Your Profile

Name
Address
City
State and Zip
Phone Number
Email
Date of birth
Spouses name
Branch of service
Stationed where
Dates of Duty
When diagnosed with PD
Where are you being treated
Comments

If you would please fill out the form and email them back I will put them in your file.

This very useful when go and talk to the senators and congressmen and women.

Syrbr
Chemical exposures and Parkinson's disease: a population-based case-control study.


Division of Epidemiology, Department of Health Sciences Research, Mayo College of Medicine, Rochester, Minnesota 55905, USA.

The putative association between pesticide exposures and Parkinson's disease (PD) remains controversial. We identified all subjects who developed PD in Olmsted County, Minnesota, from 1976 through 1995, and matched them by age (+/- 1 year) and sex to general population controls. We assessed exposures to chemical products by means of telephone interview with cases, controls, or their proxies (149 cases; 129 controls). Exposure to pesticides related or unrelated to farming was associated with PD in men (odds ratio, 2.4; 95% confidence interval, 1.1-5.4; P = 0.04). The association remained significant after adjustment for education or smoking. Analyses for the other six categories of industrial and household chemicals were all nonsignificant. This population-based study suggests a link between pesticides use and PD that is restricted to men. Pesticides may interact with other genetic or nongenetic factors that are different in men and women.

PMID: 16773614 [PubMed - indexed for MEDLINE]
On appeal from the Department of Veterans Affairs Regional Office in Winston-Salem, North Carolina

THE ISSUE

Entitlement to service connection for Parkinson's disease, to include as a result of exposure to an herbicide agent.

REPRESENTATION

Appellant represented by: Sharon A. Hatton, Attorney at Law

ATTORNEY FOR THE BOARD

E. Pomeranz, Counsel

INTRODUCTION

The appellant had active military service from July 1970 to April 1972.

This matter comes before the Board of Veterans' Appeals (Board) on appeal of a June 2002 rating action by the Department of Veterans Affairs (VA) Regional Office (RO) located in Winston-Salem, North Carolina.

FINDING OF FACT

The appellant's Parkinson's disease is related to his in-service herbicide exposure.

CONCLUSION OF LAW

Parkinson's disease was incurred in active military service. 38 U.S.C.A. § 1110 (West 2002); 38 C.F.R. § 3.303 (2004).

REASONS AND BASES FOR FINDING AND CONCLUSION
I. Veterans Claims Assistance Act of 2000

In November 2000, the Veterans Claims Assistance Act of 2000 (VCAA) was signed into law. See 38 U.S.C.A. §§ 5100, 5102, 5103, 5103A, 5106, 5107, 5126 (West 2002). Regulations implementing the VCAA are applicable to the appellant's claim. 38 C.F.R. §§ 3.102, 3.156(a), 3.159, 3.326 (2004).

With respect to VA's duty to notify, the RO sent the appellant a letter in May 2002, prior to the initial rating decision with regard to the issue on appeal, in which the appellant was notified of the types of evidence he needed to submit, and the development the VA would undertake. See Quartuccio v. Principi, 16 Vet. App. 183 (2002). The letter specifically informed the appellant what was needed from him and what VA would obtain on his behalf. Id. The appellant was also informed of the elements needed to substantiate a service connection claim, including requirements specific to Agent Orange claims. In addition, the Board observes that the October 2002 statement of the case provided the appellant with the text of the relevant portions of the VCAA, as well as the implementing regulations. The Board further notes that there is no indication that there is additional evidence that has not been obtained and that would be pertinent to the present claim. The appellant has been notified of the applicable laws and regulations pertinent to his service connection claim. Moreover, the appellant has been afforded the opportunity to present evidence and argument in support of the claim. Id. Thus, VA's duty to notify has been fulfilled.

VA also has a duty to assist the appellant in obtaining evidence necessary to substantiate a claim. 38 C.F.R. § 3.159(c). The duty to assist includes providing a medical examination or obtaining a medical opinion when such is necessary to make a decision on the claim. In this regard, the Board notes that in August 2004, the appellant underwent a VA examination which was pertinent to his service connection claim. In addition, in February 2004, the Board referred this case for a medical opinion from a Veterans Health Administration (VHA) physician. The Board further observes that in this case, there is no outstanding evidence to be obtained, either by VA or the appellant. Consequently, given the standard of the new regulation, the Board finds that VA did not have a duty to assist that was unmet. The Board also finds, in light of the above, that the facts relevant to this appeal have been fully developed and there is no further action to be undertaken to comply with the provisions of the regulations implementing the VCAA. Therefore, and in light of the decision herein, the appellant will not be prejudiced as a result of the Board proceeding to the merits of the claim. See Bernard v. Brown, 4 Vet. App. 384, 392-94 (1993); see also Mayfield v. Nicholson, No. 02-1077 (U.S. Vet.App. April 14, 2005).
II. Factual Background

The appellant's DD 214, Armed Forces of the United States Report of Transfer or Discharge, shows that he served in the United States Army from July 1970 to April 1972. The appellant's DD 214 also reflects that he served in the Republic of Vietnam from July 1971 to March 1972.

The appellant's service medical records are negative for any complaints or findings of Parkinson's disease. The records show that in March 1972, the appellant underwent a separation examination. At that time, the appellant was clinically evaluated as "normal" for neurologic purposes.

In March 2002, the appellant filed a claim for entitlement to service connection for Parkinson's disease. At that time, he maintained that his currently diagnosed Parkinson's disease was due to his exposure to Agent Orange while he was serving in the Republic of Vietnam.

In June 2002, the RO received private medical records, from February 2000 to June 2001. The records show that in March 2001, the appellant sought treatment from Valerie A. Lasko, M.D., for complaints of a tremor. At that time, Dr. Lasko noted that according to the appellant, he first noticed his tremor in approximately January 2000. When asked about exposure to heavy metals, the appellant reported extensive involvement with lead while working in a remodeling and restoration business. The appellant also stated that he had used pesticides extensively. The assessment was Parkinson's disease and Dr. Lasko indicated that in light of the appellant's history of metal exposure, she would request a heavy metal screen. The records reflect that in June 2001, Dr. Lasko reported that the appellant's heavy metal screen from March 2001 was within normal limits.

A private medical statement from Ellis F. Muther, M.D., dated in June 2002, shows that at that time, Dr. Muther stated that the appellant had a two-year history of Parkinson's disease. According to Dr. Muther, no explanation for the appellant's disorder had been found "except a possible exposure to Agent Orange." Dr. Muther indicated that Agent Orange had been demonstrated to be a neurotoxin, and, as such, he opined that it was highly possible that that was a contributing factor in the etiology of the appellant's Parkinson's disease.

In Fast Letter 03-20, issued by the Veterans Benefits Administration on June 25, 2003, it was noted that a study by the National Academy of Sciences found that the credible evidence against an association between herbicide exposure and Parkinson's disease outweighed the credible evidence for such an association.
In December 2003, the Board remanded this case and requested that the appellant be afforded a VA neurological examination to determine the etiology of any Parkinson's disease found. As per the Board's December 2003 remand decision, in August 2004, the appellant underwent a VA examination. Following the physical examination and a review of the appellant's claims file, the examiner diagnosed the appellant with Parkinson's disease, with a predominant tremor. The examiner noted that in private medical records from Dr. Lasko, Dr. Lasko had referred to the appellant's occupation of remodeling rental homes and the possibility that the appellant was exposed to lead based paints. However, the examiner reported that the appellant's heavy metal screens came back negative which meant that that could be "safely eliminated" as a cause of the appellant's Parkinson's disease. Next, in order to answer the question as to whether Agent Orange caused the appellant's Parkinson's disease, the examiner noted that an extensive three-day literature review was conducted. The examiner listed numerous medical articles which addressed the relationship between herbicide agents and neurological disorders, including Parkinson's disease. Following a review of the medical literature obtained, the examiner opined that it was at least as likely as not that the appellant's Parkinson's disease may be related to exposure to Agent Orange or other herbicide exposure in Vietnam.

A private medical statement from Joel C. Morgenlander, M.D., Associate Professor of Medicine (Neurology), dated in October 2004, shows that at that time, Dr. Morgenlander stated that he had first seen the appellant in October 2001 and had diagnosed him with probable Parkinson's disease. According to Dr. Morgenlander, the appellant's symptoms began in approximately 2000 or 2001.

In February 2004, the Board referred this case for a medical opinion from a VHA medical doctor with the necessary expertise in the treatment of Parkinson's disease. The Board noted that due to the appellant's service in the Republic of Vietnam during the Vietnam era, he was presumed to have been exposed to herbicide agents, including Agent Orange, during his period of active military service. Thus, the Board requested that the VHA physician, after reviewing the appellant's claims file, offer an opinion with respect to the following question: Whether it was at least as likely as not that the appellant's Parkinson's disease was related to his period of military service, to specifically include his presumed exposure to herbicide, including Agent Orange, while in Vietnam.

A VHA opinion from the Chief, Neurology Service, was provided on March 30, 2005. In the opinion, the VHA neurologist stated that the 2002 Update "Veterans and Agent Orange" published by the Institute of Medicine summarized several epidemiologic studies, most of which suggested a mildly increased risk of Parkinson's disease in individuals "with many years of occupational exposure" to herbicides or
pesticides. According to the VHA neurologist, no particular association had been demonstrated for any single chemical or class of compounds, and no association with exposure to "2,4-D, 2,4,5-T, or TCDD" had been published. The VHA neurologist stated that the Institute of Medicine concluded that although an etiologic connection between pesticide/herbicide exposure was "biologically plausible," there was insufficient evidence at present to support a definite association between Parkinson's disease and "2,4-D, 2,4,5-T, or TCDD." According to the VHA neurologist, to his knowledge, since the 2002 Update from the Institute of Medicine, there had been no more recent epidemiologic or biochemical studies indicating a definite association between Parkinson's disease and "2,4-D, 2,4,5-T, or TCDD." Thus, in reviewing the appellant's medical records, the VHA neurologist stated that he did not find any details of the appellant's particular military service or of his neurologic condition to lead to any conclusion different from that of the Institute of Medicine, namely that there was no definite etiologic link between Agent Orange exposure and subsequent Parkinson's disease.

III. Analysis

The law provides that service connection may be established for chronic disability resulting from disease or injury incurred in or aggravated by service. 38 U.S.C.A. § 1110; 38 C.F.R. § 3.303. In addition, service connection may be granted for any disease diagnosed after discharge, when all the evidence, including that pertinent to service, establishes that the disease was incurred in service. 38 C.F.R. § 3.303(d).

The Board notes that a change in the law has taken place with respect to the adjudication of claims based upon exposure to Agent Orange during service in Vietnam. On December 27, 2001, the Veterans Education and Benefits Expansion Act of 2001 (VEBEA), Pub. L. No. 107-113, 115 Stat. 976 (2001) was signed into law. That new statute, in pertinent part, redesignated and amended 38 U.S.C.A. § 1116(f) to provide that, for purposes of establishing service connection for a disability or death resulting from exposure to an herbicide agent, including a presumption of service connection under this section, a veteran who, during active military, naval, or air service, served in Vietnam during the period beginning on January 9, 1962, and ending on May 7, 1975, shall be presumed to have been exposed during such service to an herbicide agent of the kind specified in section 1116, unless there is affirmative evidence to establish that the veteran was not exposed to any such agent during that service. See 38 U.S.C.A. § 1116 (West 2002).

If a veteran was exposed to an herbicide agent during active military, naval, or air service, the following diseases shall be service-connected if the requirements of 38
U.S.C.A. § 1116 and 38 C.F.R. § 3.307(a)(6)(iii) are met, even though there is no record of such disease during service, provided further that the rebuttable presumption provisions of 38 U.S.C.A. § 1113 and 38 C.F.R. § 3.307(d) are also satisfied: chloracne or other acneform disease consistent with chloracne; type II diabetes mellitus; Hodgkin's disease; multiple myeloma; non-Hodgkin's lymphoma; acute and subacute peripheral neuropathy; porphyria cutanea tarda (PCT); prostate cancer; respiratory cancers (cancer of the lung, bronchus, larynx, or trachea); and soft-tissue sarcomas (other than osteosarcoma, chondrosarcoma, Kaposi's sarcoma, or mesothelioma). 38 C.F.R. § 3.309(e) (2004); see also 38 U.S.C.A. § 1113 (West 2002); 38 C.F.R. § 3.307 (2004).

VA has determined that a presumption of service connection based on exposure to herbicides used in the Republic of Vietnam during the Vietnam era is not warranted for any condition for which VA has not specifically determined a presumption of service connection is warranted. See 59 Fed. Reg. 341-46 (1994); 61 Fed. Reg. 414421 (1996); see also 64 Fed. Reg. 59232 (1999); 67 Fed. Reg. 42600-42608 (2002). More recently, VA clarified that a presumption of service connection based on exposure to herbicides used in the Republic of Vietnam during the Vietnam Era is not warranted for the following conditions: hepatobiliary cancers, nasopharyngeal cancer, bone and joint cancer, breast cancer, cancers of the female reproductive system, urinary bladder cancer, renal cancer, testicular cancer, leukemia (other than CLL), abnormal sperm parameters and infertility, Parkinson's disease and parkinsonism, amyotrophic lateral sclerosis (ALS), chronic persistent peripheral neuropathy, lipid and lipoprotein disorders, gastrointestinal and digestive disease, immune system disorders, circulatory disorders, respiratory disorders (other than certain respiratory cancers), skin cancer, cognitive and neuropsychiatric effects, gastrointestinal tract tumors, brain tumors, light chain-associated (AL) amyloidosis, endometriosis, adverse effects on thyroid homeostasis, and any other condition for which the Secretary has not specifically determined a presumption of service connection is warranted. See 68 Fed. Reg. 27,630-41 (May 20, 2003).

Following consideration of the evidence of record, the Board finds that entitlement to service connection for Parkinson's disease is warranted. Initially, it is noted that the appellant's DD 214 confirms that the appellant had active service in Vietnam during the Vietnam era. Therefore, the appellant is presumed to have been exposed to herbicides in service. 38 U.S.C.A. § 1116(f). However, the Board also observes that Parkinson's disease is not among the disabilities listed in 38 C.F.R. § 3.309(e). Thus, the appellant may not receive the benefit of a rebuttable presumption that his Parkinson's disease was caused by exposure to Agent Orange. 38 C.F.R. §§ 3.307, 3.309 (2004). Nevertheless, as indicated above, the appellant is not precluded from establishing service connection on a direct basis. See Combee v. Brown, 34 F.3d 1039 (Fed.Cir. 1994); McCartt v. West, 12 Vet. App. 164, 167 (1999).

Upon a review of the evidence of record, the evidence taken as a whole tends toward the conclusion that the appellant's Parkinson's disease was caused by his herbicide exposure while in service. The Board recognizes that, as previously stated, in Fast Letter 03-20, issued by the Veterans Benefits Administration on June 25, 2003, it was noted that a study by the National Academy of Sciences study found that the credible evidence against an association between herbicide exposure and Parkinson's disease outweighed the credible evidence for such an association. However, in support of the appellant's contention that his Parkinson's disease was due to his exposure to Agent Orange while he was serving in the Republic of Vietnam, the appellant has submitted a private medical statement from Dr. Muther, dated in June 2002. In the June 2002 statement, Dr. Muther indicated that no explanation for the appellant's diagnosed Parkinson's disease had been found "except a possible exposure to Agent Orange." Dr. Muther further noted that Agent Orange had been demonstrated to be a neurotoxin, and, as such, he opined that it was highly possible that that was a contributing factor in the etiology of the appellant's Parkinson's disease. In addition, in the appellant's August 2004 VA examination, the examiner stated that although it was possible that the appellant was exposed to lead based paints due to his occupation of remodeling rental homes, in light of the fact that the appellant's heavy metal screens came back negative, such exposure could be "safely eliminated" as a cause of the appellant's Parkinson's disease. Moreover, following a review of pertinent medical literature, the examiner opined that it was "at least as likely as not" that the appellant's Parkinson's disease may be related to exposure to Agent Orange or other herbicide exposure in Vietnam. Furthermore, in the March 2005 opinion from the VHA neurologist, although he stated that his conclusion was no different from that of the Institute of Medicine, namely that there was no definite etiologic link between Agent Orange exposure and subsequent
Parkinson's disease, the VHA neurologist also noted that the Institute of Medicine had concluded that an etiologic connection between pesticide/herbicide exposure was "biologically plausible."

The medical opinions in this case are less than absolute in their conclusions. However, given the nature of cases such as this one, in which most causes of the claimed disability are idiopathic and the passage of a significant amount of time between separation from service and the filing of a claim with VA, and the fact that medicine is still a somewhat inexact science, the Board must resign itself to dealing with medical opinion evidence couched in terms such as "highly possible," "at least as likely as not," and "biologically plausible," rather than absolutes. See Lathan v. Brown, 7 Vet. App. 359, 366 (1995) (medicine is more art than exact science). Although none of the medical opinions of record are couched in terms of absolute certainty, none have to be.

In any event, the standard of review which must be applied by the Board is found in 38 U.S.C.A. § 5107(b). Under the benefit-of-the-doubt rule, in order for a claimant to prevail, there need not be a preponderance of the evidence in the veteran's favor, but only an approximate balance of the positive and negative evidence. In other words, the preponderance of the evidence must be against the claim for the benefit to be denied. Gilbert v. Derwinski, 1 Vet. App. 49, 54 (1990). Thus, in consideration of the aforementioned evidence, the Board finds that the evidence for and against the appellant's claim for service connection for Parkinson's disease is in a state of relative equipoise. With reasonable doubt resolved in the appellant's favor, the Board concludes that service connection is warranted.

