U. S. DEPARTMENT OF LABOR

Employees' Compensation Appeals Board

In the Matter of JAMES H. TATUM and U.S. POSTAL SERVICE,
POST OFFICE, Stockton, CA

Docket No. 98-1836; Submitted on the Record;
Issued March 20, 2000

DECISION and ORDER

Before GEORGE E. RIVERS, DAVID S. GERSON,
MICHAEL E. GROOM

The issue is whether the Office of Workers' Compensation Programs met its burden of proof to terminate appellant's compensation benefits on November 11, 1995.

On May 9, 1994 appellant, then a 44-year-old manager of customer service, filed a claim for an occupational disease, Form CA-2, alleging that he sustained Leber's optic neuropathy due to stress at work. In October 1993 the employing establishment underwent downsizing which resulted in appellant being assigned additional work including that of three other supervisors whose positions were eliminated. In March 1994, due to appellant's problems with his vision, the additional duties were removed. On August 8, 1994 the Office accepted appellant's claim for bilateral aggravation of ischemic optic neuropathy. Appellant began receiving compensation as of May 23, 1994. He retired on December 23, 1994.

By decision dated November 8, 1995, the Office terminated compensation benefits, effective November 8, 1995, finding that appellant's continuing disability was not related to factors of federal employment. The Office found that a conflict which existed between appellant's treating physician, Dr. Galen E. Winegardner, a Board-certified ophthalmologist, and a referral physician, Dr. Barrett Katz, a Board-certified ophthalmologist, as to whether appellant's disability of optic neuropathy was work related was resolved by the impartial medical specialist, Dr. Sharon Lutosky, a Board-certified ophthalmologist, who opined appellant's current disability was not work related.

In a report dated March 25, 1994, Dr. Theresa S. de Barros, a Board-certified ophthalmologist, diagnosed Leber's optic neuropathy. She stated:

> "This is a genetic degeneration of the optic nerve, which could, theoretically be precipitated by extreme fatigue and stress as well as many other factors. The person with his disease has a hereditary predisposition to develop the condition but other external influences are believed to contribute to the appearance of the

> atrophy. Apparently, appellant was doing the work of three people in the months just prior to the onset of his symptoms and was under a great deal of work-related stress."

In his July 10, 1995 report, Dr. Winegardner considered appellant's history of injury, performed a physical examination, and diagnosed lateral anterior ischemic optic nerve neuropathy, "which is presumed labors hereditary optic neuropathy." He stated that upon appellant's return to work, "it was not simply coincidence and that because of the continued stress at work he did continue to deteriorate until taking leave of his duties at the [employing establishment]." Dr. Winegardner attached a letter from Dr. William H. Hoyt, a Board-certified opthalmologist, dated November 16, 1993 in which he diagnosed anterior ischemic optic nerve neuropathy.

In his report dated May 24, 1995, Dr. Katz considered appellant's history of injury, performed a physical examination, and noted that appellant had deoxyribonucleic acid (DNA) testing done which revealed mutations at 13708 and 15257 positions. He diagnosed, *inter alia*, vision of 20/70 *oculus dexter*, *i.e.*, right eye, 20/200, *oculus sinister*, *i.e.*, left eye, dyschromatopsia observation unit (OU), dyschromatopsia OU, sluggish pupillary response without obvious afferent defect OU, diffuse pallor of each optic nerve head associated with surface gliosis, and central scotoma on visual field testing. He stated:

> "It is my supposition that this man has Leber's hereditary optic neuropathy. There is a genetic defect that was programmed to go off; it went off this past year. I can in no good conscience consider this a work-related event; I suspect what happened to him would have happened to him on an island in the South Pacific as it happened here, with the job he had.... While stress makes everything worse, I do not think it all appropriate or fair to consider this a stress-related event. And so this is not an occupational disease, it was not aggravated by his work situation, it was not in all medical probability accelerated or precipitated by his employment."

In her October 31, 1995 report, Dr. Lutosky considered appellant's history of injury, the DNA testing, and performed a physical examination. She diagnosed post-papillitic Leber's hereditary optic neuropathy (LHON). Dr. Lutosky admitted that she was not a specialized researcher on LHON and that such a person would best be able to speculate on what actually triggers the asymptomatic onset of LHON disease. She stated that so far there had not been enough data "to clearly associate stress as a triggering factor to LHON decompensation." Further, Dr. Lutosky stated that there were no provable facts to uphold appellant's belief that his job stress caused his visual decompensation. In addressing what caused LHON, she stated:

> "No one can argue the temporal association between job stress and [appellant's] illness. In science, however, temporal association does not necessarily imply a cause and effect relationship. [F]or now I do n[o]t think anyone can say with certainty -- based on currently available scientific knowledge -- that job stress can be said to have precipitated or aggravated [appellant's] condition."

Dr. Lutosky stated that appellant had suffered markedly from LHON and was legally blind. She concluded that it was not possible to state with certainty that job stress aggravated appellant's visual decompensation.

