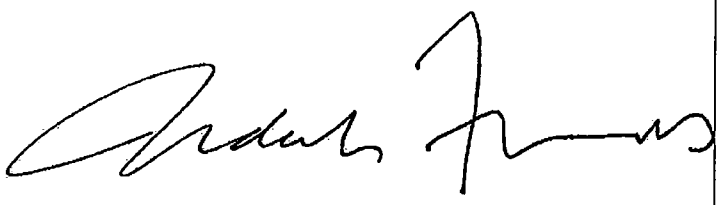
7. Within a degree of medical probability, did the nature of Mr. Palumbo's employment create an increased risk or hazard of contacting glioblastoma multiforme in a greater degree and in a different manner than the general public?

Because Mr. Palumbo was not exposed to ionizing radiation during his duties as a firefighter, and that the above mentioned alleged exposures are not proven to cause or contribute to glioblastoma, there is no degree of medical probability that his employment as a firefighter increased his risk or hazard of contracting glioblastoma more so than the general public.

8. Within a degree of medical probability did Mr. Palumbo's glioblastoma develop as a result of the alleged exposure to carcinogens through his duties as a firefighter.

There is no degree of medical probability that Mr. Palumbo's glioblastoma developed from any exposures through his official duties as a firefighter.

Thank you kindly for your consideration



Judah Friedman, MD

Mark T. Finneran, M.D., FAADEP

Medical Disability Evaluations

July 25, 2018

Attorney John Saccoccia
Dinsmore & Shohl, LLP
191 West Nationwide Boulevard, Suite 300
Columbus, Ohio 43215

Re:	Michael L. Palumbo, Jr.
Claim#:	17-142995
DOB:	12/26/1967
Date of Death:	May 24, 2017
Date of Diagnosis:	October 2015
Cause of Death:	Glioblastoma multiforme, WHO grade IV
Employers:	City of Willowick, City of Beachwood
Occupation:	Firefighter

Dear Attorney Saccoccia:

At your request, I performed a medical record review on Mr. Michael L. Palumbo, Jr. In rendering my opinion, I reviewed each and every one of the approximately 1500 pages of medical records contained in the two binders accompanying your letter. I received reviewed and discuss three documents submitted by claimant's counsel Bradley E. Elzeer, II to the Industrial Commission on July 18, 2018. I accept the objective medical evidence provided by the examining physicians and limited license practitioners, however, not necessarily their conclusions. I have not provided care for Mr. Palumbo and I do not have a doctor patient relationship with this claimant. I am an independent medical examiner and I have formulated my opinions and diagnoses independent of referral source, remuneration, others opinions or personal bias.

History: Mr. Michael L. Palumbo, Jr. worked as a firefighter since January 01, 1990. On Sunday, August 30, 2015, Mr. Palumbo developed blurred vision. As he drove to church that day, he drove left of center. After church, he and his family went to a park and Mr. Palumbo forgot to take off his seatbelt. While in the park, Mr. Palumbo walked into a tree. Emergency medical services were called and Mr. Palumbo was transported to TriPoint Hospital. He went into a generalized tonic clonic seizure and cardiopulmonary arrest. He was intubated, stabilized and a preliminary evaluation was completed.

An MRI of the brain revealed two rounded enhancing masses within the right parietal lobe, the first measuring 1.1 cm in diameter with peripheral enhancement. The second lesion measured 0.7 cm with increased T1 signal suggesting calcium and blood products. There was adjacent cortical thickening with increased T2/flair signal and mild sulcal effacement in the right parietal lobe.

Mr. Palumbo was life-flighted to the Cleveland Clinic Foundation where he underwent brain surgery and a biopsy was collected. Histological analysis revealed a WHO grade IV glioblastoma with small cell features. Microscopically, the tumor showed hypercellularity with atypical infiltrating cells containing hyperchromatic and angulated elongated nuclei. Gemistocytic cells were abundant. Perinuclear clearing with round cell morphology was observed and active mitotic figures were present. There was microvascular proliferation and pseudopalisading necrosis. Immunohistochemistry revealed p53 strong nuclear positivity at 5% with a Ki-67 proliferative index up to 50%.

Mr. Palumbo was thereafter treated in the usual fashion. He underwent three open surgical procedures followed by standard chemotherapy and radiation therapy. Mr. Palumbo expired on May 24, 2017.

A First Report of Injury was completed on May 05, 2017 and a statement of causation was provided by Jeremy Rich, M.D. from the Cleveland Clinic. The diagnosis on the FROI is listed as glioblastoma. The causal relationship boxes were left blank.

Dr. Rich authored a brief note dated April 26, 2017. He states that he was involved in and directed Mr. Palumbo's care from the time of diagnosis in October 2015. He states "in my professional medical opinion Michael's work and job-related duties as a firefighter, for many years, and the exposures to carcinogens in that role, contributed to and more likely than not caused his cancer." Dr. Rich does not identify the carcinogenic exposures that caused Mr. Palumbo's Glioblastoma Multiforme. Neither does he identify the specific carcinogen responsible for the development of Mr. Palumbo's Glioblastoma Multiforme.

A C-265 Presumption of Causation for Firefighter Cancer was completed by the claimant's wife on May 02, 2017. This document notes Mr. Palumbo's last date of service was in July 2016. Exposure information included: arsenic and inorganic arsenic compounds; asbestos; benzene; diesel engine exhaust; dioxin; formaldehyde; shift work; and radio tower frequency electromagnetic fields. These eight exposures are the agents allegedly responsible for Mr. Palumbo's Glioblastoma Multiforme.

In the medical records is an Infectious Exposure Form for Mr. Palumbo. On September 26, 2014, Mr. Palumbo was working as a captain for the City of Beachwood. While he was on the roof of a building, walking to a penthouse elevator to remove a victim from the elevator, Mr. Palumbo "noticed a warning sign that said do not pass." He had already walked past that point to exit the roof before seeing the signs. There is no further information as to what was on the roof of this building.

Contained in the medical records are fire run sheet comparisons from the City of Beachwood and the City of Willowick for run dates going back to June 07, 2002 for the City of Willowick. The last run occurred on July 24, 2015 for the City of Beachwood. Mr. Palumbo responded to 102 fires for the City of Beachwood during the course of his employment. He responded to 65 fires for the City of Willowick.

Discussion: Glioblastoma Multiforme (GBM) is a type of malignant brain tumor that is idiopathic and GBM has no known cause. Glial cells in the Glioblastoma are the support cells of the central nervous system. They surround and support the 5 billion neurons in the brain as they make 10 trillion synaptic connections. There are two primary types of glial cells and these are microglia, or "small cells" and astroglia, or "star-shaped" cells.

A Glioblastoma Multiforme develops spontaneously when, for unknown reasons, repressor genes are turned off and the glial cells begin to replicate (undergo mitosis). It is impossible to know precisely when this spontaneous genetic anomaly takes place because of the hydraulic nature of the human brain. The human brain is well protected inside the bony calvarium or skull, and it is bathed on the inside and outside by a sea-water-like solution called cerebrospinal fluid (CSF).

Approximately 500 ml, 17 ounces, of CSF is manufactured every day from the choroid plexus located in the lateral ventricles deep within the brain. The cerebrospinal fluid circulates through the lateral ventricles, the third ventricle, the fourth ventricle and out of the ventricles at the base of the brain to bathe the surface of the brain and the entirety of the spinal cord. When a brain tumor develops, depending upon its rate of growth, the increase in intracranial tissue is accommodated by a reduction in the amount of circulating cerebrospinal fluid. However, because the cerebrospinal fluid is essential to the metabolism and normal function of the billions of nerves in the brain, only so much CSF loss can be clinically tolerated. When that threshold is exceeded, and when the growing tumor disrupts critical neural circuits, individuals become clinically symptomatic. Such was the case with Mr. Palumbo.

By August 30, 2015, when Mr. Palumbo clinically deteriorated and developed generalized tonic clonic seizures with respiratory failure, he had reached that critical threshold. An MRI performed on that date revealed two well established lesions in his right parietal lobe, measuring 1.1 cm and 0.7 cm respectively. These tumors were surrounded by vasogenic edema and the second tumor contained central blood products. Looking at the tumor under the microscope tells us that this tumor was aggressive, made so by its polymorphonuclear mitotic activity, hence the name Multiforme.

Unlike other cancers with known etiologies and caused by an exposure to toxins, a Glioblastoma Multiforme is unique in that it has a multitude of different cell types that makes the tumor nearly impossible to cure. When one tumor-cell type is destroyed, another tumor-cell type resumes the uncontrolled growth. Glioblastoma Multiforme is malignant and 99.9% of individuals diagnosed with the condition die within two years of the date of diagnosis. Glioblastoma Multiforme has no known cause. Glioblastoma Multiforme cannot be experimentally induced with any agent, compound, substance or exposure in any living organism. Glioblastoma Multiforme is completely idiopathic.