ORDER

Entitlement to service connection for Parkinson's disease is granted.

____________________________________________
JOY A. MCDONALD
Veterans Law Judge, Board of Veterans' Appeals

Department of Veterans Affairs
On appeal from the Department of Veterans Affairs (VA) Regional Office (RO) in Winston-Salem, North Carolina

THE ISSUE

Entitlement to service connection for a neurological disorder, claimed as due to in-service herbicide exposure.

REPRESENTATION

Appellant represented by: The American Legion

WITNESSES AT HEARING ON APPEAL

The veteran and his son.

ATTORNEY FOR THE BOARD

L. Cryan, Counsel

INTRODUCTION

The veteran had active service from June 1966 to October 1969, with approximately four months of additional prior service.

This matter comes before the Board of Veterans' Appeals (Board) from a March 1994 rating decision of the RO, which denied the veteran's claim seeking entitlement to service connection for a neurological disorder, claimed as peripheral neuropathy, due to alleged exposure to Agent Orange while in Vietnam. The veteran submitted a notice of disagreement with that rating decision in May 1994. In July 1994, he was provided with a statement of the case. His substantive appeal was received in September 1994.

The Board notes that the veteran had previously claimed entitlement to service connection for a neurological disorder, claimed as Parkinson's disease, due to alleged exposure to Agent Orange while in Vietnam, which was denied by an October 1988 rating decision. The veteran submitted a notice of disagreement with that rating decision in January 1989. In February 1989, he was provided with a statement of the case. His substantive appeal was received in March 1989.
The matter was received at the Board in October 1989 but was referred back to the RO pending review and revision of herbicide regulations. The RO then also deferred a decision on the claim pending updated proposed regulations.

As noted in a June 1999 remand by the Board, the RO, in the currently appealed March 1994 rating decision essentially considered both the claimed peripheral neuropathy and the claimed Parkinson's disease. Given that the veteran has claimed service connection for a neurological disorder, initially claimed as Parkinson's disease and subsequently claimed as peripheral neuropathy, and given that the veteran's claims were essentially one continuous claim for the same neurological disorder, the Board has simply characterized the veteran's claim as entitlement to service connection for a neurological disorder, claimed as due to Agent Orange exposure. The issue has been so identified on the title page hereinabove.

The veteran testified at a personal hearing before the undersigned Veterans Law Judge, sitting at the RO in September 1997. A transcript of his testimony is associated with the claims file.

Finally, it is noted that the case was previously twice before the Board and was remanded to the RO in January 1998 and June 1999 for additional evidentiary development. Following compliance with the Board's directives on Remand, the case is now returned to the Board for further appellate consideration.

FINDINGS OF FACT

1. The veteran had active military service in the Republic of Vietnam during the Vietnam era, and is therefore presumed to have been exposed to herbicide agents in service.

2. The veteran has a currently diagnosed neurological disorder with Parkinson-like characteristics, also referred to as Parkinsonism.

3. The veteran's neurological disorder may not be presumptively service connected under the provisions of 38 C.F.R. § 3.309(e).

4. The competent and probative medical opinions of record have determined that the veteran's currently diagnosed neurological disorder is at least as likely as not due to in-service exposure to Agent Orange.

CONCLUSION OF LAW

With resolution of all doubt in the veteran's favor, the veteran's currently diagnosed neurological disorder, referred to as Parkinsonism and Parkinson-like syndrome, was incurred

REASONS AND BASES FOR FINDINGS AND CONCLUSION

The veteran asserts that service connection is warranted for his neurological disorder with symptomatology which mirrors that of Parkinson's disease, which he claims is due to in-service herbicide exposure in the Republic of Vietnam.

I. Duties to Notify and Assist

At the outset, the Board notes that on November 9, 2000, the Veterans Claims Assistance Act of 2000 (VCAA) was enacted. See 38 U.S.C.A. §§ 5103, 5103A (West 2002). Among other things, the VCAA amended 38 U.S.C.A. § 5103 to clarify VA's duty to notify claimants and their representatives of any information and evidence that is necessary to substantiate the claim for benefits. The VCAA also created 38 U.S.C.A. § 5103A, which codifies VA's duty to assist, and essentially states that VA will make reasonable efforts to assist a claimant in obtaining evidence necessary to substantiate a claim. Implementing regulations for the VCAA were subsequently enacted, which were also made effective November 9, 2000, for the most part. See 66 Fed. Reg. 45,620 (Aug. 29, 2001) (codified at 38 C.F.R. §§ 3.102, 3.159). The intended effect of the implementing regulations was to establish clear guidelines consistent with the intent of Congress regarding the timing and scope of assistance VA will provide to claimants who file a claim for benefits. See 66 Fed. Reg. 45,620 (Aug. 29, 2001). Both the VCAA and the implementing regulations are applicable in the present case, and will be collectively referred to as "the VCAA."

Pertinent to the merits of the veteran's claim of entitlement to service connection for a neurological disorder, the Board finds that the provisions of the VCAA have been complied with. In light of the complete grant of benefits sought on appeal (entitlement to service connection for a neurological disorder), no further evidence is necessary to substantiate the veteran's claim for service connection. See 38 U.S.C.A. § 5103(a) (West 2002). In this veteran's case, there is no reasonable possibility that further assistance would aid in substantiating the claim for VA compensation benefits. See 38 U.S.C.A. § 5103A(a)(1),(2) (West 2002). Also, further notice to the veteran concerning the evidence necessary to substantiate his claim or regarding responsibilities in obtaining evidence would serve no useful purpose.

II. Factual Background

The veteran's service medical records are negative for complaints, findings, or diagnosis of a neurological disorder of any kind.
A review of the post-service evidentiary record reveals numerous medical records clearly indicating that the veteran has been diagnosed with a neurological disorder, although there has been some degree of variance in the precise nature of that diagnosis.

A September 1982 private neurology consultation report prepared by Dr. K, indicated that the veteran was experiencing progressive weakness of the left side of the body and noted that he had a history of having polio of the left arm and left leg when he was age 22 months. There was also a history of having served two tours of duty in Vietnam. Other numerous subsequent private medical records from the neurological offices of Dr. L as well as from a Duke University Medical Center doctor indicated that the veteran began experiencing left sided numbness in 1982 and there are several diagnoses of Parkinsonism shown from 1983. At one time, it was thought that the veteran's neurological symptoms could be attributed to basal ganglia disease. Other doctors noted that the veteran may not have a pure form of Parkinsonism, but instead, a Parkinson-plus syndrome such as a progressive supranuclear palsy.

The veteran underwent a VA neurological examination in July 1988, and he gave a history of having been exposed to dioxins in service. The veteran reported symptoms to include slowness of movement, muscle stiffness, poor coordination, slurred speech, excessive salivation, muscle twitching, muscle cramps, tremor, and involuntary movements. The examiner noted that the veteran had many features of Parkinson's Disease, but noted that his picture was unusual. First, the examiner pointed out that the veteran was young. Next, the examiner noted that he could never see the true resting tremor, and muscle tone was not significantly increased on testing. On the other hand, he showed a lot of the variability of muscle function that one did see in Parkinsonism, and he could never convince himself that the veteran was functional. Therefore, the examiner diagnosed a neurological problem that was similar to Parkinson's and may be a variety of such. He further stated that he was not aware of the veteran's picture being seen with dioxin exposure, but would defer that question to those with more knowledge in that field.

Amongst other medical evidence of record is a July 1990 letter from Dr. R, a PhD and toxicologist, with the State of North Carolina, Department of Environment, Health, and Natural Resources, Division of Epidemiology. Dr. R referred to various scientific literature indicating a possible relationship between dioxin exposure and various neurological disorders, indicating that one cannot rule out the possible role of dioxin as a causal agent for various neurological disorders, including Parkinson's disease. Dr. R noted that recent scientific literature had brought to light the possibility of environmental causes of neurological disorders, as opposed to genetic causes, and that this may be the reason why more young people were developing that
Also of record is a report of Dr. R, dated February 1991 and titled "[veteran's name] - A Possible Association Between His Current Medical Problems and Exposure to Agent Orange in Vietnam." This 21 page report, plus attached appendices, contained references to numerous scientific studies and literature discussing potential neurological health effects of dioxin exposure. Dr. R further indicated that it did not appear that the veteran had true Parkinson's but instead had Parkinson like symptoms and based upon an evaluation of scientific literature, Dr. R stated that it was possible, indeed quite probable, that the veteran's condition may stem from past Agent Orange exposure. It is noted that Dr. R's opinion was based upon the veteran's history of having been stationed in Dong Ha and Quang Tri and having been subject to significant amounts of Agent Orange spraying in those areas. Dr. R elsewhere stated that the veteran "was in an area that was subject to extensive Agent Orange treatment and exposure for an extended period of time."

In December 1991, Dr. L prepared a memorandum in which he notes that the sum total of all of the information provided by Dr. R's report suggested an association between the veteran's exposure to dioxin and possible development of neurological symptoms.

At his personal hearing before the undersigned Veterans Law Judge in September 1997, the veteran testified that he was in good health at the time he was discharged from service. The veteran testified that he could find no family history of a neurological disorder, and that he had never had any kind of traumatic injuries that may have stimulated a central nervous system dysfunction. Furthermore, the veteran testified that he never worked in any type of environment where he was exposed to excessive chemicals.

Following the Board's January 1998 Remand, the veteran underwent a VA neurological examination in September 1998. It was noted that the veteran was in a wheelchair due to an inability to control his movements, especially on the left. Movements of his face were symmetric but limited in excursion. He could move his tongue perhaps half of the normal range. He was constantly writhing. He had normal strength in the deltoids, triceps and biceps, but the testing was interrupted by constant twisting, as severe as the examiner had seen in many years. The examiner found it interesting that sensation was within normal limits, including superficial sensation, traced figures, and vibration in four extremities, and joint sense in the lower extremities. The provided a history of dioxin exposure in service. The diagnosis was that of chronic severe generalized choreoathetosis with preserved reflexes, strength, sensation, and intellect. The examiner specifically found that this was not an acute or sub-acute peripheral neuropathy. It was also stated that there was a moderate to strong possibility that the veteran's bizarre
movements were a result of exposure to some chemical during his service.

In the June 1999 remand, the Board pointed out that the aforementioned report of Dr. R and the most recent VA examination provided clear medical evidence of a current diagnosis of a neurological disorder, and further supported a nexus between the current diagnosis and chemical exposure in service, more specifically dioxin exposure. However, at the time of the June 1999 remand, VA laws and regulations did not permit a presumption of exposure to Agent Orange based solely on having served in-country during the Vietnam Era. As such, the case was remanded for development, specifically to determine if the veteran was likely exposed to Agent Orange during service in Vietnam.

In the meantime, it was confirmed that the veteran served in Vietnam from April 28, 1967 to December 3, 1967 and from June 26, 1969 to September 26, 1969.

In March 2001, the case was referred to the VA Under Secretary for Health for review and preparation of a medical opinion. In response, VA's Chief Public Health and Environmental Hazards Officer first noted that the veteran had documented exposure to herbicides based on Department of Defense records of Agent Orange spraying. The opinion also noted that in its most recent report, Veterans and Agent Orange Update 1998, the Institute of Medicine (IOM) committee concluded that there was inadequate/insufficient evidence to determine whether an association exists between exposure used in Vietnam and motor/coordination dysfunction. Parkinson's Disease was included in this group of disorders by the IOM. In light of the foregoing, the Chief Public Health and Environmental Hazards Officer opined that it was possible that the veteran's neurological disorder could be attributed to exposure to herbicides; however, she could not state that Agent Orange exposure was likely or at least as likely as not to be responsible.

In response to the opinion from the Under Secretary of Health, the RO opined, as a result of the opinion by the Under Secretary, and following review of the evidence in its entirety, the veteran's disability was not the result of Agent Orange exposure.

In a December 2001 memorandum, Dr. G, of the Washington Hospital Center, noted a possible etiology of the veteran's neurological disorder. Dr. G noted the veteran's history of Parkinson-like symptoms, Parkinsonism, and the lack of a clearly defined neurological syndrome dating back to 1982.

Dr. G noted that the initial medical evaluations of the veteran's condition performed by several neurologists seemed to be suggestive of a neurological syndrome that had some similarities to Parkinson's disease, but in some respects was quite different. Dr. G found that such a neurological condition, initially diagnosed at a young age raised several
questions concerning a possible etiology. Dr. G found that the significant evidence provided by Dr. R strongly suggested that the offending agent may have been dioxin found in Agent Orange preparation. Dr. G found that Dr. R's research in that subject provided overwhelming support of a probable nexus between exposure to those compounds and the manifestations of acute and chronic neurological damage as well as the myriad symptoms experienced by the veteran. In conclusion, Dr. G opined that it was as likely as not that the highly unusual symptomatology observed in the veteran at an extremely young age was related to his exposures during service.

In response to Dr. G's medical opinion, the Chief Officer of the Office of Public Health and Environmental Hazards prepared another opinion, in March 2002, regarding the possible etiology of the veteran's neurological disorder. The opinion noted that the most recent report from the National Academy of Sciences "Veterans and Agent Orange-Update 2000" reaffirmed their earlier conclusions on the lack of an association between herbicide exposure and Parkinson's or related neurological diseases. It was noted that the National Academy of Sciences further stated that in the future, as diagnostic accuracy for Parkinson's disease improved, and when herbicide exposure assessment is quantitated with specific biomarkers, and further research confirms the gene-toxicant interaction in larger prospective studies of Parkinson's disease, the evidence for association may change. The Chief Officer of the Office of Public Health and Environmental Hazards found, in essence, that Dr. R's 1991 opinion was outdated, and not supported by National Academy of Sciences current review of medical and scientific evidence. It was noted that VA relied extensively upon the independent and highly credible medical and scientific analysis provided by the NAS in establishing associations between herbicide exposure and health effects in veterans. Therefore, she again opined that it was possible that the Parkinson's or Parkinson's-like disease diagnosed in the veteran could be related to exposure to herbicide in service. However, they could not state that it was as likely, or more likely than not, that his disease was the result of herbicide exposure.

In light of the aforementioned March 2002 opinion, the RO found that there was no reasonable possibility that the veteran's Parkinson's or Parkinson's-like disease resulted from exposure to herbicides in service.

After the case was returned to the Board, the Board determined that an Independent Medical Opinion might be useful in determining the likely etiology of the veteran's neurological disability.

In the meantime, both Dr. R and Dr. G responded to the March 2002 opinion by the Under Secretary of Health. Dr. R provided a lengthy report nearly 25 pages long noting a more likely than not association between the veteran's current
medical problem and exposure to Agent Orange during service in Vietnam. In essence, the report finds an environmental link to Parkinson-like symptoms illustrated by numerous physicians in medical and scientific literature cited to by Dr. R in his report. Moreover, Dr. R noted that the National Academy of Sciences, in their report "Veterans and Agent Orange-Update 2002, were in general agreement with the information presented in his report. Dr. R concluded that because the evidence shows that the veteran was directly exposed to Agent Orange, and because the new medical peer-reviewed literature evidence presented in the report and the strength of the individual epidemiological studies relating the veteran's exposure to Agent Orange in Vietnam, it was more likely than not that the Vietnam exposure to Agent Orange is responsible for his present medical condition; and that the new and existing information in the report, more likely than not links the veteran's Agent Orange exposure to his current neurological problems.

In a January 2005 response to the Board's request for an independent medical opinion, a doctor from the Washington University School of Medicine noted that he reviewed the medical record and did not feel that he could render an opinion regarding the relationship between the veteran's exposure to Agent Orange and his Parkinsonion syndrome with the available information. The doctor noted that he would have to perform a history and examination of the veteran to determine the likely cause of his Parkinsonism.

Dr. G noted that the evidence indicated that the veteran was exposed to Agent Orange during service, and also noted Dr. R's more recent medical data supporting a strong association between dioxins and subsequent manifestations of acute and chronic neurological damage as well as the myriad symptoms experienced by the veteran. Dr. G therefore concluded that the additional data gave further support to the case, and opined that it was as likely as not that the highly unusual symptomatology observed in the veteran at an extremely young age is related to his Agent Orange exposure during service.

III. Legal Criteria and Analysis

Under the relevant regulations, service connection may be granted for a disability resulting from disease or injury incurred in or aggravated by active service. 38 U.S.C.A. § 1110 (West 2002). If a chronic disease is shown in service, subsequent manifestations of the same chronic disease at any later date, however remote, may be service connected, unless clearly attributable to intercurrent causes. 38 C.F.R. § 3.303(b) (2004). However, continuity of symptoms is required where the condition in service is not, in fact, chronic or where diagnosis of chronicity may be legitimately questioned. 38 C.F.R. § 3.303(b) (2004).

Further, service connection may also be granted for any disease diagnosed after discharge, when all the evidence,
including that pertinent to service, establishes that the
disease was incurred in service. 38 U.S.C.A. § 1113(b) (West
2002); 38 C.F.R. § 3.303(d) (2004). The Board must determine
whether the evidence supports the claim or is in relative
equipoise, with the appellant prevailing in either case, or
whether the preponderance of the evidence is against the
claim, in which case, service connection must be denied.

Additionally, where a veteran served continuously 90 days or
more during a period of war or during peacetime service after
December 31, 1946, and an organic disease of the nervous
system becomes manifest to a degree of at least 10 percent
within one year from the date of termination of service, such
a disease shall be presumed to have been incurred in or
aggravated by service, even though there is no evidence of
such a disorder during the period of service. 38 U.S.C.A. §§
1101, 1112, 1113 (West 2002); 38 C.F.R. §§ 3.307, 3.309
(2004).

