

Dr. Licensed Physician

[Specialty]

October 8, 2026

Robert A. Sample

DOB: 03/14/1968

Clinical Background of the Authoring Physician:

I am a [board certification withheld in this sample] [Specialty] physician. I completed my residency in [Specialty] at [institution] in [year], and I have practiced as an attending physician in [Specialty] since that time.

Medical Records Reviewed:

I have reviewed the entirety of the records submitted for Mr. Sample in connection with this independent medical opinion. These records consist of his service treatment records for the period of August 3, 1988 through July 29, 1994, comprising 112 pages, including his enlistment examination of May 23, 1988, the troop medical clinic note of June 11, 1991 documenting an acute lumbar injury and the follow-up notes that came after it, and his separation examination and Report of Medical History of May 12, 1994; his DD Form 214 and Official Military Personnel File materials, comprising 41 pages; his Department of Veterans Affairs claims file comprising 1,286 pages, including the rating decisions of February 7, 1995, March 20, 2012, May 2, 2013, October 16, 2017 and August 21, 2025 with their reasons for decision, and the VA contract examination and medical opinions of July 9, 2025; the veteran's VA Blue Button medical record generated September 14, 2026 comprising 418 pages, covering his VA primary care, mental health, physical medicine and rehabilitation, physical therapy, MOVE! Weight Management Program, nutrition, podiatry, sleep medicine and neurology care, including the attended in-laboratory polysomnography of February 19, 2025 and the nerve conduction study and electromyography of August 6, 2025; 64 pages of records from his private family medicine practice for the period of March 2009 through August 2012; 22 pages of private orthopedic records, including magnetic resonance imaging of the lumbar spine performed November 15, 2011; and lay statements from Mr. Sample dated August 30, 2026 and from his wife, Linda M. Sample, dated September 2, 2026.

Overview of Case:

Mr. Sample is a 58-year-old veteran of the United States Army who served on active duty from August 3, 1988 to July 29, 1994 with the military occupational specialty of Cannon Crewmember, 13B. He completed basic combat training and advanced individual training at Fort Sill, Oklahoma, and served the remainder of his enlistment with a 155 millimeter towed howitzer battalion at the same installation, rising to the rank of Sergeant and serving as a howitzer gunner. His characterization of service was honorable. His DD Form 214 records no foreign service and nothing in his personnel file places him in the Southwest Asia theater of operations, so the presumptive provisions of 38 CFR section 3.317 do not apply, and this opinion proceeds on a secondary basis under 38 CFR section 3.310.

His service treatment records document the injury from which the rest of this case follows. On June 11, 1991, during a field training exercise, he presented to the troop medical clinic reporting that he had felt a "pop" in his low back while lifting a 155 millimeter projectile onto the loading tray of his howitzer. A 155 millimeter high explosive projectile weighs approximately 95 pounds. The physician assistant who saw him, R. Delgado, PA-C, recorded spasm of the muscles alongside the lumbar spine, forward bending limited by pain and a negative straight leg raise, and entered an assessment of "acute lumbosacral strain." A fourteen-day physical profile prohibited lifting more than 20 pounds. He returned for low back pain on September 23, 1991, February 4, 1992 and January 11, 1993, when the assessment was recorded as "chronic LBP, mechanical," and he completed six sessions of physical therapy in the spring of 1993. On the Report of Medical History at his separation examination of May 12, 1994 he checked "Yes" to recurrent back pain, and the examining physician wrote "LBP since 6/91, recurrent, no radicular sx." His recorded height at separation was 72 inches and his weight 178 pounds, a body mass index of 24.1, which is in the normal range.

The Department of Veterans Affairs has recognized the residuals of that injury since 1995. The rating decision of February 7, 1995 granted service connection for lumbosacral strain at 10 percent, effective the day after his separation. The rating decision of March 20, 2012 increased that evaluation to 20 percent. The rating decision of May 2, 2013 granted service connection for major depressive disorder at 50 percent as secondary to the service-connected lumbar spine disability, and separately granted service connection for tinnitus at 10 percent on the basis of his artillery noise exposure. The rating decision of October 16, 2017 recharacterized the spine disability as degenerative disc disease of the lumbar spine, increased it to 40 percent on a measured forward flexion of 25 degrees, and granted service connection for radiculopathy of the left lower extremity at 10 percent

under Diagnostic Code 8520. In its decision of May 2, 2013 the Department wrote that his "chronic pain and the resulting loss of employment and physical function" were "the precipitating factors in the onset of [his] depressive disorder." The Department has therefore already found, in its own words, that this veteran's service-connected back took away his physical function and his work. That finding is the first link in every opinion that follows.

