Parrillo, Jeffrey M. (VACO)

From: (b) (6)
Sent: Wednesday, October 15, 2014 9:12 AM
To: VHA CO CLCW SME
Cc: (b) (6) (b) (6) (b) (6) (b) (6) (b) (6)
Subject: CLCW contamination time frame
Attachments: CampLejeuneVALetter011713

There has been ongoing questions regarding the time frame for the contamination of the water supply at Camp Lejeune.

The attached letter from the ATSDR, dated January 2013 provides updated information on the contaminated water supply at Camp Lejeune. This letter had previously been circulated and can be found in the shared documents on the SharePoint site.

Based on the water modeling from the ATSDR, the water modeling projected that the water supply at CL exceed current recommended levels as early as August 1953.

Please also be advised that any statement on the 2507 regarding any of the science of CLCW is only informational and SHOULD NOT be used in our determinations. The definitive information regarding the science and toxicology of the chemical found in the water at Camp Lejeune is found in the SharePoint site and what is found in the current medical literature as well as what the ATSDR has published. As Independent Medical Examiners, it is our duty to provide an independent review without bias from the legal arbitrators of the final rating decision. The 2507 states the legal contention file by the claimant, it verifies that claimant has meet the legal bar to proceed with the IME and provided the requisite "burden of proof" to be used during the review. The scientific review is the sole responsibility of the SME reviewer.

If there are any questions, please give me a call.

We continue to do very important work. As a group, and under direction of the SecVA, we have set a high bar for quality and completeness of the medical legal evaluation of cases generated by exposure to CLCW. We need to remain committed to these standards as we continue to be watch closely. As the case load grows we need to explore any efficiencies we can find, however, timeliness without quality is a hollow" victory."

This project is one that I am proud of, and that pride comes from your hard work and dedication!

Thanks.

(b) (6)

Parrillo, Jeffrey M. (VACO)

From: (b) (6)
Sent: Friday, September 12, 2014 6:05 PM
To: (b) (6); (b) (6); (b) (6); (b) (6); (b) (6); (b) (6); (b) (6)
Cc: (b) (6); (b) (6); (b) (6); (b) (6); (b) (6); (b) (6); (b) (6)
Subject: CLCW training

I want to thank you for stepping up to the challenges inherent in providing Advisory Medical Opinions for veterans who have served at Camp Lejeune!

You're in sites and probing questions were not only welcomed but necessary for this program to continue to mature. You are not alone as you proceed through cases. Please feel free to call and of the SME for questions. We have the monthly call for SME which is also a chance to seek any help you may find valuable.

We have added some of the templates to the SharePoint site and will continue to build out library.

Thanks again!!!

(b) (6)

(b) (6)

Parrillo, Jeffrey M. (VACO)

From: (b) (6)
Sent: Monday, June 16, 2014 10:17 PM
To: VHA CO CLCW SME
Subject: cml 6.1.2014
Attachments: cml 6.1.2014.docx

Fyi CML

Disease Specific Discussion, Chronic Lymphocytic leukemia (CML)

Disease description:

Adult Leukemia is divided into four categories based the appearance of the cells and the type of cells which make up the leukemia. The appearance is acute or chronic and the types of cells are lymphocytic or myelogenous. Therefore, the four adult leukemia are acute lymphocytic leukemia (ALL), chronic Lymphocytic leukemia (CLL), acute myelogenous leukemia (AML) and chronic myelogenous leukemia (CML).

Incidence:

Cause/Risk Factors:

Cause of CLL is unknown.
Risk factors include

- Age, more common in people over 60 years of age, unusual before 45 years of age, median age of onset 70 years of age
- Caucasian race compared to other races
- Family history of CLL
- Male have a greater risk than women, 1.7:1

NB--CLL is included in the list of disease presumed to be related to AO per the Department of Veteran Affairs.

Scientific Review:

NRC 2008 report:

The water supply at Camp Lejeune was contaminated with benzene, vinyl chloride, tetracholoethylene (PERC) and trichloroethylene (TCE). The NRC report found "inadequate/insufficient evidence to determine whether an association exists" between TCE/PCE exposures to Camp Lejeune contaminated water supply "adult leukemia"; and limited association with solvent mixtures with adult leukemia.

Environmental Exposure literature Review:

Occupational Exposure Literature Review:

NRC 2008 report:

The water supply at Camp Lejeune was contaminated with benzene, vinyl chloride, tetracholoethylene (PERC) and trichloroethylene (TCE). The NRC report found "inadequate/insufficient evidence to determine whether an association exists" between TCE/PCE exposures to Camp Lejeune contaminated water supply "adult leukemia"; and limited association with solvent mixtures with adult leukemia.

Abdul Khalade et al; **Exposure to benzene at work and the risk of leukemia: a systematic review and meta-analysis,** Environ Health. 2010; 9: 31. Published online 2010 June 28. doi: 10.1186/1476-069X-9-31

TCE exposure and risk of NHL, RR 1.23 (CI 1.07-1.42) "overall exposure" RR 1.43 (CI 1.13 to 1.82) "highest exposure"

"Our study provides consistent evidence that exposure to benzene at work increases the risk of leukemia with a dose-response pattern. There was some evidence of an increased risk of AML and CLL. The meta-analysis indicated a lack of association between benzene exposure and the risk of CML."

Hansen, Johnni et al, **Rick of Cancer Among Workers Exposed to Trichloroethylene: Analysis of Three Nordic Cohort Studies,** JNCI, 2013; 105:869-877

TCE exposure and risk of NHL, SIR 1.26 (CI 0.89 – 1.73) included individuals with exposure of greater than 50 U-TCA (mg/L) measure in their urine

TCE exposure and risk of Leukemia, SIR 1.19 (CI 0.72 – 1.86)

Cocco P, et al; **Occupational exposure to solvents and risk of lymphoma subtypes: results from the Epilymph case-control study.** Occup Environ Med. 2010 May;67(5):341-7. doi: 10.1136/oem.2009.046839.

CONCLUSION:

This analysis of a large European dataset confirms a role of occupational exposure to solvents in the aetiology of B-NHL, and particularly, CLL. It is suggested that benzene is most likely to be implicated, but we cannot exclude the possibility of a role for other solvents in relation to other lymphoma subtypes, such as follicular lymphoma. No association with risk of T-cell lymphoma and Hodgkin's lymphoma was shown.