In addition to the regulations governing entitlement to
service connection outlined above, 38 C.F.R. § 3.309(e)
provides that if a veteran was exposed to an herbicide agent
during active military, naval, or air service, the diseases
set forth in 38 C.F.R. § 3.309(e) shall be service-connected
if the requirements of 38 C.F.R. § 3.307(a)(6) are met even
though there is no record of such disease during service,
provided that the rebuttable presumption provisions of 38
C.F.R. § 3.307(d) are also satisfied. These diseases include
chloracne, Type 2 diabetes (also known as Type II diabetes
mellitus or adult-onset diabetes), Hodgkin's disease,
multiple myeloma, Non-Hodgkin's lymphoma, acute and subacute
peripheral neuropathy, porphyria cutanea tarda, prostate
cancer, respiratory cancers (including cancer of the lung,
bronchus, larynx, or trachea), and soft-tissue sarcoma. 38
U.S.C.A. § 1116(a)(2) (West 2002); 38 C.F.R. § 3.309(e)
(2004). For purposes of this section, the term acute and
subacute peripheral neuropathy means transient peripheral
neuropathy that appears within weeks or months of exposure to
an herbicide agent and resolves within two years of the date
of onset. 38 C.F.R. § 3.309(e), Note 2 (2004).

A veteran who, during active military, naval, or air service,
served in the Republic of Vietnam during the period beginning
on January 9, 1962, and ending on May 7, 1975, shall be
presumed to have been exposed during such service to an
herbicide agent, unless there is affirmative evidence to
establish that the veteran was not exposed to any such agent
during that service. The last date on which such a veteran
shall be presumed to have been exposed to an herbicide agent
shall be the last date on which he served in the Republic of
Vietnam during the Vietnam era. 38 U.S.C.A. § 1116 (West
2002); 38 C.F.R. § 3.307(a)(6)(iii) (2004). The Board notes
that in June 2003, 38 C.F.R. § 3.307(a)(6)(iii) was amended
to expand the presumption of exposure to herbicides to
include all Vietnam veterans, not just those who have a
disease on the presumptive list in 38 U.S.C.A. § 1116(a)(2)
Despite the presumption of in-service herbicide exposure in Vietnam, the Board is not in a position to grant service connection for the veteran's neurological disorder on a presumptive basis as due to herbicide agent exposure, as the veteran's neurological disorder, manifested by Parkinson-like symptomatology, did not "[appear] within weeks or months of exposure to a herbicide agent and [resolve] within two years of onset." 38 C.F.R. § 3.309(e), Note 2. However, in the case of Combee v. Brown, 34 F. 3d 1039 (Fed Cir. 1994), the United States Court of Appeals for the Federal Circuit (Federal Circuit) held that a veteran was not precluded from presenting proof of direct service connection between a disorder and exposure even if the disability in question was not among conditions enumerated under the Veterans' Dioxin and Radiation Exposure Compensation Standards Act, the presumption not being the sole method for showing causation.

Hence the veteran may establish service connection for a neurological disorder by presenting evidence establishing that it is at least as likely as not that his neurological disorder, also referred to as Parkinsonism and Parkinson-like symptoms, was caused by his presumed in-service herbicide agent exposure. 38 U.S.C.A. § 5107(b) (West 2002); 38 C.F.R. § 3.303 (2004); Gilbert v. Derwinski, 1 Vet. App. 49 (1990).

In this case, the medical evidence of record tends to support the veteran's assertions that his neurological disorder was caused by in-service herbicide exposure. More specifically, the medical opinions of Drs. L, R, and G have found an association between the veteran's unusual neurological disability and exposure to Agent Orange in service. These doctors have repeatedly noted the unusual symptomatology, similar to, yet different in some respects, from Parkinson's Disease. These doctors have also repeatedly noted that the onset of the veteran's symptoms at a young age was unique, supporting the notion that exposure to herbicides during service as likely as not led to the veteran's neurological disorder. Furthermore, Dr. R, who specializes in toxicology, provided a complete rationale for his opinion, providing a report of extensive research which cited multiple medical and scientific data.

Moreover, the VA physicians who examined the veteran during the pendency of this appeal also provided opinions that it was at least as likely as not that the veteran's presumed in-service herbicide exposure led to the current neurological disorder.

The Board is mindful that VA's Chief Public Health and Environmental Hazards Officer provided opinions in 2001 and 2002 in which she stated that there was inadequate/insufficient medical/scientific evidence to
determine whether an association existed between exposure to herbicides and neurological dysfunction. She also noted that Dr. R's 1991 opinion was based on outdated research.

However, Dr. R provided a second opinion noting a likely association between the veteran's in-service herbicide exposure and his neurological disorder, based on updated scientific and medical information.

In sum, several private and VA doctors have found an as likely as not association between the veteran's in-service herbicide exposure and his current neurological disorder. Although VA's Chief Public Health and Environmental Hazards Officer could not state that it was at least as likely as not that the veteran's neurological disorder was caused by in-service herbicide exposure, she did state that it was possible.

In light of the foregoing, the Board finds that the preponderance of the competent and probative evidence supports the veteran's claim because all of the opinions of record find either that it is possible, or at least as likely as not, that the veteran's neurological disorder resulted from in-service herbicide exposure. In the absence of clear contrary medical evidence, the opinion of the Chief Officer of the Office of Public Health and Environmental Hazards, coupled with the multiple positive opinions by Drs. L, R, G and the VA doctors, provide a preponderance of the evidence supporting the claim for entitlement to service connection for a neurological disorder, referred to as Parkinsonism and Parkinson-like syndrome. Accordingly, a grant of service connection is warranted. 38 U.S.C.A. §§ 1110, 5107(b) (West 2002); 38 C.F.R. § 3.303(d) (2004).

ORDER

Service connection for a neurological disorder, referred to as Parkinsonism and Parkinson-like syndrome, is granted.

____________________________________________
BARBARA B. COPELAND
Veterans Law Judge, Board of Veterans' Appeals

Department of Veterans Affairs
Sentry enzyme blocks two paths to Parkinson's disease

St. Jude study shows the enzyme GST pi stands at the crossroads of several pathways that lead to the death of dopaminergic neurons and prevents both cell degeneration and cell suicide

The degeneration of brain cells that occurs in Parkinson's disease may be caused by either externally provoked cell death or internally initiated suicide when the molecule that normally prevents these fatal alternatives is missing, according to studies in mouse models by investigators at St. Jude Children's Research Hospital.

Parkinson's disease is a disease in which nerve cells in part of the brain called the substantia nigra die, resulting in the loss of dopamine, a nerve-signaling molecule that helps control muscle movement. The absence of dopamine from these cells, called dopaminergic neurons, causes a loss of muscle control, trembling and lack of coordination.

The molecule that prevents damage to the substantia nigra is an enzyme called GST pi ("pie"). This molecule stands like a sentry at the crossroads of several biochemical pathways, any one of which can lead to Parkinson's disease, the researchers reported in an article in the Feb. 1 early online edition of Proceedings of the National Academy of Sciences.

The job of the antioxidant GST pi is to protect the cell from death caused by either environmental toxins (externally evoked cell death), such as herbicides and pesticides, or a self-destruction process called apoptosis (cell suicide), triggered by certain stressful conditions in the cell. If GST pi levels are reduced or this enzyme is overwhelmed by toxins, these nerves are at increased risk of death. Previous research has shown that the ability of GST pi to protect cells against toxic molecules is directly linked to the ability of cancer cells with excessive amounts of this enzyme to reduce the effectiveness of chemotherapy.

The finding that GST pi plays a key role in preventing Parkinson's disease suggests that measuring levels of this enzyme might be an effective way to determine individuals at risk for developing this disease, according to Richard Smeyne, Ph.D., an associate member of the Department of Developmental Neurobiology at St. Jude. "In the future, treatments that increase GST pi levels in the substantia nigra might help to prevent or delay the onset of Parkinson's disease or reduce its severity," said Smeyne, the report's senior author.

GST pi is one of a family of similar enzymes that eliminate free radicals generated by pesticides and other chemicals. Two members of this family are present in the brain, but only one of them, GST pi, is found in the dopaminergic neurons of the substantia nigra. Free radicals are highly unstable molecules that readily interact with other molecules, causing cell damage.
The study sheds light on the cause of most cases of Parkinson's disease, which currently are unexplained. "The majority of these cases of Parkinson's disease appear to arise because individuals who have a genetic susceptibility to the disease are exposed to environmental toxins such as pesticides and herbicides, which trigger the formation of free radicals that kill dopaminergic neurons in the substantia nigra," Smeyne said. "We also know that GST pi blocks the process of cell suicide triggered by stresses that the cell can't overcome, such as an increase in the presence of free radicals or a loss of the cell's ability to produce energy."

Smeyne's team showed that of the two known types of GST in the brain, only GST pi was present in the dopaminergic neurons that are lost in Parkinson's disease. The scientists then treated two different populations of mice with MPTP, a chemical that causes loss of these cells, in order to determine if levels of GST pi changed. In mice known to be sensitive to MPTP, there was a complete but transient loss of GST pi in the dopaminergic neurons of the substantia nigra, while the same area of the brain in MPTP-resistant mice never completely lost GST pi and recovered their original levels within 12 hours.

In addition, the team showed that when MPTP-resistant mice were treated with this drug, the presence of GST pi in the dopaminergic neurons prevented activation of cJUN, a molecule that triggers apoptosis. These findings are evidence that GST pi prevents apoptosis in dopaminergic neurons of the substantia nigra, Smeyne said.

The investigators also showed in cell culture studies that blocking production of GST pi in substantia nigra cells left them vulnerable to MPTP, causing a significant death rate among these cells. In addition, when the investigators blocked GST pi production in the dopaminergic neurons of the substantia nigra, about one-quarter of them died, even though they were not treated with MPTP. "This suggests that even in the absence of MPTP the enzyme GST pi plays a critical role in preventing cell death that may occur with the natural buildup of free radicals," Smeyne said.

Finally, the investigators studied the effect of MPTP on the substantia nigra of normal, "wild-type" mice and mice that lacked one (+/-) or both (-/-) genes for GST pi. Wild-type and GST pi (+/-) mice showed similar resistance to MPTP, but GST pi (-/-) mice lost slightly less than half of their dopaminergic neurons following treatment with MPTP. Six hours after MPTP treatment, the formation of free radicals increased 300 percent in the substantia nigra of GST pi (-/-) mice compared with the substantia nigra of GST pi (+/+). These results are additional evidence that GST pi may play an important role in preventing Parkinson's disease," Smeyne said.

Results of the St. Jude study showing the importance of GST pi could help to explain previous work by other researchers linking loss of this enzyme to destruction of dopaminergic neurons. For example, there is some evidence that alterations in the gene for GST pi are linked to increased risk of Parkinson's disease after pesticide exposure. Also, although most Parkinson's disease cases have no known cause, experts believe that they are caused by the interaction of genetic susceptibility to Parkinson's disease with exposure to a variety of environmental factors, such as pesticides and herbicides.

"Therefore, the new findings bring researchers a step closer to understanding why Parkinson's disease occurs and potentially how to develop more effective treatments for it," Smeyne said.
Other authors of this study include St. Jude researchers Michelle Smeyne, Justin Boyd and Kennie Ravie Shepherd, who did most of the work; Yun Jiao, Brooks Barnes Pond and Matthew Hatler (St. Jude); and Roland Wolf and Colin Henderson (Ninewells Hospital and Medical School, Dundee, UK).

The work was supported in part by the National Institutes of Health and ALSAC.

St. Jude Children’s Research Hospital

St. Jude Children’s Research Hospital is internationally recognized for its pioneering work in finding cures and saving children with cancer and other catastrophic diseases. Founded by late entertainer Danny Thomas and based in Memphis, Tenn., St. Jude freely shares its discoveries with scientific and medical communities around the world. No family ever pays for treatments not covered by insurance, and families without insurance are never asked to pay. St. Jude is financially supported by ALSAC, its fundraising organization. For more information, please visit www.stjude.org.
Filner Statement on Walter Reed Army Medical Center

Washington, D.C. – House Veterans’ Affairs Committee Chairman Bob Filner (D-CA) released the following statement today regarding the removal of the Commander of Walter Reed Army Medical Center:

“It is high time the Pentagon has admitted its mistake and taken action to improve outpatient care at Walter Reed. The removal of Army Major General George W. Weightman as the commander at Walter Reed is the first step in accepting responsibility for the treatment of our wounded soldiers.

“The events reported at Walter Reed were a break in faith with our troops. We must be able to rely on the chain of command in order to address the serious problems not only at Walter Reed, but at the military facilities across the country.

“My concerns are focused on how we serve our troops when they turn from the Pentagon to the VA for their healthcare. In order for our troops to experience the seamless transition they deserve, the bureaucratic problems that prevent many from getting the care they need must be fixed.

“I am committed to preventing similar situations to Walter Reed from striking the VA system. Clearly, we need an oversight and inspection system that is separate from the agency that is being overseen.”

Please visit our website at http://veterans.house.gov
Thanks Steve, a very valuable post with outstanding information. Hopefully all veteran's with Parkinson's disease will co-operate by posting on this topic. This is an opportunity for the Vietnam veteran and Korea DMZ ('68-'69) to get attention to a severe neurological disorder which is long over due. This has been an issue with me since 1983 and VA has never developed this issue as herbicide related. The time is now!
Although we have a topic on Agent Orange Discussion Boards Agent Orange & Parkinson, this particular topic is to entice those who have Parkinson's and were exposed to herbicides containing TCDD, to become active in the inquiry. According to all I have heard, the current Secretary of the VA, has not asked for further review of Agent Orange residual conditions. Without the Secretary of the VA making the request, AO studies will not continue.

Here is a letter to me regarding the last VAO Update 2004 (2005)

A copy of an e-mail to me Posted Mon 28 March 2005 12:52 AM:
Dear Mr. Barker,
Dr. Catlin forwarded me your message, because I have recently taken over responsibility for IOM's on-going reviews of scientific literature related to health effects and herbicide exposure from her.

The prepublication version of Update 2004 was delivered to VA at the beginning of this month. We have exhausted our supply of printed copies, but it is posted for viewing on our website (http://veterans.iom.edu). If you follow that web site's link to Vietnam War, at the head of the list of studies, you will see this most recent report. If you click on the book cover, you will go to a summary. If you click again on "Read and Purchase," you can scan the final draft of the report. The final bound books should be available in June.

Although Update 2004 did not result in any new health conditions being added to the list of those with enough ("limited or suggestive" or "sufficient") evidence available to support an association with exposure to the herbicides used in Vietnam, I hope the committee's clarification (p.
To answer your question: This is a one case decision, it is not an US Veterans Appeals Court Decision and has no binding effect on any other cases.

From the BVA decision "Despite the presumption of in-service herbicide exposure in Vietnam, the Board is not in a position to grant service connection for the veteran's neurological disorder on a presumptive basis as due to herbicide agent exposure, as the veteran's neurological disorder, manifested by Parkinson-like symptomatology, did not "[appear] within weeks or months of exposure to a herbicide agent and [resolve] within two years of onset."

In order to be fair and address the issue for all victims of Agent Orange related Parkinson's disease, we need to have the condition as a presumptive condition. Studies need to be continued.
That was the Board of Veterans Appeals. The Board of Veterans' Appeals (also known as "BVA" or "the Board") is a part of the VA, located in Washington, D.C.

Members of the Board review benefit claims determinations made by local VA offices and issue decision on appeals. These Law Judges, attorneys experienced in veterans law and in reviewing benefit claims, are the only ones who can issue Board decisions.

The U.S. Court of Veterans Appeals was created by Congress in 1988 to exercise exclusive jurisdiction over the decisions of the Board of Veterans' Appeals on the motion of claimants. Such cases include all types of veterans' and survivors' benefits, mainly disability benefits, and also loan eligibility and educational benefits. Its decisions are subject to limited review by the U.S. Court of Appeals for the Federal Circuit.

The court has seven judgeships. The judges of the court are appointed by the President with the advice and consent of the Senate. The court is based in Washington, D.C., but as a national court, it may sit anywhere in the United States.

The first is an administrative legal panel that is part of an Executive agency to review internal decisions. The later is a Federal Court, part of the judicial branch under the Constitution.

The decision of the Court has the effect of law. The decisions of the Board are subject to be Executive discretion of the Secretary and the President. In other words the secretary under his legal rule making power and the president under executive order can change the rules so that another veteran will not get the same outcome. For the court, only legislation by Congress can change the decision.
I do not understand what you mean by it being a one case decision because it was The Board of Veterans Appeals that approved this case.

To what purpose? Closing another avenue to basic justice???

Elaborate...or let's just wait and see the outcome...RE budget vs basic justice...shall we???
DaveM. "The first is an administrative legal panel that is part of an Executive agency to review internal decisions. The later is a Federal Court, part of the judicial branch under the Constitution.

The decision of the Court has the effect of law. The decisions of the Board are subject to be Executive discretion of the Secretary and the President. In other words the secretary under his legal rule making power and the president under executive order can change the rules so that another veteran will not get the same outcome. For the court, only legislation by Congress can change the decision."

From one Dave to another, thanks. That was a proper response and timely. On weekends my family keeps me busy throughout the day.

I wish to add giving an example. Recently the U.S. Court of Veterans appeals ruled in favor of Blue Water Navy Agent Orange claims. The Secretary of VA, Mr. James Nicholson, appealed that decision to the next level.

The NVLSP has released the following statement.

On March 15, 2007, the U.S. Court of Appeals for the Federal Circuit granted the government’s March 2, 2007 motion for an enlargement of time within which to file their brief. The VA has now filed their brief, so our brief is due on April 19, 2007. As the government did, we will probably need to ask the Court for an extension of this deadline, so that we can prepare as strong a response as possible to the government’s 45-page brief.