The conditions at issue are obstructive sleep apnea, type 2 diabetes mellitus, hypertension, peripheral neuropathy of both lower extremities, and erectile dysfunction. The first three were denied in the rating decision of August 21, 2025, which relied on a VA contract examination of July 9, 2025 that I address condition by condition below. Obesity is not claimed as a disability and I do not treat it as one. It appears in this opinion only as an intermediate step in the chain of causation, which the Department's General Counsel has expressly permitted.

The framework I apply to the first three conditions is the analysis set out in VAOPGCPREC 1-2017, a precedent opinion of the Department's General Counsel dated January 6, 2017. That opinion holds that obesity is not itself a disease or a disability for compensation purposes, but that it "may be an 'intermediate step' between a service-connected disability and a current disability that may be service connected on a secondary basis." It requires three determinations: first, whether the service-connected disability caused the veteran to become obese; second, whether the obesity resulting from the service-connected disability was a substantial factor in causing the claimed condition; and third, whether the claimed condition would not have occurred but for obesity caused by the service-connected disability. I note that the example the General Counsel chose to illustrate the analysis was a veteran whose "service-connected back disability causes obesity due to lack of exercise, which leads to hypertension." That is very nearly a description of Mr. Sample. Where a condition is instead caused directly by another service-connected condition or by the treatment of one, I apply 38 CFR section 3.310(a) without the intermediate step, and where a condition has been worsened by a service-connected condition I apply the aggravation provision of 38 CFR section 3.310(b). Finally, although 38 CFR section 3.102 requires that reasonable doubt be resolved in the veteran's favor when the evidence is in approximate balance, I do not rely on that provision for any opinion in this report. I hold each of them to the standard of more likely than not.

Obesity as the intermediate step:

The weight record is long and it is consistent. He separated at 178 pounds in July of 1994. When his private family medicine records begin in March of 2009 he weighed 207 pounds, a body mass index of 28.1, which is overweight but not obese. That interval of fifteen years represents a gain of roughly two pounds a year, and I want to be fair about it. He smoked about half a pack of cigarettes a day from the age of eighteen until he quit in March of 2006, and a meta-analysis published in the BMJ by Aubin and colleagues in 2012 found that smokers gain a mean of 4 to 5 kilograms, about 10 pounds, in the year after quitting. Some of that early gain is therefore ordinary and I do not attribute it to service. Throughout those years he worked full time as a building maintenance technician, a job that kept him on his feet, climbing and lifting, and his weight changed little.

The point at which that changed is documented. In October of 2011 his back worsened to the point that his private orthopedic surgeon obtained magnetic resonance imaging of the lumbar spine, which on November 15, 2011 demonstrated degenerative disc disease at L4-L5 and L5-S1 with a left-sided disc protrusion at L5-S1 abutting the left S1 nerve root. The orthopedic note of March 27, 2012 records that he was "unable to return to work requiring ladders, prolonged standing or lifting over 25 lbs," and he stopped working the following month. His VA psychiatrist, Dr. L. Brennan, diagnosed major depressive disorder on September 24, 2012 with a PHQ-9 score of 19, which falls in the moderately severe range, and her note attributes the episode to "chronic pain, loss of job and loss of identity as a provider." He weighed 219 pounds that day, a body mass index of 29.7. From that point the weight curve changes shape. He weighed 236 pounds on January 12, 2016, 263 pounds on March 7, 2017, 271 pounds on February 10, 2020, and reached his peak of 284 pounds on June 3, 2024, a body mass index of 38.5. He weighed 279 pounds at his most recent primary care visit of August 18, 2026. Having gained about 29 pounds in the fifteen years after service, he gained 65 pounds in the twelve years after his back took him out of work and his depression began.

Three drivers of that gain appear in the record, and each is either a service-connected disability or the treatment of one. The first is the loss of physical activity imposed by the service-connected spine. The VA physical medicine and rehabilitation note of February 13, 2018 records that he could walk "approximately one block" before he had to stop and could stand for about ten minutes. On a six-minute walk test performed by VA physical therapy on May 8, 2019 he covered 190 meters with two rest stops for back pain, less than half the distance expected of a healthy man of his age and size, and he was issued a cane that month. Physical activity is the part of a person's daily energy expenditure that is

under voluntary control, and a man whose spine limits him to a block of walking has lost most of it. His own statement of August 30, 2026, that he "used to hunt every fall and walk the fields all day" and has not been able to do so in more than ten years, is consistent with what his clinicians recorded.

The second driver is the service-connected major depressive disorder itself. In a meta-analysis of fifteen longitudinal studies involving 58,745 participants, Luppino and colleagues found that depression increased the odds of later developing obesity, with an odds ratio of 1.58. I want to state that finding precisely, because the same study found that the relationship runs in both directions, and that obesity also increased the odds of later depression. The direction therefore matters, and in this record it is fixed by the dates. Mr. Sample was not obese when his depression was diagnosed in September of 2012. He became obese after it. His mental health notes repeatedly document eating driven by his mood, for example on April 15, 2015, "eating at night when he can't sleep, eating out of boredom and low mood," and his depression has never remitted; his PHQ-9 scores have ranged from 12 to 19 throughout his treatment.