By letter dated November 16, 1995, appellant requested an oral hearing before an Office hearing representative which was held on June 19, 1996. At the hearing, appellant explained that in October 1992, the employing establishment underwent a reorganization which resulted in his being assigned three additional jobs and he began to lose vision in both eyes and stopped working on May 23, 1994. Appellant testified that he retired based on the recommendation of his doctors, the employing establishment's inability to find him suitable work and the fact that his claim was accepted.

Appellant also submitted a letter from Janey Youngblom, a Ph.D in genetics at California State University, dated June 17, 1996. In her letter, Dr. Youngblom explained that LHON is an inherited disease that involves a mutation in one or more genes in the mitochondria. She stated that it had been suggested by several researchers that environmental factors might play a role in triggering the onset of the disease. Dr. Youngblom stated:

> "Factors such as excess tobacco and alcohol consumption, head trauma and excessive occupational exposure to toxic substances that stress or directly inhibit the respiratory capacity of the body have all been cited as potential environmental contributors to the initiation or progression of the disease. It is consistent with this train of thinking that excessive mental stress on the job may also compromise the normal cellular function and capabilities of the mitochondria."
>
> "It is noteworthy that the onset of [appellant's] visual problems coincided with his job restructuring, which consequently subject[ed] him to excessive work overload and stress. His condition continued to deteriorate while employed in his position, and then abruptly stabilized when he left his job. His eyes have maintained their stability to the present time. Given the nature of this disease, the particular circumstances of this case, and the evidence that already exists for environmental contributing factors in this disease, it is my professional opinion that work-related stress was a strong contributing factor in [appellant's] situation."

Appellant also submitted an article, "Bureaucratic Misuse of Genetic Information," by Scott Winokur.

By decision dated August 29, 1996, the Office hearing representative affirmed the Office's November 8, 1995 decision.

By letter dated June 3, 1997, appellant requested reconsideration of the Office's decision and submitted additional evidence consisting of a letter from Dr. Youngblom dated May 19, 1997, a letter from Daniel A Peterson, Ph.D, from the Salk Institute for Biological Studies, a report from Dr. Dana A. Jungschaffer, an ophthalmologist, dated January 31, 1997, a letter from Ruth Hubbard, a biology professor from Harvard University, dated May 14, 1997 and General Service Administration regulations pertaining to smoking and a sketch of the offices at his

workplace. Appellant stated that he was exposed to second hand smoke at work particularly in November 1992 when his Office designated the area where he worked as a smoking area.

In his May 19, 1997 report, Dr. Youngblom reiterated her opinion that work-related stress "probably" was a strong contributing factor in the onset of appellant's LHON. She concluded that the combination of work-related stress, *i.e.*, the downsizing and reorganization appellant's office underwent, and exposure to second hand smoke played a critical role in the onset of appellant's LHON disease.

In his January 8, 1997 report, Dr. Peterson explained that the late onset of appellant's LHON, *i.e.*, in his forties instead of his twenties, might be attributed to "epigenetic factors, that is, environmental factors beyond the actual mutation causing the disease to be manifested." He stated that "since [appellant] reported both psychological stress due to work load with concomitant environmental stressors such as tobacco smoke, these elements should be considered as material in attributing the late onset manifestation of his LHON."

In his January 31, 1997 report, Dr. Jungschaffer performed a physical examination and stated:

> "Although [LHON] is hereditary and [appellant] has had DNA testing documenting specific mutations consistent with Leber's hereditary optic neuropathy, environmental factors such as stress or tobacco smoke could well have contributed to his disease process. It is well known that stress, both psychological as well as physical stressors such as nicotine can and do increase circulating levels of catecholamines. These corticosteroids may play a role in the expression of his disease."

Referring to a couple of authors, including Dr. Peterson, Dr. Jungschaffer stated that they suggested that epigenetic factors might be responsible for the disease. He concluded that "given the onset of [appellant's] visual loss coinciding with an extremely stressful time at his workplace," it was reasonable to conclude that environmental factors contributed to appellant's disease.

In her May 14, 1997 report, Ms. Hubbard stated that she was familiar with the medical and employment situation of appellant and stated:

> "[I]t was impossible to predict that [appellant's condition] would have occurred irrespective of the stress he experienced due to excessive work pressures and his having to work in a designated smoking area. Quite the contrary, it is very likely that these conditions helped bring on his condition or, at the very least, hastened its onset."

By letter dated June 19, 1997, the Office denied modification of its prior decision.

By letter dated September 23, 1997, appellant requested reconsideration and submitted an article by Joel Deane.

By decision dated April 23, 1998, the Office denied modification of its prior decisions.

The Board finds that the Office met its burden of proof to terminate appellant's compensation benefits effective November 8, 1995.