So TerryNckl although we have won a court case and that is a step in the right direction, we have a long way to go to obtain justice. An appealed case which is won by the veteran, at the BVA, only affects that one case. An appealed case to the U.S. Court of Veterans appeals may be president, if the Secretary does not appeal it to the higher level, which he did in the Haas case.

Justice does not come easy for veterans.
Just to add a bit to understanding, how a case moves through the appeal process.

1. Decision by regional office  
2. Board of Veterans Appeals  
3. U.S. Court of Veterans Appeals  
4. US Court of Appeals for the Federal Circuit  
5. Supreme Court

A good document from the VA in PDF.  

And a note on the Federal Circuit.

The Federal Circuit is unique among the thirteen Circuit Courts of Appeals. It has nationwide jurisdiction in a variety of subject areas, including international trade, government contracts, patents, trademarks, certain money claims against the United States government, federal personnel, and The Federal Circuit is unique among the thirteen Circuit Courts of Appeals. It has nationwide jurisdiction in a variety of subject areas, including international trade, government contracts, patents, trademarks, certain money claims against the United States government, federal personnel, and veterans' benefits.
ROCHESTER, Minn.--Mayo Clinic researchers have found that using pesticides for farming or other purposes increases the risk of developing Parkinson's disease for men. Pesticide exposure did not increase the risk of Parkinson's in women, and no other household or industrial chemicals were significantly linked to the disease in either men or women.

Findings will be published in the June issue of the journal Movement Disorders. "This confirms what has been found in previous studies: that occupational or other exposure to herbicides, insecticides and other pesticides increases risk for Parkinson's," says Jim Maraganore, M.D., Mayo Clinic neurologist and study investigator. "What we think may be happening is that pesticide use combines with other risk factors in men's environment or genetic makeup, causing them to cross over the threshold into developing the disease. By contrast, estrogen may protect women from the toxic effects of pesticides."

The investigators identified all those in Olmsted County, Minn., home of Mayo Clinic,
who had developed Parkinson's disease between 1976 and 1995. Each person with Parkinson's disease was matched for comparison to someone similar in age and gender who did not have the disease. The researchers conducted telephone interviews with 149 of those with Parkinson's and 129 of those who did not have the disease, or a proxy for these people, to assess exposure to chemical products via farming occupation, non-farming occupation or hobbies. The investigators were unable to determine through these interviews the exact exposure levels of these individuals or the cumulative lifetime exposure to pesticides.

Overall, the study found that the men with Parkinson's were 2.4 times more likely to have had exposure to pesticides than those who did not have Parkinson's. Women who had Parkinson's, on the other hand, had a far lower frequency of exposure to pesticides than men with the disease.

This study was undertaken due to conflicting results from previous studies of pesticides and other chemical products and risk for Parkinson's.

Funding for the study is from two grants from the National Institutes of Health. The medical-records linkage system of the Rochester Epidemiology Project also made this study possible.
Calling All Vietnam Veterans with Parkinson’s Disease!

Governments lie to their citizens. It is the norm. We have come to expect it. Sometimes we like it that way. Lies can provide psychological comfort.

Lies are particularly well-absorbed in matters dealing with war and the military. Armed forces recruitment would be seriously impaired if we asked young men and women to bear arms for the economic benefit of Chevron, the American Israel Public Affairs Committee, Haliburton International, and unnamed global investors. It is better to ask that life and limb be forfeited for the furtherance of democracy and protection of the homeland.

It is also better to promise recruits that we will provide financial subsistence in the event they are injured or become ill in the service of their country. Promises, however, require funding—funding from federal tax revenues, which conflicts with the most-welcomed political promise of all—“I will not raise taxes!”

A senior legal helpline caller has encountered the contradiction of promises versus funding. He is a 62-year-old Vietnam veteran who served with the United States Marine Corp at the Chu Lai Air Base. “We were all exposed to Agent Orange during our tour of duty there,” he said. “I found out in 2000 that I have Parkinson’s Disease. I filed a claim in 2001 and was turned down.”

The Veterans Administration ruled that “Despite the presumption of in-service herbicide exposure in Vietnam, the Board is not in a position to grant service connection because the veteran’s neurological disorder did not appear within weeks or months of exposure to herbicide agent and resolved within two years of onset.”

Mayo Clinic physicians believe the caller’s Parkinson’s is the result of his exposure to Agent Orange while serving in the Republic of Vietnam. “Mayo researchers have found that using pesticides for farming or other purposes increases the risk of developing Parkinson’s disease for men,” according to the June 2007 issue of Movement Disorders. Veteran’s law judges in two cases have found an Agent Orange-Parkinson’s connection, but their rulings are not binding on the Board of Veterans’ Appeals.

It is unlikely the current administration will add Parkinson’s to the list of Agent Orange residual conditions. A well-executed legal and political strategy is needed. If you or a person you know has Parkinson’s and served in Vietnam, contact me at 1-605-677-6343, or email at mmyers@usd.edu.

(Pro bono legal information and advice is available to persons 55 and older through the USD Senior Legal Helpline, 1-800-747-1895; mmyers@usd.edu. Opinions are solely those of the author and not the University of South Dakota).
Types of Herbicides Used in Vietnam and Manufacturers

Name [N]: Afalon
CLASS/LABEL [C/L]: HERB (CAUTION)
CHEMICAL NAME [CN]: 50% N-(3,4 dichlorophenyl)-N-methoxy mentheusea.
USED FOR [UF]: Selective weed control
MANUFACTURER [MFG]:
USED BETWEEN [UB]: 1967 Australian tests
AMOUNT USED [AU]: Under 5 gals

N: AGENT BLUE
C/L: HERB (CAUTION)
CN: Hydroxydimethyarsin Oxide (cacodylate adic)
UF: Rice crop destruction and to kill grass and bamboo
MFG: Ansul Company
UB: 64-71
AU: 2,166,656 gals

N: AGENT GREEN
C/L: HERB (WARNING)
CN: n-butyl ester of 2,4,5-T
UF: Defoliation of jungle cover
MFG: Bayer A G, Dow Chem, Union Carbide
UB: 62-64
AU: 8,208 gals

N: Agent Orange
C/L: HERBCIDE (Caution)
CN: A 50/50 mixture of n-butyl ester of 2,4,-dichlorophenoxy acetic acid
and 2,4,5,-Trichlorophenoxy Acetic acid
UF: Defoliation of jungle cover
MFG: Hercules Inc, Dow Chem, Diamond Shamrock, TH Agriculture & Nutrition
Co, Thompson Chem Corp. Mansanto Co, Uniroyal Inc
UB: 1964 and 4-70
ANT USED: 10,316,929 gals

N: Agent Orange II
C/L: Herb (Caution)
CN: A 50/50 mixture of n-butyl esters of 2,4-dichlorophenoxy acetic acid
and isosocyt ester of 2,4,5-T
UF: Defoliation of jungle cover
MFG: Miller Chem & Fertilizer, Union Carbide, Vertac, Dow Chemical,
Diamond
Shamrock, ICIANZ
UB: 68-69
AU: 950,000 gals

N: Agent Pink
C/L: Herb (Caution)
CN: 2,4,5-Trichlorophenoxy acetic acid
UF: Defoliation of jungle cover
MFG: Dow Chem, Union Carbide  
UB: 62-64  
AU: 122,800 gal

N: Agent Purple  
C/L: Herb (Caution)  
CN: A 50/30/20 mixture of n-butyl ester of 2,4-D, n-butyl, N-butyl 2,4,5-T, and Iso-butyl 2,4,5-T  
UF: Forest defoliation  
UB: 1962-64  
MFG: Union Carb, Diamond Shamrock, Dow Chem  
AU: 145,000 gal

N: Agent White  
C/L: Herb (Caution)  
CN: a 20/80 mixture of the triisopropanolimine salts of picloram and 2,4-D acid  
UF: Forest defoliation  
MFG: Dow Chem, Union Carbide  
UB: 66-71  
AU: 5,264,129 gal

N: Agral  
C/L: Wetting agent (None)  
CN: Nonylphenolethylene oxide condensate  
UF: A wetting and spreading agent for herb's  
MFG: ICI Plant Protection Div  
UB: 67  
AU: 12,500 gal

N: Aldrin  
C/L: Insecticide (Warning)  
CN: 1,2,3,4,10,10-hexocholor-1,4,4a,5,8,8a,-hexahydroexo 1-4-endo-5,8,-dimethanonnaphthalene not less the 95%  
UF: primarily to control insects  
MFG: Shell Chem  
UB: 72-73  
AU: 15,400 pounds [lbs]

N: Axodrin - 5  
C/L: Insect (Danger, poison)  
CN: Dimethyl phosphate of 3-hydroxy-N-methyl-cis-croto-namide, or 0,0-dimethyl-O-(2 methylcarbamoyl-1-methyl-vinyl) phosphate, or Phosphoric acid, dimethyl 1-methyl-E-methylamino)-3-oxo-1-propenyl ester  
UF: USAID fram prog insct used on potatoes, cotton, rice, a very stable toxic mat'1  
UF: control of flies, thrips, bollworms, and aphids  
MFG: Crystal Chem Inter-America  
UB: 72-73  
AU: 213,850 lbs

N: Danex
C/L: Herb (Caution)
CN: 20% solution of 2-methoxy 3-6-dichlorobenzoic acid
UF: Nonselective grass and weed killer
MFG: ICIANZ
UB: 67 Australian test
AU: under 5 gal

N: BHC
C/L: Insect (Warning)
CN: 1,2,3,4,5,6-Hexahlorocyclohexane; aka benzene hexachloride
UF: Control of pests in cereals and sugar beets. Production or use of BHC is prohibited in the US by the EPA
MFG: Hooker Chem; Woolfolk Chem
UB: 72-73
AU: 275,575 lbs

N: Bidran
C/L: Insect (Danger/Poison)
CN: Phosphoric acid, di-methyl 1-methyl-n, n-(dimethyl-amion)-3-oxo-1-ppropenyl ester, (E)-isomer-(9CI); or dimethyl phosphate of 3-hydroxy-N-N-dimethyl-cis-crotonamide; or 2-dimethyl cis-2 dimethyl-carbamol-1-methylvinyl phosphate
UF: USAID farm prog insect used for the control of pests on cotton and control of ticks
MFG: Shell Chem
UB: 72-73
AU: 84,800 lbs

N: Binocat
C/L: Medication (Prescription)
CN: A headache medication containing 50 meg of amytal (tradename for amobarbital, a sedative and hypnotic) and 70mg of seconal (tradename for secobarbital, a short acting barbitrate)
UF: A French headache medicine, available without prescription in V'nam: when abused, it led to persistent neurological disorders, sudden aggression, personality changes, and disorientation
MFG: A French product
UB: 62 and 75
AU: unk

N: Borate Chlorate
C/L: Herb (Warning/Danger)
CN: Sodium tetraborate dechydrate and Na Cl O2
UF: A non-selective herb and soil sterilant
MFG: Kerr-McGee Chem Corp (Australian)
UB: 67
AU: 4,500 lbs (estimated)

N: Bromacil
C/L: Herb (Caution/Warning)
CN: 5-Bromo-3-sec-butyl-6-methyuracil
UF: weed and brush killer
MFG: Hopkins Agriculture
N: Butoxone 80  
C/L: Herb (Caution)  
CN: 80% butyl ester of 2,3,5-trichlorophenoxy acetic acid  
UF: Defoliant  
MFG: ICIANZ  
UB: 67 Australian tests  
AU: 16,500 lbs

N: Cacodylic Acid (Phytar 138)  
C/L: Herb (Caution)  
CN: Hydroxydimethylarsin oxide or dimethylarsinic acid  
UF: grass, bamboo, and rice crop destruction  
MFG: Ansul Co  
UB: 62-64  
AU: 10,000 lbs

N: Carbaryl (Selvin)  
C/L: Insect (Caution/Poison)  
CN: 1-Naphthyl N-methylcarbamate  
UF: control of insect pests on field crops, trees, poultry and pets  
MFG: Union Carbide Agriculture Products Co, Inc  
UB: 72-73  
AU: 72,750 lbs

N: Chlorfurazole 8  
C/L: Herb (Caution)  
CN: 20% sodium salt of 4,5-dichloro-2-trifluoro-methyl benzimidazole  
UF: Herb  
MFG: Fisons  
UB: 67 Australian tests  
AU: under 50 gals

N: Chlordane  
C/L: Insect (Warning)  
CN: 1,2,4,5,6,7,8,8-Octachlor-2,3a,4,7,7a-hexahydro-4,6-methanoindane  
UF: Sprayed for cockroach, termite, tick, and flea control, Banned by the EPA in 75 for all domestic use  
MFG: PPG Industries  
UB: 62-72  
AU: 50,000 gals

N: Cresote (Coal Tar)  
C/L: Wood preservative (Caution)  
Chem Properties: Relatively insoluble in H2O  
UF: wood preservative possessing a high toxicity to wood destroying organisms and a low rate of evaporation  
MFG: Crowley Tar Products Co; Koopers Co Inc Organic Mat'ls; Los Angeles Chemical Co  
UB: 62-71  
AU: Unk
N: 2,4, D Acid  
C/L: Herb (Warning)  
CN: 2,4-Dichlorophenoxyacetic acid  
UF: Defoliant; also used in Agents Orange, White, Purple, and as a separate defoliant  
MFG: Miller Chem & Fertilizer; Union Carbide; Vertac; Dow Chem; Diamond Shamrock  
UB: 72-73  
AU: 815,000 lbs

N: 2,4-D Ester  
C/L: Herb (Warning)  
CN: A compound formed by the union of an organic acid and an organic base (an alcohol). An example is 2,4-D & isooctyl alcohol to form the isooctyl ester of 2,4-D  
UF: Defoliation of forest canopy  
MFG: Miller Chem & Fertilizer; Union Carbide; Vertac; Dow Chem; Diamond Shamrock; ICIANZ  
UB: 62-70 in AO mixes; 72-73 as a separate herb; 67 Australian tests  
AU: 224,870 lbs (72-73)

N: DDT  
C/L: Insect (Caution)  
CN: Dichloro, Diphenyl, Trichloroethane  
UF: Dusted to control rat fleas in quarters  
MFG: Montrose Chemical; Diamond Shamrock  
UB: 72-73  
AU: 2,253,100 lbs

N: DDVP  
C/L: Insect (Danger/Poison)  
CN: 2,2-Dichlorovinyl dimethyl phosphate  
UF: Dusted to control rat fleas in quarters and pests in stored grain  
MFG: Hopkins Agriculture  
UB: 72-73  
AU: 674,600 lbs

N: Dalapon  
C/L: Herb (Caution)  
CN: 2,2-Dichloropropionic acid  
UF: Effective against annual grasses and brushes  
MFG: Dow Chem; Crystal Chem; Bayer  
UB: 72-73; 67 Australian tests  
AU: 121,250 lbs

N: Dapsone  
C/L: Medication  
CN: 4-4' Diraminodipheylsulfon  
UF: An experimental anti-malaria drug used in combat areas in 67-69. Dapsone is the drug of choice for treatment of Hansens disease (Leprosy)  
MFG:  
UB: 67-69
AU: Unk

N: DEET
C/L: Insect Repellant (Caution)
CN: N,N-Diethyl-m-Toluamide
UF: Insect repellant, personal and clothing
MFG: Chemical Formulators; Hercules
UB: 62-73
AU: Unk

N: Del Defoliant 21
C/L: Herb
UF: A defoliant preparation intended for causing leaves to drop from crop plants such as cotton, soybeans or tomatoes, usually to facilitate harvest. Used as a fire base defoliant
MFG: Australian
UB: 67-69
AU: over 44,100 lbs

N: Diazinoh
C/L: Insect (Caution)
CN: O,O-Diethyl O (2-isopropyl-4-methyl-6-pyrimidiny1) phosphorothioate
UF: Principality used for control of soil insects, also in rice pesticide control
MFG: Ciba-Geigy Corp Agricultural Div
UB: 72-73
AU: 1,016,320 lbs

N: Dibrom
C/L: Insect (Danger)
CN: 1,2-Dibromo-2,2-Dichloroethyl dimethyl phosphate
UF: Araricide insect used on numerous crops, flies, and in food processing plants
MFG: Chevron Chem Co; Ortho Agricultural
UB: 72-73
AU: 520,285

N: Dieldrin
C/L: Insect (Warning)
CN: Hexachloro-epoxy-octahydroendo,exo-dimethanonaph thalene (principal constituent, known as HEOD), with not over 15% related compounds
UF: Insect control around quarters
MFG: Shell Internat'l
UB: 64-71
AU: 11,025 lbs

N: Diquat
C/L: Herb (Warning)
CN: 1,1' -2-2'-Bipyridylium ion: or 6, 7-dihydropyridyl (1,2-a:2,'1'-c) pyrazdiumion present in formulation as the dibromide monohydrate salt
UF: Defoliant - aquatic weed control
MFG: ICI Plant Protection Div
UB: 67-70; 67 Australian tests
AU: 40,000 lbs

N: Diuron
C/L: Herb (Caution)
CN: 3-(3-4-Dichlorophenyl)-1,1-dimethylurea
UF: selective broadleaf herb and soil sterilant
MFG: Hopkins Agriculture
UB: 64
AU: under 5 gals

N: EPN
C/L: Insect (Danger)
CN: O-Ethyl O(4-nitrophenyl) phenylophosholhioat on ethyl p-nitrophenyl
theonobenzenephospho
UF: insect control such as rice stem borer and boll weevil
MFG: EI duPont de Nemours & Co Inc
UB: 72-73
AU: 26,450 lbs