Risk of B-NHL for ever exposure to solvents was not elevated (OR=1.1, 95% CI 1.0 to 1.3), and that for CLL and follicular lymphoma was 1.3 (95% CI 1.1 to 1.6) and 1.3 (95% CI 1.0 to 1.7), respectively. Exposure to benzene accounted, at least partially, for the association observed with CLL risk. Hodgkin's lymphoma and T-cell lymphoma did not show an association with solvent exposure

Karami S, Bassig B, Stewart PA, Lee KM, Rothman N, Moore LE, Lan Q. **Occupational trichloroethylene exposure and risk of lymphatic and haematopoietic cancers: a meta-analysis.** Occup Environ Med. 2013 Aug;70(8):591-9. doi: 10.1136/oemed-2012-101212. Epub 2013 May 30.

The carcinogenic potential of trichloroethylene (TCE) continues to generate much controversy, even after the US Environmental Protection Agency raised its classification to 'carcinogenic to humans'. We conducted a meta-analysis of published cohort and case-control studies exploring occupational TCE exposure in relation to five different lymphatic and haematopoietic cancers: non-Hodgkin's lymphoma (NHL, N=24), Hodgkin's lymphoma (HL, N=13), multiple myeloma (MM, N=11), leukaemia (N=12) and chronic/small lymphocytic leukaemia (CLL/SLL, N=7). Studies published between 1950 and 2011 were identified through a PubMed Medline search. All studies included in analyses were classified as those that assessed either occupational TCE exposure specifically ('TCE-exposure' studies) or a broader classification of all chlorinated solvents ('chlorinated solvent-exposure' studies). A significantly raised summary estimate for NHL was seen for all cohort and case-control 'TCE-exposure' studies combined (N=19; relative risk (RR)=1.32, 95% CI 1.14 to 1.54; I(2)=25.20; p-heterogeneity=0.12) and for cohort 'TCE-exposure' studies (N=10; RR=1.52, 95% CI 1.29 to 1.79; I(2)=7.09; p-heterogeneity=0.63). A non-significant but raised summary estimate was seen for NHL case-control 'TCE-exposure' studies. No significant association with NHL risk was detected overall for any 'chlorinated solvent-exposure' studies. Summary estimates for occupational TCE exposure were not associated with risk of HL, MM, leukaemia or CLL/SLL. Our updated meta-analysis of NHL, which incorporates new analytical results from three cohort and four case-control studies, supports an association between occupational TCE exposure and NHL.

Lamm SH, et al, .**Chronic myelogenous leukemia and benzene exposure: a systematic review and meta-analysis of the case-control literature.** Chem Biol Interact. 2009 Dec 10;182(2-3):93-7. doi: 10.1016/j.cbi.2009.08.010. Epub 2009 Aug 18.

Benzene exposure is well demonstrated as a cause of acute myelogenous leukemia, but not of chronic myelogenous leukemia. Previous literature reviews based on case series and cohort studies have not shown an association. We have now conducted a literature search for case-control studies that examine the association between benzene exposure and chronic myelogenous leukemia. Six case-control studies have been found. These derive from occupational groups, cancer registries, and a clinical laboratory. Their exposure ascertainments are all based on job histories, job-exposure matricies, or industrial hygiene data. The odds ratios (ORs) for individual studies range from 0.73 to

1.2. The pooled OR is 1.003 with 95% confidence interval (CI) of 0.94-1.07 (p=0.98) for both a fixed effects model and a random effects model. The case-control literature indicates that chronic myelogenous leukemia does not appear to be related to benzene exposure.

Gulf War and Health:

Volume 2. Insecticides and Solvents (2003)

6. Cancer and Exposure to Solvents ." *Gulf War and Health: Volume 2. Insecticides and Solvents* . Washington, DC: The National Academies Press, 2003

Summary and Conclusion

Although there is a substantial body of literature on the association between exposure to specific organic solvents and solvent mixtures and risk of NHL, most studies are based on small numbers of exposed cases. An association between comparatively high relative risks of NHL and exposure to benzene was seen consistently in a number of cohort studies. The studies on exposure to benzene and NHL provide consistently positive findings because the populations or groups had known exposure and there was evidence of exposure-response relationships.

The committee concludes, from its assessment of the epidemiologic literature, that there is limited/suggestive evidence of an association between chronic exposure to benzene and non-Hodgkin's lymphoma.

Summary and Conclusion

Overall, the studies reviewed by the committee did not show any persuasive evidence of associations between HD and exposure to specific solvents or solvent mixtures. Although many of the studies of exposure to mixtures of solvents yielded increased risk estimates, there was considerable statistical variability in them. The incidence of HD is low, and most studies had small numbers of exposed cases to evaluate. Having such small numbers may lead to spuriously increased relative risks when the null hypothesis of no association is true. That limitation is reflected in the wide CIs observed in most of the studies. The lack of specific or validated exposure-assessment information and the impact of bias are other limitations that the committee considered in drawing its conclusion. Table 6.36 identifies the key studies reviewed for each exposure and the data points evaluated by the committee. Unless indicated in the tables, the study populations include both men and women.

The committee concludes, from its assessment of the epidemiologic literature, that there is inadequate/insufficient evidence to determine whether an association exists between chronic exposure to solvents under review and Hodgkin's disease.

Summary and Conclusion

IARC and the US Environmental Protection Agency (EPA) have determined that benzene is carcinogenic in humans on the basis of both animal and human studies (ATSDR, 1997a; IARC, 1987; NTP, 2001). That determination was based primarily on the findings on leukemia defined broadly. In addition, epidemiologic studies of occupations exposed to mixtures of solvents, including benzene have shown increased risks of developing cancer. Among those at risk are rubber workers, mechanics, and some groups of chemical workers, printers and paper-industry workers, and shoe and leather workers (IARC, 1987, 1989).

Based on its review of the literature on exposure to benzene, the committee found that the combination of consistently positive findings in the cohort of workers with known exposures to benzene and evidence of a dose-response relationship fulfilled the criteria for a conclusion of sufficient evidence of an association between exposure to benzene and adult leukemia. However, the committee decided that the evidence of an association between exposure to benzene and adult leukemia was not as strong as that for acute leukemia. Thus, it did not warrant a conclusion of causality. The findings, although mostly positive, are not as consistent and statistically precise as the findings on acute leukemia. Most likely, the positive studies on adult leukemia and exposure to benzene include cases of acute leukemia. However, they may also include cases of chronic leukemia, lymphatic leukemia, and hairy cell leukemia, for which the existence of associations is not as clear. On the basis of the studies reviewed, the committee believes that the evidence on exposure to benzene and adult leukemia, defined broadly, met the definition of sufficient evidence of an association but not sufficient evidence of a causal relationship.