In the C-256 application eight putative causative exposures were checked. These included:

1. Arsenic: Arsenic and inorganic arsenical compounds are not known to cause Glioblastoma Multiforme (GBM).
2. Asbestos: Asbestos is known to cause mesothelioma of the lung. Asbestos is not known to cause GBM.
3. Benzene: Benzene is not known to cause GBM.

4. Diesel engine exhaust: Diesel engine exhaust is not known to cause GBM.
5. Dioxin: Dioxin is not known to cause GBM.
6. Formaldehyde: Formaldehyde is not known to cause GBM.
7. Shift work: Shift work and circadian disruption is not known to cause GBM.
8. Radio tower frequency, electromagnetic fields: There is no evidence to support the contention that radio tower frequency or electromagnetic fields cause GBM.

Glioblastoma Multiforme cannot be experimentally induced, it has no known cause, it is completely idiopathic.

Discussion of Medical Literature: On July 18, 2018 claimant's counsel Bradley E. Elzeer, II submitted three documents to the Industrial Commission in anticipation of a Hearing on July 30, 2018 at 1:30 – Room 10. These documents include a copy of the November 2006 article in the Journal of Occupational and Environmental Medicine, Volume 48, Number 11, by Grace K. LeMasters, et. al. entitled; Cancer Risk Among Firefighters: A Review and Meta-Analysis of 32 studies. Nowhere in the LeMaster study is there any evidence whatsoever that any exposure of any type will increase the hazard of, or produce an increased risk of, developing a malignant Glioblastoma Multiforme brain tumor.

Two PowerPoint presentations were also included. The first is entitled Fire Service Occupational Cancer Symposium, presented by Robert D. Daniels, MD on September 7-8, 2017 in Phoenix Arizona. Nowhere in Dr. Daniels' PowerPoint is there any evidence whatsoever that any exposure of any type will increase the hazard of, or produce an increased risk of, developing a malignant Glioblastoma Multiforme brain tumor.

The second PowerPoint presentation is also from the Fire Service Occupational Cancer Symposium on September 7-8, 2017 in Phoenix Arizona and was presented by Kenneth Fent, PhD. Nowhere in Dr. Fent's PowerPoint is there any evidence whatsoever that any exposure of any type will increase the hazard of, or produce an increased risk of, developing a malignant Glioblastoma Multiforme brain tumor.

A final pertinent paper, not found in claimant's counsel's offering, is the original article by Robert D. Daniels, et. al., published in OEM online and entitled; "Mortality and cancer incidence in a cohort of US firefighters from San Francisco, Chicago and Philadelphia (1950-2009)" on September 23, 2013. Nowhere in Dr. Daniels' original paper is there any evidence whatsoever that any exposure of any type will increase the hazard of, or produce an increased risk of, developing a Glioblastoma Multiforme brain tumor.

On July 25, 2018 this author performed a review of the world literature on the etiology of Glioblastoma Multiforme. It remains a matter of fact that Glioblastoma Multiforme has no known cause. Glioblastoma Multiforme cannot be made to develop in any organism by any agent or exposure. Glioblastoma Multiforme is entirely idiopathic.

Conclusions: All questions are answered in my professional opinion within a reasonable degree of medical certainty, based on the available medical records and the scientific literature.

1. Based on your review of the records provided, what was the cause of Mr. Palumbo's death?

Mr. Michael L. Palumbo, Jr. developed an idiopathic malignant Glioblastoma Multiforme, World Health Organization Grade IV, and this tumor presented itself on August 30, 2015. A histopathological diagnosis was made in October 2015. The date of onset of this tumor is unknown and the date of onset is impossible to predict with standard radiographic imaging, historical and/or histological techniques.

2. In your medical opinion, was the cause of death in any way related to Mr. Palumbo's employment at the City of Willowick?

Glioblastoma Multiforme has no known cause, it cannot be induced in the laboratory or made to develop in any organism by any agent or exposure and it is entirely idiopathic. Mr. Palumbo's fatal Glioblastoma Multiforme had no relationship to his employment at the City of Willowick or any of his alleged exposures at work including exposures to arsenic, asbestos, benzene, diesel engine exhaust, dioxin, formaldehyde, shift work, radiofrequency or electromagnetic fields, under any theory of causation.

3. In your medical opinion, did the nature of Mr. Palumbo's employment at the City of Willowick create an increased hazard of contracting Glioblastoma Multiforme different from employment generally?

Mr. Palumbo's employment at the City of Willowick did not create an increased hazard of contracting Glioblastoma Multiforme different from employment generally. Glioblastoma Multiforme has no known cause, it cannot be induced in the laboratory or made to develop in any organism by any agent or exposure and it is entirely idiopathic. There are no known agents or exposures of any sort that will increase the hazard of contracting Glioblastoma Multiforme for any human being in any occupation.

4. In your medical opinion, did Mr. Palumbo's employment at the City of Willowick create a risk of contracting Glioblastoma Multiforme in greater degree and in a different manner than the general public?

Mr. Palumbo's employment at the City of Willowick did not create a risk of contracting Glioblastoma Multiforme in greater degree or in a different manner than the general public. Glioblastoma multiforme has no known cause, it cannot be induced in the laboratory, it cannot be made to develop in any organism by any agent or exposure, and it is entirely idiopathic. There are no known agents or exposures of any sort that will create a risk of contracting a Glioblastoma Multiforme in greater degree and in a different manner than the general public.

5. Mrs. Palumbo simply alleged, in general terms, exposure to "Arsenic, Asbestos, Benzene, Diesel engine exhaust, Dioxin, Formaldehyde, Shiftwork that involves circadian disruption, Radiofrequency or electromagnetic fields (Please refer to the C-265 form.)

    a. Do you find sufficient evidence, according to your review of the file, that Mr. Palumbo was exposed to the alleged agents?

The medical records do not provide us with objective evidence of an exposure to any of these eight alleged agents. Jeremy Rich, M.D. authored a brief note dated April 26, 2017 wherein he states that he was involved in and directed Mr. Palumbo's care from the time of diagnosis in October 2015. He states "in my professional medical opinion Michael's work and job-related duties as a firefighter, for many years, and the exposures to carcinogens in that role, contributed to and more likely than not caused his cancer." Dr. Rich does not identify the alleged exposures that caused Mr. Palumbo's Glioblastoma Multiforme. Neither does he identify the carcinogenic agent that was responsible for the development of Mr. Palumbo's Glioblastoma Multiforme. On the claimant's First Report of Injury the diagnosis is listed as "glioblastoma." The causal relationship boxes were left blank.

    b. Please explain in detail whether the alleged exposures would or would not be expected to cause Mr. Palumbo's Glioblastoma Multiforme?

There is no known dose-response relationship between any of the eight alleged exposures and the development of a Glioblastoma Multiforme. A review of the world literature performed on July 25, 2018 searching for a single known cause, or multiple known causes, for the etiology of Glioblastoma Multiforme tells us once again that GBM has no known cause. Glioblastoma Multiforme cannot be made to develop in any organism of any type by any agent or agents or any exposure or exposures. Glioblastoma Multiforme is entirely idiopathic.

Even if Mr. Palumbo were exposed to any, or all, of these eight alleged causative agents, none of the alleged agents, singly or in combination, are known to cause Glioblastoma Multiforme. There is no known dose-response relationship between any of these alleged agents and the development of a Glioblastoma Multiforme. That is, even if Mr. Palumbo were exposed to all of the alleged agents repeatedly, that exposure, and those exposures, would not have been sufficient to cause Mr. Palumbo's Glioblastoma Multiforme. Glioblastoma Multiforme is completely idiopathic, it has no known cause.

If you have any questions regarding this review, or my conclusions, please do not hesitate to contact me.

Respectfully submitted,

M.D.

Mark T. Finneran, M.D., FAADEP
MTF\Pts

Physician Review

Customer service office Toledo	Claim number 17-142995
Injured worker's name Michael L. Palumbo Jr.	Date of injury 04-26-17
Allowed conditions: Alleged Conditions: GLIOBLASTOMA	
The attached claim is being referred to you on 10-26-18 by Mary Beth C. RN (phone: 419-245-2420).	
Question(s) to be addressed: (1) Please explain in detail, within reasonable medical probability, was the injured worker's death on 05-24-17 a direct or proximate result of his occupation? (2) If not, what was the cause of the injured worker's demise? Please state your answer in detail and within a reasonable medical probability. (3) Is there sufficient evidence to establish that exposure to heat, smoke, toxic gasses, chemical fumes , and other toxic substances were the proximate cause of the glioblastoma? In other words, is there evidence that the documented exposure to these substances caused the condition have occurred?	