The third driver is the medication prescribed to treat that depression. On January 12, 2016 Dr. Brennan added mirtazapine 15 milligrams at bedtime to his venlafaxine for insomnia and residual depressive symptoms, and increased it to 30 milligrams in June of 2016. In a comprehensive meta-analysis of antidepressants and body weight, Serretti and Mandelli identified mirtazapine as one of three antidepressants associated with a greater risk of weight gain. Mr. Sample weighed 236 pounds the day it was started and 263 pounds fourteen months later. Dr. Brennan's note of March 7, 2017 records "wt gain 27 lbs since mirtazapine started, pt distressed about weight," and records that the medication was continued because two attempts to taper it had produced a return of severe insomnia and worsening mood. I would emphasize that this drug was selected by the Department's own treating psychiatrist to treat a service-connected illness, and was continued by her, for sound clinical reasons, with full knowledge of its effect on his weight.

The record also answers the question of whether this weight gain reflects a lack of effort on his part. It does not. Mr. Sample enrolled in the VA MOVE! Weight Management Program in March of 2018. The registered dietitian documented that he was "unable to meet physical activity goals due to chronic low back pain" and could tolerate only seated exercise. He lost 9 pounds by September of 2018 and then regained it. A program whose physical activity component he could not perform was unlikely to succeed, and it did not.

I have looked for other causes of his weight gain, and the record does not supply one. His thyroid stimulating hormone has been normal on every measurement, most recently 2.1

on January 9, 2025. He has never received systemic corticosteroids for any sustained period. His alcohol screening (the AUDIT-C questionnaire administered annually by VA) has scored 1 to 2 throughout his VA care, which is low risk. Setting aside the weight he gained after he stopped smoking, which I have conceded above, there is no explanation in this record for the gain of 65 pounds between 2012 and 2024 other than the loss of activity imposed by his spine, the depression his spine caused, and the drug used to treat that depression.

His primary care physician reached the same conclusion years before any claim was filed. Dr. Castillo's assessment of February 10, 2020 reads "Obesity, BMI 36.8, multifactorial: inactivity d/t chronic LBP, mirtazapine-assoc wt gain." On the first question posed by VAOPGCPREC 1-2017 I therefore find that Mr. Sample's service-connected lumbar spine disability, together with the major depressive disorder the Department has already found to be secondary to it and the medication prescribed for that disorder, caused him to become obese. It is more likely than not that his obesity resulted from his service-connected disabilities.

Obstructive sleep apnea:

Mr. Sample was seen in VA sleep medicine by Dr. J. Whitaker on January 14, 2025. Dr. Whitaker recorded an Epworth Sleepiness Scale score of 15 out of 24, which indicates excessive daytime sleepiness, a neck circumference of 18.5 inches, a crowded throat on examination (Mallampati class III, meaning that only the soft palate and the base of the uvula can be seen past the tongue), and a history from his wife of "loud nightly snoring and witnessed apneas for about ten years." Attended in-laboratory polysomnography on February 19, 2025 recorded an apnea-hypopnea index (the number of times per hour that breathing stops or becomes shallow during sleep) of 34.6 events per hour, rising to 51.2 when he slept on his back and 47.9 in dream (REM) sleep, with his oxygen saturation falling as low as 81 percent and remaining below 90 percent for 14.2 percent of the night. An index of 30 or more is severe obstructive sleep apnea. The central apnea index was 0.3 per hour, which excludes a meaningful central component, and he takes no opioid medication. A titration study on March 18, 2025 established an effective pressure of 11 centimeters of water, and he has used continuous positive airway pressure (CPAP) since April of 2025. His device download of August 2025 shows use on 88 percent of nights for four hours or more with a residual index of 3.1 events per hour, and his Epworth score has fallen to 8.

The date of the sleep study is not the date the disease began. Mrs. Sample's statement of September 2, 2026 places the onset of his snoring "not long after he stopped working and

started putting on the weight," and describes him stopping breathing in his sleep for "at least ten years." The contemporaneous record corroborates her. On November 6, 2017 his VA primary care physician, Dr. M. Castillo, recorded daytime fatigue and that his "wife reports heavy snoring," and attributed the fatigue to mirtazapine; no sleep study was ordered at that time. He weighed 266 pounds at that visit. Nothing in his private records of 2009 through 2012, or in his VA records before 2017, documents snoring, witnessed apneas or excessive sleepiness. I find it more likely than not that his obstructive sleep apnea was present and clinically significant by 2017, eight years before it was diagnosed, and I find Mrs. Sample's account consistent with the clinical record in every particular I was able to check.