N: Endrin
C/L: Insect (Danger)
CN: Hexachloroepoxyactah-endo,endoendimethanona-phthalene (principal
constituent)
UF: Insect control on cotton and small grains
MFG: Velsicol Chem Corp
UB: 72-73
AU: 451,940 lbs

N: Furadan
C/L: Insect (Warning/Danger)
CN: 2,3-Dihydro-2,2-dimethyl-7-benzofuranyl methylcarbamate
UF: insect control in living quarters
MFG: Pillar Internat'l; FMC agricultural Chem Group
UB: 73
AU: 103,620 lbs

N: Gardona
C/L: Insect (Caution)
CN: (Z) isomer of the compound 2-chloro-1-(2,4,5 trichloropheny) vinyl
dimethyl phosphate
UF: major pests on cotton, corn, and stored grain
MFG: Shell Internat'l Chem Co
UB: 72-73
AU: 26,450 lbs

N: Lindane
C/L: Insect (Warning)
CN: Gamma isomer of 1,2,3,4,5,6-hexachlorocy-clohexane
UF: a powder used in living quarters for roaches and around rat burrows for fleas
MFG: Celamerck Gmbh & Co KG
UB: 72-73
AU: 46,300 lbs
N: Malathion
C/L: Insect (Caution)
CN: O,O-dimethyl phosphorodithioate of diethylmercapto succinate or diethylmercaptosucc, S-ester with O,O-dimethyl phosphorodithioate
UF: Roach and pest control in living quarters
MFG: American Cyanamid Co
UB: 72-73
AU: 1,380,080 lbs

N: Maneb
C/L: Fungicide (Caution)
CN: Manganese ethylenebisdithiocarb
UF: Blight on tomatoes and potatoes
MFG: Crystal Chem Inter-American; Cumberland Internat'l Co
UB: 72-73
AU: 485,000 lbs

N: Methyl Parathion
C/L: Insect (Danger)
CN: O,O-dimethyl-O-p-nitrophenyl phosphorothioato
UF: Broad use insect control
MFG: Kerr McGee Chem
UB: 72-73
AU: 343,920 lbs

N: Mirex
C/L: Insect (Warning)
CN: Dodecachlorooctahydr-1,3,4-metheno-2 N-cyclobuta (cd) pentalene
UF: General purpose insect
MFG: Allied Chem Corp
UB:
AU:

N: Nonuron
C/L: Herb (Caution)
CN: 3-(p-chlorophenyl)-1,1-dimethylurea
UF: Soil Sterilant
MFG: Hopkins Agricultural chem Co
UB:
AU:

N: Paraquat
C/L: Herb (Danger/Poison)
CN: 1,1-dimethyl-4,4'-bipyridinium ion; present as the dichloride salt (ICI/Chevron) or dimethyl sulfate salt
UF: Contract herb and dessicant (Extremely toxic)
MFG: Ortho
UB: 67
AU: 330 lbs

N: Phosphamidon
C/L: Insect (Poison)  
CN: O,O-dimethyl O-(2-chloro-2-diethylcarbamoyl-1-methyl-vinyl) phosphate  
UF: Insect control in quarters, stores, etc  
MFG: Chevron Chem Co; Ortho Agricultural Chems Div  
UB: 72-73  
AU: 41,900 lbs

N: Phosvel  
C/L: Insect (Warning)  
CN: O-(4-Bromo-2,5-dichlorophenyl) O-methylphenylphosphonothioate  
UF: Insect control in grain storage and warehouses  
MFG: Velsicol Chem Corp  
UB: 72-73  
AU: 41,900 lbs

N: Icloram  
C/L: Herb (Caution/Warning)  
CN: 4-amino-3,5,6-trichloropicolinic acid  
UF: Herb against a wide variety of deep rooted herbaceous weeds and woody plants, also used in Agent White  
MFG: Dow Chem Co  
UB: 66-71  
AU: 5,274,129 gals (2,650 lbs in 68)

N: Polybor Chlorate  
C/L: Herb (Caution)  
CN: 73% disodium octaborate tetrahydrate and 25% sodium chlorate  
UF: Non-selective weed and grass killer  
MFG: US Borax  
UB: 66-67  
AU: 68,500 lbs

N: Primatol A  
C/L: Herb (Caution)  
CN: 2-chloro-4-ethylamino-6 isopropylamino-1:3:5-trazine  
UF: Selective herb  
MFG: Geigy  
UB: 67 Australian tests  
AU: under 5 gals

N: Pyrethrins  
C/L: Insect (Caution)  
CN: a botanical insect with active principles of pyrethrins, cinerins, and jasmolins I and II  
UF: Stock, sprays, pet sprays, and food warehouses  
MFG: Fairfield American Corp, McLaughlin Gormley King Co; Printiss Drug & Chem Co  
UB:  
AU:  

N: Tandex  
C/L: Herb (Caution)  
CN: m-(3,3-dimethylureido) phenyl-tert-butylcarbamate
UF: Broad spectrum defoliant for weeds and brush
MFG: FMC Corp
UB:
AU:

N: Trinoxol
C/L: Herb (Caution)
CN: 2,4,5-Trichlorophenoxy acetic acid, and 49% butoxy ethanol ester of 2,4,5-T
UF: Selective herb for brush control
MFG: Union Carbide
UB:
AU:

N: Urox 22
C/L: Herb (Caution/Warning)
CN: 3-(p-chlorophenyl)-1,1-dimethylurea trichlor
UF: soil sterilant - long lasting
MFG: Hopkins Agricultural Chem Co
UB:
AU:

N: Weedazole
C/L: Herb (Caution)
CN: 3-Amino-1,2,4-triazole
UF: control of annual grasses and broadleaf leaves
MFG: Geigy
UB: 67 Australian tests
AU: under 5 gals

N: Zinc Phosphide
C/L: Rodenticide (Danger/Warning)
CN: Zn3 P2
UF: Mice, rat, rodent control
MFG: Hopkins; Bell Laboratories Inc
UB: 72-73
AU: 99,200

N: Zineb
C/L: Fungicide (Caution)
CN: Zinc ethylenebisdithiocarb
UF: control of mites on citrus
MFG: Bayer AG; Farmoplant SPA; FMC Corp Agricultural Chem Group
UB: 72-73
AU: 257,940 lbs
Research suggests link between pesticides and brain disease

Jul 28, 2006 | Minnesota Public Radio

Researchers at the University of North Dakota are quick to point out these are preliminary results covering one year of a planned four-year study. But Dr. Patrick Carr says there's clear evidence pesticide exposure at relatively low doses affect brain cells.

"Some areas of the brain displayed what I would call physical changes in other words, a loss of neurons in particular regions of the brain," says Carr. "In other regions of the brain you wouldn't notice a change in the number of cells present there, but now the cells that are present there are expressing chemicals in different amounts, compared to normal rats."

As an example, Carr found cells responsible for production of a substance called myelin were damaged or destroyed. Myelin is a substance made up of fats and proteins that encloses nerves. It helps transmit signals along the nerves. Loss of myelin causes nerve damage in neurological diseases such as multiple sclerosis.

Researchers studied six common pesticides. Carr says some rats were given a single large dose, while others were injected with small doses over a nine-month period.

"It's hard to then correlate that to what the average person that's working with pesticides would be exposed to," says Carr. "We're not at that position yet, where we can say this is comparable to what these people working with pesticides, short term or long term, are exposed to."

Dr. Carr hopes to have his results completely analyzed by next spring.

Gerald Groenwald, director of energy and environmental research, says there's clearly a need to continue and expand the research.

"What this research says is that we have started to open some doors and shine some light in a very objective fashion, a very comprehensive fashion, on this group of questions," says Groenwald. "And it says, more than ever, that this research is extremely important not only here in the Red River Valley, but basically globally."

Groenwald says other researchers are also looking at ways people are exposed to pesticides. He says people commonly think of being exposed to pesticides through contaminated water or food. But he believes the most efficient means of exposure is through tiny airborne particles of pollen.

Groenwald says some beneficial drugs are delivered as tiny particles, which are inhaled deep into the lungs.

He says researchers found tiny bits of pollen carried on the wind carry with them a load of pesticide.
"Frankly, if there is a link between pesticides and these diseases, I think the very fine pollen is the transport mechanism, and is in some cases you might say the smoking gun," he says.

Groenwald says because there are relatively few competing airborne pollutants in the Red River Valley, it's a perfect place to study airborne pesticide pollution.

Groenwald hopes to continue and expand the study over the next three years, depending on how much funding the research receives.

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**Sentry enzyme blocks two paths to Parkinson's disease**

St. Jude study shows the enzyme GST pi stands at the crossroads of several pathways that lead to the death of dopaminergic neurons and prevents both cell degeneration and cell suicide

The degeneration of brain cells that occurs in Parkinson's disease may be caused by either externally provoked cell death or internally initiated suicide when the molecule that normally prevents these fatal alternatives is missing, according to studies in mouse models by investigators at St. Jude Children's Research Hospital.

Parkinson's disease is a disease in which nerve cells in part of the brain called the substantia nigra die, resulting in the loss of dopamine, a nerve-signaling molecule that helps control muscle movement. The absence of dopamine from these cells, called dopaminergic neurons, causes a loss of muscle control, trembling and lack of coordination.

The molecule that prevents damage to the substantia nigra is an enzyme called GST pi ("pie"). This molecule stands like a sentry at the crossroads of several biochemical pathways, any one of which can lead to Parkinson's disease, the researchers reported in an article in the Feb. 1 early online edition of Proceedings of the National Academy of Sciences.
The job of the antioxidant GST pi is to protect the cell from death caused by either environmental toxins (externally evoked cell death), such as herbicides and pesticides, or a self-destruction process called apoptosis (cell suicide), triggered by certain stressful conditions in the cell. If GST pi levels are reduced or this enzyme is overwhelmed by toxins, these nerves are at increased risk of death. Previous research has shown that the ability of GST pi to protect cells against toxic molecules is directly linked to the ability of cancer cells with excessive amounts of this enzyme to reduce the effectiveness of chemotherapy.

The finding that GST pi plays a key role in preventing Parkinson's disease suggests that measuring levels of this enzyme might be an effective way to determine individuals at risk for developing this disease, according to Richard Smeyne, Ph.D., an associate member of the Department of Developmental Neurobiology at St. Jude. "In the future, treatments that increase GST pi levels in the substantia nigra might help to prevent or delay the onset of Parkinson's disease or reduce its severity," said Smeyne, the report's senior author.

GST pi is one of a family of similar enzymes that eliminate free radicals generated by pesticides and other chemicals. Two members of this family are present in the brain, but only one of them, GST pi, is found in the dopaminergic neurons of the substantia nigra. Free radicals are highly unstable molecules that readily interact with other molecules, causing cell damage.

The study sheds light on the cause of most cases of Parkinson's disease, which currently are unexplained. "The majority of these cases of Parkinson's disease appear to arise because individuals who have a genetic susceptibility to the disease are exposed to environmental toxins such as pesticides and herbicides, which trigger the formation of free radicals that kill dopaminergic neurons in the substantia nigra," Smeyne said. "We also know that GST pi blocks the process of cell suicide triggered by stresses that the cell can't overcome, such as an increase in the presence of free radicals or a loss of the cell's ability to produce energy."

Smeyne's team showed that of the two known types of GST in the brain, only GST pi was present in the dopaminergic neurons that are lost in Parkinson's disease. The scientists then treated two different populations of mice with MPTP, a chemical that causes loss of these cells, in order to determine if levels of GST pi changed. In mice known to be sensitive to MPTP, there was a complete but transient loss of GST pi in the dopaminergic neurons of the substantia nigra, while the same area of the brain in MPTP-resistant mice never completely lost GST pi and recovered their original levels within 12 hours.

In addition, the team showed that when MPTP-resistant mice were treated with this drug, the presence of GST pi in the dopaminergic neurons prevented activation of cJUN, a molecule that triggers apoptosis. These findings are evidence that GST pi prevents apoptosis in dopaminergic neurons of the substantia nigra, Smeyne said.

The investigators also showed in cell culture studies that blocking production of GST pi in substantia nigra cells left them vulnerable to MPTP, causing a significant death rate among these cells. In addition, when the investigators blocked GST pi production in the dopaminergic neurons of the substantia nigra, about one-quarter of them died, even though they were not treated with MPTP. "This suggests that even in the absence of MPTP the enzyme GST pi plays a critical role in preventing cell death that may occur with the natural buildup of free radicals," Smeyne said.
Finally, the investigators studied the effect of MPTP on the substantia nigra of normal, "wild-type" mice and mice that lacked one (+/-) or both (-/-) genes for GST pi. Wild-type and GST pi (+/-) mice showed similar resistance to MPTP, but GST pi (-/-) mice lost slightly less than half of their dopaminergic neurons following treatment with MPTP. Six hours after MPTP treatment, the formation of free radicals increased 300 percent in the substantia nigra of GST pi (-/-) mice compared with the substantia nigra of GST pi (+/+) mice. These results are additional evidence that GST pi may play an important role in preventing Parkinson's disease," Smeyne said.

Results of the St. Jude study showing the importance of GST pi could help to explain previous work by other researchers linking loss of this enzyme to destruction of dopaminergic neurons. For example, there is some evidence that alterations in the gene for GSP pi are linked to increased risk of Parkinson's disease after pesticide exposure. Also, although most Parkinson's disease cases have no known cause, experts believe that they are caused by the interaction of genetic susceptibility to Parkinson's disease with exposure to a variety of environmental factors, such as pesticides and herbicides.

"Therefore, the new findings bring researchers a step closer to understanding why Parkinson's disease occurs and potentially how to develop more effective treatments for it," Smeyne said.

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Other authors of this study include St. Jude researchers Michelle Smeyne, Justin Boyd and Kennie Raviie Shepherd, who did most of the work; Yun Jiao, Brooks Barnes Pond and Matthew Hatler (St. Jude); and Roland Wolf and Colin Henderson (Ninewells Hospital and Medical School, Dundee, UK).

The work was supported in part by the National Institutes of Health and ALSAC.

**St. Jude Children's Research Hospital**

St. Jude Children's Research Hospital is internationally recognized for its pioneering work in finding cures and saving children with cancer and other catastrophic diseases. Founded by late entertainer Danny Thomas and based in Memphis, Tenn., St. Jude freely shares its discoveries with scientific and medical communities around the world. No family ever pays for treatments not covered by insurance, and families without insurance are never asked to pay. St. Jude is financially supported by ALSAC, its fundraising organization. For more information, please visit [www.stjude.org](http://www.stjude.org).
Vietnam Veterans with Diagnosed Parkinson’s or Parkinson like symptoms.

Hi to all,

Shelia sent me the following data that is very important to those Vietnam Veterans and families that have been fighting the VA for service-connected disability associated with Parkinson’s and/or Parkinson like neurological symptoms.

You will find below a write up by Mayo Clinic that clearly points to a connection in males that were exposed to herbicides, a request for contact data, as well as two Board of Veterans Appeals cases awarding neurological issues associated to Parkinson’s and/or Parkinson like neurological disorders.

Now they do not differentiate between the massive amounts of militarized herbicides used with the unprecedented toxic TCDD levels nor the 6 to 25 the normal recommended dose rate that was used in Vietnam from the normal farmer or the rail road worker usage, etc.

At this time in our legacy, VA and IOM have both refused to associate military service in the herbicides as a presumptive disorder. Nevertheless, you will also find references of IOM stating the possibility of a connection.

In researching for my book, I also found many references to this neurological disorder as associated to these pesticides and/or herbicides.

The Office of Technology Assessment (OTA) was commissioned by congress in 1991 to get a nationwide consensus on toxic chemical damages. The Department of Veterans Affairs, for the most part, ignored most, if not all of these findings. It also seems congress did not take the data and “apply it” to those Veterans that said and experienced the same issues as OTA had found and pointed out.

The VA's proposal to compensate Vietnam veterans for peripheral neuropathy, as related to exposure to herbicides containing dioxin (Agent Orange), excludes neuropathies under a wide range of conditions. The proposed compensable neuropathies include only those manifested not later than 10 years following the date of exposure, and excludes those related to the effects of aging, alcohol abuse, trauma, other diseases known to be associated with peripheral neuropathy such as diabetes and Parkinson's disease, and exposure to other toxicants known to produce peripheral neuropathy. In making such exclusions, however, the VACEH did not take into consideration relevant information on neurological damage.

Because of a concern over the wide range of neurotoxic effects being induced in our population by manmade chemicals, the 101st Congress of the United States commissioned the Office of Technology Assessment to prepare a scientific consensus
document in 1991 (OTA, 1990). As explained below, the VACEH basis for excluding peripheral neuropathies under its conditions contradicts the OTA's findings on the biological mechanism of neurotoxic damage such as peripheral neuropathy.

The VA proposed exclusion of peripheral neuropathies that only become evident 10 or more years after service in Vietnam, on the assumption that such a neuropathy could not be associated with Agent Orange exposure, due to the long interval from exposure. This assumption contradicts the findings of the OTA, which found that neurological damage is not always detectable clinically, or noticeable by, the sufferer after exposure to a neurotoxic substance such as dioxin. As time progresses or old age approaches, the rate of natural neuronal cell death accelerates, and the results of earlier neurological damage may first become evident, or unmasked (OTA, 1990). The availability of alternate neuronal pathways is reduced, which were formerly responsible for compensating for earlier toxic damage. The OTA specifically noted the importance of research showing the possibility that neurotoxic substances were important in Alzheimer's disease, the degenerative brain disease of old age.