The committee concludes, from its assessment of the epidemiologic literature, that there is sufficient evidence of an association between chronic exposure to benzene and adult leukemia.

For exposure to other solvents, such as trichloroethylene and toluene, the overall paucity of studies and the lack of consistently positive findings limits the evidence that the committee had to review.

The committee concludes, from its assessment of the epidemiologic literature, that there is inadequate/insufficient evidence to determine whether an association exists between chronic exposure to specific organic solvents under review, other than

In contrast, the findings for unspecified mixtures of organic solvents and adult leukemia showed increased relative risks, including two studies that provided evidence for a dose-response relationship with increasing levels of exposure. Table 6.40 identifies the key studies reviewed by the committee on adult leukemia. Unless indicated in the table, the study populations include both men and women.**The committee concludes, from its assessment of the epidemiologic literature, that there is limited/suggestive evidence of association between chronic exposure to unspecified mixtures of organic solvents and adult leukemia.**

Summary and Conclusion

On the basis of the consistently high relative risks in studies in which the exposure to benzene is well known, the committee decided that the evidence meets the requirement for a conclusion of causality between chronic exposure to benzene and acute leukemia. Furthermore, given the strong positive associations in the cohort studies of highly exposed subjects, it is likely that

confounding and selection bias do not account for the findings. Experimental evidence supports a biologic mechanism that strengthens the conclusion. The details of that experimental evidence are provided in Chapter 4 and discussed below.

The metabolism of benzene, which occurs in the liver and to a smaller extent in the bone marrow, plays an important role in its toxicity. Benzene is metabolized to benzene oxide, an epoxide, through an oxidation reaction catalyzed primarily by cytochrome P450 2E1. Benzene oxide can be metabolized to various compounds, including o-benzoquinone and *p*-benzoquinone, which are thought to be the two main metabolites that mediate the toxicity of benzene. Data on laboratory animals show that benzene affects the bone marrow in a dose-dependent manner, causing anemia, leukopenia, and thrombocytopenia; continued exposure causes aplasia and pancytopenia (Bruckner and Warren, 2001). Benzene also has carcinogenic properties. In experimental animals, increases in incidence of malignant lymphoma and some solid tumors have been seen after exposure to high doses of benzene.

The committee concludes, from its assessment of the epidemiologic and experimental literature, that there is sufficient evidence of a causal relationship between chronic exposure to benzene and acute leukemia.

The committee drew no conclusion on an association between exposure to benzene or unspecified mixtures of organic solvents and chronic leukemia. Table 6.42 identifies the studies reviewed by the committee on chronic leukemia. Unless indicated in the table, the study populations include both men and women.

Parrillo, Jeffrey M. (VACO)

From: (b) (6)
Sent: Tuesday, June 17, 2014 5:00 PM
To: VHA CO CLCW SME
Subject: FW: benzene/smoking

Categories: Red Category

Fyi per our discussion on this noon calls

From: (b) (6)
Sent: Tuesday, June 17, 2014 11:59 AM
To: (b) (6)
Subject: FW: benzene/smoking

Here's (b) (6) helpful analysis

From: (b) (6)
Sent: Thursday, February 20, 2014 8:48 AM
To: (b) (6)
Cc: (b) (6)
Subject: benzene/smoking

1) The ATSDR Tox Guide for Benzene states: "About 50% of the entire nationwide exposure to benzene results from smoking tobacco or from exposure to tobacco smoke."

The ATSDR Public Health Statement says: "The average smoker (32 cigarettes per day) takes in about 1.8 milligrams (mg) of benzene per day. This amount is about 10 times the average daily intake of benzene by nonsmokers."

The maximum benzene measured in the Hadnot Point water supply was 720 ug/l. This is .7 ppm. If a Veteran drank 8 liters per day, that would be 5760 ug/day of Benzene exposure. Smoking exposure is approximately 1.8 mg per day. This converts to 1800 micrograms per day. If he were at CL for 201 days, he would have been exposed to a total of 1157760 micrograms, or 1157 milligrams total Benzene. If he smoked daily for 50 years, he would have been exposed to 32850 milligrams from smoking. His Benzene exposure from 50 years of smoking would have vastly outweighed any potential exposure from contaminated ground water, even if we were to add in inhalation or dermal exposure. The numbers are drawn from an average smoker of 32 cigarettes per day. If he smoked 20 per day this number would be decreased some but still not coming near that of groundwater exposure and therefore not near a 50:50 level.

Another example- With regard to potential benzene exposure, The maximum benzene measured in the Hadnot Point water supply was 720 ug/l. This is .7 ppm. If a Veteran drank 8 liters per day, that would be 5.760 mg/day of Benzene exposure. Smoking exposure is approximately 1.8 mg per day. Even if he were at CL for 6 years (2190 days), he would have been exposed to a total of 12614milligrams total Benzene. If he smoked 32 cigs. daily for 60 years he would have been exposed to 39420 (over 3x that

of groundwater) milligrams total from smoking. His Benzene exposure from 2 ppd for 60 years of smoking would have vastly outweighed any potential exposure from contaminated ground water, even if we were to add in inhalation or dermal exposure. This would not come near a 50:50 level.

ToxGuide™ for Benzene C_6H_6 CAS# 71-43-2
ATSDR Tox Guide for Benzene and Public Health Statement

2) The ATSDR Tox Guide for Benzene states: "About 50% of the entire nationwide exposure to benzene results from smoking tobacco or from exposure to tobacco smoke."

The ATSDR Public Health Statement says: "The average smoker (32 cigarettes per day) takes in about 1.8 milligrams (mg) of benzene per day. This amount is about 10 times the average daily intake of benzene by nonsmokers."

The maximum benzene measured in the Hadnot Point water supply was 720 ug/l. This is .7 ppm. If a Veteran drank 8 liters per day, that would be 5760 ug/day – 5.76 mg- of Benzene exposure. Smoking exposure is approximately 1.8 mg per day based on smoking 32 cigarettes per day. If he were at CL for 1035 days (appx. Based on dates provided, he would have been exposed to a total of 5961 milligrams total Benzene. If he 90 pack years of smoking, he would have been exposed to 59130 milligrams from smoking. His Benzene exposure smoking would have vastly outweighed any potential exposure from contaminated ground water, even if we were to add in inhalation or dermal exposure. Even if the estimates above are not exact, the benzene exposure from smoking would still be much greater that of groundwater exposure and therefore not near a 50:50 level.