Physician's narrative

Analysis: Michael Palumbo, Jr. was a 47 year-old male Firefighter for the City of Willowick Fire Department when on 8/30/15 he noted onset of some blurred vision. While driving to church that day he went left of center, and later that afternoon he was with his family at a city park when he walked into a tree and became confused. He was transported to TriPoint Hospital by EMS, and subsequently suffered a generalized seizure with cardiopulmonary arrest. He was resuscitated and stabilized. An MRI of the brain demonstrated masses in the right parietal lobe. Mr. Palumbo was LifeFlighted to the Cleveland Clinic. The evaluation there included a surgical biopsy which revealed a Grade IV Glioglastoma with small cell features. He was subsequently treated with a combination of surgery, radiation, and chemotherapy. Unfortunately, his clinical status gradually and progressively declined. He was admitted into Hospice Care in the Spring of 2017, and expired on 5/24/2017. Mr. Palumbo was only 49 years-old at the time of his untimely death. A FROI, C-265, and request for death benefits were subsequently filed. In a letter of support of causation dated 4/26/17 Dr. Jeremy Rich, who supervised the medical management of Mr. Palumbo's brain cancer, stated that "It is my professional medical opinion that Michael's work and job related duties as a firefighter for many years, and the exposures to carcinogens in that role, contributed to and more likely than not caused his cancer." Dr. Rich provided no medical evidence in support of his opinion (such as Scientific papers, expert consensus opinions, etc.).

Discussion:

An Independent Medical Review was performed by Dr. Mark Finneran on 7/25/18. In his excellent, detailed report Dr. Finneran discussed in significant detail the circumstances of Mr. Palumbo's diagnosis and treatment. Dr. Finneran also responded to Dr. Rich's aforementioned letter, as well as a C-265 Presumption of Causation for FireFighter Cancer completed on 5/2/17 by Mr. Palumbo's wife. Dr. Finneran pointed out that none of the potential carcinogens to which Mr. Palumbo would possibly have been exposed to during the course of performing his duties as a FireFighter have been demonstrated to have any causative relationship to the development of Glioblastomas. He documented that Dr. Rich did not provide any medical evidence, or identify any specific carcinogen to which Mr. Palumbo was exposed to that has any evidence-based support for causality in the literature. Dr. Finneran also discussed the fact that Glioblastomas are very malignant and uniformly fatal. He further documented that none of the carcinogenic toxic materials listed on the C- 265 are known to cause Glioblastoma Multiforme. He summarized by making the important point that "Glioblastoma Multiforme cannot be experimentally induced, it has no known cause, and it is completely idiopathic." Dr. Finneran concluded that the cause of death was Glioblastoma Multiforme, and that this condition "had no relationship to his (Mr. Palumbo's) employment at the City of Willowick or any of his alleged exposures at work..." On 8/31/18 an interlocutory order confirmed that Mr. Palumbo's widow had withdrawn the C-265 Presumption of Causation for FireFighter Cancer, as well as "any request to have her C-5 application for Death Benefits allowed as a result of this presumption." Given these facts, the body of evidence on file clearly supports that Mr. Palumbo died of Gliolastoma Multiforme, as well as the fact

that there was no causal relationship between his employment as a FireFighter for the City of Willowick and his death due to Glioblastoma Multiforme on 5/24/17.
Conclusion: My conclusions, based on my review of the medical evidence on file, and within the realm of reasonable medical probability, are as follows: (1) Michael Palumbo, Jr.'s death on 5/24/2017 was not causally related to his occupation as a FireFighter for the City of Willowick, Ohio (reference above "Discussion" section of report). (2) The cause of Mr. Palumbo's death on 5/24/2017 was Glioblastoma Multiforme. (3) There is not sufficient evidence to establish that exposure to heat, smoke, toxic gases, chemical fumes, and other toxic substances were the proximate cause of Mr. Palumbo's Glioblastoma Multiforme (reference above "Discussion" section of report). In fact, there is no evidence in the file to support the conclusion that the documented exposure to these substances caused Mr. Palumbo's condition of Glioblastoma Multiforme to occur. Thank you for this very interesting referral. Respectfully submitted,
Physician name (please print or type) Rohn T. Kennington, M.D.

Physician's signature	Date	Time
	11/1/2018	120 minutes

Sarah
ER Dr -
No experience
in Glioblastoma

FIRE RUN COMPARISON - Beachwood and Willowick

Description	Type Code	# of incidents – Beachwood	Last	# of incidents – Willowick	Last
Structure fire	NA	1	7/15/00	0	0
Building fire	111	16	12/22/14	25	5/2/15
Fires in structure other than building	112	2	5/29/07	3	10/6/08
Cooking fire, confined to container	113	27	6/16/15	11	5/24/13
Fuel burner/boiler malfunction	116	1	3/28/10	2	10/30/12
Trash/rubbish, contained	118	4	12/18/13	1	6/7/02
Fire in mobile home used as fixed residence	121	1	2/6/10	1	3/20/14
Passenger vehicle fire	131	16	5/28/15	2	12/17/13
Natural vegetation fire, other	140	9	4/7/13	5	4/23/15
Brush and grass mixture fire	142	12	5/19/12	4	9/5/13
Grass fire	143	3	7/23/10	1	5/9/06
Outside rubbish, trash or waste fire	151	3	1/14/14	5	5/2/13
Dumpster or other outside trash receptacle fire	154	1	10/26/09	3	1/14/15
Special outside fire, other	160	1	4/14/06	2	8/4/09
Road freight or transportation vehicle fire	NA	2	7/24/15	0	0
Off-road vehicle/heavy equipment fire	NA	1	5/14/13	0	0
Outside equipment	NA	1	9/4/11	0	0
Special outside fire, including mulch fires	160	1	4/17/06	0	0
TOTAL:		**102**		**65**	

17-142990

SAMPLE MISSING (1992-7/14/00) 8 yrs.

Beachwood Fire & Rescue

Structure Fire Runs
(01/01/2000 through 12/31/2015)

Regarding: **Michael Palumbo, Jr.**

Incident Type	Date	Time	Hours
Structure Fire	07/15/00	14:18	0.57
Building Fire	01/27/01	03:24	3.37
Building Fire	02/20/01	03:06	1.05
Building Fire	07/01/01	11:36	2.17
Building Fire	04/04/07	12:38	2.85
Building Fire	10/23/07	05:01	0
Building Fire	01/14/08	22:26	2.78
Building Fire	05/30/10	10:51:06	1.05
Building Fire	11/09/11	22:09:41	0.79
Building Fire	05/28/12	18:40:47	1.22
Building Fire	07/15/12	14:44:04	0.62
Building Fire	01/01/13	15:28:22	1.11
Building Fire	02/22/13	08:00:13	1.56
Building Fire	04/07/13	13:49:40	1.93
Building Fire	05/31/13	20:57:59	2.56
Building Fire	10/29/14	15:33:07	2.19
Building Fire	12/22/14	21:15:46	1.76
Fires in Structures Other Than in a Building	08/18/03	18:47	1.71
Fires in Structures Other Than in a Building	05/29/07	02:43	2.7
Cooking Fire, Confined to Container	01/05/01	13:10	0.28
Cooking Fire, Confined to Container	09/02/01	23:56	0.33
Cooking Fire, Confined to Container	09/14/01	13:50	0.28
Cooking Fire, Confined to Container	05/22/02	21:10	0.54
Cooking Fire, Confined to Container	07/20/02	12:06	0.56
Cooking Fire, Confined to Container	01/17/03	19:01	0.75
Cooking Fire, Confined to Container	07/07/03	20:46	0.78
Cooking Fire, Confined to Container	04/12/04	17:20	0.3
Cooking Fire, Confined to Container	12/17/04	15:11	0.35
Cooking Fire, Confined to Container	04/01/07	17:54	0.12
Cooking Fire, Confined to Container	09/08/07	15:24	0.71
Cooking Fire, Confined to Container	03/20/08	09:11	0.73
Cooking Fire, Confined to Container	01/21/09	05:21:29	0.23
Cooking Fire, Confined to Container	10/17/09	18:00:00	0.08
Cooking Fire, Confined to Container	11/10/09	17:25:07	0.06
Cooking Fire, Confined to Container	01/21/10	17:23:00	0.32
Cooking Fire, Confined to Container	06/17/10	16:14:00	0.67