The relationship between body weight and sleep apnea is among the best established in pulmonary medicine, and it is a causal relationship rather than a mere association. Fat deposited in the tissues surrounding the upper airway narrows it, and fat in the chest and abdomen reduces the lung volume that helps hold the airway open, so that when the muscles of the throat relax in sleep the airway collapses. In the Wisconsin Sleep Cohort, Peppard and colleagues studied 690 adults with polysomnography repeated at four-year intervals and found that a 10 percent gain in weight predicted an approximately 32 percent increase in the apnea-hypopnea index and a six-fold increase in the odds of developing moderate to severe sleep-disordered breathing, while a 10 percent loss of weight predicted a 26 percent decrease in the index. Mr. Sample's weight rose 27 percent between 2009 and 2017 and 37 percent between 2009 and 2024.

Male sex and age also raise the risk of sleep apnea, and I do not ignore them. They were, however, the same for this man at 207 pounds as at 284 pounds, and his airway symptoms appeared only after the weight gain I have described. His upper airway was never examined for sleep purposes before 2025, so I cannot exclude some degree of anatomic predisposition. But anatomic predisposition is precisely the circumstance in which weight gain converts a narrow airway into a collapsing one.

Applying the three-part analysis: his service-connected disabilities caused his obesity, as set out above; the obesity was a substantial factor in causing his obstructive sleep apnea, as the longitudinal data and his own history demonstrate; and it is more likely than not that he would not have developed obstructive sleep apnea of this severity but for that obesity, because his symptoms appeared only after the weight gain and because a gain of the magnitude he experienced is associated with a six-fold increase in the odds of the disease.

I must respectfully disagree with the VA contract examiner of July 9, 2025, whose rationale on this condition reads in full: "The veteran's OSA is most likely due to his obesity (BMI 38.5). Obesity is the strongest risk factor for OSA. Obesity is not a service-connected disability. Medical literature does not support a causal relationship between degenerative disc disease of the lumbar spine or major depressive disorder and obstructive sleep apnea." I agree that obesity caused his sleep apnea. But the examiner stopped at the point where VAOPGCPREC 1-2017 requires the inquiry to begin. Having identified obesity as the cause, the examiner never asked what caused the obesity, and the opinion does not mention the General Counsel's analysis at all. The statement that no literature links disc disease directly to sleep apnea is accurate and beside the point, because no one contends that a degenerated disc obstructs an airway. The examiner also answered the separate question of aggravation in a single sentence, that the condition "is stable on current treatment." Whether a condition is stable on treatment is not the question that 38 CFR section 3.310(b) asks.

Type 2 diabetes mellitus:

Mr. Sample's hemoglobin A1c (a blood test that reflects average blood sugar over the preceding three months) has tracked his weight with unusual fidelity. It was 5.4 percent in March of 2009, when he weighed 207 pounds; 5.7 percent in April of 2013; 5.9 percent in January of 2016; 6.2 percent in February of 2020, when prediabetes (R73.03) was entered on his problem list; and 6.4 percent in July of 2023. On October 2, 2024 it was 6.9 percent with a fasting glucose of 138 milligrams per deciliter, confirmed at 7.0 percent on October 28, 2024, and Dr. Castillo diagnosed type 2 diabetes mellitus. A value of 6.5 percent or higher is diagnostic. He was started on metformin, now 1,000 milligrams twice daily, and on November 14, 2024 a VA registered dietitian prescribed a carbohydrate-controlled diet, which his primary care notes have reinforced at every visit since. His most recent value, on August 18, 2026, was 7.4 percent.

Excess body fat, and in particular fat stored within the abdomen, makes the body's tissues resistant to insulin, and when the pancreas can no longer secrete enough insulin to overcome that resistance, blood sugar rises. This is taught in medical school physiology and pathology classes all over the United States, and it is why body weight is the dominant modifiable cause of type 2 diabetes. The strongest evidence that the relationship is causal comes from the Diabetes Prevention Program, reported by Knowler and colleagues in the New England Journal of Medicine in 2002. Among 3,234 people with elevated blood sugar who did not yet have diabetes, a lifestyle program whose goals were a weight loss of at least 7 percent and at least 150 minutes of physical activity a week reduced the incidence of diabetes by 58 percent. When removing an exposure

removes most of the disease, the exposure is a cause. I would add an observation specific to this veteran. The two levers the Diabetes Prevention Program used were weight loss and physical activity. His service-connected spine has taken away the second and, through the mechanisms described above, defeated the first.

The contract examiner of July 9, 2025 relied on a family history of diabetes in his mother, and I agree that the history is real; his VA primary care intake records that his mother developed type 2 diabetes in her sixties. A family history means that he carried an inherited susceptibility. It does not mean he was destined to develop the disease at 56, and his own laboratory record is the best evidence on the point: he carried exactly the same genes in 2009, when his hemoglobin A1c was 5.4 percent. The participants in the Diabetes Prevention Program were themselves a high-risk group, and weight loss still prevented most of their diabetes. Susceptibility explains why weight gain produced diabetes in this man. It does not explain away the weight gain.