From: (b) (6)
Sent: Wednesday, February 19, 2014 5:41 PM
To: (b) (6)
Subject: RE:

Hi, (b) (6)
I am off –site this week too.
So, No rush-whenever you can-I don't need it for a specific case- but your approach just sounded so clever and right on target-a really creative yet quantitative way to make the necessary points. Something we all should be thinking about!
Thanks for sharing your ideas and I really look forward to reading it -but again, no rush.
(b) (6)

From: (b) (6)
Sent: Wednesday, February 19, 2014 9:41 AM
To: (b) (6)
Subject: RE:

Sure. Can I email it tomorrow when I am onsite? Or do you have a case you need it for today? I can send today thru VPN if you need it today.

From: (b) (6)
Sent: Tuesday, February 18, 2014 5:51 PM
To: (b) (6)

Cc: (b) (6)
Subject:

Hi, (b) (6)
On the CLCW conference call today, (b) (6) mentioned that you did a really creative and clever write- up in calculating actual benzene levels from a smoking history and then comparing it to exposure numbers at Camp Lejuene.
Can I see how you wrote this up?
Thanks,
(b) (6)

Parrillo, Jeffrey M. (VACO)

From: (b) (6)
Sent: Friday, November 14, 2014 5:20 PM
To: (b) (6)
Cc: (b) (6); (b) (6); (b) (6)
Subject: RE: CLCW: Dec Training

I am OK with rescheduling training.

How are we to get the cases completed. Is there the possibility of having some of the SME keep this trip and just do a bunch of cases?

You know better than I but we are getting further behind by the week.

(b) (6)

From: (b) (6)
Sent: Thursday, November 13, 2014 7:45 AM
To: (b) (6)
Cc: (b) (6); (b) (6); (b) (6)
Subject: CLCW: Dec Training

(b) (6)

I think we should look into rescheduling the training.

- Given the reduced travel budget, I think we can only complete 1 CLCW training for FY 15
  - We have 4 potential SMEs committed to attending in DEC
  - I have at least 4 others that are not able to attend
- The computer room at Louisville is now unavailable on Tuesday-Thursday of our training week
  - Most of the doctors do not have personal laptops to bring

Please advise.

(b) (6)

From: (b) (6)
Sent: Friday, November 07, 2014 2:27 PM
To: (b) (6)
Cc: (b) (6); (b) (6); (b) (6)
Subject: RE: Laptops

(b) (6)– The information I have is that test dates would be 12/9 and 12/11- The test is a 6 hour test. With an hour lunch break.

From: (b) (6)
Sent: Friday, November 07, 2014 2:00 PM
To: (b) (6)

Cc: (b) (6) (b) (6) (b) (6)
Subject: RE: Laptops

Good Afternoon (b) (6)

Can you be more specific about the timeframe where we would not be able to use the computer room? We were planning for Monday 12/8 afternoon to Friday 12/12 noon. Please advise.

(b) (6)

From: (b) (6)
Sent: Friday, November 07, 2014 1:47 PM
To: (b) (6)
Cc: (b) (6) (b) (6) (b) (6)
Subject: Laptops
Importance: High

Hi, (b) (6) Hope you are doing well.

During the week of the December CLCW SME training, Louisville must now hold a mandatory skills certification test for our journey-level RVSRs. This has put a slight kink in the training room availability. Is it possible to have the SME trainees bring laptops with them so we can assign them to another training room? Depending upon the length of tests, we may be able to move them back in the computer training room for the remainder of their visit.

Please let me know if this is feasible.

Thanks,
(b) (6)

Parrillo, Jeffrey M. (VACO)

From: (b) (6)
Sent: Monday, September 08, 2014 10:06 AM
To: (b) (6); (b) (6); (b) (6); (b) (6)
Cc: (b) (6); (b) (6); (b) (6)
Subject: RE: CLCW question

I assume the TL 11-03 is a VBA training letter.

Non Hodgkin Lymphoma has been part of the "list" included with the health care law since the original training. I believe it is this list that has been used by the VBA to determine which cases have merit to forward to the SME. Happy to work with whomever is in charge of VBA training letters to help clarify this issue.

As always, the health care law may provide guidance as to what conditions may or may not have face validity in advancing a claim. However it is not the end all, for instance, Parkinson's is not on the "list" however, we have opinioned some cases as being related to CLCW dependent on high level of exposures.

There may be some value to have a VBA/SME meeting to refine the approach to CLCW cases.

(b) (6)

From: (b) (6)
Sent: Monday, September 08, 2014 8:22 AM
To: (b) (6); (b) (6); (b) (6); (b) (6)
Cc: (b) (6); (b) (6)
Subject: FW: CLCW question

Please see the question below from the RO.

From: (b) (6)
Sent: Friday, September 05, 2014 2:58 PM
To: (b) (6)
Cc: (b) (6)
Subject: FW: CLCW question

Hello (b) (6)

Has this condition been added to the official list?

Thanks,

(b) (6)

From: (b) (6)
Sent: Friday, September 05, 2014 2:24 PM
To: (b) (6)
Subject: FW: CLCW question

Hey (b) (6) I was wondering if you had any contact with the CLCW SMEs. It seems that our list of recognized conditions differs from the one on the website. I was wondering if there is a way that we can have the SMEs maybe draft a revised version of the conceded conditions? I don't know, it is just a thought!

Thanks,

(b) (6)

From: (b) (6)
Sent: Friday, September 05, 2014 12:28 PM
To: (b) (6)
Subject: FW: CLCW question

Any ideas?

From: (b) (6)
Sent: Friday, September 05, 2014 10:47 AM
To: (b) (6)
Cc: (b) (6) (b) (6)
Subject: CLCW question