Cooking Fire, Confined to Container	03/11/11	12:26:40	0.13
Cooking Fire, Confined to Container	06/18/11	14:37:57	0.36
Cooking Fire, Confined to Container	09/03/12	12:04:30	0.27
Cooking Fire, Confined to Container	05/21/13	18:41:01	0.28
Cooking Fire, Confined to Container	11/29/13	12:15:27	0.25
Cooking Fire, Confined to Container	12/27/13	21:51:12	0.48
Cooking Fire, Confined to Container	02/04/14	11:18:21	0.43
Cooking Fire, Confined to Container	05/23/14	20:16:49	0.53
Cooking Fire, Confined to Container	09/20/14	12:34:20	0.74
Cooking Fire, Confined to Container	06/16/15	12:04:44	0.43
Fuel Burner / Boiler Malfunction, Fire Confined	03/28/10	22:10:50	0.4
Trash / Rubbish Fire in a Structure No Flame Damage	05/19/07	11:13	0.39
Trash / Rubbish Fire in a Structure No Flame Damage	11/18/07	20:55	0.32
Trash / Rubbish Fire in a Structure No Flame Damage	07/13/08	05:08	1.5
Trash / Rubbish Fire in a Structure No Flame Damage	12/18/13	21:14:20	0.16

Beachwood Fire & Rescue

Fire – Mobile Property (Vehicle) Fire Runs
(01/01/2000 through 12/31/2015)

Regarding: **Michael Palumbo, Jr.**

Incident Type	Date	Time	Hours
Mobile Property (Vehicle) Fire	01/28/03	17:56	0.44
Mobile Property (Vehicle) Fire	02/06/10	01:26:31	0.51
Passenger Vehicle Fire	01/09/02	17:42	0.63
Passenger Vehicle Fire	02/26/02	21:18	0.38
Passenger Vehicle Fire	07/09/03	16:03	0.35
Passenger Vehicle Fire	09/23/04	06:54	0.41
Passenger Vehicle Fire	09/17/06	12:14	0.47
Passenger Vehicle Fire	08/02/07	21:34	0.29
Passenger Vehicle Fire	12/18/07	17:30	0.44
Passenger Vehicle Fire	03/17/08	12:52	0.14
Passenger Vehicle Fire	05/25/08	13:56	0.34
Passenger Vehicle Fire	12/12/08	15:01:34	0.06
Passenger Vehicle Fire	09/03/10	13:38:59	0.25
Passenger Vehicle Fire	01/13/11	21:53:50	0.22
Passenger Vehicle Fire	06/14/12	08:20:28	0.33
Passenger Vehicle Fire	07/15/13	16:55:46	0.07
Passenger Vehicle Fire	09/22/13	17:10:07	0.56
Passenger Vehicle Fire	05/28/15	08:17:27	0.38
Road Freight or Transport Vehicle Fire	12/17/03	16:09	0.33
Road Freight or Transport Vehicle Fire	07/24/15	21:01:53	0.12
Off-road Vehicle or Heavy Equipment Fire	05/14/13	02:25:52	2.27

Beachwood Fire & Rescue

Fire – Natural Vegetation Fire Runs
(01/01/2000 through 12/31/2015)

Regarding: **Michael Palumbo, Jr.**

Incident Type	Date	Time	Hours
Natural Vegetation Fire	04/01/00	11:54	0.57
Natural Vegetation Fire	07/03/00	20:56	0.78
Natural Vegetation Fire	06/24/07	15:38	0.1
Natural Vegetation Fire	06/27/07	13:03	0.22
Natural Vegetation Fire	06/09/11	19:14:16	0.14
Natural Vegetation Fire	06/27/11	18:41:20	0.08
Natural Vegetation Fire	07/09/11	22:15:58	0.12
Natural Vegetation Fire	07/15/11	16:59:09	0.09
Natural Vegetation Fire	04/07/13	10:34:41	0.81
Brush or Brush-Grass Mixture Fire	04/23/01	17:11	0.13
Brush or Brush-Grass Mixture Fire	04/24/01	00:34	0.17
Brush or Brush-Grass Mixture Fire	07/03/02	20:30	0.17
Brush or Brush-Grass Mixture Fire	05/07/07	17:57	0.14
Brush or Brush-Grass Mixture Fire	08/23/08	14:58	0.1
Brush or Brush-Grass Mixture Fire	04/12/10	18:01:40	0.24
Brush or Brush-Grass Mixture Fire	04/15/10	16:01:44	0.21
Brush or Brush-Grass Mixture Fire	05/30/10	09:59:42	0.09
Brush or Brush-Grass Mixture Fire	07/02/10	13:01:44	0.1
Brush or Brush-Grass Mixture Fire	09/09/10	14:12:56	0.27
Brush or Brush-Grass Mixture Fire	05/04/12	21:39:17	0.34
Brush or Brush-Grass Mixture Fire	05/19/12	18:00:18	0.35
Grass Fire	07/18/08	16:28	0.08
Grass Fire	03/21/09	17:02:59	0.15
Grass Fire	07/23/10	13:35:11	0.09

Beachwood Fire & Rescue

Fire – Outside Rubbish Fire Runs
(01/01/2000 through 12/31/2015)

Regarding: **Michael Palumbo, Jr.**

Incident Type	Date	Time	Hours
Outside Rubbish / Trash / Waste Fire, Inc Brush Piles	09/12/04	16:58	0.1
Outside Rubbish / Trash / Waste Fire, Inc Brush Piles	12/09/08	12:28:58	0.07
Outside Rubbish / Trash / Waste Fire, Inc Brush Piles	01/14/14	18:22:38	0.12
Dumpster or Other Outside Trash Receptacle Fire	10/26/09	09:28:47	0.92

Beachwood Fire & Rescue

Fire – Special Outside Fire Runs
(01/01/2000 through 12/31/2015)

Regarding: **Michael Palumbo, Jr.**

Incident Type	Date	Time	Hours
Special Outside Fire, Inc Mulch Fires	04/17/06	08:02	0.28
Outside Equipment Fire	09/04/11	15:00:44	0.31

City of Willowick Fire Department

Structure Fire Runs
(01/01/2000 through 12/31/2015)

Some Missing Runs (1980-2000) Hours

Regarding: Michael Palumbo, Jr.

Incident Type	Date	Time	Hours
Building fire	4/28/01	NA	0.4
Building fire	3/21/02	03:00	3.5
Building fire	7/22/02	13:30	0.48
Building fire	11/25/04	11:40	1.32
Building fire	12/28/04	01:16	2.77
Building fire	7/13/05	19:20	0.54
Building fire	12/2/05	16:31	0.48
Building fire	1/12/06	11:31	1.57
Building fire	3/17/06	01:08	1.33
Building fire	11/14/06	21:36	1.65
Building fire	11/18/06	05:01	3.98
Building fire	11/27/06	12:42	1.09
Building fire	11/7/07	08:57	0.83
Building fire	10/13/09	10:35:21	1.08
Building fire	6/16/10	17:39:00	2.82
Building fire	10/14/10	06:55:28	0.69
Building fire	6/13/12	03:24:59	0.88
Building fire	10/11/12	11:12:02	8.34
Building fire	12/21/12	19:04:07	0.89
Building fire	6/26/13	00:49:02	0.64
Building fire	8/12/13	21:24:00	0.54
Building fire	12/10/13	17:57:41	2.7
Building fire	5/28/14	00:54:50	1.22
Building fire	8/10/14	20:56:34	0.61
Building fire	5/2/15	18:21:19	1.8
Fires in structure other than in a building	5/9/07	09:34	0.45
Fires in structure other than in a building	11/17/07	01:02	0.44
Fires in structure other than in a building	10/6/08	08:22:00	0.58
Cooking fire, confined to container	1/26/04	19:41	0.37
Cooking fire, confined to container	2/17/04	16:26	0.35
Cooking fire, confined to container	6/30/06	08:37	0.34
Cooking fire, confined to container	10/12/06	16:11	0.72
Cooking fire, confined to container	5/4/07	16:35	0
Cooking fire, confined to container	11/7/07	19:42	0.26
Cooking fire, confined to container	10/18/08	21:39:02	0.2
Cooking fire, confined to container	11/9/10	18:57:00	0.25

Cooking fire, confined to container	8/3/11	20:15:14	0.16
Cooking fire, confined to container	3/31/12	17:58:24	0.27
Cooking fire, confined to container	5/24/13	02:12:34	0.42
Fuel burner/boiler malfunction, fire confined	1/27/03	11:46	0.38
Fuel burner/boiler malfunction, fire confined	10/30/12	00:34:56	0.36
Trash or rubbish fire, contained	6/7/02	22:58	2.12

City of Willowick Fire Department

Fire – Mobile Property (Vehicle) Fire Runs
(01/01/2000 through 12/31/2015)

Regarding: Michael Palumbo, Jr.