Once the Office accepts a claim, it has the burden of justifying termination or modification of compensation benefits. After it has determined that an employee has disability causally related to his or her federal employment, the Office may not terminate compensation without establishing that the disabling condition has ceased or that it is no longer related to the employment.[1] The Office's burden of proof includes the necessity of furnishing rationalized medical evidence based on a medical background.[2]

In situations where there are opposing medical reports of virtually equal weight and rationale, and the case is referred to an impartial medical specialist for the purpose of resolving the conflict, the opinion of such specialist, if sufficiently well rationalized and based on a proper factual background, must be given special weight.[3] In the present case, the Office referred appellant to the impartial medical specialist, Dr. Lutosky, a Board-certified ophthalmologist, to resolve the conflict in the evidence between Dr. Winegarden's July 10, 1995 opinion that appellant's LHON was related to stress at work and Dr. Katz's May 24, 1995 opinion that appellant's LHON was not related to stress at work. In her October 31, 1995 report, Dr. Lutosky reviewed appellant's history of injury, the DNA testing showing mutant chromosomes and performed a physical examination. She admitted that she was not a specialized researcher on LHON but stated that there were no provable facts to establish that appellant's job stress caused his visual decompensation. She noted the temporal connection between the onset of appellant's LHON and the stress at work, but concluded that it was not possible to state with certainty that the stress at work caused or contributed to appellant's LHON because there was not enough scientific data to establish that stress triggered LHON. Dr. Lutosky's opinion is sufficiently rationalized to establish that appellant's current disability of LHON is not related to stress at work. Therefore, as an impartial medical specialist, Dr. Lutosky's opinion constitutes the weight of the evidence.

The Federal Employees' Compensation Act[4] and the regulations interpreting the Act[5] make clear that the term "physician" includes only physicians who have an M.D. or O.D. degree, surgeons, podiatrists, dentists, clinical psychologist, optometrists and chiropractors within the scope of their practice as defined by state law.[6] Thus, despite the obvious expertise and well-reasoned letters of Dr. Youngblum dated June 17, 1996 and May 19, 1997 in which she

[1] *Patricia M. Mitchell*, 48 ECAB 371 (1987); *Patricia A. Keller*, 45 ECAB 278 (1993).

[2] *Larry Warner*, 43 ECAB 1027 (1992); *see Del K. Rykert*, 40 ECAB 284, 295-96 (1988).

[3] *Kathryn Haggerty*, 45 ECAB 383, 389 (1994); *Jane B. Roanhaus*, 42 ECAB 288 (1990).

[4] 5 U.S.C. § 8101(2).

[5] 20 C.F.R. § 10.400(a).

[6] *Sheila A. Johnson*, 46 ECAB 323, 326 (1994).

attributed appellant's LHON to his stress at work, she does not qualify as a physician within the meaning of the Act. Her opinion on causation therefore does not have probative medical value in this case. Similarly, the letters from Dr. Peterson dated January 8, 1997 and Ms. Hubbard dated May 14, 1997 are not medically probative because Dr. Peterson and Ms. Hubbard do not qualify as physicians within the meaning of the Act. Dr. Billings' opinion, which appellant presented secondhand through testimony and an article "When Science and Society Collide" is not sufficiently specific to appellant's case within the meaning of the Act to be probative, that is, despite appellant asserting that Dr. Billings reviewed his case record, appellant did not submit a report from Dr. Billings containing his review of the record and his opinion specifically based on that information.[7]

The articles appellant submitted, "Bureaucratic Misuse of Genetic Information," and "When Science and Society Collide" are not probative as the Board has held that textual evidence or articles from publications are of little probative value unless a physician shows the applicability of the general medical principles discussed in the text to the specific factual situation at issue in the case.[8]

Moreover, while Dr. Jungschaffer in her January 31, 1997 report concluded that environmental factors contributed to appellant's LHON, her rationale in her opinion that environmental factors such as stress or tobacco smoke "could well have contributed" to his disease process, that stress, both psychological as well as physical stressors such as nicotine "can and do increase circulating levels of catecholamines," and corticosteroids "may play a role" in the expression of the disease is vague and speculative. Her opinion therefore is insufficient to establish that appellant's LHON was related to stress at work.[9] Similarly, Dr. de Barros' March 25, 1994 opinion that genetic degeneration of the optic nerve "could" theoretically be precipitated by extreme fatigue and stress as well as many other factors is vague and speculative and does not specifically relate appellant's LHON to stress. Her opinion therefore is also not probative.

Inasmuch as Dr. Lutosky's opinion that appellant's LHON could not conclusively be related to appellant's stress at work constitutes the weight of the evidence, her opinion justified the Office's termination of benefits. Appellant did not submit additional evidence that negated her opinion.

[7] *See Durwood H. Nolin*, 46 ECAB 818, 821-22 (1995); *Ruby I. Fish*, 46 ECAB 276, 282 (1994).

[8] *Id.*

[9] *See Alberta S. Williamson*, 47 ECAB 569, 573-74 (1996); *William S. Wright*, 45 ECAB 498, 503-04 (1994).

The decisions of the Office of Workers' Compensation Programs dated April 23, 1998 and June 19, 1997 are hereby affirmed.

Dated, Washington, D.C.
March 20, 2000

George E. Rivers
Member

David S. Gerson
Member

Michael E. Groom
Alternate Member