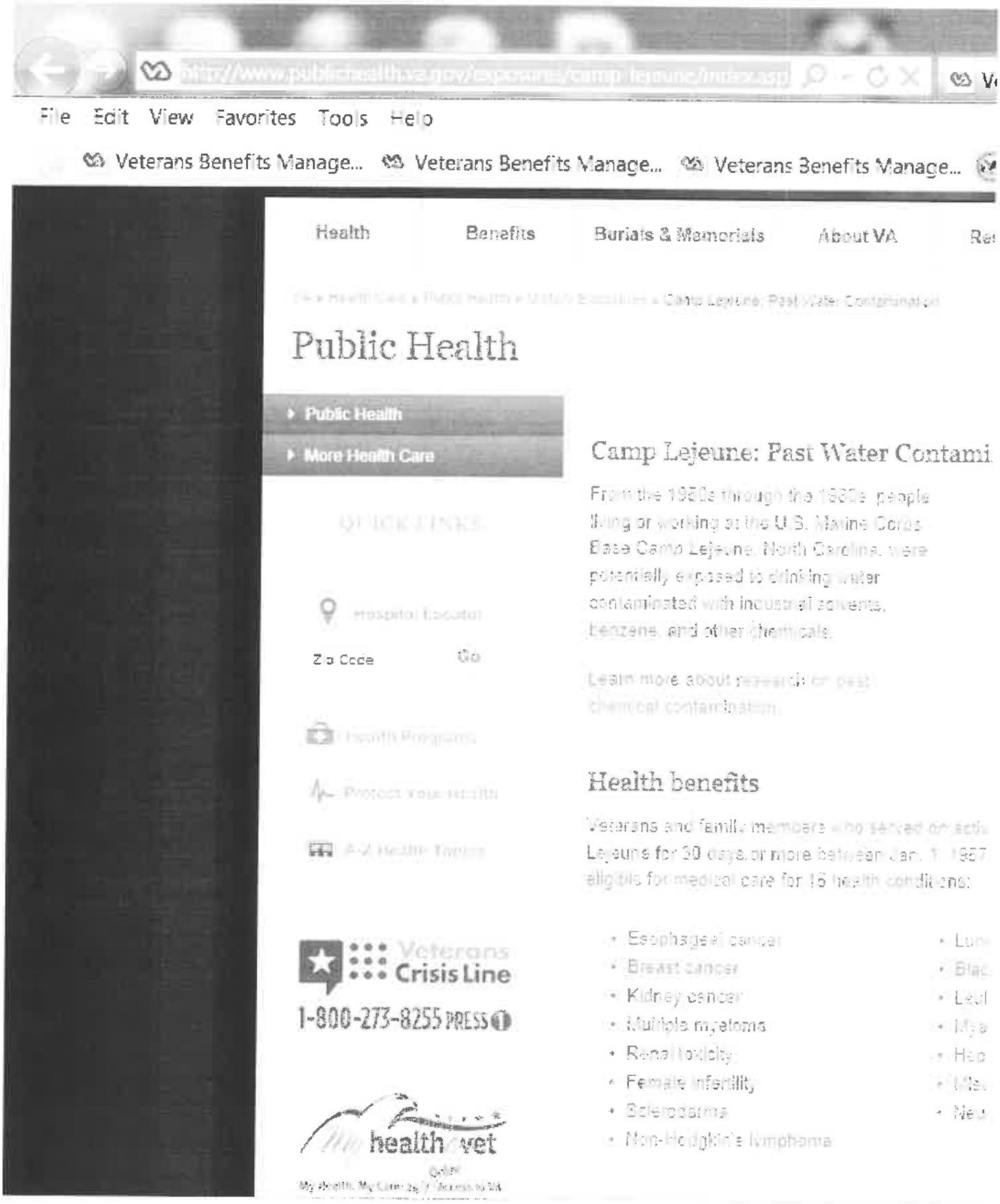
(b) (6)

I have asked several raters and I have not been able to find the answer to the following question:

Non-Hodgkin's Lymphoma is not listed as one of the conditions in the current TL 11-03 (revised). However, it is listed on the below va.gov website.

Question: Is there a reason that it is not on TL 11-03, but it is listed on the website?

http://www.publichealth.va.gov/exposures/camp-lejeune/index.asp


File Edit View Favorites Tools Help
Veterans Benefits Manage...
Veterans Benefits Manage...
Veterans Benefits Manage...
Health
Benefits
Burials & Memorials
About VA
Public Health
Public Health
More Health Care
Camp Lejeune: Past Water Contami
From the 1950s through the 1980s, people
living or working at the U.S. Marine Corps
Base Camp Lejeune, North Carolina, were
potentially exposed to drinking water
contaminated with industrial solvents,
benzene, and other chemicals.
Learn more about research on past
chemical contamination.
Health benefits
Veterans and family members who served on activ
Lejeune for 30 days or more between Jan. 1, 1957
eligible for medical care for 16 health conditions:
Esophageal cancer
Breast cancer
Kidney cancer
Multiple myeloma
Renal toxicity
Female infertility
Scleroderma
Non-Hodgkin's lymphoma
Lung
Blad
Leuk
Mye
Hep
Mes
Neu
Veterans Crisis Line
1-800-273-8255 PRESS 1
health.vet

(b) (6)

Dept of Veteran Affairs
VARO Louisville, KY
Express Team

(b) (6)

Parrillo, Jeffrey M. (VACO)

From: (b) (6)
Sent: Tuesday, May 20, 2014 10:17 AM
To: (b) (6) VHA CO CLCW SME; (b) (6)
Cc: (b) (6); (b) (6); (b) (6); (b) (6); (b) (6); (b) (6); (b) (6)
Subject: RE: Monthly CLCW Update--VANTS 1-800-767-1750 Code71193

Categories: Red Category


PARKINSON.doc


Colon-lit review final.docx


Draft.doc


Generic Template - Copy.dotm

Some draft for discussion for the noon meeting

(b) (6)

-----Original Appointment-----
From: (b) (6)
Sent: Thursday, May 15, 2014 10:44 AM
To: VHA CO CLCW SME; (b) (6)
Cc: (b) (6); (b) (6); (b) (6); (b) (6); (b) (6); (b) (6)
E.
Subject: Monthly CLCW Update--VANTS 1-800-767-1750 Code71193
When: Occurs the third Tuesday of every 1 month effective 2/18/2014 until 3/17/2015 from 12:00 PM to 1:00 PM (UTC-05:00) Eastern Time (US & Canada).
Where: VANTS 1-800-767-1750 Code71193

Join online meeting

https://meet.RTC.VA.GOV/(b) (6)TT0PPB05

(b) (6) has invited you to a conference call: Monthly CLCW Update.

This conference is scheduled to begin on February 18 2014 at 12:00 PM EST+ DST (New York).
This is a recurring conference. It occurs each Tuesday on the 3 week of each month.
This conference is scheduled to last for 60 minutes.
To access this conference via telephone, please call the following number: 8007671750, when prompted for your access code, please enter 71193 on your telephone keypad followed by the # key.

Please do not disclose this information to any unauthorized parties, as the privacy of your conference may be compromised.

We hope you enjoy your conference experience.