The VA also proposed exclusion of peripheral neuropathies which could only be attributed to diseases associated with neurological deficits (diabetes, Parkinson's disease) or alcohol abuse, under the assumption that the disease or alcohol abuse, and not dioxin, was the cause of neuropathy, similarly contradicts the findings of the OTA. The OTA found that damage to the nervous system from toxicants may first be unmasked by other conditions, such as diseases associated with neurological disorders or the voluntary intake of substances capable of neurological damage (alcohol, prescription drugs).

The OTA cited evidence that toxic chemicals might even be the sole causative agents in some cases of Parkinson's disease, since onset in certain families was at similar ages, and since Parkinson's disease has increased significantly from 1962 to 1984 along with exposures to toxic chemicals. The OTA also cited evidence that the substantial increase in the incidence of motor neuron disease and amyotrophic lateral sclerosis (Lou Gehrig's disease) between 1962 and 1984 was due to environmental exposures to neurotoxic chemicals.

It is therefore scientifically probable that in the future a higher incidence than normal of peripheral neuropathy will be experienced by Vietnam veterans due to Agent Orange exposure, despite the fact that the peripheral neuropathy was not detected in the 10-year interval after exposure in Vietnam. The degree or incidence of neurological damage in those Vietnam veterans suffering from diabetes or Parkinson's disease is also predicted to be higher than others suffering from diabetes or Parkinson's disease, due to earlier aggravating exposures to Agent Orange.
For the same reasons, there is no scientific basis for presuming that alcohol abuse is the only cause of any peripheral neuropathy in a Vietnam veteran. The Secretary of Veterans' Affairs, therefore, should not place any limitations or exclusions on compensation for peripheral neuropathy, and following the congressional mandate of providing the benefit of the doubt to the Vietnam veteran. This so-called mandate as we all know is a joke, A SICK GOVERNMENT JOKE.

The exclusion of peripheral neuropathy associated with diabetes as a compensable disease is flawed for another reason. This is because the VACEH conveniently neglected the results of the Ranch Hand study (Roegner, et al., 1991) and other epidemiological research, which shows a dose-related significant association between diabetes and dioxin exposure. The minutes of the May 23, 1991 VACEH meeting discussed in detail the correlation between serum dioxin in Ranch Hand veterans and increasing diabetic rates, with Dr. Lathrop stating, "these are figures which support an association.” For the VA to now exclude peripheral neuropathy associated with diabetes, when the VA has not been able to exclude diabetes itself as being caused by dioxin, is spurious.

It is important to add here that Secretary Derwinski took the credibility of ‘any scientific conclusions being valid’ to a new low in medical history. What was a proposed as a 10-year inclusion was reduced to a one (1) year inclusion by Veterans Affairs. This should mean to anyone in science or the medical field that the Derwinski and the VACEH had no idea of the etiology of what was going on with the association; only mandates to make sure that:

A. Veterans would not be able to qualify to the nonsensical time requirements and the two-year time limit to resolution of the neurological disorder(s).
B. If Veterans Affairs admitted a CNS>PNS effect causation then the door would be wide open to even more long-term neurological disorders as well as neuro-psychiatric / neuro-psychological disorders.

NEGLECT OF CENTRAL NERVOUS SYSTEM (CNS) EFFECTS

The controlling majority of the VACEH, in making its recommendations to compensate Vietnam veterans for peripheral neuropathy, neglected to evaluate dioxin's central nervous system (CNS) effects. * Because the available evidence for CNS damage by dioxin outweighs that for peripheral nervous system (PNS) among Vietnam veterans, and because of the inseparable relationship between the biological mechanisms by with dioxin exerts both CNS and PNS effects, this failure of the VACEH is indefensible.

* The CNS consists of the neurological apparatus of the brain and spinal cord (including motor neurons), while the peripheral nervous system (PNS) consists of those nerves in the extremities of the body (arms, legs, etc.). Peripheral neuropathies are one result of damage to the PNS.
CNS damage by fat soluble (lipophilic) neurotoxicants such as dioxin has always been found to accompany, and usually precede, any peripheral nervous system (PNS) damage such as peripheral neuropathy. See the discussions of relevant studies in the attached affidavit (Jenkins, 1991). The prestigious International Agency for Research on Cancer (IARC) concluded as early as 1977 that human CNS damage was associated with dioxin exposures (IARC, 1977a, 1977b). In 1986 the IARC clearly restated its’ finding that dioxin was associated with both peripheral neuropathies and personality changes, a neuropsychological consequence of CNS damage (IARC, 1986).

Since the IARC evaluations, many new epidemiological investigations have established an even stronger causal relationship between dioxin and CNS damage, including the Air Force investigations of veterans of Operation Ranch Hand.

**TOXICOLOGICAL BASIS FOR CNS DAMAGE BY DIOXIN**

A discussion of the biological basis for dioxin's neurological is relevant in demonstrating the inseparability of dioxin's effects on both the CNS and PNS. Neurotoxic substances may exert their effects by several mechanisms (Anthony and Graham, 1991). Chemical attack of whole nerve cell structures may result in cell injury or death (neuropathy). Chemical attack may be specifically on the axon (long nerve fiber) (axonopathy), or the myelin sheath of the axon (myelinopathy). Neurotoxicants may also damage or alter the neurotransmitter system, damage the glial cells, which support the primary neurons, or damage the blood vessels supplying the nervous system.

The OTA found that degeneration of the axon (axonopathy) is one of the most frequently determined neurological effects from neurotoxic chemicals (OTA, 1990). If the axon of a nerve cell dies back, it no longer reaches the next nerve cell, muscle, etc., and cannot transmit any message. Because the longer axons have more targets (larger surface area) for toxic damage, it is predicted that the longer axons found in CNS are more effected by neurotoxicants (Anthony and Graham, 1991), assuming the neurotoxicant is sufficiently lipophilic to cross the blood-brain barrier. A critical difference between nerve cell damage in the CNS compared to the PNS is that PNS nerve cells can regenerate, while those of the CNS cannot. Thus, any toxic damage to the CNS is permanent.

Although the mechanism by which dioxin exerts its neurotoxic effects, have yet to be fully elucidated, the CNS effects are consistent with destruction of the nerve axons (axonopathy). Because of the extreme toxicity of dioxin and the wide range of biological affects, however, the mechanisms of dioxin's neurotoxicity may not be limited to axonopathies. The hypothesis that dioxin damages the CNS and PNS by destruction of axons is supported by the similarity of the neurological symptoms caused by dioxin and many other lipophilic neurotoxicants causing both CNS and PNS axonopathies, including
carbon disulfide, hexane, methyl n-butyl ketone, trichloroethylene, polybrominated biphenyls, and polychlorinated biphenyls (Anthony and Graham, 1991), discusses the enduring CNS deficits found among populations exposed to these other lipophilic neurotoxicants.

Lipophilic toxicants such as dioxin are able to cross the blood-brain barrier to affect the CNS. In addition, since the brain is 50 percent lipid (dry weight), compared to 6 to 20 percent lipid in other organs (OTA, 1990), the brain may be particularly vulnerable to accumulating dioxin into its fat content. Nervous system tissue itself, with its high lipid content, will also act as a selective repository for dioxin. In addition, the low elimination rate of dioxin from the body will contribute to its ability to reach equilibrium concentrations in lipid-rich nervous system tissue itself, with its high lipid content, will also act as a selective repository for dioxin. In addition, the low elimination rate of dioxin from the body will contribute to its ability to reach equilibrium concentrations in lipid-rich nervous system tissue itself, with its high lipid content, will also act as a selective repository for dioxin. In addition, the low elimination rate of dioxin from the body will contribute to its ability to reach equilibrium concentrations in lipid-rich nervous system.

The mechanism by which dioxin exerts its neurotoxic effects may differ from that of 2,4-D alone (Agent White). The higher polarity of 2,4-D (less lipophilic) compared to dioxin suggests that it would be less capable of penetrating the blood-brain.

Neuropsychological damage may be one of the most significant consequences of exposure to Agent Orange. The Office of Technology Assessment (OTA, 1990) concluded that neurotoxic chemicals play a significant causal role in development of psychiatric as well as neurological disorders. Even minor changes in the structure or function of the nervous system were found to have profound consequences for behavioral and other neurological functions.

The OTA found that neurotoxic chemicals can cause or exacerbate anxiety, depression, mania, and psychosis.

It is simply amazing how our congress cannot put two and two together and come up with the correct answer even when they paid for the study results. I guess they figure THEY did the study; WHAT MORE DO YOU VETERANS WANT! JUSTICE AND GOVERNMENT ACCOUNTABLY; ----- SURLY YOU JEST!!!!!

Mayo Clinic in Rochester
Wednesday, June 14, 2006
Study Concludes that Pesticide Use Increases Risk of Parkinson's in Men

For appointments or more information, call the Central Appointment Office at 507-284-2111.
ROCHESTER, Minn.--Mayo Clinic researchers have found that using pesticides for farming or other purposes increases the risk of developing Parkinson's disease for men. Pesticide exposure did not increase the risk of Parkinson's in women, and no other household or industrial chemicals were significantly linked to the disease in either men or women.

Findings will be published in the June issue of the journal Movement Disorders.

"This confirms what has been found in previous studies: that occupational or other exposure to herbicides, insecticides and other pesticides increases risk for Parkinson's," says Jim Maraganore, M.D., Mayo Clinic neurologist and study investigator. "What we think may be happening is that pesticide use combines with other risk factors in men's environment or genetic makeup, causing them to cross over the threshold into developing the disease. By contrast, estrogen may protect women from the toxic effects of pesticides."

The investigators identified all those in Olmsted County, Minn., home of Mayo Clinic, who had developed Parkinson's disease between 1976 and 1995. Each person with Parkinson's disease was matched for comparison to someone similar in age and gender who did not have the disease. The researchers conducted telephone interviews with 149 of those with Parkinson's and 129 of those who did not have the disease, or a proxy for these people, to assess exposure to chemical products via farming occupation, non-farming occupation or hobbies. The investigators were unable to determine through these interviews the exact exposure levels of these individuals or the cumulative lifetime exposure to pesticides.

Overall, the study found that the men with Parkinson's were 2.4 times more likely to have had exposure to pesticides than those who did not have Parkinson's. Women who had Parkinson's, on the other hand, had a far lower frequency of exposure to pesticides than men with the disease.

This study was undertaken due to conflicting results from previous studies of pesticides and other chemical products and risk for Parkinson's.

Funding for the study is from two grants from the National Institutes of Health.
The medical-records linkage system of the Rochester Epidemiology Project also made this study possible.

Jun 08, 2007

The Elderlaw Forum: Calling all Vietnam veterans with Parkinson’s disease!

By Professor Michael Myers

Governments lie to their citizens. It is the norm. We have come to expect it. Sometimes we like it that way. Lies can provide psychological comfort.

A senior legal helpline caller has encountered the contradiction of promises versus funding. He is a 62-year-old Vietnam veteran who served with the United States Marine Corp at the Chu Lai Air Base. “We were all exposed to Agent Orange during our tour of duty there,” he said. “I found out in 2000 that I have Parkinson’s disease. I filed a claim in 2001 and was turned down.”

The Veterans Administration ruled “Despite the presumption of in-service herbicide exposure in Vietnam, the Board is not in a position to grant service connection because the veteran’s neurological disorder did not appear within weeks or months of exposure to herbicide agent and resolved within two years of onset.”

Mayo Clinic physicians believe the caller’s Parkinson’s is the result of his exposure to Agent Orange while serving in the Republic of Vietnam. “Mayo researchers have found that using pesticides for farming or other purposes increases the risk of developing Parkinson’s disease for men,” according to the June 2007 issue of Movement Disorders. Veteran’s law judges in two cases have found an Agent Orange-Parkinson’s connection, but their rulings are not binding on the Board of Veterans’ Appeals.

It is unlikely the current administration will add Parkinson’s to the list of Agent Orange residual conditions. A well-executed legal and political strategy is needed. If you or a person you know has Parkinson’s and served in Vietnam, contact me at 1-605-677-6343, or email at mmyers@usd.edu.
BVA REVIEWS

Citation Nr: 0519813
Decision Date: 07/21/05    Archive Date: 08/03/05

DOCKET NO. 94-37 191 ) DATE

On appeal from the
Department of Veterans Affairs (VA) Regional Office (RO)
in Winston-Salem, North Carolina

THE ISSUE

Entitlement to service connection for a neurological disorder, claimed as due to in-service herbicide exposure.

REPRESENTATION

Appellant represented by: The American Legion

WITNESSES AT HEARING ON APPEAL

The veteran and his son.

ATTORNEY FOR THE BOARD

L. Cryan, Counsel
INTRODUCTION

The veteran had active service from June 1966 to October 1969, with approximately four months of additional prior service.

This matter comes before the Board of Veterans' Appeals (Board) from a March 1994 rating decision of the RO, which denied the veteran's claim seeking entitlement to service connection for a neurological disorder, claimed as peripheral neuropathy, due to alleged exposure to Agent Orange while in Vietnam. The veteran submitted a notice of disagreement with that rating decision in May 1994. In July 1994, he was provided with a statement of the case. His substantive appeal was received in September 1994.

The Board notes that the veteran had previously claimed entitlement to service connection for a neurological disorder, claimed as Parkinson's disease, due to alleged exposure to Agent Orange while in Vietnam, which was denied by an October 1988 rating decision. The veteran submitted a notice of disagreement with that rating decision in January 1989. In February 1989, he was provided with a statement of the case. His substantive appeal was received in March 1989. The matter was received at the Board in October 1989 but was referred back to the RO pending review and revision of herbicide regulations. The RO then also deferred a decision on the claim pending updated proposed regulations.

As noted in a June 1999 remand by the Board, the RO, in the currently appealed March 1994 rating decision essentially considered both the claimed peripheral neuropathy and the claimed Parkinson's disease. Given that the veteran has claimed service connection for a neurological disorder, initially claimed as Parkinson's disease and subsequently claimed as peripheral neuropathy, and given that the veteran's claims were essentially one continuous claim for the same neurological disorder, the Board has simply characterized the veteran's claim as entitlement to service connection for a neurological disorder, claimed as due to Agent Orange exposure. The issue has been so identified on the title page hereinafore.

The veteran testified at a personal hearing before the undersigned Veterans Law Judge, sitting at the RO in September 1997. A transcript of his testimony is associated
with the claims file.

Finally, it is noted that the case was previously twice before the Board and was remanded to the RO in January 1998 and June 1999 for additional evidentiary development. Following compliance with the Board's directives on Remand, the case is now returned to the Board for further appellate consideration.

FINDINGS OF FACT

1. The veteran had active military service in the Republic of Vietnam during the Vietnam era, and is therefore presumed to have been exposed to herbicide agents in service.

2. The veteran has a currently diagnosed neurological disorder with Parkinson-like characteristics, also referred to as Parkinsonism.

3. The veteran's neurological disorder may not be presumptively service connected under the provisions of 38 C.F.R. § 3.309(e).

4. The competent and probative medical opinions of record have determined that the veteran's currently diagnosed neurological disorder is at least as likely as not due to in-service exposure to Agent Orange.

CONCLUSION OF LAW

With resolution of all doubt in the veteran's favor, the veteran's currently diagnosed neurological disorder, referred to as Parkinsonism and Parkinson-like syndrome, was incurred in service as a result of in-service herbicide exposure in Vietnam. 38 U.S.C.A. §§ 1110, 5103, 5103A, 5107 (West 2002); 38 C.F.R. §§ 3.159, 3.303 3.304 (2004).

REASONS AND BASES FOR FINDINGS AND CONCLUSION

The veteran asserts that service connection is warranted for his neurological disorder with symptomatology which mirrors that of Parkinson's disease, which he claims is due to in-service herbicide exposure in the Republic of Vietnam.
I. Duties to Notify and Assist

At the outset, the Board notes that on November 9, 2000, the Veterans Claims Assistance Act of 2000 (VCAA) was enacted. See 38 U.S.C.A. §§ 5103, 5103A (West 2002). Among other things, the VCAA amended 38 U.S.C.A. § 5103 to clarify VA's duty to notify claimants and their representatives of any information and evidence that is necessary to substantiate the claim for benefits. The VCAA also created 38 U.S.C.A. § 5103A, which codifies VA's duty to assist, and essentially states that VA will make reasonable efforts to assist a claimant in obtaining evidence necessary to substantiate a claim. Implementing regulations for the VCAA were subsequently enacted, which were also made effective November 9, 2000, for the most part. See 66 Fed. Reg. 45,620 (Aug. 29, 2001) (codified at 38 C.F.R. §§ 3.102, 3.159). The intended effect of the implementing regulations was to establish clear guidelines consistent with the intent of Congress regarding the timing and scope of assistance VA will provide to claimants who file a claim for benefits. See 66 Fed. Reg. 45,620 (Aug. 29, 2001). Both the VCAA and the implementing regulations are applicable in the present case, and will be collectively referred to as "the VCAA."

Pertinent to the merits of the veteran's claim of entitlement to service connection for a neurological disorder, the Board finds that the provisions of the VCAA have been complied with. In light of the complete grant of benefits sought on appeal (entitlement to service connection for a neurological disorder), no further evidence is necessary to substantiate the veteran's claim for service connection. See 38 U.S.C.A. § 5103(a) (West 2002). In this veteran's case, there is no reasonable possibility that further assistance would aid in substantiating the claim for VA compensation benefits. See 38 U.S.C.A. § 5103A(a)(1),(2) (West 2002). Also, further notice to the veteran concerning the evidence necessary to substantiate his claim or regarding responsibilities in obtaining evidence would serve no useful purpose.

