



DEPARTMENT OF VETERANS AFFAIRS  
Office of Inspector General  
PO Box 50410  
Washington DC 20091-0410

December 28, 2015

Dear Mr. Bergh,

We encourage you to read this message in its entirety because it contains answers to questions you may have about your submission. The U.S. Department of Veterans Affairs Office of Inspector General (OIG) Hotline received your submission on December 23, 2015; the reference number in our system is 2016-9743.

**WHAT HAPPENS NEXT? WILL I BE CONTACTED AGAIN BY THE OIG?**

Within the next 6 weeks your information will be carefully reviewed by our Hotline Staff and other OIG subject matter experts to determine the appropriate course of action. *We will contact you again only if we open a case or need additional information; therefore this may be the only correspondence you receive from our office.*

**Acceptance Criteria** -The Hotline accepts tips or complaints that, on a select basis, result in reviews of:

- VA-related criminal activity
- Systematic patient safety issues
- Gross mismanagement of waste of VA resources
- Misconduct by senior VA officials

We recommend that if you are having ongoing health care, disability claim, or business disputes with VA, continue working with the responsible Department office while waiting to see if the OIG will open a case on your complaint.

**CAN I CHECK THE STATUS OF MY SUBMISSION?**

We cannot provide status reports or information regarding the disposition of submissions that do not result in cases. However, complaints that do not result in formal cases may be used in planning future OIG inspections and audits, or, if not confidential, referred to VA officials for their information.

**IT HAS BEEN 6 WEEKS SINCE I SUBMITTED MY COMPLAINT AND I HAVE NOT HEARD BACK FROM THE OIG. WHY WASN'T MY COMPLAINT ACCEPTED? WHAT CAN I DO NOW?**

If we have not contacted you in 6 weeks, your complaint was not accepted for review because it does not meet one of the four criteria for acceptance or because another Government agency is better positioned to resolve the issue. Contact information for VA and other Government agencies that can help you is located on our website at <http://www.va.gov/oig/hotline/faq.asp>.

The OIG does not investigate complaints that are unrelated to VA programs and operations or issues, such as those listed below, that are addressed in other legal or administrative forums:

- Allegations of whistleblower retaliation (This is because the OIG cannot provide direct relief to complainants in contrast to the U.S. Office of Special Counsel which can. Further information is available at <https://osc.gov/Pages/WhatWeDo.aspx> or 1-800-572-2249.)
- Claims for VA disability and pension benefits, and ratings, appeals, or home loan issues
- Claims for VA education benefits
- Tort claims or other legal issue/case/claims
- Litigation matters
- Employee grievances, unfair labor practices, union matters



U.S. OFFICE OF SPECIAL COUNSEL

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JUN 18 2014

Mr. David M. Bergh  
855 N. River Ave, Apt #5  
Sauk Rapids, MN 56379

[Addendum (7/9/14)]: See NAS retrovirologist Peter  
Duesberg ([www.duesberg.com](http://www.duesberg.com)) and his 1996 book,  
Inventing The AIDS Virus. -David M. Bergh]

Re: OSC File No. MA-14-3224

Dear Mr. Bergh:

This letter is in response to the complaint that you recently filed with the U.S. Office of Special Counsel (OSC). In your complaint, you state that you are trying to reopen a VA claim. You also request that we send you a copy of NIEHS Director David Rall's letter dated 4/3/87. The Complaints Examining Unit has carefully reviewed the information that you have provided. Based on this information, we have made a determination to close your file in this matter.

Your complaint does not allege a prohibited personnel practice or other prohibited activity. Rather, you request that OSC send you copies of Director Rall's letter dated 4/3/87. It appears that you will need to contact NIEHS to request a copy of this letter. OSC does not have the authority to request copies of documents from other agencies on your behalf. However, OSC has the authority to investigate allegations of prohibited personnel practices and certain activities prohibited by civil service law, rule or regulation. 5 U.S.C. §§ 1214(a)(1)(A), 1216(a) and 2302(b). The provisions of 5 U.S.C. § 2302(b) specifically define thirteen prohibited personnel practices for which we have jurisdiction to investigate. However, you have not alleged, nor is there any information in your complaint which indicates, that an official has taken an action that constitutes any of the thirteen personnel practices prohibited by section 2302(b), or any other prohibited activity within the Special Counsel's investigative jurisdiction.

Further, our authority to investigate allegations of prohibited personnel practices extends only to employees, former employees, or applicants for employment to competitive or excepted service positions in the Executive Branch departments and agencies of the federal government. The information you provided indicates that you are not an employee as defined in the provisions of 5 U.S.C. § 2105. Thus, we have no authority to assist you in this matter.

Sincerely,

A handwritten signature in black ink, appearing to read "Chauncey Lawson". The signature is stylized and cursive.