PARKINSON'S DISEASE AND ORGANIC SOLVENTS

Human studies: epidemiology

Trichloroethylene (TCE) has been widely used in the workplace, in drycleaning and degreasing, and in environmental exposures, including in typewriter fluids, adhesives, paints, carpet cleaners, spot removers. In 1977 FDA banned its use as an anesthetic and decaffeinating agent.

Since 1981, a robust body of literature has explored the relationship between exposure to organic solvents and Parkinson's disease (PD). At least nine case control studies expored the relationship of solvents, pesticides and PD. Six showed a clear relationship between exposure to organic solvents and the development of disease. The "better" the exposure assessment techniques the more likely associations are to be evident. Two studies failed to distinguish pesticide from solvent exposure and found only elevated risks associated with pesticides. One study failed to find an increased risk of PD after exposure to solvents.

Several formal cohort studies failed to show an increased rate of disease, but PD is only rarely the actual cause of death so that these are not ideal approaches to identifying such relationships. The one cross-sectional study conducted in the workplace identified a strong relationship between the degree of exposure to trichloroethylene and PD or, at lower levels of exposure, early signs of basal ganglion involvement

Mechanistic studies of solvents in general and TCE in particular identified ways that general damage in dopaminergic neurons in the brainstem.

Few studies provide approximate exposure data, much less information allowing precise dose estimates. The most pertinent and useful study was conducted by Goldman et al: the World War II Veterans Twins study. An exposure to TCE of at least one hour a day or 2% of the work day for at least six months was associated with a six-fold risk of disease; PERC in that same exposure definition was associated with a 10-fold risk.

Additional pertinent considerations include other known risk factors for Parkinson's disease. Smoking is known to be protective, with consistent relative risks of 0.5 – 0.6; similarly, coffee drinking reduces the risk. No explanation exists for these two factors.

Several other exposures are known to increase the risk of Parkinson's disease, most prominently, managanese, carbon monoxide, and pesticides. Carbon monoxide represents a known risk factor, but that onset of disease is relatively prompt after over-exposure. Common events leading to onset include suicidal gestures with combustion products, "accidental" overexposures from use of internal combustion (indoor chain saws, gas-powered buffers, and entrainment of grill exhaust). Manganese in mining and manufacturing and in welding is clearly associated with disease. Examiners should make efforts to identify an acute onset of disease (CO) or exposures to pesticides (farming, pesticide application) or welding

Exposures and Dose Extrapolation

1. ***Inhalation exposures per day***

Showering for 15 minutes, @ 12 breaths / minutes and 530 cc tidal volume: 95.4 liters of breathed air, with typical absorption of 55% in inhalation exposures ( ) per day: spreadsheet calculation supports modification of parameters to achieve a total dose by inhalation for median and maximum concentration levels

2. ***Ingestion:*** Approximately 90 percent of TCE is absorbed from the gut. Spreadsheet calculation 2 supports calculation of a total absorbed dose for median and maximum concentration levels

3. ***Comparisons with critical levels***: The ATSD Toxicologic Profile ( ) provides information on various categories of serious and less serious effects for humans and for animals for short/acute, intermediate, and long-term exposures for both inhalation and ingestion.

PARKSON REFERENCES

CASE-CONTROL STUDIES

Smargiassi A, Mutti A, De Rosa A, De Palma G, Negrotti A, Calzetti S. A case-control study of occupational and environmental risk factors for Parkinson's disease in the Emilia-Romagna region of Italy. Neurotoxicology. 1998 Aug-Oct;19(4-5):709-12.

Ohlson CG, Hogstedt C. Parkinson's disease and occupational exposure to organic solvents, agricultural chemicals and mercury--a case-referent study. Scand J Work Environ Health. 1981 Dec;7(4):252-6.

Goldman SM, Quinlan PJ, Ross GW, Marras C, Meng C, Bhudhikanok GS, Comyns K, Korell M, Chade AR, Kasten M, Priestley B, Chou KL, Fernandez HH, Cambi F, Langston JW, Tanner CM. Solvent exposures and Parkinson disease risk in twins. Ann Neurol. 2012 Jun;71(6):776-84. doi: 10.1002/ana.22629

Pezzoli G, Canesi M, Antonini A, Righini A, Perbellini L, Barichella M, Mariani CB, Tenconi F, Tesei S, Zecchinelli A, Leenders KL. Hydrocarbon exposure and Parkinson's disease. Neurology. 2000 Sep 12;55(5):667-73. (earlier onset, more severe symptoms)

McDonnell L, Maginnis C, Lewis S, Pickering N, Antoniak M, Hubbard R, Lawson I, Britton J. Occupational exposure to solvents and metals and Parkinson's disease. Neurology. 2003 Sep 9;61(5):716-7.

Tanner CM, Ross GW, Jewell SA, Hauser RA, Jankovic J, Factor SA, Bressman S, Deligtisch A, Marras C, Lyons KE, Bhudhikanok GS, Roucoux DF, Meng C, Abbott RD, Langston JW. Occupation and risk of parkinsonism: a multicenter case-control study. Arch Neurol. 2009 Sep;66(9):1106-13. (pesticides, no solvents)

Dick FD, De Palma G, Ahmadi A, Scott NW, Prescott GJ, Bennett J, Semple S, Dick S, Counsell C, Mozzoni P, Haites N, Wettinger SB, Mutti A, Otelea M, Seaton A, Söderkvist P.

Felice A; Geoparkinson study group. Environmental risk factors for Parkinson's disease and parkinsonism: the Geoparkinson study. Occup Environ Med. 2007 Oct;64(10):666-72. Epub 2007 Mar 1. (pesticides, no solvents)

Firestone JA, Lundin JI, Powers KM, Smith-Weller T, Franklin GM, Swanson PD, Longstreth WT Jr, Checkoway H. Occupational factors and risk of Parkinson's disease: A population-based case-control study. Am J Ind Med. 2010 Mar;53(3):217-23. (no effect of solvents)

Seidler A, Hellenbrand W, Robra BP, Vieregge P, Nischan P, Joerg J, Oertel WH, Ulm G, Schneider E. Possible environmental, occupational, and other etiologic factors for Parkinson's disease: a case-control study in Germany. Neurology. 1996 May;46(5):1275-84. (solvents by self-report but not by JEM)

COHORT AND CROSS-SECTIONAL STUDIES

Feldman AL, Johansson AL, Nise G, Gatz M, Pedersen NL, Wirdefeldt K. Occupational exposure in parkinsonian disorders: a 43-year prospective cohort study in men. Parkinsonism Relat Disord. 2011 Nov;17(9):677-82. Epub 2011 Jul 5.