Incident Type	Date	Time	Hours
Fire in mobile home used as fixed residence	3/20/14	18:44:07	1.67
Passenger vehicle fire	4/15/11	11:40:39	0.22
Passenger vehicle fire	12/17/13	08:39:58	0.4

City of Willowick Fire Department

Fire – Natural Vegetation Fire Runs
(01/01/2000 through 12/31/2015)

Regarding: Michael Palumbo, Jr.

Incident Type	Date	Time	Hours
Natural vegetation fire, Other	11/10/04	11:13	0.12
Natural vegetation fire, Other	6/2/05	12:58	0.1
Natural vegetation fire, Other	11/28/05	22:06	0.38
Natural vegetation fire, Other	4/19/06	15:06	0.37
Natural vegetation fire, Other	4/23/15	11:40:43	0.14
Brush or brush-and-grass mixture fire	6/12/01	10:44	0.23
Brush or brush-and-grass mixture fire	6/28/04	07:07	0.25
Brush or brush-and-grass mixture fire	5/19/09	10:02:20	0.24
Brush or brush-and-grass mixture fire	9/5/13	18:12:52	0.18
Grass fire	5/9/06	13:41	0.22

City of Willowick Fire Department

Fire – Outside Rubbish Fire Runs
(01/01/2000 through 12/31/2015)

Regarding: Michael Palumbo, Jr.

Incident Type	Date	Time	Hours
Outside rubbish, trash or waste fire	4/19/01	10:10	0.42
Outside rubbish, trash or waste fire	5/14/02	02:05	0.17
Outside rubbish, trash or waste fire	6/18/10	19:38:54	0.13
Outside rubbish, trash or waste fire	5/21/12	11:22:39	0.11
Outside rubbish, trash or waste fire	5/2/13	01:58:13	0.24
Dumpster or other outside trash receptacle fire	4/16/08	11:08	0.35
Dumpster or other outside trash receptacle fire	1/2/15	00:50:13	0.22
Dumpster or other outside trash receptacle fire	1/14/15	03:06:56	0.23

City of Willowick Fire Department

Fire – Special Outside Fire Runs
(01/01/2000 through 12/31/2015)

Regarding: Michael Palumbo, Jr.

Incident Type	Date	Time	Hours
Special outside fire, Other	8/18/08	21:30	1.1
Special outside fire, Other	8/4/09	21:32:00	1.97

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FAX COVER SHEET

DATE: **January 23, 2019** **FAX: (216) 787-5289**

TO: **Industrial Commission of Ohio**
Cleveland Service Office

CC: **Lisa Gattozzi, Esq.**
Dinsmore & Shohl
Via fax: (216) 413-3839

FROM: **Bradley E. Elzeer II**

RE:
Claimant: **Michael Palumbo**
Claim No.: **17-142995 - Willowick**
Date of Hearing: **1/25/2019 hearings - Room 1**

MEMO: **Enclosed please find the study relative to potential risk factors for Glioblastoma multiforme which we respectfully submit for your consideration at the above captioned hearing.**

Thank you.

Total number of pages including cover: 14

Please contact this office if the total number of pages are not received.

17-142990
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SAME / SEE P. 14

Potential risk factors for incident glioblastoma multiforme: the Honolulu Heart Program and Honolulu-Asia Aging Study

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Abstract

Glioblastoma multiforme (GBM) is the most common adult primary malignant brain tumor. Ninety percent of adult GBM patients die within 24 months after diagnosis. The etiology of GBM is unknown. The Honolulu Heart Program (HHP) and Honolulu-Asia Aging Study (HAAS) are prospective, cohort studies of cardiovascular and neurodegenerative disease based on 8,006 Japanese–American men followed since 1965. The Japan Hawaii Cancer Study provides data on incident cancer cases in the HHP/HAAS cohort. We used data from these studies to obtain epidemiologic information about GBM. GBM cases were identified by searching the 1965–1998 databases using International Classification of Diseases (ICD-9) codes. Nine histologically confirmed GBM cases, 58–80 years old, were identified. The incidence rate was 6.2/100,000 person–years. Records of each case were reviewed. Selected variables from the first three examinations (1965–1968; 1968–1970; 1971–1974) were used to identify potential candidate GBM risk factors. A multivariate Cox proportional hazards model showed sugar intake and occupational exposure to carbon tetrachloride were independently and significantly associated with development of GBM.

Keywords: Keywords Brain tumor, Cancer, Epidemiology, Glioma, Risk factors

Introduction

Glioblastoma multiforme (GBM) is the most common adult primary malignant brain tumor with a peak incidence between 55 and 84 years of age [1]. Fifty percent of adult GBM patients die within 10–12 months after diagnosis. Approximately 10 % of adult patients survive 24 months after diagnosis [2]. The causes of most glioblastomas are unknown. Established risk factors include inherited genetic syndromes and therapeutic ionizing radiation but account for only a small fraction of cases [3].

Analytic epidemiologic studies of glioblastoma compare the risk of developing GBM in persons with and without certain characteristics (cohort studies) or compare the histories of persons with and without GBM (case–control studies) to identify possible risk factors including lifestyle habits, environmental

and occupational exposures to toxic agents, genetic factors and infections. Cohort studies are preferred because they ensure exposure occurs before disease develops and permit risk estimates based on exposure status. Accrual of statistically robust numbers of cases for a cohort study of GBM risk factors, however, is time consuming and difficult because the tumor is relatively rare. Most epidemiologic studies of GBM are case–control studies. Accrual of adequate numbers of cases is quicker in these studies. Interpretation of data from case control studies is more difficult because of potential problems such as recall bias, difficulty in selecting an unbiased control group, and lack of adequate exposure information.

The Honolulu Heart Program (HHP) is a prospective cohort study of cardiovascular disease based on the island of Oahu, Hawaii that began in 1965 with enrollment of 8,006 men of Japanese ancestry living on the island of Oahu, Hawaii. Research on neurodegenerative diseases of aging began in 1991 with establishment of the Honolulu- Asia Aging Study (HAAS). Data on incident cancer cases in the cohort is provided by the Japan Hawaii Cancer Study. We used the HHP/HAAS database to investigate the incidence of GBM in this population and identify potential GBM risk factors.

Materials and methods

Study population

The HHP/HAAS is a cohort study of cardiovascular disease and aging among 8,006 men of Japanese ancestry who were born between 1900 and 1919 and were 45–68 years of age at the time of their initial examination between 1965 and 1968. The cohort was identified through selective service records from World War II, and men were located through searches of telephone, business, and state agency records. Of the estimated 14,426 men born in 1900–1919 and believed to be residents of Oahu, 11,148 were located; 8,006 completed the baseline examination [4]. Thus the study population represents over 70 % of the target population.

Glioblastoma multiforme

Since the initial examination (1965–1968), the cohort has been followed through a series of six follow-up examinations performed in 1968–1970; 1971–1974; 1991–1993; 1994–1996; 1997–1999; and 1999–2000. In addition to the periodic examinations the cohort is followed through rigorous surveillance of hospital admissions and records, obituaries in local newspapers, and death certificates. Deaths are ascertained by daily review of newspaper obituaries, telephone calls to relatives, mortuaries, hospitals, and the medical examiner's office and searches of the National Death Index (NDI). The Japan-Hawaii Cancer Study provides information on incident cancer cases in the cohort through surveillance of hospital discharge records on Oahu and through links with the Hawaii Tumor Registry for cases in other parts of the state. Information on participants living on the mainland is obtained from local relatives, interviews during island visits of participants, and the NDI, Out-migration from Oahu has been less than one per 1,000 per year and only five participants have been lost to follow-up. The final cause of death is determined by an expert panel of three physicians based on all available information obtained from follow-up examinations, surveillance of hospital discharge records, autopsy reports, and death certificates. Protocol autopsies are obtained on approximately 25 % of cohort deaths.

All primary and secondary CNS tumors were identified through searching the entire cohort database for the years 1965–1998 using International Classification of Diseases (ICD 9) codes for oncology. The original records of each of these cases were reviewed by one of the authors (JSN) to identify cases of GBM.