Chauncey Lawson  
Complaints Examiner



## STATEMENT IN SUPPORT OF CLAIM

**PRIVACY ACT INFORMATION:** The law authorizes us to request the information we are asking you to provide on this form (38 U.S.C. 501(a) and (b)). The responses you submit are considered confidential (38 U.S.C. 5701). They may be disclosed outside the Department of Veterans Affairs (VA) only if the disclosure is authorized under the Privacy Act, including the routine uses identified in the VA system of records, 58VA21/22, Compensation, Pension, Education and Rehabilitation Records - VA, published in the Federal Register. The requested information is considered relevant and necessary to determine maximum benefits under the law. Information submitted is subject to verification through computer matching programs with other agencies.

**RESPONDENT BURDEN:** VA may not conduct or sponsor, and respondent is not required to respond to this collection of information unless it displays a valid OMB Control Number. Public reporting burden for this collection of information is estimated to average 15 minutes per response, including the time for reviewing instructions, searching existing data sources, gathering and maintaining the data needed, and completing and reviewing the collection of information. If you have comments regarding this burden estimate or any other aspect of this collection of information, call 1-800-827-1000 for mailing information on where to send your comments.

FIRST NAME - MIDDLE NAME - LAST NAME OF VETERAN ( <i>Type or print</i> )	SOCIAL SECURITY NO.	VA FILE NO.
David M Bergh		C/CSS .25 300 414

The following statement is made in connection with a claim for benefits in the case of the above-named veteran:

This is in furtherance of reopening this claim for HIV/EBV-free chemical AIDS (CAIDS) secondary to Agent Orange (AO) exposure, as expressed in my 21-4138s dated 1/13/14, 12/18/13, 12/8/13, and 11/23/13.

I have previously asserted that my Nehmer v. DVA status means this claim will continue in perpetuity. This claim file may not attain the International Criminal Court legal standing it warrants, but I at least want posterity to know about connections between this claim and the events of 9/11/01 (9.11). I have previously submitted my 4/1/04 letter to the 9.11 Commission to VARO twice in its original handwritten form. The last time, I added the typewritten notation, "DVA OIG ref. # (53E/DS) 2008-6746"

What follows is an exact replication of the body of my 4/1/04 letter to the 9.11 Commission:

**The Institute of Medicine of the National Academy of Sciences has confirmed that Vietnam War veterans by the thousands have incurred HIV-free Chemical AIDS. Our exposure to toxic chemicals, including the notorious "Agent Orange", has been shown to be the cause. The HIV theory of AIDS causation has been disproved.**

**But the U.S. has exported such dangerous chemicals (as well as drugs and radioactive materials) throughout the world. The vaunted Centers for Disease Control and Prevention was primarily responsible for providing DDT to sub-Saharan Africa. This "AIDS belt" coincides with the malaria belt there.**

**The United States Government is CAUSING "AIDS" on a world-wide scale. I suspect that AT LEAST the events of "9.11" and the embassy bombing in Nairobi are Muslim World retribution.**

This is a shortened version. See the entire letter (copy attached).

(CONTINUE ON REVERSE)

I CERTIFY THAT the statements on this form are true and correct to the best of my knowledge and belief.

SIGNATURE	DATE SIGNED
	2/9/14
ADDRESS	TELEPHONE NUMBERS ( <i>Include Area Code</i> )
855 N River Av. Apt. 5 Sauk Rapids MN 56379	DAYTIME 320.203.9455
EVENING	

**PENALTY:** The law provides severe penalties which include fine or imprisonment, or both, for the willful submission of any statement or evidence of a material fact, knowing it to be false.

# Dioxin-mediated tumor progression through activation of mitochondria-to-nucleus stress signaling

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Edited by Craig B. Thompson, University of Pennsylvania, Philadelphia, PA, and approved September 25, 2007 (received for review June 30, 2007)

The environmental toxin 2,3,7,8-tetrachlorodibenzodioxin (TCDD) is a known human carcinogen; however, its precise mechanism of action remains unclear. Here we show that TCDD induces mitochondrial dysfunction, stress signaling, and tumor invasion by a mechanism similar to that described for mtDNA-depleted cells. Treatment of C2C12 cells with TCDD disrupted mitochondrial transmembrane potential in a time-dependent fashion and inhibited mitochondrial transcription and translation. TCDD also increased cytosolic  $[Ca^{2+}]_c$  and RyR1-specific  $Ca^{2+}$  release. These changes were associated with increased calcineurin (CnA) levels and activation of CnA-sensitive NF- $\kappa$ B/Rel (I $\kappa$ B $\beta$ -dependent) factors. Cells treated with TCDD displayed resistance to apoptosis, increased expression of the tumor marker cathepsin L, and a high degree of invasiveness as tested by the Matrigel membrane invasion assay. These effects were reversed by the CnA inhibitor FK506, and CnA mRNA silencing suggesting that TCDD triggers a signaling pathway similar to mtDNA depletion. Taken together, these results reveal that TCDD may promote tumor progression *in vivo* by directly targeting mitochondrial transcription and induction of mitochondrial stress signaling.

calcineurin | mitochondrial transcription | 2,3,7,8-tetrachlorodibenzodioxin (TCDD) | tumor invasion | transmembrane potential