His age is also a risk factor, but the steepest part of his glucose curve came between 2016 and 2024, in the same years his weight rose from 236 to 284 pounds. He has never taken a corticosteroid or an antipsychotic medication for any sustained period, both of which can raise blood sugar, and his records contain no disease of the pancreas.

Applying the three-part analysis: his service-connected disabilities caused his obesity; the obesity was a substantial factor in causing his diabetes, as both the physiology and his own parallel weight and hemoglobin A1c curves demonstrate; and it is more likely than not that the diabetes would not have occurred but for that obesity, because he was normoglycemic at a lower weight with the same family history, and because the best controlled evidence available shows that weight reduction prevents most cases of the disease in people at exactly his level of risk. The examiner's statement that "there is no medical literature to support that a lumbar spine condition causes diabetes mellitus" is, again, accurate and beside the point. No one contends that the spine causes diabetes directly. The contention is the one the General Counsel described, and the examiner did not address it.

Hypertension:

His blood pressure was 118/74 in March of 2009 and 124/80 in September of 2012. It was 134/86 on January 12, 2016 and 142/90 on November 6, 2017, the visit at which his snoring was first recorded. Dr. Castillo then obtained two readings on each of three separate days: 152/102 and 150/100 on November 4, 2019, 148/100 and 146/98 on November 18, 2019, and 154/104 and 150/102 on December 2, 2019, and diagnosed

hypertension (I10). He was started on lisinopril, amlodipine was added in March of 2021, and by June of 2024 he required lisinopril 40 milligrams and amlodipine 10 milligrams daily, both at the top of their usual dosage ranges, with readings that month of 152/94, 148/96 and 150/94.

In my opinion the principal cause of his hypertension is his obstructive sleep apnea, and that relationship does not depend on the intermediate step. Each time the airway closes during sleep, the oxygen level falls and the brain briefly rouses the sleeper to reopen it, and each of those events is accompanied by a surge of adrenaline-like activity from the sympathetic nervous system and a spike in blood pressure. Repeated hundreds of times a night, year after year, that surge persists into the waking day. The Wisconsin Sleep Cohort again provides prospective evidence. Peppard and colleagues, reporting in the New England Journal of Medicine in 2000, found a dose-response relationship between sleep apnea at baseline and hypertension four years later, with an odds ratio of 2.89 at an apnea-hypopnea index of 15 or more, and that relationship held after adjustment for body mass index, neck and waist circumference, age, sex, alcohol and smoking. Marin and colleagues followed 1,889 adults for a median of 12.2 years and found that patients who declined CPAP for their sleep apnea had nearly twice the risk of developing hypertension (hazard ratio 1.96) after adjustment for changes in body mass index, while patients whose sleep apnea was treated with CPAP had no excess risk at all. Sleep apnea is therefore an independent cause of hypertension and not merely a marker of the weight that accompanies it.

The timing in this record fits that mechanism. His snoring was documented in November of 2017, his hypertension was diagnosed two years later, and it escalated to two drugs at full doses while his sleep apnea remained undiagnosed and untreated. Since he began CPAP in April of 2025 his readings have improved, to 138/86 on December 9, 2025 and 136/84 on August 18, 2026, with no change in his antihypertensive regimen. That response to treatment is what the Marin data would predict.

A second service-connected contributor deserves mention. From October of 2011 until June of 2023, Mr. Sample took meloxicam 15 milligrams daily, a nonsteroidal anti-inflammatory drug prescribed for his service-connected spine. Dr. Castillo advised him to stop it on December 2, 2019 because of his blood pressure, and a trial off the drug in March of 2021 failed because of back pain. In a meta-analysis of randomized trials, Johnson and colleagues found that nonsteroidal anti-inflammatory drugs raised supine mean blood pressure by an average of 5.0 millimeters of mercury and could blunt the effect of antihypertensive medication; the agent with the most marked effect in that analysis was piroxicam, which belongs to the same chemical family as meloxicam. For

nearly twelve years the treatment of his service-connected back pushed his blood pressure upward and worked against the drugs used to lower it. I regard this as aggravation within the meaning of 38 CFR section 3.310(b), in addition to the causal pathway through his sleep apnea.

I have also considered his venlafaxine, which can raise diastolic blood pressure, and I decline to rely on it. In a meta-analysis of original data from 3,744 patients, Thase found that effect to be dose-dependent and clinically significant only at doses above 300 milligrams a day. Mr. Sample's dose has never exceeded 225 milligrams. I mention it so that the rating activity knows it was considered.

His father had hypertension, his age increases his risk, and obesity raises blood pressure on its own, which in this case is a further service-connected pathway rather than an alternative to one. None of this alters my conclusion. The contract examiner characterized his condition as "essential hypertension, which is the most common type and has no single identifiable cause." Essential hypertension is a diagnosis of exclusion. It is the name given to high blood pressure when no cause can be found. In this case a cause has been found, it was documented by attended polysomnography five months before the examination, and the examiner's opinion does not mention it.