II. Factual Background

The veteran's service medical records are negative for complaints, findings, or diagnosis of a neurological disorder of any kind.
A review of the post-service evidentiary record reveals numerous medical records clearly indicating that the veteran has been diagnosed with a neurological disorder, although there has been some degree of variance in the precise nature of that diagnosis.

A September 1982 private neurology consultation report prepared by Dr. K, indicated that the veteran was experiencing progressive weakness of the left side of the body and noted that he had a history of having polio of the left arm and left leg when he was age 22 months. There was also a history of having served two tours of duty in Vietnam. Other numerous subsequent private medical records from the neurological offices of Dr. L as well as from a Duke University Medical Center doctor indicated that the veteran began experiencing left sided numbness in 1982 and there are several diagnoses of Parkinsonism shown from 1983. At one time, it was thought that the veteran's neurological symptoms could be attributed to basal ganglia disease. Other doctors noted that the veteran may not have a pure form of Parkinsonism, but instead, a Parkinson-plus syndrome such as a progressive supranuclear palsy.

The veteran underwent a VA neurological examination in July 1988, and he gave a history of having been exposed to dioxins in service. The veteran reported symptoms to include slowness of movement, muscle stiffness, poor coordination, slurred speech, excessive salivation, muscle twitching, muscle cramps, tremor, and involuntary movements. The examiner noted that the veteran had many features of Parkinson's Disease, but noted that his picture was unusual. First, the examiner pointed out that the veteran was young. Next, the examiner noted that he could never see the true resting tremor, and muscle tone was not significantly increased on testing. On the other hand, he showed a lot of the variability of muscle function that one did see in Parkinsonism, and he could never convince himself that the veteran was functional. Therefore, the examiner diagnosed a neurological problem that was similar to Parkinson's and may be a variety of such. He further stated that he was not aware of the veteran's picture being seen with dioxin exposure, but would defer that question to those with more knowledge in that field.

Amongst other medical evidence of record is a July 1990 letter from Dr. R, a PhD and toxicologist, with the State of
North Carolina, Department of Environment, Health, and Natural Resources, Division of Epidemiology. Dr. R referred to various scientific literature indicating a possible relationship between dioxin exposure and various neurological disorders, indicating that one cannot rule out the possible role of dioxin as a causal agent for various neurological disorders, including Parkinson's disease. Dr. R noted that recent scientific literature had brought to light the possibility of environmental causes of neurological disorders, as opposed to genetic causes, and that this may be the reason why more young people were developing that disease.

Also of record is a report of Dr. R, dated February 1991 and titled "[veteran's name] - A Possible Association Between His Current Medical Problems and Exposure to Agent Orange in Vietnam." This 21 page report, plus attached appendices, contained references to numerous scientific studies and literature discussing potential neurological health effects of dioxin exposure. Dr. R further indicated that it did not appear that the veteran had true Parkinson's but instead had Parkinson like symptoms and based upon an evaluation of scientific literature, Dr. R stated that it was possible, indeed quite probable, that the veteran's condition may stem from past Agent Orange exposure. It is noted that Dr. R's opinion was based upon the veteran's history of having been stationed in Dong Ha and Quang Tri and having been subject to significant amounts of Agent Orange spraying in those areas. Dr. R elsewhere stated that the veteran "was in an area that was subject to extensive Agent Orange treatment and exposure for an extended period of time."

In December 1991, Dr. L prepared a memorandum in which he notes that the sum total of all of the information provided by Dr. R's report suggested an association between the veteran's exposure to dioxin and possible development of neurological symptoms.

At his personal hearing before the undersigned Veterans Law Judge in September 1997, the veteran testified that he was in good health at the time he was discharged from service. The veteran testified that he could find no family history of a neurological disorder, and that he had never had any kind of traumatic injuries that may have stimulated a central nervous system dysfunction. Furthermore, the veteran testified that he never worked in any type of environment where he was
exposed to excessive chemicals.

Following the Board's January 1998 Remand, the veteran underwent a VA neurological examination in September 1998. It was noted that the veteran was in a wheelchair due to an inability to control his movements, especially on the left. Movements of his face were symmetric but limited in excursion. He could move his tongue perhaps half of the normal range. He was constantly writhing. He had normal strength in the deltoids, triceps and biceps, but the testing was interrupted by constant twisting, as severe as the examiner had seen in many years. The examiner found it interesting that sensation was within normal limits, including superficial sensation, traced figures, and vibration in four extremities, and joint sense in the lower extremities. The provided a history of dioxin exposure in service. The diagnosis was that of chronic severe generalized choreoathetosis with preserved reflexes, strength, sensation, and intellect. The examiner specifically found that this was not an acute or sub-acute peripheral neuropathy. It was also stated that there was a moderate to strong possibility that the veteran's bizarre movements were a result of exposure to some chemical during his service.

In the June 1999 remand, the Board pointed out that the aforementioned report of Dr. R and the most recent VA examination provided clear medical evidence of a current diagnosis of a neurological disorder, and further supported a nexus between the current diagnosis and chemical exposure in service, more specifically dioxin exposure. However, at the time of the June 1999 remand, VA laws and regulations did not permit a presumption of exposure to Agent Orange based solely on having served in-country during the Vietnam Era. As such, the case was remanded for development, specifically to determine if the veteran was likely exposed to Agent Orange during service in Vietnam.

In the meantime, it was confirmed that the veteran served in Vietnam from April 28, 1967 to December 3, 1967 and from June 26, 1969 to September 26, 1969.

In March 2001, the case was referred to the VA Under Secretary for Health for review and preparation of a medical opinion. In response, VA's Chief Public Health and Environmental Hazards Officer first noted that the veteran
had documented exposure to herbicides based on Department of Defense records of Agent Orange spraying. The opinion also noted that in its most recent report, Veterans and Agent Orange Update 1998, the Institute of Medicine (IOM) committee concluded that there was inadequate/insufficient evidence to determine whether an association exists between exposure used in Vietnam and motor/coordination dysfunction. Parkinson's Disease was included in this group of disorders by the IOM. In light of the foregoing, the Chief Public Health and Environmental Hazards Officer opined that it was possible that the veteran's neurological disorder could be attributed to exposure to herbicides; however, she could not state that Agent Orange exposure was likely or at least as likely as not to be responsible.

In response to the opinion from the Under Secretary of Health, the RO opined, as a result of the opinion by the Under Secretary, and following review of the evidence in its entirety, the veteran's disability was not the result of Agent Orange exposure.

In a December 2001 memorandum, Dr. G, of the Washington Hospital Center, noted a possible etiology of the veteran's neurological disorder. Dr. G noted the veteran's history of Parkinson-like symptoms, Parkinsonism, and the lack of a clearly defined neurological syndrome dating back to 1982. Dr. G noted that the initial medical evaluations of the veteran's condition performed by several neurologists seemed to be suggestive of a neurological syndrome that had some similarities to Parkinson's disease, but in some respects was quite different. Dr. G found that such a neurological condition, initially diagnosed at a young age raised several questions concerning a possible etiology. Dr. G found that the significant evidence provided by Dr. R strongly suggested that the offending agent may have been dioxin found in Agent Orange preparation. Dr. G found that Dr. R's research in that subject provided overwhelming support of a probable nexus between exposure to those compounds and the manifestations of acute and chronic neurological damage as well as the myriad symptoms experienced by the veteran. In conclusion, Dr. G opined that it was as likely as not that the highly unusual symptomatology observed in the veteran at an extremely young age was related to his exposures during service.
In response to Dr. G's medical opinion, the Chief Officer of the Office of Public Health and Environmental Hazards prepared another opinion, in March 2002, regarding the possible etiology of the veteran's neurological disorder. The opinion noted that the most recent report from the National Academy of Sciences "Veterans and Agent Orange-Update 2000" reaffirmed their earlier conclusions on the lack of an association between herbicide exposure and Parkinson's or related neurological diseases. It was noted that the National Academy of Sciences further stated that in the future, as diagnostic accuracy for Parkinson's disease improved, and when herbicide exposure assessment is quantitated with specific biomarkers, and further research confirms the gene-toxicant interaction in larger prospective studies of Parkinson's disease, the evidence for association may change. The Chief Officer of the Office of Public Health and Environmental Hazards found, in essence, that Dr. R's 1991 opinion was outdated, and not supported by National Academy of Sciences current review of medical and scientific evidence. It was noted that VA relied extensively upon the independent and highly credible medical and scientific analysis provided by the NAS in establishing associations between herbicide exposure and health effects in veterans.

We shall see just how independent NAS/IOM is on July 18th at the Disability Commission meeting. From my research, it looks as if they are caught between a rock and hard place as our legal “judge and jurors.” Scientific associations to cause and effect (Absolutes) are not the same as legal “as least as likely as not associations; or highly plausible; or biologically plausible associations.” Including lets not forget there is a government contract involved here in which its mandates or either secret or the team of VA/NAS-IOM does not want to come forward with the real requirements to mandate a presumption. No different than the VACEH from 1979 to 1991 that operated with extreme bias and even possible conflict of prior professional interest and publicly pre-stated opinions. Not exactly a non-biased scientific endeavourer for our dead and dying Vetearns.

Therefore, she again opined that it was possible that the Parkinson's or Parkinson's-like disease diagnosed in the veteran could be related to exposure to herbicide in service. However, they could not state that it was as likely, or more likely than not, that his disease was the result of herbicide exposure.

In light of the aforementioned March 2002 opinion, the RO found that there was no reasonable possibility that the veteran's Parkinson's or Parkinson's-like disease resulted from exposure to herbicides in service.
After the case was returned to the Board, the Board determined that an Independent Medical Opinion might be useful in determining the likely etiology of the veteran's neurological disability.

In the meantime, both Dr. R and Dr. G responded to the March 2002 opinion by the Under Secretary of Health. Dr. R provided a lengthy report nearly 25 pages long noting a more likely than not association between the veteran's current medical problem and exposure to Agent Orange during service in Vietnam. In essence, the report finds an environmental link to Parkinson-like symptoms illustrated by numerous physicians in medical and scientific literature cited to by Dr. R in his report. Moreover, Dr. R noted that the National Academy of Sciences, in their report "Veterans and Agent Orange-Update 2002, were in general agreement with the information presented in his report. Dr. R concluded that because the evidence shows that the veteran was directly exposed to Agent Orange, and because the new medical peer-reviewed literature evidence presented in the report and the strength of the individual epidemiological studies relating the veteran's exposure to Agent Orange in Vietnam, it was more likely than not that the Vietnam exposure to Agent Orange is responsible for his present medical condition; and that the new and existing information in the report, more likely than not links the veteran's Agent Orange exposure to his current neurological problems.

In a January 2005 response to the Board's request for an independent medical opinion, a doctor from the Washington University School of Medicine noted that he reviewed the medical record and did not feel that he could render an opinion regarding the relationship between the veteran's exposure to Agent Orange and his Parkinsonion syndrome with the available information. The doctor noted that he would have to perform a history and examination of the veteran to determine the likely cause of his Parkinsonism.

Dr. G noted that the evidence indicated that the veteran was exposed to Agent Orange during service, and also noted Dr. R's more recent medical data supporting a strong association between dioxins and subsequent manifestations of acute and chronic neurological damage as well as the myriad symptoms experienced by the veteran. Dr. G therefore concluded that the additional data gave further support to the case, and
opined that it was as likely as not that the highly unusual symptomatology observed in the veteran at an extremely young age is related to his Agent Orange exposure during service.

III. Legal Criteria and Analysis

Under the relevant regulations, service connection may be granted for a disability resulting from disease or injury incurred in or aggravated by active service. 38 U.S.C.A. § 1110 (West 2002). If a chronic disease is shown in service, subsequent manifestations of the same chronic disease at any later date, however remote, may be service connected, unless clearly attributable to intercurrent causes. 38 C.F.R. § 3.303(b) (2004). However, continuity of symptoms is required where the condition in service is not, in fact, chronic or where diagnosis of chronicity may be legitimately questioned. 38 C.F.R. § 3.303(b) (2004).

Further, service connection may also be granted for any disease diagnosed after discharge, when all the evidence, including that pertinent to service, establishes that the disease was incurred in service. 38 U.S.C.A. § 1113(b) (West 2002); 38 C.F.R. § 3.303(d) (2004). The Board must determine whether the evidence supports the claim or is in relative equipoise, with the appellant prevailing in either case, or whether the preponderance of the evidence is against the claim, in which case, service connection must be denied. Gilbert v. Derwinski, 1 Vet. App. 49 (1990).

Additionally, where a veteran served continuously 90 days or more during a period of war or during peacetime service after December 31, 1946, and an organic disease of the nervous system becomes manifest to a degree of at least 10 percent within one year from the date of termination of service, such a disease shall be presumed to have been incurred in or aggravated by service, even though there is no evidence of such a disorder during the period of service. 38 U.S.C.A. §§ 1101, 1112, 1113 (West 2002); 38 C.F.R. §§ 3.307, 3.309 (2004).

In addition to the regulations governing entitlement to service connection outlined above, 38 C.F.R. § 3.309(e) provides that if a veteran was exposed to an herbicide agent during active military, naval, or air service, the diseases set forth in 38 C.F.R. § 3.309(e) shall be service-connected.
if the requirements of 38 C.F.R. § 3.307(a)(6) are met even though there is no record of such disease during service, provided that the rebuttable presumption provisions of 38 C.F.R. § 3.307(d) are also satisfied. These diseases include chloracne, Type 2 diabetes (also known as Type II diabetes mellitus or adult-onset diabetes), Hodgkin's disease, multiple myeloma, Non-Hodgkin's lymphoma, acute and subacute peripheral neuropathy, porphyria cutanea tarda, prostate cancer, respiratory cancers (including cancer of the lung, bronchus, larynx, or trachea), and soft-tissue sarcoma. 38 U.S.C.A. § 1116(a)(2) (West 2002); 38 C.F.R. § 3.309(e) (2004). For purposes of this section, the term acute and subacute peripheral neuropathy means transient peripheral neuropathy that appears within weeks or months of exposure to an herbicide agent and resolves within two years of the date of onset. 38 C.F.R. § 3.309(e), Note 2 (2004).

A veteran who, during active military, naval, or air service, served in the Republic of Vietnam during the period beginning on January 9, 1962, and ending on May 7, 1975, shall be presumed to have been exposed during such service to an herbicide agent, unless there is affirmative evidence to establish that the veteran was not exposed to any such agent during that service. The last date on which such a veteran shall be presumed to have been exposed to an herbicide agent shall be the last date on which he served in the Republic of Vietnam during the Vietnam era. 38 U.S.C.A. § 1116 (West 2002); 38 C.F.R. § 3.307(a)(6)(iii) (2004). The Board notes that in June 2003, 38 C.F.R. § 3.307(a)(6)(iii) was amended to expand the presumption of exposure to herbicides to include all Vietnam veterans, not just those who have a disease on the presumptive list in 38 U.S.C.A. § 1116(a)(2) and 38 C.F.R. § 3.309(e). See 68 Fed. Reg. 34539, 34541 (June 10, 2003). Thus, the veteran is presumed to have been exposed to herbicides because he served in Vietnam during the Vietnam era.

Despite the presumption of in-service herbicide exposure in Vietnam, the Board is not in a position to grant service connection for the veteran's neurological disorder on a presumptive basis as due to herbicide agent exposure, as the veteran's neurological disorder, manifested by Parkinson-like symptomatology, did not "[appear] within weeks or months of exposure to a herbicide agent and [resolve] within two years of onset." 38 C.F.R. § 3.309(e), Note 2. However, in the case of Combee v. Brown, 34 F. 3d 1039 (Fed Cir. 1994), the
United States Court of Appeals for the Federal Circuit (Federal Circuit) held that a veteran was not precluded from presenting proof of direct service connection between a disorder and exposure even if the disability in question was not among conditions enumerated under the Veterans' Dioxin and Radiation Exposure Compensation Standards Act, the presumption not being the sole method for showing causation.

Hence the veteran may establish service connection for a neurological disorder by presenting evidence establishing that it is at least as likely as not that his neurological disorder, also referred to as Parkinsonism and Parkinson-like symptoms, was caused by his presumed in-service herbicide agent exposure. 38 U.S.C.A. § 5107(b) (West 2002); 38 C.F.R. § 3.303 (2004); Gilbert v. Derwinski, 1 Vet. App. 49 (1990).

In this case, the medical evidence of record tends to support the veteran's assertions that his neurological disorder was caused by in-service herbicide exposure. More specifically, the medical opinions of Drs. L, R, and G have found an association between the veteran's unusual neurological disability and exposure to Agent Orange in service. These doctors have repeatedly noted the unusual symptomatology, similar to, yet different in some respects, from Parkinson's Disease. These doctors have also repeatedly noted that the onset of the veteran's symptoms at a young age was unique, supporting the notion that exposure to herbicides during service as likely as not led to the veteran's neurological disorder. Furthermore, Dr. R, who specializes in toxicology, provided a complete rationale for his opinion, providing a report of extensive research which cited multiple medical and scientific data.

Moreover, the VA physicians who examined the veteran during the pendency of this appeal also provided opinions that it was at least as likely as not that the veteran's presumed in-service herbicide exposure led to the current neurological disorder.

The Board is mindful that VA's Chief Public Health and Environmental Hazards Officer provided opinions in 2001 and 2002 in which she stated that there was inadequate/insufficient medical/scientific evidence to determine whether an association existed between exposure to herbicides and neurological dysfunction. She also noted that Dr. R's 1991 opinion was based on outdated research.
However, Dr. R provided a second opinion noting a likely association between the veteran's in-service herbicide exposure and his neurological disorder, based on updated scientific and medical information.