Risk factors

Selected variables from the first three examinations (1965–1968; 1968–1970; and 1971–1974) were used to identify potential candidate GBM risk factors. These examinations included questionnaires that provided basic demographic, occupational and socioeconomic data, medical history, and lifestyle factors including usual physical activity, smoking habits, alcohol intake, and dietary habits [5]. Specific questions that were available included whether they ever had chest surgery, any blood transfusion and recurrent herpes. Other procedures included an electrocardiogram, grip strength assessment, pulmonary function testing, blood measurements including hematocrit, glucose (1-h post 50-gram load), cholesterol, and triglycerides, seated blood pressure determination, and various anthropometric measures such as height, weight, and subscapular and triceps skinfold thickness. Self-reported weight gain from age 25 was also ascertained. Separate evaluation of each man's diet was carried out by a dietician, using the 24-hour recall method [6]. A common food grouping system was used to estimate nutrient composition. Dietary data were also validated by repeat 24 h recall interviews and 7-day dietary records in a subset of 329 men examined 2 years later [7]..

Occupational exposure data were available from the entire cohort based on information collected at the first and third examinations. Participants were asked what their present and usual jobs were and how many years they worked in these jobs at both examinations. Their jobs were assigned two three-digit occupation and industry codes according to the U.S. Bureau of the Census [8]. All unique occupation/industry combinations were identified and independently assessed by three industrial hygienists for the likelihood of exposure to pesticides (insecticides, herbicides and fungicides), metals (manganese, mercury and iron) and solvents (carbon tetrachloride and carbon disulfide) [9, 10]. Likelihood of exposure was assigned by consensus as none, low, medium, and high. An intensity score was calculated by multiplying the likelihood of exposure by the number of years worked. Usual occupation was used in these analyses.

Statistical analysis

Follow-up for incident GBM among the 8,006 men occurred over the approximate 30-year period from the time of study initiation through the end of 1998. Incidence of GBM was calculated using person–years of follow-up. Participants were considered at risk of developing GBM from the date of their initial examination until onset of this disease for cases and date of death, date lost to follow-up or the end of the follow-up period for non-cases. Corresponding 95 % confidence interval (CI) for incidence of GBM was computed assuming the number of cases (events) follows a Poisson distribution. Data analysis included comparing mean values of selected continuous variables for GBM cases and non-cases; comparing percentages of participants with a specified variable in GBM cases and non-cases by levels of selected categorical variables; and computing GBM incidence rates and associated 95 % CIs by categories of selected variables. The Wilcoxon rank sum test was used to compare the distribution of continuous variables between GBM cases and non-cases, while the Fisher's exact test was used to compare categorical variables. Due to small sample size (GBM cases) exact *P* values were preferred. Instead the Monte Carlo method was used to obtain an empirical *P* value that approximates the exact *P* value without relying on asymptotic distributional theory or exhaustive enumeration. The GENMOD procedure in SAS was used to estimate incidence rates of glioblastoma and associated 95 % CI by assuming a Poisson distribution and specifying person–years at risk as the offset. Exact logistic regression analysis relating GBM to each potential risk factor was performed to obtain exact p-values which were later used to assess the linear trend in incidence rates of GBM across categories of the predictor variables [11]. A multivariate analysis to estimate hazard ratio (HR) associated with each risk factor was performed using the Cox proportional hazards regression model.

Results

GBM incidence

Nine participants, 58–80 years of age at diagnosis (mean age = 66.8 years), developed GBM during the follow-up period between 1974 and 1995. All cases were confirmed by histological examination. Average age at death was 67.6 years for GBM cases and 77.6 years for non-GBM cases. All tumors were supratentorial. The incident GBM cases occurred during 144,405 person–years of follow-up, yielding an overall incidence rate of 6.2 per 100,000 person–years (95 % CI = 2.8–11.8).

Risk factors

Comparisons of participants who developed GBM and did not develop GBM revealed several characteristics that differed significantly or were of borderline significance (Table 1). Participants developing GBM were significantly more educated and more likely to have a usual job that involved high carbon tetrachloride exposure. Although differences were of borderline statistical significance, compared with participants who did not develop GBM, those who developed GBM gained twice as much weight since age 25 (10.6 vs. 20.9 lbs), had larger triceps skinfold thickness (8.0 vs. 10.0 mm), consumed a greater percentage of calories from carbohydrate (46.4 vs. 53.3 %) and a 1.5-fold greater amount of sugar in the diet (45.2 vs. 70.0 g), and reported more frequent chest surgery (0.9 vs. 11.1 %) and blood transfusion (13.7 vs. 37.5 %), respectively. No significant differences were observed for age, body mass index, physical activity index, recurrent herpes labialis, smoking, total calories, coffee and tea consumption, and level of solvent exposure at one's usual job. In addition, no differences were found for pesticide or metal exposure at work (data not shown).

Table 1

Characteristics of incident glioblastoma multiforme cases and non-cases, 1965–1974

Characteristics[a]	GBM cases ($n = 9$)	Non-cases ($n = 7997$)	P value[b]
Age, years	52.8 (2.9)	54.5 (5.6)	0.584
Education, %			0.038
<High school	33.3	51.0	
High school	22.2	34.7	
Technical	22.2	4.0	
University	22.2	10.4	
Weight gain from age 25, lbs	20.9 (20.6)	10.6 (17.8)	0.096
Body mass index, kg/m^2	24.7 (2.9)	23.8 (3.1)	0.341
Triceps skinfold, mm	10.0 (3.8)	8.0 (3.4)	0.084
Physical activity index	311.4 (31.1)	328.1 (45.4)	0.286
Current smoking, %	22.2	43.8	0.315
Total calories	2301.1 (736.1)	2273.6 (738.9)	0.934
Calories from carbohydrate, %	53.3 (13.8)	46.4 (11.0)	0.081
Sugar, g	70.0 (47.5)	45.2 (36.8)	0.097
Coffee-4 oz	3.0 (3.0)	3.4 (3.2)	0.770
Tea-4 oz	2.4 (3.4)	1.8 (2.3)	0.895
Chest surgery, %	11.1	0.9	0.080
Blood transfusion, %	37.5	13.7	0.085
Recurrent herpes, %	12.5	16.0	1.000
Usual job included solvents, %			0.300
None	55.6	48.6	
Low	11.1	30.6	
Medium	0.0	6.4	
High	33.3	14.5	
Usual job included carbon tetrachloride, %			0.008
None	77.8	74.5	
Low	0.0	22.9	
Medium	11.1	1.9	
High	11.1	0.8	

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Note Values are means (SD) or percentages

[a]Assessed at Exam I (1965–1968) except for the following: chest surgery and blood transfusion (Exam II, 1968–1970), recurrent herpes (Exam III, 1971–1974), and occupational exposure (Exam I, 1965–1968 and Exam III, 1971–1974)

[b]Based on Monte Carlo estimates for the Wilcoxon rank sum test for continuous variables and Fisher's exact test for categorical variables

Incidence rates of GBM are presented in Table 2 across levels of categorical variables and across tertiles of continuous variables that reached significant or borderline significant levels. Although tests for trend tended not to be significant for most of the continuous variables, incidence rates increased with increasing tertiles of triceps skinfold and percentage of calories from carbohydrate, decreased with increasing coffee consumption, or were highest in the highest tertile of weight gain from age 25 and dietary intake of sugar. Incidence rates tended to be higher in those who had a technical or university education. Although the number of participants was limited, incidence rates were significantly elevated in those who had chest surgery, a blood transfusion and a usual job that involved exposure to medium or high levels of carbon tetrachloride. Results using the composite intensity score based on level and duration of usual job exposure to carbon tetrachloride were similar (data not shown).