Peripheral neuropathy of both lower extremities:

On August 21, 2023, fourteen months before his diabetes was diagnosed, Mr. Sample told Dr. Castillo of "burning and numbness in both feet x several months, worse at night." His hemoglobin A1c the month before had been 6.4 percent. At his VA podiatry diabetic foot examination of November 20, 2024 he could not feel a 10-gram monofilament (a standardized nylon filament used to test protective sensation) at the first, third and fifth metatarsal heads of either foot, vibration sense was absent at both great toes, and the ankle reflexes were absent on both sides. He has no weakness of the feet or ankles and no muscle wasting. He was started on gabapentin in September of 2025.

Diabetic neuropathy is a diagnosis of exclusion, as the American Diabetes Association states expressly in its 2017 position statement on the subject, and the workup in this record is appropriate to that standard. His vitamin B12 level was 512 picograms per milliliter on January 9, 2025, which matters because metformin can lower it; his thyroid function was normal; serum protein electrophoresis showed no abnormal monoclonal protein; hepatitis C antibody was negative; and his alcohol screening has been low risk throughout. No other cause of a polyneuropathy has been identified.

The most important document on this condition is the nerve conduction study and electromyography performed by Dr. T. Huang of VA neurology on August 6, 2025, because Mr. Sample already holds a service-connected evaluation for radiculopathy of the left lower extremity, and the rating activity is entitled to know whether the new complaint is the same thing. It is not, and the study shows why. A radiculopathy is an injury to a single nerve root where it leaves the spine. It produces findings on one side, in the muscles that root supplies, and it characteristically spares the sensory nerve responses measured at the ankle, because the root is injured above the point where the sensory nerve cell bodies sit, so the distal sensory fibers stay connected to their cells and continue to conduct. A polyneuropathy is a disease of the nerves themselves, longest fibers first, and it produces symmetric loss of exactly those distal sensory responses. Dr. Huang found both. The sural sensory responses were absent on both sides, the superficial peroneal sensory responses were absent on the right and reduced to 3 microvolts on the left, and the peroneal motor responses were symmetrically reduced at 1.6 and 1.8 millivolts, findings that a left S1 radiculopathy cannot produce in the right leg. Separately, the needle examination showed chronic changes confined to the left medial gastrocnemius and left gluteus maximus, both supplied by the S1 root, with normal findings in the same muscles on the right. The impression reads: "1. Length-dependent, sensory-predominant, axonal sensorimotor polyneuropathy, consistent with diabetic polyneuropathy. 2. Chronic left S1 radiculopathy without active denervation." These are two different diseases with two different causes, and I identify them separately so that the rating activity can apply 38 CFR section 4.14 without evaluating the same manifestation twice.

Because his foot symptoms began before his diabetes was diagnosed, I want to answer directly the objection that the neuropathy came first. The American Diabetes Association recommends that every patient be assessed for neuropathy starting at the diagnosis of type 2 diabetes, a recommendation that presupposes the neuropathy may already be present by then, and its 2017 position statement records emerging evidence that this form of neuropathy may be present in 10 to 30 percent of people with prediabetes. Singleton and colleagues, studying patients with otherwise unexplained neuropathy, found impaired glucose tolerance at nearly three times the expected rate, and noted that 10 percent of patients with diabetes already have neuropathy when the diabetes is diagnosed. Mr. Sample had laboratory prediabetes from at least February of 2020. His neuropathy appeared on the rising limb of the same glucose curve that became diabetes, and it is a product of the same disease.

I find that the peripheral neuropathy of both lower extremities is more likely than not proximately due to his type 2 diabetes mellitus and the dysglycemia that preceded its diagnosis, which are themselves secondary to his service-connected disabilities through the intermediate step described above.

Erectile dysfunction:

This condition stands on a more direct footing than any other in this opinion. It began before his diabetes and before his hypertension, and it began on a documented date in direct response to the treatment of a service-connected condition. On February 19, 2013, five months after Dr. Brennan started him on sertraline, her note records "decreased libido and difficulty maintaining erections since starting sertraline." On May 6, 2014 she changed his antidepressant to venlafaxine, citing a partial response and "persistent sexual side effects," and on August 12, 2014 she recorded that his erectile function was "unchanged." Dr. Castillo prescribed sildenafil in June of 2016 with a partial response and increased the dose to 100 milligrams in December of 2024. His total testosterone was normal at 412 nanograms per deciliter in June of 2016, which excludes low testosterone as the cause. The private family medicine records of 2009 through 2012 contain no complaint of erectile dysfunction.