In sum, several private and VA doctors have found an as likely as not association between the veteran's in-service herbicide exposure and his current neurological disorder. Although VA's Chief Public Health and Environmental Hazards Officer could not state that it was at least as likely as not that the veteran's neurological disorder was caused by in-service herbicide exposure, she did state that it was possible.

In light of the foregoing, the Board finds that the preponderance of the competent and probative evidence supports the veteran's claim because all of the opinions of record find either that it is possible, or at least as likely as not, that the veteran's neurological disorder resulted from in-service herbicide exposure. In the absence of clear contrary medical evidence, the opinion of the Chief Officer of the Office of Public Health and Environmental Hazards, coupled with the multiple positive opinions by Drs. L, R, G and the VA doctors, provide a preponderance of the evidence supporting the claim for entitlement to service connection for a neurological disorder, referred to as Parkinsonism and Parkinson-like syndrome. Accordingly, a grant of service connection is warranted. 38 U.S.C.A. §§ 1110, 5107(b) (West 2002); 38 C.F.R. § 3.303(d) (2004).

ORDER

Service connection for a neurological disorder, referred to as Parkinsonism and Parkinson-like syndrome, is granted.

____________________________________________
BARBARA B. COPELAND
Veterans Law Judge, Board of Veterans' Appeals
THE ISSUE

Entitlement to service connection for Parkinson's disease, to include as a result of exposure to an herbicide agent.

REPRESENTATION

Appellant represented by: Sharon A. Hatton, Attorney at Law

ATTORNEY FOR THE BOARD

E. Pomeranz, Counsel

INTRODUCTION

The appellant had active military service from July 1970 to April 1972.

This matter comes before the Board of Veterans' Appeals (Board) on appeal of a June 2002 rating action by the Department of Veterans Affairs (VA) Regional Office (RO) located in Winston-Salem, North Carolina.

FINDING OF FACT

The appellant's Parkinson's disease is related to his in-
service herbicide exposure.

CONCLUSION OF LAW

Parkinson's disease was incurred in active military service. 38 U.S.C.A. § 1110 (West 2002); 38 C.F.R. § 3.303 (2004).

REASONS AND BASES FOR FINDING AND CONCLUSION

I. Veterans Claims Assistance Act of 2000

In November 2000, the Veterans Claims Assistance Act of 2000 (VCAA) was signed into law. See 38 U.S.C.A. §§ 5100, 5102, 5103, 5103A, 5106, 5107, 5126 (West 2002). Regulations implementing the VCAA are applicable to the appellant's claim. 38 C.F.R. §§ 3.102, 3.156(a), 3.159, 3.326 (2004).

With respect to VA's duty to notify, the RO sent the appellant a letter in May 2002, prior to the initial rating decision with regard to the issue on appeal, in which the appellant was notified of the types of evidence he needed to submit, and the development the VA would undertake. See Quartuccio v. Principi, 16 Vet. App. 183 (2002). The letter specifically informed the appellant what was needed from him and what VA would obtain on his behalf. Id. The appellant was also informed of the elements needed to substantiate a service connection claim, including requirements specific to Agent Orange claims. In addition, the Board observes that the October 2002 statement of the case provided the appellant with the text of the relevant portions of the VCAA, as well as the implementing regulations. The Board further notes that there is no indication that there is additional evidence that has not been obtained and that would be pertinent to the present claim. The appellant has been notified of the applicable laws and regulations pertinent to his service connection claim. Moreover, the appellant has been afforded the opportunity to present evidence and argument in support of the claim. Id. Thus, VA's duty to notify has been fulfilled.

VA also has a duty to assist the appellant in obtaining evidence necessary to substantiate a claim. 38 C.F.R. § 3.159(c). The duty to assist includes providing a medical examination or obtaining a medical opinion when such is
necessary to make a decision on the claim. In this regard, the Board notes that in August 2004, the appellant underwent a VA examination which was pertinent to his service connection claim. In addition, in February 2004, the Board referred this case for a medical opinion from a Veterans Health Administration (VHA) physician. The Board further observes that in this case, there is no outstanding evidence to be obtained, either by VA or the appellant. Consequently, given the standard of the new regulation, the Board finds that VA did not have a duty to assist that was unmet. The Board also finds, in light of the above, that the facts relevant to this appeal have been fully developed and there is no further action to be undertaken to comply with the provisions of the regulations implementing the VCAA. Therefore, and in light of the decision herein, the appellant will not be prejudiced as a result of the Board proceeding to the merits of the claim. See Bernard v. Brown, 4 Vet. App. 384, 392-94 (1993); see also Mayfield v. Nicholson, No. 02-1077 (U.S. Vet.App. April 14, 2005).

II. Factual Background

The appellant's DD 214, Armed Forces of the United States Report of Transfer or Discharge, shows that he served in the United States Army from July 1970 to April 1972. The appellant's DD 214 also reflects that he served in the Republic of Vietnam from July 1971 to March 1972.

The appellant's service medical records are negative for any complaints or findings of Parkinson's disease. The records show that in March 1972, the appellant underwent a separation examination. At that time, the appellant was clinically evaluated as "normal" for neurologic purposes.

In March 2002, the appellant filed a claim for entitlement to service connection for Parkinson's disease. At that time, he maintained that his currently diagnosed Parkinson's disease was due to his exposure to Agent Orange while he was serving in the Republic of Vietnam.

In June 2002, the RO received private medical records, from February 2000 to June 2001. The records show that in March 2001, the appellant sought treatment from Valerie A. Lasko, M.D., for complaints of a tremor. At that time, Dr. Lasko noted that according to the appellant, he first noticed his tremor in approximately January 2000. When asked about
exposure to heavy metals, the appellant reported extensive involvement with lead while working in a remodeling and restoration business. The appellant also stated that he had used pesticides extensively. The assessment was Parkinson's disease and Dr. Lasko indicated that in light of the appellant's history of metal exposure, she would request a heavy metal screen. The records reflect that in June 2001, Dr. Lasko reported that the appellant's heavy metal screen from March 2001 was within normal limits.

A private medical statement from Ellis F. Muther, M.D., dated in June 2002, shows that at that time, Dr. Muther stated that the appellant had a two-year history of Parkinson's disease. According to Dr. Muther, no explanation for the appellant's disorder had been found "except a possible exposure to Agent Orange." Dr. Muther indicated that Agent Orange had been demonstrated to be a neurotoxin, and, as such, he opined that it was highly possible that that was a contributing factor in the etiology of the appellant's Parkinson's disease.

In Fast Letter 03-20, issued by the Veterans Benefits Administration on June 25, 2003, it was noted that a study by the National Academy of Sciences found that the credible evidence against an association between herbicide exposure and Parkinson's disease outweighed the credible evidence for such an association.

In December 2003, the Board remanded this case and requested that the appellant be afforded a VA neurological examination to determine the etiology of any Parkinson's disease found. As per the Board's December 2003 remand decision, in August 2004, the appellant underwent a VA examination. Following the physical examination and a review of the appellant's claims file, the examiner diagnosed the appellant with Parkinson's disease, with a predominant tremor. The examiner noted that in private medical records from Dr. Lasko, Dr. Lasko had referred to the appellant's occupation of remodeling rental homes and the possibility that the appellant was exposed to lead based paints. However, the examiner reported that the appellant's heavy metal screens came back negative which meant that that could be "safely eliminated" as a cause of the appellant's Parkinson's disease. Next, in order to answer the question as to whether Agent Orange caused the appellant's Parkinson's disease, the examiner noted that an extensive three-day literature review
was conducted. The examiner listed numerous medical articles which addressed the relationship between herbicide agents and neurological disorders, including Parkinson's disease. Following a review of the medical literature obtained, the examiner opined that it was at least as likely as not that the appellant's Parkinson's disease may be related to exposure to Agent Orange or other herbicide exposure in Vietnam.

A private medical statement from Joel C. Morgenlander, M.D., Associate Professor of Medicine (Neurology), dated in October 2004, shows that at that time, Dr. Morgenlander stated that he had first seen the appellant in October 2001 and had diagnosed him with probable Parkinson's disease. According to Dr. Morgenlander, the appellant's symptoms began in approximately 2000 or 2001.

In February 2004, the Board referred this case for a medical opinion from a VHA medical doctor with the necessary expertise in the treatment of Parkinson's disease. The Board noted that due to the appellant's service in the Republic of Vietnam during the Vietnam era, he was presumed to have been exposed to herbicide agents, including Agent Orange, during his period of active military service. Thus, the Board requested that the VHA physician, after reviewing the appellant's claims file, offer an opinion with respect to the following question: Whether it was at least as likely as not that the appellant's Parkinson's disease was related to his period of military service, to specifically include his presumed exposure to herbicide, including Agent Orange, while in Vietnam.

A VHA opinion from the Chief, Neurology Service, was provided on March 30, 2005. In the opinion, the VHA neurologist stated that the 2002 Update "Veterans and Agent Orange" published by the Institute of Medicine summarized several epidemiologic studies, most of which suggested a mildly increased risk of Parkinson's disease in individuals "with many years of occupational exposure" to herbicides or pesticides. According to the VHA neurologist, no particular association had been demonstrated for any single chemical or class of compounds, and no association with exposure to "2,4-D, 2,4,5-T, or TCDD" had been published. The VHA neurologist stated that the Institute of Medicine concluded that although an etiologic connection between pesticide/herbicide exposure was "biologically plausible,"
there was insufficient evidence at present to support a definite association between Parkinson's disease and "2,4-D, 2,4,5-T, or TCDD." According to the VHA neurologist, to his knowledge, since the 2002 Update from the Institute of Medicine, there had been no more recent epidemiologic or biochemical studies indicating a definite association between Parkinson's disease and "2,4-D, 2,4,5-T, or TCDD." Thus, in reviewing the appellant's medical records, the VHA neurologist stated that he did not find any details of the appellant's particular military service or of his neurologic condition to lead to any conclusion different from that of the Institute of Medicine, namely that there was no definite etiologic link between Agent Orange exposure and subsequent Parkinson's disease.

III. Analysis

The law provides that service connection may be established for chronic disability resulting from disease or injury incurred in or aggravated by service. 38 U.S.C.A. § 1110; 38 C.F.R. § 3.303. In addition, service connection may be granted for any disease diagnosed after discharge, when all the evidence, including that pertinent to service, establishes that the disease was incurred in service. 38 C.F.R. § 3.303(d).

The Board notes that a change in the law has taken place with respect to the adjudication of claims based upon exposure to Agent Orange during service in Vietnam. On December 27, 2001, the Veterans Education and Benefits Expansion Act of 2001 (VEBEA), Pub. L. No. 107-113, 115 Stat. 976 (2001) was signed into law. That new statute, in pertinent part, redesignated and amended 38 U.S.C.A. § 1116(f) to provide that, for purposes of establishing service connection for a disability or death resulting from exposure to an herbicide agent, including a presumption of service connection under this section, a veteran who, during active military, naval, or air service, served in Vietnam during the period beginning on January 9, 1962, and ending on May 7, 1975, shall be presumed to have been exposed during such service to an herbicide agent of the kind specified in section 1116, unless there is affirmative evidence to establish that the veteran was not exposed to any such agent during that service. See 38 U.S.C.A. § 1116 (West 2002).

If a veteran was exposed to an herbicide agent during active
military, naval, or air service, the following diseases shall be service-connected if the requirements of 38 U.S.C.A. § 1116 and 38 C.F.R. § 3.307(a)(6)(iii) are met, even though there is no record of such disease during service, provided further that the rebuttable presumption provisions of 38 U.S.C.A. § 1113 and 38 C.F.R. § 3.307(d) are also satisfied: chloracne or other acneform disease consistent with chloracne; type II diabetes mellitus; Hodgkin's disease; multiple myeloma; non-Hodgkin's lymphoma; acute and subacute peripheral neuropathy; porphyria cutanea tarda (PCT); prostate cancer; respiratory cancers (cancer of the lung, bronchus, larynx, or trachea); and soft-tissue sarcomas (other than osteosarcoma, chondrosarcoma, Kaposi's sarcoma, or mesothelioma). 38 C.F.R. § 3.309(e) (2004); see also 38 U.S.C.A. § 1113 (West 2002); 38 C.F.R. § 3.307 (2004).

VA has determined that a presumption of service connection based on exposure to herbicides used in the Republic of Vietnam during the Vietnam era is not warranted for any condition for which VA has not specifically determined a presumption of service connection is warranted. See 59 Fed. Reg. 341-46 (1994); 61 Fed. Reg. 414421 (1996); see also 64 Fed. Reg. 59232 (1999); 67 Fed. Reg. 42600-42608 (2002). More recently, VA clarified that a presumption of service connection based on exposure to herbicides used in the Republic of Vietnam during the Vietnam Era is not warranted for the following conditions: hepatobiliary cancers, nasopharyngeal cancer, bone and joint cancer, breast cancer, cancers of the female reproductive system, urinary bladder cancer, renal cancer, testicular cancer, leukemia (other than CLL), abnormal sperm parameters and infertility, Parkinson's disease and parkinsonism, amyotrophic lateral sclerosis (ALS), chronic persistent peripheral neuropathy, lipid and lipoprotein disorders, gastrointestinal and digestive disease, immune system disorders, circulatory disorders, respiratory disorders (other than certain respiratory cancers), skin cancer, cognitive and neuropsychiatric effects, gastrointestinal tract tumors, brain tumors, light chain-associated (AL) amyloidosis, endometriosis, adverse effects on thyroid homeostasis, and any other condition for which the Secretary has not specifically determined a presumption of service connection is warranted. See 68 Fed. Reg. 27,630-41 (May 20, 2003).

Notwithstanding the foregoing, the United States Court of

Following consideration of the evidence of record, the Board finds that entitlement to service connection for Parkinson's disease is warranted. Initially, it is noted that the appellant's DD 214 confirms that the appellant had active service in Vietnam during the Vietnam era. Therefore, the appellant is presumed to have been exposed to herbicides in service. 38 U.S.C.A. § 1116(f). However, the Board also observes that Parkinson's disease is not among the disabilities listed in 38 C.F.R. § 3.309(e). Thus, the appellant may not receive the benefit of a rebuttable presumption that his Parkinson's disease was caused by exposure to Agent Orange. 38 C.F.R. §§ 3.307, 3.309 (2004). Nevertheless, as indicated above, the appellant is not precluded from establishing service connection on a direct basis. See Combee v. Brown, 34 F.3d 1039 (Fed.Cir. 1994); McCartt v. West, 12 Vet. App. 164, 167 (1999).

Upon a review of the evidence of record, the evidence taken as a whole tends toward the conclusion that the appellant's Parkinson's disease was caused by his herbicide exposure while in service. The Board recognizes that, as previously stated, in Fast Letter 03-20, issued by the Veterans Benefits Administration on June 25, 2003, it was noted that a study by the National Academy of Sciences study found that the credible evidence against an association between herbicide exposure and Parkinson's disease outweighed the credible evidence for such an association. However, in support of the appellant's contention that his Parkinson's disease was due to his exposure to Agent Orange while he was serving in the Republic of Vietnam, the appellant has submitted a private medical statement from Dr. Muther, dated in June 2002. In the June 2002 statement, Dr. Muther indicated that no explanation for the appellant's diagnosed Parkinson's disease had been found "except a possible exposure to Agent Orange."  Dr. Muther
further noted that Agent Orange had been demonstrated to be a neurotoxin, and, as such, he opined that it was highly possible that that was a contributing factor in the etiology of the appellant's Parkinson's disease. In addition, in the appellant's August 2004 VA examination, the examiner stated that although it was possible that the appellant was exposed to lead based paints due to his occupation of remodeling rental homes, in light of the fact that the appellant's heavy metal screens came back negative, such exposure could be "safely eliminated" as a cause of the appellant's Parkinson's disease. Moreover, following a review of pertinent medical literature, the examiner opined that it was "at least as likely as not" that the appellant's Parkinson's disease may be related to exposure to Agent Orange or other herbicide exposure in Vietnam. Furthermore, in the March 2005 opinion from the VHA neurologist, although he stated that his conclusion was no different from that of the Institute of Medicine, namely that there was no definite etiologic link between Agent Orange exposure and subsequent Parkinson's disease, the VHA neurologist also noted that the Institute of Medicine had concluded that an etiologic connection between pesticide/herbicide exposure was "biologically plausible."

The medical opinions in this case are less than absolute in their conclusions. However, given the nature of cases such as this one, in which most causes of the claimed disability are idiopathic and the passage of a significant amount of time between separation from service and the filing of a claim with VA, and the fact that medicine is still a somewhat inexact science, the Board must resign itself to dealing with medical opinion evidence couched in terms such as "highly possible," "at least as likely as not," and "biologically plausible," rather than absolutes. See Lathan v. Brown, 7 Vet. App. 359, 366 (1995) (medicine is more art than exact science). Although none of the medical opinions of record are couched in terms of absolute certainty, none have to be.

In any event, the standard of review which must be applied by the Board is found in 38 U.S.C.A. § 5107(b). Under the benefit-of-the-doubt rule, in order for a claimant to prevail, there need not be a preponderance of the evidence in the veteran's favor, but only an approximate balance of the positive and negative evidence. In other words, the preponderance of the evidence must be against the claim for the benefit to be denied. Gilbert v. Derwinski, 1 Vet. App.
49, 54 (1990). Thus, in consideration of the aforementioned evidence, the Board finds that the evidence for and against the appellant's claim for service connection for Parkinson's disease is in a state of relative equipoise. With reasonable doubt resolved in the appellant's favor, the Board concludes that service connection is warranted.

ORDER

Entitlement to service connection for Parkinson's disease is granted.

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JOY A. MCDONALD
Veterans Law Judge, Board of Veterans' Appeals

Department of Veterans Affairs