Table 2

Incidence rates of glioblastoma by levels of selected characteristics, 1965–1998

Characteristic[a]	Level	No. of cases/ no. of subjects at risk	Person-years at risk	Rate per 100,000 person-years	95% CI	P value[b]
Education	<High school	3/4,077	97,172	3.1	0.6–9.0	0.137
	High school	2/2,773	70,526	2.8	0.3–10.2	
	Tech or Univ	4/1,153	29,414	13.6	3.7–34.8	
Wt gain from age 25, lbs	−81–2	2/2,613	60,859	3.3	0.4–11.9	0.192
	3–18	2/2,737	69,166	2.9	0.4–10.5	
	19–98	5/2,565	65,099	7.7	2.5–17.9	
Triceps, mm	1–6	1/2,896	70,789	1.4	0.04–7.9	0.073
	7–8	3/2,240	55,907	5.4	1.1–15.7	
	9–32	5/2,865	70,454	7.1	2.3–16.5	
Calories from CHO, %	0–41.8	1/2,662	65,686	1.5	0.04–8.5	0.055
	41.9–51.0	3/2,683	66,772	4.5	0.9–13.1	
	51.1–96.9	5/2,656	64,595	7.7	2.5–18.0	
Sugar, g	0–23.9	2/2,645	61,882	3.2	0.4–11.7	0.043
	24.0–53.2	2/2,693	67,167	3.0	0.4–10.8	
	53.3–337.1	5/2,668	68,151	7.3	2.4–17.1	
Coffee-4 oz	0	2/1,287	31,936	6.3	1.57–25.04	0.721
	1–3	5/3,733	90,926	5.5	0.27–113.3	
	≥4	2/2,986	74,337	2.7	0.09–76.37	
Tea-4 oz	0	5/3,770	92,081	5.4	2.3–13.0	0.430
	1–3	2/2,502	61,459	3.3	0.26–40.29	
	≥4	2/1,734	43,659	4.6	0.37–56.73	

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[a]Assessed at Exam I (1965–1968) except for the following: chest surgery and blood transfusion (Exam II, 1968–1970), and occupational exposure (Exam I, 1965–1968 and Exam III, 1971–1974)
[b]Tests for linear trend in incidence rates for continuous variables and differences in incidence rates for dichotomous variables using exact logistic regression

Results from a multivariate Cox proportional hazards model are shown in Table 3. Age, education, triceps skinfold thickness, coffee and tea consumption, chest surgery, and having a blood transfusion were not independently associated with development of GBM. Dietary sugar intake and a usual job that involving high carbon tetrachloride exposure were independently associated with risk of GBM. A 10 g increase in dietary intake of sugar was associated with a 15 % increase in the risk of GBM (HR = 1.15, 95 % CI = 1.00–1.32) after adjusting for other variables in the model. Having a job with high carbon tetrachloride exposure was also independently associated with a significantly elevated risk of developing GBM. For the latter potential risk factor, wide confidence intervals reflect some instability.

Table 3

Hazard ratios for risk of glioblastoma, 1965–1998

Potential risk factor	Hazard ratio[a]	95 % CI	*P* value
Age, years	0.55	0.11–2.69	0.459
Education, %			
<High school	1.00		
High school	0.78	0.13–4.76	0.786
Tech or university	2.62	0.49–13.90	0.258
Triceps skinfold, mm	2.26	0.36–14.11	0.384
Sugar, g	1.15	1.00–1.32	0.046
Coffee-4 oz			
0	1.00		
1–3	1.83	0.21–16.25	0.589
≥4	0.89	0.08–10.02	0.924
Tea-4 oz			
0	1.00		
1–3	0.79	0.14–4.47	0.789
≥4	1.21	0.22–6.76	0.827
Chest surgery, %	8.60	0.86–85.92	0.067
Blood transfusion, %	3.79	0.80–18.06	0.094
Usual job included carbon tetrachloride, %			
None	1.00		
Low or medium	0.62	0.07–5.44	0.669
High	26.59	2.90–243.50	0.004

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[a]Hazard ratios and 95 % CIs are determined from a multivariate Cox proportional hazards model and are based on a 10-unit change in the continuous independent variables

Discussion

Based on US Central Brain Tumor Registry data, glioblastomas account for 18.5 % of reported brain tumors. They represent approximately 1.42 % of all primary malignant cancers expected to be diagnosed in the United States in 2007. The US age-adjusted incidence rate for all reported glioblastomas is 3.09 cases per 100,000 person–years [1]. GBM incidence is higher in males than in females, 3.94 versus 2.38 [1, 3]. The incidence rate for GBM in this study of 6.2 per 100,000 person–years is lower than that reported for US men and women in the 65–74 year age group (12.47 per 100,000 person–years) [1, 12, 13]. Comparable data from Japan are limited. One study reports an age-adjusted incidence rate of 2.8/100,000 person–years for Japanese men over the age of 70 years [14]. The mean age at diagnosis was 67 years in this study which was similar but slightly older than the average of 64 years reported for men and women combined in the US [1].

The only established risk factors for glioblastoma are ionizing radiation and certain inherited genetic disorders [3]. A number of other possibilities have been explored including, but not limited to, occupational exposures, infectious agents, medications, head trauma, blood transfusion, surgery, anesthesia, and lifestyle factors such as diet, however, the evidence for their role as GBM risk factors is inconclusive [3, 12, 13, 15, 16, 17].

Although this study has a relatively small number of incident GBM cases and resultant statistical power is low, results indicate that at least two variables are worthy of further investigation, intensity of carbon tetrachloride exposure and dietary levels of glucose. A usual job with medium or high exposure to carbon tetrachloride was independently associated with development of GBM; examples of such occupations included dry cleaners, firemen, chemists, machinists, and radio/TV repairmen. The association of carbon tetrachloride and other chlorinated aliphatic hydrocarbons has been reported previously [18]. An epidemiologic investigation showed elevated brain cancer mortality was associated with cumulative exposure to chlorinated hydrocarbons [19]. Dietary factors have been associated with cured meat consumption and low intake of foods high in vitamin C [20, 21]. Consumption of caffeinated beverages, especially coffee and tea, has been associated with a reduced risk of glioma [22, 23]. Although we observed a slight inverse trend of decreasing incidence with increasing coffee intake, neither coffee nor tea consumption was independently associated with GBM incidence. The biological basis for a possible association of high levels of dietary glucose with GBM risk is uncertain. Glucose and other reducing sugars in foods react with asparagine and other amino acids also in foods during cooking especially at temperatures above 120 °C to form acrylamide [24, 25]. Female rats given acrylamide in the drinking water have an increased incidence of tumors including CNS tumors [26]. On the basis of animal studies acrylamide is considered by the WHO as a probable human carcinogen. The link between acrylamide and human cancer, however, is unproven and the oncogenic risk of dietary acrylamide is controversial [27].

Although some studies have reported associations between infectious agents and GBM [28, 29], we did not observe an association with self-reported recurrent herpes infection. We observed an elevated incidence of GBM in those who reported a blood transfusion which could be linked with an increased likelihood of exposure to infectious agents, diagnostic radiation or exposure to anesthesia in a hospital setting. However, this association with blood transfusion was not independent of other potential risk factors. Although chest surgery was associated with borderline statistical significance in the current study and others have suggested this could be related to halogenated hydrocarbons found in anesthesia [12, 30, 31] chest surgery was relatively rare in this population (0.8 %) and associated GBM confidence intervals for the incidence rates were therefore imprecise.

Limitations of this study include the small number of participants who developed GBM and the resultant instability of hazard ratio estimates for some risk factors. Adjustment for multiple comparisons was not made given that the objective was to generate hypotheses. Some misclassification

of occupational exposure is possible due to changes in a job over time; however, there was a high degree of concordance (89–99 %) between job related exposure estimates available at the first and third examinations. If exposure misclassification were present, it is likely this would have been non-differential and thus would have minimized the associations observed. Competing risks for mortality may have made it more difficult to identify potential risk factors. In addition, since this study was not initially designed to identify risk factors for this cancer, risk factors that could be important may not have been ascertained.

Strengths of this study include its prospective design, the large sample size of at risk individuals, histologic confirmation of all GBM cases, and population-based investigation of a relatively homogeneous sample. Ascertainment of all cases of GBM was enhanced because of the comprehensive surveillance, review of detailed hospital records, tumor registry reports, and autopsy reports, and assessment of underlying cause of death. Baseline risk factors were measured before onset of clinical disease providing estimates of risk based on exposure status that would not be influenced by recall bias.

Conclusion

In this long-term prospective epidemiologic study of Japanese–American men the incidence rate of glioblastoma multiforme was 6.2 per 100,000 person–years. Dietary intake of sugar and having a usual job with medium or high carbon tetrachloride exposure were identified as factors independently associated with its development.

Acknowledgments

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Footnotes

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References

1. CBTRUS. Statistical report: primary brain tumors in the United States. Chicago: The Central Brain Tumor Registry of the United States; 2008.

2. Laws ER, Parney IF, Huang W, Anderson F, Morris AM, Asher A, Lillehei KO, Bernstein M, Brem H, Sloan A, Berger MS, Chang S. Survival following surgery and prognostic factors for recently diagnosed malignant glioma: data from the Glioma Outcomes Project. J Neurosurg. 2003;99:467–473. [PubMed]

3. Wrensch M, Minn Y, Chew T, Bondy, Berger MS. Epidemiology of primary brain tumors: current concepts and review of the literature. Neuro-Oncology. 2002;4:278–299. [PMC free article] [PubMed]

4. Worth RM, Kagan A. Ascertainment of men of Japanese ancestry in Hawaii through World War II selective service registration. J Chronic Dis. 1970;23:389–397. [PubMed]

5. Yano K, Reed DM, McGee DL. Ten-year incidence of coronary heart disease in the Honolulu Heart Program: relationship to biologic and lifestyle characteristics. Am J Epidemiol. 1984;119:653–666. [PubMed]

6. Tillotson JL, Kato H, Nichaman MZ, Miller DC, Gay ML, Johnson KG, Rhoads GG. Epidemiology of coronary heart disease and stroke in Japanese men living in Japan, Hawaii, and California: methodology for comparison of diet. Am J Clin Nutr. 1973;26:177–184. [PubMed]

7. McGee D, Roads G, Hankin J, Yano K, Tillotson J. Within-person variability of nutrient intake in a group of Hawaiian men of Japanese ancestry. Am J Clin Nutr. 1982;36:657–663. [PubMed]

8. U.S. Bureau of the Census. Washington, DC: US Government Printing Office; 1971. Alphabetical Index of Industries and Occupations. 1970 Census of Population.