In a meta-analysis of studies in which sexual function was specifically assessed, Serretti and Chiesa found treatment-emergent sexual dysfunction significantly more often than with placebo for a list of antidepressants headed, in decreasing order of impact, by sertraline and venlafaxine, the two drugs Mr. Sample has taken. The same analysis found no significant difference from placebo for mirtazapine, and I therefore do not attribute his erectile dysfunction to that drug. Depression is also an independent cause; in the Massachusetts Male Aging Study, Araujo and colleagues found depressive symptoms associated with erectile dysfunction with an odds ratio of 1.82 after controlling for aging and other confounding factors.

His condition has since been compounded by diabetes and by hypertension, both of which I have found to be secondary to service. In a meta-analysis of 145 studies, Kouidrat and colleagues found erectile dysfunction in 52.5 percent of men with diabetes overall and in about two thirds of men with type 2 diabetes. He also smoked about half a pack of cigarettes a day for roughly twenty years, a recognized vascular risk factor for erectile dysfunction, and I do not dismiss it. But he had quit seven years before his symptoms began, and the onset of those symptoms is fixed in the record to the months after his first antidepressant was started.

I find that his erectile dysfunction is more likely than not proximately due to his service-connected major depressive disorder and to the antidepressant medications prescribed to treat it, and that it has since been aggravated by his secondary diabetes and hypertension.

Medical Opinion:

Based on my training, experience, and professional qualifications and under penalty of perjury, it is more likely than not that the following conditions are primarily etiologically service connected on a secondary basis under 38 CFR section 3.310, arising from the service-connected degenerative disc disease of the lumbar spine and the service-connected major depressive disorder that is secondary to it:

1. Obstructive sleep apnea. Per VA Diagnostic Code 6847, service connection should be established for Mr. Sample's obstructive sleep apnea as secondary to his service-connected lumbar spine disability and major depressive disorder, through obesity as an intermediate step under VAOPGCPREC 1-2017. Severe obstructive sleep apnea was confirmed by attended polysomnography on February 19, 2025, with an apnea-hypopnea index of 34.6 events per hour and an oxygen saturation nadir of 81 percent, and it requires the use of a continuous positive airway pressure machine, which he has used nightly since April of 2025. That meets the criteria for a 50 percent evaluation.
2. Type 2 diabetes mellitus. Per VA Diagnostic Code 7913, service connection should be established for Mr. Sample's type 2 diabetes mellitus as secondary to his service-connected lumbar spine disability and major depressive disorder, through obesity as an intermediate step under VAOPGCPREC 1-2017. He requires an oral hypoglycemic agent, metformin, and a restricted diet prescribed by a VA registered dietitian on November 14, 2024, which meets the criteria for a 20 percent evaluation. He does not require insulin, and I do not suggest a higher evaluation. Under Note (1) to Diagnostic Code 7913, his compensable complications should be evaluated separately.
3. Hypertension. Per VA Diagnostic Code 7101, service connection should be established for Mr. Sample's hypertension as proximately due to his obstructive sleep apnea, and in the alternative as aggravated beyond its natural progression by that sleep apnea and by the meloxicam prescribed for his service-connected spine under 38 CFR section 3.310(b). His readings at diagnosis, taken twice on each of three different days in November and December of 2019, showed diastolic pressures of 98 to 104, and he has required continuous medication for control since that time. That history supports at least the 10 percent minimum evaluation provided for an individual with a history of diastolic pressure predominantly 100 or more who requires continuous medication for control.

Whether his diastolic pressure was predominantly at that level is a determination for the rating activity on the full set of readings.

4. Peripheral neuropathy of the right and left lower extremities. Per VA Diagnostic Code 8520, or such other lower extremity peripheral nerve code as best fits the distribution, service connection should be established for the peripheral neuropathy of both lower extremities as secondary to Mr. Sample's type 2 diabetes mellitus. The involvement is clinically sensory, with absent monofilament and vibration sensation and absent ankle reflexes but no weakness or wasting, and 38 CFR section 4.124a directs that wholly sensory involvement be rated at the mild or, at most, the moderate degree. I would recommend an evaluation of not less than mild incomplete paralysis, 10 percent, for each lower extremity. This polyneuropathy is a separate disease from his service-connected left S1 radiculopathy, as the electrodiagnostic study of August 6, 2025 demonstrates, and the left-sided evaluations should be coordinated so that no manifestation is evaluated twice under 38 CFR section 4.14.

5. Erectile dysfunction. Per VA Diagnostic Code 7522, service connection should be established for Mr. Sample's erectile dysfunction as secondary to his service-connected major depressive disorder and the sertraline and venlafaxine prescribed to treat it, with subsequent aggravation by his secondary diabetes and hypertension. Diagnostic Code 7522 carries a noncompensable evaluation, and the rating activity should consider special monthly compensation for loss of use of a creative organ under 38 CFR section 3.350(a).