Mitochondrial dysfunction is associated with a myriad of pathologies, including diabetes, heart disease, blindness, deafness, kidney disease, obesity, and neurodegenerative diseases, as well as aging (1). Mitochondrial dysfunction is also associated with cancer and has been reported to play a role in carcinogenesis (2–4). Diverse stimuli, including environmental toxins, drugs, ionophores, hypoxia, and mtDNA mutations/deletions are known to cause mitochondrial dysfunction (2, 4–7). Recent studies from our and others' laboratories have shown that, in a number of cell lines, mitochondrial dysfunction induced by partial depletion of mtDNA or by mitochondrial inhibitors elicits mitochondria-to-nucleus stress signaling that is propagated through activation of calcineurin (CnA) and other factors (7–11). Moreover, activation of mitochondrial stress signaling in C2C12 rhabdomyoblasts and A549 lung carcinoma cells induces invasive phenotypes that are resistant to apoptotic stimuli (8, 9, 12, 13).

Although various causes of mitochondrial dysfunction by physiological processes are relatively better understood, the environmental factors that affect mitochondrial function and lead to mtDNA mutations are much less clear. The environmental toxin 2,3,7,8-tetrachlorodibenzodioxin (TCDD), a member of a family of halogenated aromatic hydrocarbons known as dioxins, is a known carcinogenic and teratogenic agent. TCDD has deleterious effects on human as well as wildlife health. Wasting (cachexia), thymic involution, tumor promotion, hepatotoxicity, developmental toxicity, and immunosuppression are a few of the pathological effects of TCDD (14, 15). TCDD is also known to induce oxidative stress, production of superoxide and peroxide radicals, and DNA single-strand breaks (16–18). However, the cellular and molecular mechanisms of TCDD-mediated pathologies are poorly understood.

TCDD and related dioxins are well established ligands for aryl hydrocarbon receptor (AhR), which modulates transcriptional ac-

tivation of many genes, including those involved in fatty acid metabolism (18), cell cycle regulation, immune response, and xenobiotic metabolism. Binding of TCDD to AhR triggers AhR nuclear translocation and its heterodimerization with AhR nuclear translocator (Arnt). The AhR–Arnt complex activates transcription by binding to dioxin-responsive elements, although some studies question the absolute requirement of AhR for transactivation of TCDD-responsive genes and hint at the existence of alternate, AhR-independent pathways (19, 20).

Here we show that exposure of C2C12 myocytes to TCDD results in inhibition of mitochondrial transcription, disruption of mitochondrial transmembrane potential ( $\Delta\Psi_m$ ), and altered  $Ca^{2+}$  homeostasis. TCDD-treated C2C12 cells also developed resistance to apoptosis and acquired highly invasive phenotypes. Notably, these effects are dependent on CnA but not on AhR–Arnt factors. These findings suggest that TCDD may promote tumor progression by directly targeting mitochondrial function and triggering mitochondria-to-nucleus stress signaling.

## Results

**TCDD Induces Mitochondrial Dysfunction in C2C12 Cells.** Exposure of C2C12 cells to the mitochondrial respiratory inhibitors such as carbonyl cyanide *m*-chlorophenylhydrazone (CCCP) or depletion of mtDNA induces mitochondrial stress signaling, resulting in the activation of a number of nuclear genes and the development of invasive phenotypes (3, 7, 10). Therefore, we examined whether TCDD that induces mitochondrial damage (16) also induces stress signaling. TCDD treatment (10 nM) for 4 h resulted in a dissipation of  $\Delta\Psi_m$  as measured by MitoTracker Orange dye uptake (Fig. 1A). This effect was similar to that seen in mtDNA-depleted or CCCP-treated cells. Mitochondria from treated cells produced a higher level of reactive oxygen species [supporting information (SI) Fig. 6] possibly because of its secondary effects on membrane complexes or matrix enzymes.

TCDD treatment also resulted in a marked increase in caffeine-mediated  $Ca^{2+}$  release and a concomitant reduction in acetylcholine-mediated  $Ca^{2+}$  release in C2C12 cells (Fig. 1B and C). These results were similar to those obtained with mtDNA-depleted cells. Interestingly, acetylcholine-mediated  $Ca^{2+}$  release, signifying inositol trisphosphate channel activity, is prominent in

Author contributions: G.B. and N.G.A. designed research; G.B., S.S., and H.K.A. performed research; G.B. and N.G.A. analyzed data; and G.B. and N.G.A. wrote the paper.

The authors declare no conflict of interest.

This article is a PNAS Direct Submission.

Abbreviations: AhR, aryl hydrocarbon receptor;  $\alpha$ -NF,  $\alpha$ -naphthoflavone; Arnt, AhR nuclear translocator; CCCP, carbonyl cyanide *m*-chlorophenylhydrazone; CnA, calcineurin; CREB, cAMP response element-binding;  $\Delta\Psi_m$ , mitochondrial transmembrane potential; MEF, mouse embryonic fibroblast; siArnt, Arnt-silenced; STP, staurosporine; TCDD, 2,3,7,8-tetrachlorodibenzodioxin.

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This article contains supporting information online at [www.pnas.org/cgi/content/full/0706183104/DC1](http://www.pnas.org/cgi/content/full/0706183104/DC1).

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