9. Abbott RD, Ross GW, White LR, Sanderson WT, Burchfiel CM, Kashon M, Sharp DS, Masaki KH, Curb JD, Petrovitch H. Environmental, life-style, and physical precursors of clinical Parkinson's disease: recent findings from the Honolulu-Asia Aging Study. J Neurol. 2003;250(Suppl 3):30–39. [PubMed]

10. Charles LE, Burchfiel CM, Fekedulegn D, Kashon ML, Ross GW, Petrovitch H, Sanderson WT. Occupational exposures and movement abnormalities among Japanese–American men: the Honolulu-Asia Aging Study (HAAS) Neuroepidemiol. 2006;26:130–139. [PubMed]

11. Mehta CR, Patel NR. Exact logistic regression: theory and examples. Statist Med. 1995;14:2143–2160. [PubMed]

12. Inskip PD, Linet MS, Heineman EF. Etiology of brain tumors in adults. Epidemiol Rev. 1995;17:382–414. [PubMed]

13. Wrensch M, Minn Y, Chew T, Bondy M. Intracranial neoplasms. In: Nelson LM, Tanner CM, Van Den Eeden SK, McGuire VM, editors. Neuroepidemiology: from principles to practice. New York: Oxford University Press; 2004.

14. Kuratsu J, Ushio Y. Epidemiological study of primary intracranial tumours in elderly people. J Neurol Neurosurg Psychiatry. 1997;63:116–118. [PMC free article] [PubMed]

15. Savitz D, Trichopoulos D. Brain cancer. In: Adami H-O, Hunter D, Trichopoulos D, editors. Textbook of cancer epidemiology. New York: Oxford University Press; 2002.

16. Thomas TL, Waxweiler RJ. Brain tumors and occupational risk factors. Scand J Work Environ Health. 1986;12:1–15.

17. Krishnan G, Felini M, Carozza SE, Miike R, Chew T, Wrensch M. Occupation and adult gliomas in the San Francisco Bay Area. J Occup Environ Med. 2003;45:639–647. [PubMed]

18. Heineman EF, Cocco P, Gomez MR, Dosemeci M, Stewart PA, Hayes RB, Zahm SH, Thomas TL, Blair A. Occupational exposure to chlorinated aliphatic hydrocarbons and risk of astrocytic brain cancer. Am J Ind Med. 1994;26:155–169. [PubMed]

19. Park RM, Silverstein MA, Green MA, Miner FE. Brain cancer mortality at a manufacturer of aerospace electrochemical systems. Am J Ind Med. 1990;17:537–552. [PubMed]

20. Lee M, Wrensch M, Miike R. Dietary and tobacco risk factors for adult onset glioma in the San Francisco Bay Area (California, USA) Cancer Causes Control. 1997;8:13–24. [PubMed]

21. Blowers L, Preston-Martin S, Mack WJ. Dietary and other lifestyle factors of women with brain gliomas in Los Angeles County (California, USA) Cancer Causes Control. 1997;8:5–12. [PubMed]

22. Michaud DS, Gallo V, Schlehofer B, Tjønneland A, Olsen A, Overvad K, et al. Coffee and tea intake and risk of brain tumors in the European Prospective Investigation into Cancer and Nutrition (EPIC) cohort study. Am J Clin Nutr. 2010;92:1145–1150. [PubMed]

23. Holick CN, Smith SG, Giovannucci E, Michaud DS. Coffee, tea, caffeine intake, and risk of adult glioma in three prospective cohort studies. Cancer Epidemiol Biomarkers Prev. 2010;19:39–47. [PMC free article] [PubMed]

24. Mottram DS, Wedzicha BL, Dodson AT. Acrylamide is formed in the Maillard reaction. Nature. 2002;419:448–449. [PubMed]

25. Stadler RH, Blank I, Varga N, Robert F, Hau J, Guy PA, Robert MC, Riediker S. Acrylamide from Maillard reaction products. Nature. 2002;419:449–450. [PubMed]

26. Johnson KA, Gorzinski SJ, Bodner KM, Campbell RA, Wolf CH, Friedman MA, Mast RW. Chronic toxicity and oncogenicity study on acrylamide incorporated in the drinking water of Fischer 344 rats. Toxicol Appl Pharmacol. 1986;85:154–168. [PubMed]

27. Mitka M. Fear of frying. Is acrylamide in foods a cancer risk. JAMA. 2002;288:2105–2106. [PubMed]

28. Wrensch M, Weinberg A, Wiencke J, Miike R, Barger G, Kelsey K. Prevalence of antibodies to four herpesviruses among adults with glioma and controls. Am J Epidemiol. 2001;154:161–165. [PubMed]

29. Wrensch M, Weinberg A, Wiencke J, Miike R, Sison J, Wiemels J, Barger G, DeLorenze G, Aldape K, Kelsey K. History of chickenpox and shingles and prevalence of antibodies to varicella-zoster virus and three other herpes viruses among adults with glioma and controls. Am J Epidemiol. 2005;161:929–938. [PubMed]

30. McCredie M, Little J, Cotton S, Mueller B, Peris-Bonet R, Choi NW, Cordier S, Filippini G, Holly EA, Modan B, Arslan A, Preston-Martin S. SEARCH international case-control study of childhood brain tumours: role of index pregnancy and birth, and mother's reproductive history. Paediatr Perinat Epidemiol. 1999;13:325–341. [PubMed]

31. Thomas TL, Stolley PD, Stemhagen A, Fontham ET, Bleecker ML, Stewart PA, Hoover RN. Brain tumor mortality risk among men with electrical and electronics jobs: a case-control study. JNCI. 1987;79:233–238. [PubMed]

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RE:
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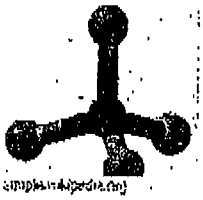

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History and synthesis · Properties · Uses · Historic uses

ATSDR - Toxic Substances - Carbon Tetrachloride
https://www.atsdr.cdc.gov/substances/toxsubstance.asp?toxid=35 ▾
Summary: Carbon tetrachloride is a manufactured chemical that does not occur naturally. It is a clear liquid with a sweet smell that can be detected at low levels. It is also called carbon chloride, methane tetrachloride, perchloromethane, tetrachloroethane, or benziform.

ATSDR - Public Health Statement: Carbon Tetrachloride
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Carbon tetrachloride is a clear liquid that evaporates very easily. Most carbon tetrachloride that escapes to the environment is found as a gas in the atmosphere.

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Chemical compound

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Formula: CCl_4
IUPAC ID: Tetrachloromethane
Molar mass: 153.82 g/mol
Boiling point: 170.1°F (76.72°C)
Melting point: -9.256°F (-22.92°C)
Soluble in: Chloroform, Benzene, Carbon disulfi acid

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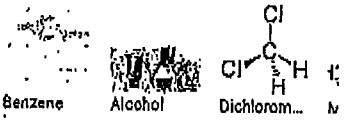

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carbon tetrachloride

n.

A poisonous, nonflammable, colorless liquid, CCl_4, used in fire extinguishers and as a dry-cleaning fluid.

carbon tetrachloride

n

(Elements & Compounds) a colourless volatile nonflammable sparingly soluble liquid made from chlorine and carbon disulphide; tetrachloromethane. It is used as a solvent, cleaning fluid, and insecticide. Formula: CCl_4

car'bon tetrachlo'ride

n.

a colorless, nonflammable, vaporous, toxic liquid, CCl_4, used mainly as a refrigerant, fire extinguisher, cleaning fluid, solvent, and insecticide.

[1900–05]

Thesaurus

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≡ **carbon tet, perchloromethane, tetrachloromethane**

↔ **dissolvent, dissolver, dissolving agent, resolvent, solvent** - a liquid substance capable of dissolving other substances; "the solvent does not change its state in forming a solution"