These conditions did not arise independently of one another. Each traces to the injury recorded on June 11, 1991. The back injured that day took away his work and most of his physical activity, caused a depression the Department has already service connected, and was treated for nearly twelve years with a drug that raised his blood pressure. The depression and the medications prescribed for it caused his erectile dysfunction and drove his weight upward, and the weight produced his sleep apnea and his diabetes, from which his hypertension and his neuropathy in turn followed. Where the evidence supports a finding less strongly than I would wish, I have said so, and where I considered a possible cause and rejected it, I have said that as well.

Sources used (not available online):

Mr. Sample's service treatment records for the period of August 3, 1988 through July 29, 1994, 112 pages, including the enlistment examination of May 23, 1988, the troop medical clinic note of June 11, 1991 by R. Delgado, PA-C, the follow-up notes of September 23, 1991, February 4, 1992 and January 11, 1993, the physical therapy notes

of 1993, and the separation examination and Report of Medical History of May 12, 1994. His DD Form 214 and Official Military Personnel File materials, 41 pages. His Department of Veterans Affairs claims file, 1,286 pages, including the rating decisions of February 7, 1995, March 20, 2012, May 2, 2013, October 16, 2017 and August 21, 2025, and the VA contract examination and medical opinions of July 9, 2025. VA Blue Button medical record of September 14, 2026, 418 pages, including the mental health notes of Dr. L. Brennan from September 24, 2012 forward, the primary care notes of Dr. M. Castillo, the physical medicine and rehabilitation note of February 13, 2018, the physical therapy six-minute walk test of May 8, 2019, the MOVE! Weight Management Program notes of 2018 and the nutrition note of November 14, 2024, the podiatry examination of November 20, 2024, the sleep medicine consultation of January 14, 2025 by Dr. J. Whitaker, the polysomnography of February 19, 2025 and titration study of March 18, 2025, and the nerve conduction study and electromyography of August 6, 2025 by Dr. T. Huang. Private family medicine records for the period of March 2009 through August 2012, 64 pages. Private orthopedic records, 22 pages, including magnetic resonance imaging of the lumbar spine of November 15, 2011 and the office note of March 27, 2012. Lay statements of Robert A. Sample dated August 30, 2026 and of Linda M. Sample dated September 2, 2026. Reference texts: Harrison's Principles of Internal Medicine, 21st Edition; Cecil Essentials of Medicine, 10th Edition; Merck Manual of Diagnosis and Therapy, 19th Edition.

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hypertension. JAMA. 2012;307(20):2169 to 2176. Johnson AG, Nguyen TV, Day RO. Do nonsteroidal anti-inflammatory drugs affect blood pressure? A meta-analysis. Annals of Internal Medicine. 1994;121(4):289 to 300. Thase ME. Effects of venlafaxine on blood pressure: a meta-analysis of original data from 3744 depressed patients. Journal of Clinical Psychiatry. 1998;59(10):502 to 508. Pop-Busui R, Boulton AJ, Feldman EL, et al. Diabetic neuropathy: a position statement by the American Diabetes Association. Diabetes Care. 2017;40(1):136 to 154. Singleton JR, Smith AG, Bromberg MB. Increased prevalence of impaired glucose tolerance in patients with painful sensory neuropathy. Diabetes Care. 2001;24(8):1448 to 1453. Serretti A, Chiesa A. Treatment-emergent sexual dysfunction related to antidepressants: a meta-analysis. Journal of Clinical Psychopharmacology. 2009;29(3):259 to 266. Araujo AB, Durante R, Feldman HA, et al. The relationship between depressive symptoms and male erectile dysfunction: cross-sectional results from the Massachusetts Male Aging Study. Psychosomatic Medicine. 1998;60(4):458 to 465. Kouidrat Y, Pizzol D, Cosco T, et al. High prevalence of erectile dysfunction in diabetes: a systematic review and meta-analysis of 145 studies. Diabetic Medicine. 2017;34(9):1185 to 1192. 38 CFR section 3.102 (reasonable doubt). 38 CFR section 3.310 (disabilities that are proximately due to, or aggravated by, service-connected disease or injury). 38 CFR section 3.317 (compensation for certain disabilities occurring in Persian Gulf veterans). 38 CFR section 3.350(a) (special monthly compensation, including loss of use of a creative organ). 38 CFR section 4.14 (avoidance of pyramiding). 38 CFR section 4.97, Diagnostic Code 6847 (sleep apnea syndromes). 38 CFR section 4.104, Diagnostic Code 7101 (hypertensive vascular disease). 38 CFR section 4.115b, Diagnostic Code 7522 (erectile dysfunction). 38 CFR section 4.119, Diagnostic Code 7913 (diabetes mellitus). 38 CFR section 4.124a, Diagnostic Code 8520 (paralysis of the sciatic nerve).

Signed,

Dr. Licensed Physician

NPI: [withheld in this sample]

State Medical License: [withheld in this sample]

Expires: [withheld in this sample]