Gash DM, Rutland K, Hudson NL, Sullivan PG, Bing G, Cass WA, Pandya JD, Liu M, Choi DY, Hunter RL, Gerhardt GA, Smith CD, Slevin JT, Prince TS. Trichloroethylene: Parkinsonism and complex 1 mitochondrial neurotoxicity. Ann Neurol. 2008 Feb;63(2):184-92.

CASE REPORTS

Pezzoli G, Barbieri S, Ferrante C, Zecchinelli A, Foà V. Parkinsonism due to n-hexane exposure. Lancet. 1989 Oct 7;2(8667):874.

Tetrud JW, Langston JW, Irwin I, Snow B. Parkinsonism caused by petroleum waste ingestion. Neurology. 1994 Jun;44(6):1051-4.

Uitti RJ, Snow BJ, Shinotoh H, Vingerhoets FJ, Hayward M, Hashimoto S, Richmond J, Markey SP, Markey CJ, Calne DB. Parkinsonism induced by solvent abuse. Ann Neurol. 1994 May;35(5):616-9.

ANIMAL STUDIES AND MECHANISTIC REVIEWS

P Gash DM, Rutland K, Hudson NL, Sullivan PG, Bing G, Cass WA, Pandya JD, Liu M, Choi DY, Hunter RL, Gerhardt GA, Smith CD, Slevin JT, Prince TS. Trichloroethylene: Parkinsonism and complex 1 mitochondrial neurotoxicity. Ann Neurol. 2008 Feb;63(2):184-92.

Pezzoli G, Ricciardi S, Masotto C, Mariani CB, Carenzi A. n-hexane induces parkinsonism in rodents. Brain Res. 1990 Oct 29;531(1-2):355-7.

Mutti A, Smargiassi A. Selective vulnerability of dopaminergic systems to industrial chemicals: risk assessment of related neuroendocrine changes. Toxicol Ind Health. 1998 Jan-Apr;14(1-2):311-23.

Guehl D, Bezard E, Dovero S, Boraud T, Bioulac B, Gross C. Trichloroethylene and parkinsonism: a human and experimental observation. Eur J Neurol. 1999 Sep;6(5):609-11.

Gash DM, Rutland K, Hudson NL, Sullivan PG, Bing G, Cass WA, Pandya JD, Liu M, Choi DY, Hunter RL, Gerhardt GA, Smith CD, Slevin JT, Prince TS. Trichloroethylene:

Parkinsonism and complex 1 mitochondrial neurotoxicity. Ann Neurol. 2008 Feb;63(2):184-92.

Mutti A, Franchini I. Toxicity of metabolites to dopaminergic systems and the behavioural effects of organic solvents. Br J Ind Med. 1987 Nov;44(11):721-3. (effects from non-enzymatic condensation with many metabolites)

Last Update: 2.2.2014, MBK

Disease Specific Discussion: Colon Cancer

Disease Description:

Colorectal cancer (CRC) is defined as cancer that forms in tissues of the colon and rectum. The colon is the large intestine which is the lower part of your digestive system. Rectal cancer is cancer of the last several inches of the colon. Colon rectal cancer may spread. Common areas of spread include, lymph nodes, liver and lungs, but potentially can spread to many other areas. CRC spread, metastasis, is still colon rectal cancer despite being located in other areas of the body.

Incidence:

Colorectal cancer is the second most common cause of cancer death in the United States. In the US, in 2013, it is estimated to be 142,820 new cancer of the large bowel, including approximately, 102,840 colon cancers, and 52,390 rectal cancers.

There is an increasing incidence of colon rectal cancer with age. CRC is uncommon prior to age 50, and increases significantly each decade of life after age 50.

Men and women had significantly different incidence rates. (CDC, Colorectal cancer incidence rates)
Black male 62.0\100,000
White males 51.5\100,000
Hispanic males 44.8\100,000
Asian/Pacific Islander males 39.7\100,000
American Indian/Alaska Native males 33.5\100,000
Black women 47.1\ 100,000
White women 38.5\100,000
Hispanic women 32.6\100,000
Asian/Pacific Islander women 31.1\100,000
American Indian/Alaska Native women 28.8\100,000

Risk Factors:

CRC has numerous known risk factors to include: older age, African-American race, inflammatory intestinal conditions (ulcerative colitis, Crohn's disease), inherited familial adenomatous polyposis, family history of colon cancer and colon polyps, smoking, heavy ETOH consumption and prior history of abdominal radiation therapy for previous cancer. The majority of colon rectal cancers occur on a sporadic base with no clearly identifiable risk factors found.

Scientific review:

NCR 2008 report:
The water supply at Camp Lejeune was contaminated with benzene, vinyl chloride, tetracholoethylene (PERC) and trichloroethylene (TCE). The NCR report found "inadequate/insufficient evidence to determine whether an association exists" between exposures to Camp Lejeune contaminated water supply and colon cancer (82).

Last Update: 2.2.2014, MBK

Environmental Exposure studies:

Environmental studies evaluating possible relationship between CRC and exposure to the chemical found in CLCW are somewhat limited. The studies that have been done, such as Morgan et al, in a 2002 study, found no increased risk of colon and rectal cancers in California communities exposed to drinking water contaminated with TCE.

Occupational Exposure studies:

Paulu et al, 1999 observed the following, "The adjusted ORs for colon-rectum cancer were modestly elevated among ever-exposed subjects as more years of latency were assumed [OR and CI, 1.7 (0.8-3.8) and 2.0 (0.6-5.8) for 11 and 13 years of latency, respectively]. These elevated ORs stemmed mainly from associations with rectal cancer. Adjusted ORs for rectal cancer among ever-exposed subjects were more elevated [OR and CI, 2.6 (0. 8-6.7) and 3.1 (0.7-10.9) for 11 and 13 years of latency, respectively] than were corresponding estimates for colon cancer [OR and CI, 1.3 (0.5-3.5) and 1.5 (0.3-5.8) for 11 and 13 years of latency, respectively]." While the odds ratio are somewhat elevated the CI were below 1 which means the conclusion could be due to chance along. Therefor this data does not rise to the level of certainty to support a link between exposures to CLCW.

Lipworth et all, in 2011 publication evaluated TCE, PCE and mixed solvent exposures, concluded that there was "no consistent evidence of increased cancer" with long term exposure to the above chemical in aircraft workers.

Hansen et al, in a pooled cohort study published in 2013 study documented not increase risk of colon rectal cancer due to exposure to TCE and its metabolites.

Literature review:

1. Siegel R, Naishadham D, Jemal A. Cancer statistics, 2013. CA Cancer J Clin 2013; 63:11.
2. Epidemiologic Studies of Solvent-Contaminated Water Supplies," Contaminated Water Supplies at Camp LeJeune: assessing Potential Health Effects, National Research Council of the National Academies. The National Academies Press, Washington, D.C. 2009.
3. Mundt KA, Birk T, Burch MT. Critical review of the epidemiological literature on occupational exposure to perchloroethylene and cancer. Int Arch Occup Environ Health. 2003 Sep;76(7):473-91. Epub 2003 Jul 29.
4. Lipworth L, Sonderman JS, Mumma MT, Tarone RE, Marano DE, Boice JD Jr, McLaughlin JK. Cancer mortality among aircraft manufacturing workers: an extended follow-up. J Occup Environ Med. 2011 Sep;53(9):992-1007. doi: 10.1097/JOM.0b013e31822e0940.
5. Paulu C, Aschengrau A, Tetrachloroethylene-contaminated drinking water in Massachusetts and the risk of colon-rectum, lung, and other cancers. Ozonoff D Environ Health Perspect. 1999 Apr;107(4):265-71

Parrillo, Jeffrey M. (VACO)

From: (b) (6)
Sent: Tuesday, March 24, 2015 8:28 AM
To: (b) (6)
Subject: RE: Occcuaptaionl exposures in rare cacners-a critical review of the literature

thanks

From: (b) (6)
Sent: Monday, March 23, 2015 1:20 PM
To: (b) (6)
Subject: Occcuaptaionl exposures in rare cacners-a critical review of the literature

Parrillo, Jeffrey M. (VACO)

From: (b) (6)
Sent: Wednesday, November 05, 2014 1:30 PM
To: (b) (6); (b) (6); (b) (6); VHA CO Office of Disability and Medical Assessment (DMA)
Subject: RE: Products in support of VACAA Choice Program Rollout

This phrasing is so demeaning to the C&P community.

While it may be legally correct, I would hope we could answer the question in a more respectful manner.

(b) (6)

From: (b) (6)
Sent: Wednesday, November 05, 2014 12:09 PM
To: (b) (6); (b) (6); VHA CO Office of Disability and Medical Assessment (DMA)
Subject: Re: Products in support of VACAA Choice Program Rollout

Got a read from OCLA....

From: (b) (6)
Sent: Wednesday, November 05, 2014 12:55 PM Eastern Standard Time
To: (b) (6); VHA CO Office of Disability and Medical Assessment (DMA)
Subject: RE: Products in support of VACAA Choice Program Rollout

Additionally,

(b) (6) also got a read from OGC on VACAA as related to C&P but not sure it was included in this:

Confirmed from (b) (6) at OGC:

> C&P Exams are not considered hospital care/medical services under 38 USC 1701 and they are not part of the Medical Benefits Package at 38 CFR 17.38. For these reasons, they would not be available under the Choice Program.

From: (b) (6)
Sent: Wednesday, November 05, 2014 11:59 AM
To: VHA CO Office of Disability and Medical Assessment (DMA)
Subject: FW: Products in support of VACAA Choice Program Rollout

FYI

(b) (6)
Office of Disability and Medical Assessment (10NC8)

Department of Veterans Affairs
810 Vermont Ave., NW, Ofc 971
Washington, DC 20420
[redacted]
Fax: (202) 495-5168
[redacted]

From: [redacted]
Sent: Wednesday, November 05, 2014 11:57 AM
To: VHA CO 10NC All Staff
Subject: FW: Products in support of VACAA Choice Program Rollout

fyi

From: [redacted]
Sent: Wednesday, November 05, 2014 11:04 AM
To: VHA 10N Action; VHA 10A Action; VHA 10P Actions ; VHA 10B
Subject: FW: Products in support of VACAA Choice Program Rollout

For your information on VACAA. Please disseminate as appropriate.

Thanks, [redacted]

[redacted]

From: [redacted]
Sent: Tuesday, November 04, 2014 6:42 PM
To: [redacted]
Cc: [redacted]; [redacted]; [redacted]
Subject: Products in support of VACAA Choice Program Rollout
Importance: High

All,

As you may be aware, tomorrow VA will announce our implementation plans for the Choice Program, a new benefit that was passed into law this summer as part of the Veterans Access, Choice, and Accountability Act. Attached are documents we hope will be helpful for your field staff, call centers, and other Veteran-facing employees, as well as employees who may find themselves in the position of answering questions from Veterans.

It is important that we arm our employees with as much information as possible to help Veterans understand this new benefit, but please note that these documents will not be provided publicly until tomorrow morning at **9am Eastern time**. As you disseminate these documents to the field, please ensure they are aware of the timeline for approved release.

Attachments that are able to be printed and provided in hard copy to Veterans are: Choice Program Fact Sheet, Choice Card Quick Facts, Open Letter to Veterans. Additional products *for internal reference only* include copies of the letters

(30 day group and 40 mile group) that will be sent to eligible Veterans only, an internal Q&A document, and high level talking points. The website where the VACAA/Choice Program information is hosted is: www.va.gov/opa/choiceact

Please feel free to distribute, and let me know if you have questions.

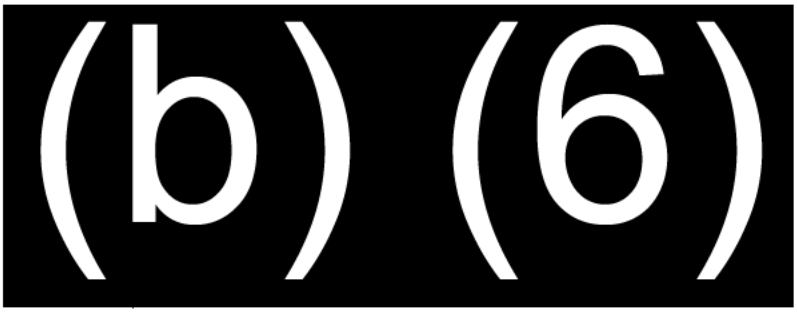
Best,

(b) (6)

(b) (6)

Parrillo, Jeffrey M. (VACO)

From: (b) (6)
Sent: Monday, July 15, 2013 11:09 AM
To: (b) (6)
Attachments: Generic Template.dotm; Prostate Cancer Template.dotm



"If you follow the counsel of those who believe that politics is only a game to be played for personal advantage, you are wasting your time." Harry S. Truman